The treatment of myofascial pain syndrome using therapeutic ultrasound, on upper trapezius trigger points: A double-blinded placebo controlled study comparing the pulsed and continuous waveforms of ultrasound.

By

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I, Magendran Ganas Pillay do hereby declare that this dissertation is representative of my own work.

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Abstract

This study was a prospective, randomised, double blinded, placebo controlled, comparative clinical trial to establish the efficacy of therapeutic ultrasound and compare the effectiveness of the two waveforms of ultrasound in the treatment of myofascial pain syndrome.

Sixty patients with active upper trapezius myofascial trigger points were randomly allocated to one, of three groups. The allocation procedure was performed by an independent party and neither the researcher nor the participants were aware of the outcome.

Group A received the continuous waveform of ultrasound while group B received the pulsed waveform. Group C the placebo controlled group received sham (detuned) ultrasound. Each participant received six minutes of ultrasound (or sham ultrasound in group C) during the four treatments, within a three-week period. An independent party set the parameters of each ultrasound treatment and kept records of the allocation.

Data collection occurred at the initial, second and after the fourth (final) treatments. Objective data was collected using the Algometer and a
Myofascial Diagnostic Scale, while the subjective data was collected using the Numerical Pain Rating Scale 101 and the Short-Form McGill Pain Questionnaire. The subjective data was collected under the supervision of the researcher.

Statistical analysis using non-parametric tests and data capturing was conducted using the SPSS statistical package. Intra-group comparisons of subjective and objective data was analysed using the Friedman's T-test while inter-group comparisons of subjective and objective data was analysed using the Kruscal-Wallis H-test. If the null hypothesis was rejected for either the Kruscal-Wallis H-test or the Friedmans T-test, then the multiple comparison Dunn procedure was applied to the tests to determine between which groups and treatments respectively there was significant difference.

The study concluded that all three groups (treatment groups A & B and placebo controlled group C) showed a significant decrease in the levels of pain perception, overall pain intensity and extent to which the patients were suffering with myofascial pain syndrome together with an overall increase in pain thresholds. When the Dunn Procedure was applied to the Kruskal-Wallis H test, significant differences were observed between the treatment groups (A and B) and the placebo controlled, group C.
Evaluation of the two waveforms revealed no statistical difference between Group A (continuous waveform) and Group B (pulsed waveform). However, there were varied responses to the different waveforms. Patient's receiving the continuous waveform, showed significant increases in pain threshold only after the second treatment as compared to the immediate (after the first treatment) effect of pulsed Ultrasound. The benefit of significantly reduced levels of pain perception tapered after the second treatment in the continuous ultrasound group while the pulsed ultrasound group B, continued to show significant improvement with subsequent treatments.

The benefits of pulsed ultrasound are clearly of important therapeutic value in the recovery of tissue especially when chronic ischemia is present, as seen in myofascial trigger points.

This study recommends the use of therapeutic ultrasound and more especially the pulsed waveform as a non-invasive therapy in the management of myofascial pain syndrome, especially with patients who prefer non-invasive therapy.
Chapter 1

1.1 Introduction

Although the musculoskeletal system comprises 40% of the human body, Gatterman (1990) reports, that the least amount of research is performed in this field. Musculoskeletal disorders were recognized by Bruce (1995) as the key occupational injury or illness challenge of the 1990’s and a major cause of concern to employers due to disability and the mounting costs of workers compensation. According to Esengel et al (2000), Myofascial Pain Syndrome (MPS) is a common muscular pain syndrome resulting from trigger points (TrPs) which they define as a hyperirritable location within a taut band of skeletal muscle, that is painful when compressed and can give rise to characteristic referred pain, tenderness and muscle tightness.

Fishbain et al (1987) reported that, among 283 consecutive admissions to a comprehensive pain center the diagnosis of myofascial pain syndrome was assigned to 85% of cases. This diagnosis was made by a Neurosurgeon and a Psychiatrist working independently, based on the diagnostic criteria of Travell and Simons (1983). Han and Harrison (1997) in a review of the epidemiology, pathogenesis and a variety of treatment methods in the treatment of myofascial pain syndrome together with Gerwin (1995) in a study of 96 subjects examined for both fibromyalgia and myofascial pain reported that the incidence of trigger points ranged from 30 to 80% of patients presenting with pain. Chaiamnuay et al (1998) in an epidemiological study of Rheumatic disease in Thailand also reported similar incidences of myofascial pain syndrome (30-80%).
Several therapies were reported by Hubbard (1993) in the treatment of myofascial pain syndrome, including cold, heat, behavioural therapy, spray and stretch, ischaemic compession and electrotherapy.

Travell and Simons (1999;146) state that many therapists find the application of ultrasound an effective means of inactivating trigger points. Esenyel et al (2000) investigated the effectiveness of ultrasound treatment in combination with neck stretching on upper trapezius myofascial trigger points. They reported that ultrasound and trigger point injection were equally effective when combined with neck stretching exercises in the treatment of myofascial pain syndrome. This studies did not differentiate between the continuous and pulse waveforms nor did they indicate which wave form was used. Rachlin (1994; 31) states that much of the recommended treatment strategies of myofascial pain syndrome remain empirical and many modalities that are often used are essentially unproven. Travell and Simons (1999) also states that controversy on the effectiveness of ultrasound in the treatment of myofascial trigger points exists due to the lack of clinically controlled studies. They recommend well designed, well-controlled experimental studies on the effect of ultrasound on competently diagnosed active trigger points be conducted to fill the void in the literature. It appears from a review of the literature that no double-blinded placebo controlled study has been conducted on the efficacy of ultrasound (US) in the treatment of myofascial pain syndrome.

In view of the recommendations of Travell and Simons (1999), the aim of this study is to establish the efficacy of ultrasound in a double-blinded placebo control study as well as to determine the relative effectiveness of the continuous and pulsed waveforms.

This study will not only help to investigate the effectiveness of therapeutic ultrasound, but possibly provide information on the correct treatment
parameters (continuous or pulsed) when using ultrasound in the management of myofascial pain syndrome.

1.2 Statement of the problem

The purpose of this study is to establish the efficacy of therapeutic ultrasound and to compare the effectiveness of the two waveforms of ultrasound (continuous and pulsed) in a double-blind placebo-controlled study in the treatment of Myofascial Pain Syndrome.

1.2.1 The first sub problem

To evaluate, the effect of the continuous waveform of therapeutic ultrasound in the treatment of myofascial pain syndrome, in terms of subjective and objective clinical findings.

1.2.2 The second sub problem

To evaluate, the effect of the pulsed waveform of therapeutic ultrasound in the treatment of myofascial pain syndrome, in terms of subjective and objective clinical findings.

1.2.3 The third sub problem

To evaluate the effect of sham (detuned) ultrasound, in the treatment of myofascial pain syndrome, in terms of objective and subjective clinical findings.
1.3 Hypotheses

1.3.1 The first hypothesis

It is hypothesized that the continuous waveform will be more effective than sham (detuned) ultrasound in the treatment of myofascial pain syndrome in terms of subjective and objective clinical findings.

1.3.2 The second hypothesis

It is hypothesized that the pulsed waveform will be more effective than the sham (detuned) ultrasound in the treatment of myofascial pain syndrome in terms of subjective and objective clinical findings.

1.3.3 The third hypothesis

It is hypothesized that the continuous waveform is more effective than the pulsed waveform of ultrasound in the treatment of myofascial pain syndrome in terms of subjective and objective clinical findings.
2.1 Introduction

Myofascial pain is described by Fricton (1990) as regional pain disorder characterized by muscle tenderness and pain and is the most common cause of persistent regional pain and tension type headaches. Auleciems (1995) states myofascial pain syndrome remains one of the least understood problems encountered in the outpatients setting. Fricton (1990) previously reported that confusion regarding the symptoms seems to stem from the lack of organic findings, the lack of a unified theory to explain it and inconsistencies in the literature's definition of the syndrome.

Auleciems (1995) reports that myofascial pain syndrome has been referred to by a variety of names, including muscular rheumatism, myalgia, myogelosis, interstitial myofibrocitis, myofacitis, etc.

Myofascial pain syndrome occurs as a result of trigger points, which are either active or latent. Baldry (1989) describes latent trigger points as those in which the degree of activation is not sufficient for them to cause pain. In contrast an active trigger point according to Baldry (1989) is one in which the activation is of such intensity that impulses from it bombarding the central nervous system cause pain to be referred locally in the vicinity of the point or at a site some distance away.

According to Auleciems (1995), myofascial pain syndrome has excellent prognosis, besides specific trigger point therapy, treatment must involve lifestyle changes and long-term treatment to prevent recurrence. A multi-
disciplinary approach is also suggested by Auleciems (1995), which involves primary care providers, physical therapists, occupational therapists and other health care professionals to correctly assess and treat myofascial pain syndrome.

2. 2 Incidence Prevalence and Epidemiology of MPS

The incidence of myofascial pain syndrome appears to vary between 30% and 85% of people presenting to pain clinics (Han and Harrison 1997, Gerwin 1995), Fishbain et al (1987) reports that in chronic pain treatment centers, myofascial pain syndrome is the cause of pain in more than half the patients. Skootsky et al (1989) conducted a study of an Internal Medicine Practice, they identified myofascial pain syndrome in 10% of all their patients and in 31% of patients presenting with a complaint of pain. In a more recent study conducted by Chaiamnuay et al (1998), 2463 rural patients were interviewed. The results of the study showed that 36.2% had musculoskeletal pain of which myofascial pain syndrome was the second most common diagnosis.

It appears from the literature that no study on the epidemiology of myofascial pain syndrome has been conducted in South Africa. However, Jansen (1999) conducted a study on the prevalence and types of headaches suffered by scholars in Afrikaans speaking high schools in the greater Durban area. She reported that myofascial pain was an important aetiology of headaches.

Han and Harrison (1997) reported the incidence of trigger points to be higher in women although it is clearly found in both sexes. In an epidemiological study conducted by Severino and Moline (1990), myofascial pain syndrome occurred in 30% of women aged 20–40 of whom 6%
presented with symptoms severe enough to require treatment. They also found that the pain increased during the second week of the menstrual cycle, which suggests a hormonal influence. Sola et al (1990) reported, that myofascial pain syndrome is less common in labourers than sedentary workers, implying a protective effect of daily vigorous activity.

Sola et al (1990) examined 204 women and 103 men. They found that the head and neck, shoulder girdle and lower back demonstrated a higher frequency of trigger points than any other region. Travell et al (1999), Sola et al (1981), Bruce(1995) and Rubin(1981) conclude that the upper trapezius is the muscle most commonly affected by myofascial trigger points.

2.3 Aetiology

Travel and Simons (1999:110-112) suggest that acute events may precipitate a sudden onset of symptoms, while chronic stress is likely to produce a gradual onset of trigger point symptoms. They separate myofascial trigger point aetiology into two groups according to the nature of onset, that is, sudden or gradual. With regard to a sudden onset they highlight the following as important; falls, wrenching movement, direct blows to muscles, fractures, joint sprains, dislocations and speed accidents. Regarding gradual onset or chronic stress aetiology, Travell and Simons (1999) reported the following as important; sustained postural overload, prolonged immobilization and poor ergonomics.

Gatterman (1990) reported that myofascial pain syndrome appears to accompany many other pain disorders, including joint pathology such as osteoarthritis. According, to Fricton (1985) myofascial pain syndrome has also been reported to be associated with systemic and local infections of
viral or bacterial origin. These include Systemic Lupus Erythematosus, Scleroderma, Rheumatoid Arthritis and along the segmental distribution of, nerve injury, nerve root compression and neuralgia, although it is unclear whether myofascial pain syndrome develops in response to the pathological conditions or is coincidental. Sola et al (1981) found that the pain initiating factors in the development of trigger points were emotional and physical stress. However, Fricton (1994), stated that it was difficult to assess the relationship between stress and myofascial pain syndrome, although, stress management intervention frequently provided significant benefits for patients suffering with myofascial pain syndrome.

2.4 Pathophysiology

Gatterman (1990) reported that the characteristic features of myofascial trigger points include; hyperirritability, a region of increased and/or decreased circulation and a palpable band. Gerwin (1994) reported that no consistent histological changes have been identified using light or electron microscopy. Kravis et al (1993) failed to identify any morphological changes in an examination of trigger points of the trapezius muscle in 18 subjects using magnetic resonance imaging.

Hong et al (1998) reported that recent studies on referred pain and the local twitch response (LTR) have supported the concept that the myofascial trigger point (MTrP) mechanism is closely related to spinal chord integration. Hong et al (1998) states that when the input from nocicepters in an original receptor field persists (pain from a trigger point), central sensitization in the spinal cord may develop and the receptive field corresponding to the original dorsal horn may be expanded (referred pain). Through this, mechanism new myofascial or satellite myofascial trigger
points may develop in the referral zone of the original myofascial trigger point.

The pathophysiology of the taut band is now much more clearly understood, Travell et al (1999), postulates that the trigger point could be the result of activation of the actin-myosin complex by calcium. Calcium could be released either by rupture of the sarcoplasmic reticulum due to stress overload or through failure to pump adenosine triphosphate (ATP) that is essential for the calcium pump. Failure to restore ATP could be the result of a local energy crisis, such as that produced by local ischaemia, this could cause the ATP dependent calcium pump to fail resulting in the inability to recapture ionised calcium and failure of the ATP dependant actin-myocin complex thereby perpetuating local muscle contraction. In either case, the contracted taut bands could persist in the absence of electrical activity through the failure to restore ATP stores.

2.5 Perpetuating Factors

Auleciems (1995) states that the event which activates MTrP is quite different from the factors which perpetuate them, these factors are often overlooked and neglected but may well be the key between successful and failed treatment and once the perpetuating factors are corrected pain associated with myofascial pain syndrome is more likely to resolve.

Mechanical stresses as described by Travell and Simons (1999) include skeletal asymmetry (short leg or small hemipelvis), disproportion (long second metatarsal and short upper arms), misfit furniture, poor posture and prolonged immobilization.

Travel and Simons (1999) reported nutritional abnormalities as perpetuating factors these included; vitamin and mineral deficiency and excessive
consumption of stimulants (including caffeine). Metabolic and endocrine disorders such as hypothyroidism, hypocalcaemia, hyperuricaemia and anaemia are also included as perpetuating factors.

Fricton (1990) also comments on psychological factors perpetuating myofascial pain syndrome. He hypothesizes, that patients who experience difficulty in verbalizing anger, hostility or experience high levels of anxiety have an increase in muscle contraction due to stress experienced through these habits. Esenyel et al (2000), also reported high anxiety scores in patients suffering with myofascial pain syndrome.

Travel and Simons (1999) also include bacterial, viral infection and parasitic infestation as perpetuation factors. Viral infections particularly herpes simplex, results in an increase in symptoms. Bacterial infections resulting in abscesses, sinusitis and chronic urinary tract infections are also implicated.

Other perpetuating factors identified by Sola et al (1981) include impaired sleep, physical and mental fatigue. Travel and Simons (1999) reported that difficulty sleeping and abnormal sleep patterns are commonly experienced by chronic myofascial pain syndrome sufferers when monitored in a sleep laboratory. They also implicate nerve impingement as another factor that may perpetuate trigger points.

2.6 Presentations and Diagnosis

Myofascial pain syndrome is defined by Fricton (1990) as a muscular pain disorder involving regional pain referred by trigger points within the myofascial structures that are either local or a distance from the site. Travell and Simons (1999) states that each muscle has a distinctive pain referral pattern specific for the trigger points in that muscle. According to Han and
Harrison (1997), patients with myofascial pain syndrome complain of persistent regional pain, most commonly located in the head, neck, shoulders and lower back. The pain varies ranging from a mild ache to excruciating pain that is either sharp or dull and often associated with general fatigue, decreased range of movement and muscle strength.

Travel and Simons (1999) lists the following as symptoms of myofascial pain syndrome:

- Myofascial pain referred from TrPs has specific patterns that are characteristic of each muscle.
- TrPs become activated directly due to acute overload, fatigue, direct trauma and by chilling of the muscle.
- Trigger points become activated indirectly by other trigger points, visceral disease, arthritic joints and emotional stress.
- Active trigger points may be transformed from a latent to an active state by any perpetuating factor.
- Active trigger points vary in pain intensity at any given time.
- Clinical features of trigger points may long outlast the initiating event.
- Phenomena other than pain such as localized vasoconstriction, sweating, salivation and pilomotor activity as well as proprioceptive disturbances including dizziness, tinnitus and imbalance may result from the activity of trigger points.

Travell and Simons (1999) defines specific criteria for the examination and diagnosis of trigger points.

- **A taut band.** A taut band is found using either flat or pincer palpation on a muscle that is slightly stretched (this slight stretch must not evoke or worsen pain).
• **Local twitch response.** A spontaneous local twitch is produced when part of the muscle containing the trigger point is rolled under the fingers.

• **Jump sign.** When sufficient pressure is applied to a trigger point the behavioural reaction may be withdrawal or a verbal response. This is characteristic of myofascial pain syndrome.

• **Referred pain.** Pressure applied to an active trigger point produces local pain over the tender spot as well as referred pain in a pattern characteristic of the muscle.

The following signs were put forward by Travell and Simons (1983:16-17) upon examination:

• Passive or active stretching of the involved muscle results in an increase in pain when trigger points are present.

• Restricted muscular stretch.

• There is an increase in pain when the affected muscle undergoes an isometric contraction and there is a decreased maximum contractile force.

• Deep tenderness and dysaesthesia are commonly found in trigger point referral pain zones.

• Muscle palpation adjacent to active myofascial trigger points feels tense.

• Disturbances of non-sensory autonomic function are sometimes induced in the zone of referral pain of myofascial trigger points.

• The myofascial TrPs are found in a taut band as a well circumscribed spot of exquisite tenderness.

• A jump sign is usually found upon digital pressure of an active trigger point.

• A local twitch response is frequently evoked by snapping palpation of an active trigger point.
• Sustained, moderate pressure on a myofascial trigger point produces or increases pain in the referred pain zone of the trigger point.
• The skin overlying active myofascial trigger points has in some patients, shown to exhibit panniculosis or dermatographia.

Identification of myofascial trigger points is essential for diagnosis and treatment of myofascial pain syndrome. Manual palpation, recognition of clinical features and patient feedback are the primary methods for the diagnosis and treatment (Fischer (1988), Travell and Simons (1999) and Sciotti et al( 2001)).

Wolf et al (1992) reported poor interrater reliability when four experienced Physicians evaluated the following characteristics of trigger points; spot tenderness, pain recognition, palpable bands, referred pain and a local twitch. Poor interrater reliability was also reported by Nice et al (1992) and Njoo (1994). Gerwin et al (1997) conducted a double-blinded study to explain why such results were obtained in previous investigations (such as Wolf et al 1992, Nice et al 1992 and Njoo (1994). The study was conducted in two parts; the first part was conducted the same way as previous studies producing similar results. The second part of the study was conducted using the same examiners, however, this was conducted following a three-hour training session. The second part of the study produced good to excellent, interrater reliability.
2.7 Treatment of Myofascial Pain Syndrome

Travell and Simons (1983) reports that uncomplicated myofascial pain syndrome is highly responsive to simple and appropriately directed treatment. According to Bruce (1995) the treatment of myofascial pain syndrome should firstly be directed at diagnosing and treating any perpetuating factors such as structural abnormalities, mechanical factors and medical conditions or psychological etiologies. Han and Harrison (1997) advocate a multidisciplinary approach to treatment, a team may include anesthesiologists, clinical psychologists, physical therapists, psychiatrists and social workers. They state that the goal of treatment is not only reducing pain but also to enable the patient to cope with pain.

2.7.1 Spray and Stretch

Fricton (1990) describes spray and stretch as one of the most common techniques in treating trigger points. Bruce (1995) reports that the spray and stretch technique is a mild application of a vapour coolant agent such as fluoromethane on the skin surface overlying the muscle in parallel sweeps with the muscle held in a passive stretch. The aim of the treatment is to decrease pain over the trigger point, restore the muscle to its length and improve range of movement.

2.7.2 Ischaemic Compression

Ischaemic compression consists of an application of sustained pressure for a long period so, as to reduce the muscle spasm and deactivate the trigger point (Gatterman 1990: 296).
Schneider (1994:27), postulated that the therapeutic benefit of ischaemic compression may be the result of the following:

- **Localized stretch**: Ischaemic compression is actually a form of intense, specific localized stretch of the contractile fibres of the taut band, it is claimed that the manual pressure over the taut band actually separated the actin-myosin cross fibre links.

- **Nerve block**: it is postulated that deep pressure causes a temporary suspension of the reflex motor neuron activity by blocking incoming sensory input, it is also postulated that the action potentials can only be propagated in the presence of oxygen and that the ischaemia produced by prolonged pressure inhibits this.

- **Reflex vasodilation**: following initial blanching and ischaemia the involved muscle region experiences a reflex vasodilation which brings fresh blood, carrying oxygen and ATP to the area, the blood flushes away metabolites and substances that maintain muscle contraction.

- **Hyperstimulation analgesia**: it is postulated that endorphins are released by the dorsal horn in response to the intense pain caused by deep sustained pressure.
2.7.3 Needling

Travell and Simons (1983:27) state that the puncture of TtPs is known to be effective whether done by injection with local anaesthetic, by injection with saline or whether done by dry needling, the procedure for all three techniques is similar and is described as follows:

The TrP is located by palpation and the needle is inserted 1-2 cm's away and directed towards the TrP such that the needle approaches at an angle of 30° to the skin. A fanning technique is used, whereby the needle is repeatedly inserted and withdrawn and then redirected to another section of the trigger point ensuring maximum coverage of the area.

Travell and Simons (1999) postulates that the mechanism of inactivation of trigger points by needling as follows:

- Mechanical disruption of muscle fibres and nerve endings.
- Mechanical disruption of muscle fibres leads to an increase in extracellular potassium with a resultant depolarisation of nerve fibres.
- The interruption of the positive feedback mechanism that is responsible for the perpetuation of pain.
- Local dilution of noiceceptive substances through the infiltration of local anaesthetic or saline solution.
- An increased removal of metabolites due to the vasodilatory effects of the local anaesthetic or saline solution.

2.7.4 Electrotherapy

Hong et al (1996) reports that electrotherapy may inactivate trigger points by stimulating fibres around TrPs and facilitating relaxation of the taut
bands, thus improving circulation. Auleciems (1995) attributed the benefit of electrotherapy in the treatment of myofascial pain syndrome to the stimulation of muscle contractions, which squeezes out edema, increases blood flow and relaxes the muscles.

Hsueh et al (1997) compared electrical muscle stimulation (EMS) to electrical nerve stimulation (ENS) in the treatment of myofascial pain syndrome. They found that ENS is far more effective for immediate relief of myofascial trigger points than EMS but, EMS has a better effect on the immediate release of muscle tightness.

Han and Harrison (1997) report that since the publication of the Gate Control Theory of pain perception by Melzack and Wall (1965), transcutaneous nerve stimulation (TENS) has become a popular therapy of acute and chronic pain conditions including myofascial trigger points. Melzack and Wall (1965) state that the low intensity stimulation of TENS selectively activates the large diameter fibres to “close the pain gate” in the dorsal horn of the spinal chord or at a higher level. Graaf- Redford et al (1989) conducted a double-blinded study on the effects of TENS on myofascial pain syndrome. Four modes of TENS and a non-stimulating control were compared, the results demonstrated that TENS can reduce myofascial pain but it may be insufficient as a long term treatment since myofascial trigger point sensitivity appears to remain unaltered.

2.8 Ultrasound

2.8.1 Introduction

Gam et al (1998) reported that ultrasound therapy has achieved recognition as a suitable method in physical medicine to treat acute and chronic
musculo-skeletal disorders. Ultrasound treatment involves the use of high-frequency acoustic energy that is generated using the reverse piezoelectric effect (Esenyel et al 2000).

Van der Windt et al (1999) in a systematic review of the literature evaluated the effectiveness of ultrasound therapy in the treatment of musculoskeletal disorders. Thirty eight studies were included in the review, which evaluated the effects of ultrasound in the treatment of, lateral epicondylitis \((n=6)\), shoulder pain \((n=7)\), degenerative rheumatic disorders \((n=10)\), ankle distortions \((n=4)\), temporomandibular pain and myofascial pain \((n=4)\) and a variety of other conditions \((n=7)\). They assigned each study a validity score according to the following criteria:

- **V1** Was a method of randomization performed?
- **V2** Was the treatment allocation sealed?
- **V3** Were the intervention groups similar at baseline regarding prognostic indicators (age, duration of symptoms etc.), and baseline scores of outcomes measures?
- **V4** Was the care provider blinded for the allocated intervention (use of a placebo)
- **V5** Were co-interventions avoided or standardized?
- **V6** Were adherence to the interventions acceptable in all groups?
- **V7** Was the patient blinded to the allocated intervention?
- **V8** Was the withdrawal/drop-out rate described and acceptable?
- **V9** Was the outcomes assessor blinded to the intervention?
- **V10** Was the timing of outcome assessment comparable in both groups.

Emphasis was placed on an adequate randomization procedure and sufficient blinding, together representing five of the ten criteria. When evaluating the four studies on temporomandibular pain and myofascial pain, Van der windt (1999) did not assign a validity score of at least five to any of
these studies and concluded that given the lack of high quality trials there is insufficient evidence for the effectiveness of ultrasound therapy for temporomandibular pain and myofascial pain. In conclusion, Van der windt (1999) reported that at the time of their study there seemed to be little evidence to support the use of ultrasound therapy in the treatment of musculoskeletal disorders.

2.8.2 Biophysical effects of therapeutic ultrasound.

The biophysical effects resulting from the interaction of US with tissue are grouped by McDiarmid et al (1987) into two categories:

- **Thermal** – these effects are produced by the ability of therapeutic ultrasound to elevate tissue temperature.

- **Non-thermal** – these effects are attributed to any mechanics other than an increase in tissue temperature.

Burns (1987) and Dyson (1983) differentiate between the thermal and non-thermal effects of ultrasound on tissue, due to the continuous and pulsed waveforms respectively.

2.8.2.1 Thermally induced therapeutic effects

Many of the therapeutic effects of ultrasound are attributed primarily to heating and are proposed by Lehman et al (1972), to include the following:
- The increased extensibility of collagen-rich structures such as tendons and joint capsules.
- A decrease in joint stiffness.
- A reduction in muscle pain and spasm.
- The production of a mild inflammatory reaction, inducing a marked increase in blood flow, which helps in the resolution of chronic inflammatory processes.

Dyson (1983) reports that the main advantage of ultrasound over a non-acoustic heating modality is the preferential heating of collagen rich tissue allowing scar tissue, joint capsules, tendons and inter-muscle surfaces to be heated within the therapeutic range without producing the damaging temperature elevations to the overlying skin, subcutaneous adipose and tissue lying adjacent to them. Abramson et al (1960) reported that the metabolic reaction due to the heating of tissue is an increase in the rate of chemical reactions and oxygen uptake by tissues. Fyfe et al (1982), Lehman et al (1990) and Baker et al (1991) report that heating and an increase in tissue temperatures is usually associated with vasodilation and thus an increase in blood flow to the area. Low and Reed (1990) state that ultrasonic heating is used therapeutically to provide analgesia and assist in the resolution of pain and muscle-guarding spasm. Wadsworth and Chanmugam (1988) earlier reported that the ability of therapeutic ultrasound to provide analgesia is due to its underlying ability to elevate pain thresholds by altering nerve conduction velocity and alter muscle spindle firing rates.

Lehmann et al (1990) reports that spasm of skeletal muscle can be the result of a reflex tonic muscle contraction. The muscle spindle afferents that alter their rate of firing normally in response to tonic or static stretch are the Type II afferents. Lehman et al (1990) reports that the elevation of muscle temperature to 42°C will decrease the rate or firing of the Type II afferents
and increase the firing rate of the Type Ib fibres from the Golgi tendon organ, a decreased firing of the alpha motoneurons is predicted, resulting in a reduced tonic extrafusal fibre activity.

2.8.2.2 Non-thermal effects

Dyson (1983) reports that the use of ultrasound at levels below those inducing the physiological significant temperature increases can be of therapeutic benefit in a number of ways in the treatment of injured tissue.

According to Hogan et al (1982) examples of the therapeutically significant non-thermal benefits of ultrasound include:

- Stimulation of tissue regeneration.
- Soft tissue repair.
- Improved blood-flow in chronically ischaemic tissue.
- Stimulation of protein synthesis.

Dyson (1983) expounds that the non-thermal, therapeutic benefits of ultrasound, involves acoustic streaming. This concept describes the unidirectional movement of fluid in an ultrasonic pressure field. Connective tissue and plasma membranes of immovable cells form boundaries within the pressure field and along these boundaries high velocity gradients develop. According to this theory enhanced microstreaming may be responsible for the observed changes in permeability of the cell membranes to ions such calcium and sodium after treatment with therapeutic ultrasound. Dyson (1983) also reports that changes in permeability to ions such as sodium may also be responsible for the altered electrical activity which has been observed in nerve and muscle following treatment with therapeutic ultrasound and such alterations may be responsible for the
reduction of muscle spasm and the relief from pain. According, to Dyson (1983) the changes in permeability to calcium ions may have a dramatic effect on cell behavior, which are clearly of important value therapeutically, in the recovery of cells and tissue from injury.

2.9 Muscle overview

2.9.1 The Trapezius muscle

The trapezius muscle is divided into upper, middle and lower sections, with trigger points occurring most commonly in the upper trapezius (Travell and Simons 1999). Sola et al (1981), Rubin (1981) and Bruce (1995) also concluded that the upper trapezius is the muscle most commonly affected by myofascial trigger points.

The following information on the upper trapezius regarding anatomical attachments, trigger point location and referral pain patterns and innervation are according to Travell and Simons (1999:278):

- **Attachments.** The upper fibres of the trapezius muscle attach superiorly to the medial third of the superior nuchal line attaching to the midline of the ligamentum nuchae and to the spinous processes of the first to the fifth cervical vertebrae. Distally the fibres converge laterally attaching to the outer third of the clavicle.

- **Trigger point location.** Tp1 is located in the upper free margin of the trapezius superiorly to both the supraspinatus muscle and the apex of the lung while Tp2 is located caudal and posterior to the free border of the upper trapezius superior to the mid-line of the scapula.
• **Referral pain pattern.** Tp1 characteristic pain is severe posterolateral neck, a temporal headache centering to the orbit. Less common presentations include pain referred to the angle of the ipsilateral, jaw, molar teeth and pinna of the ear, mimicking dental pain. Tp2 is not associated with headaches and the pain is restricted to the posterior neck, stopping at the mastoid.

• **Innervation.** The muscle is inervated by the spinal division of the XI cranial nerve, which supplies mainly motor fibres, the second to fourth cervical nerves supply mainly sensory fibres to the muscle.
CHAPTER THREE

Materials and Methods of Study

3.1 Introduction

This study was a prospective, randomized, double-blinded, placebo controlled, comparative clinical trial to establish the efficacy of therapeutic ultrasound and compare the effectiveness of the two waveforms of ultrasound in the treatment of myofascial pain syndrome.

3.2 Patient selection

Advertisements for patients suffering from neck pain and headaches were placed on notice boards at the campus of the Durban Institute of Technology, in local community newspapers and fliers were distributed in the surrounding neighborhood. Patients responding to advertisements were initially interviewed telephonically to ensure their suitability for the study and a consultation at the Chiropractic Clinic was scheduled. At this consultation, patients underwent a further detailed examination (Appendix 1,2,3) to determine eligibility (criteria for inclusion will be discussed later in the chapter).

A sample size of sixty patients was selected by means of convenience sampling.
3.3 Inclusion and exclusion criteria

3.3.1 Inclusion criteria

- Men and women between the ages of 18 and 35 were accepted, Esenyel et al (2000) suggests a relatively young population of patients to minimize pain that may be caused by accompanying degenerative disc or joint disease.

- The patients must have had active trigger points in the upper trapezius, Travell and Simons (1999), together with studies by Sola et al (1981), Bruce (1995) and Rubin (1981) concluded that this muscle is most commonly affected by myofascial trigger points.

- The diagnosis of active myofascial trigger points was based on the criteria described by Travell and Simons (1999). Patients were only accepted into the study on exhibition of varying degrees of these characteristics ranked in the Myofascial Diagnostic Scale (Appendix 4) (Chettiar, 2001). The scale was designed to assess the extent to which the patient is suffering from myofascial pain syndrome via a rating of the patient’s symptoms.

- The scoring system of the scale gives a value to each of the four signs of an active trigger point according to Travell and Simons (1999). The first three signs: focal tenderness, palpable taut bands, and a local twitch response were considered equal in importance and were therefore assigned an equal value. The fourth sign referred pain in a zone of reference was considered the strongest sign of an active trigger point and was assigned the highest value. An active
trigger point was diagnosed by a total value of nine or more, with the maximum, seventeen.

- Patients were only accepted if their initial score was nine or more.

### 3.3.2 Exclusion criteria

- Contraindications to ultrasound according to Burns (1987) includes; areas of sensory loss, tissue already treated by radiotherapy, acute infection, hemophiliacs without factor replacement and other bleeding disorders, areas with metallic implants and cardiac pacemakers
- Patients exhibiting the signs of Fibromyalgia shall be excluded from this study.

Patients found to be eligible for the study, had the study criteria described to them and received a patient information sheet (Appendix 7) outlining the nature and requirements of the study. Patients were then asked to complete an informed consent form (Appendix 8) before treatment commenced.

### 3.4 Random allocation

Once accepted into the study each of the sixty patients (60 patients chosen because of statistics and prevalence) were randomly allocated by an independent party to either group A, B or C by a draw out of a hat. The independent party also set and concealed the appropriate parameters for the ultrasound unit, exposing only the start and stop keys and kept a record of the patient/group allocations. Neither the researcher nor the patient knew the outcome of the allocation.
3.5 Interventions

The ultrasound unit used, was a Dynatron® 850plus, with a 5cm² soundhead.

**Group A Treatment.**

Group A received ultrasound with a continuous wave mode setting at a frequency of 1MHz and an intensity of 1.5w/cm² for 6 minutes. The optimal intensity and duration of ultrasound in the treatment of myofascial pain syndrome has not been established, however Esenyel et al 2000 when investigating the effectiveness of ultrasound and trigger point injections in combination with neck-stretching exercises in the treatment of myofascial pain syndrome, treated patients at an intensity of 1.5w/cm² for 6 minutes at each session.

**Group B Treatment.**

Group B received a pulsed wave 1:1 ratio (4 milliseconds on, 4 milliseconds off) of ultrasound at frequency of 1MHz at an intensity 1.5w/cm² for 6 minutes.

**Group C Treatment**

Group C, the placebo control group was treated with sham ultrasound (0w/cm²) for 6 minutes.
Groups A, B, C received four treatments over a maximum period of 3 weeks. No research has been done to establish the number of treatments that are required for a patient with myofascial pain syndrome to respond to ultrasound treatment. The treatment period and the number of treatments were based on the findings of other research using electrotherapy. Christie (1995) found significant improvement in patients receiving both interferential current and dry needling of trigger points after 4-6 treatments over 3 weeks while Hutchings (1998) found 30% of the patients receiving TENS treatment for trigger points to be asymptomatic in 5 or less treatments.

Each patient was provided with a copy of environmental, perpetuating factors (Appendix 9) that should be avoided during the course of the study. These factors have been documented by Travell and Simons (1999) to perpetuate myofascial trigger points.

Researcher/patient interaction was kept to a minimum especially during the application of Ultrasound.

### 3.6 Data collection

Patients were assessed subjectively and objectively prior to the first, second and after the forth treatment.

**Subjective measurements**

**The numerical pain rating scale-101 (NRS-101):** This questionnaire (Appendix 4) consists of a numerical scale from 0-100, with 0 representing one extreme (no pain) and 100 representing the other extreme (pain at its most). The patient indicates their pain by means of a percentage, both at its worst and least. The average of these two scores is the level of pain intensity experienced by the patient. Jensen et al (1986), examined the
usefulness of six pain intensity measures on 75 patients suffering with chronic pain. This scale was found to be the most practical index. It can be administered in a written or verbal form and is simple to score.

**Short form McGill pain questionnaire (SF-MPQ):** This questionnaire (Appendix 5) was developed by Melzack (1987), from the standard McGill pain questionnaire for the specific use in a research setting, when time for the capture of patient information regarding the sensory dimensions of pain was limited. The SF-MPQ provides valuable information on the patients sensory, affective and overall intensity. The questionnaire consists of 15 descriptions with 1-11 representing the sensory dimensions of pain and 12-15 representing the affective dimension. In the two sections, a score of 0-3 is given for each objective depending on whether the pain was ranked mild, moderate or severe respectively with none carrying a score of zero. The score is then calculated as a percentage of the maximum for the sum of all 15 categories.

The patient completed both questionnaires under the supervision of the researcher.
Objective Measures

Algometer (manufactured by Wagner instruments: P.O Box 1217, Greenwich CT 06836.)

Reeves et al (1986) conducted a study that demonstrated high inter and intra experiment reliability of the pressure algometer as a measure of myofascial trigger point sensitivity, patients with chronic myofascial head and neck pain. The procedure for the pain threshold measurement was performed as recommended by Fisher (1987). The patient was advised on the procedure and advised to say “yes” when the pain was first felt, the trigger point was marked and a measurement was recorded by slowly and gradually applying pressure until the patient said “yes.” This was repeated thrice and the average reading was used for analysis (Appendix 6).

Myofascial diagnostic scale

The myofascial diagnostic scale (Chettiar 2001)(Appendix 4) as discussed earlier, was used to assess the extent to which the patient suffers from myofascial pain syndrome. In the absence, of a satisfactory laboratory test that may be clinically utilized when assessing the efficacy of ultrasound, the myofascial diagnostic scale was used as an objective measure in this study.
3.7 Statistical Analysis

The SPSS statistical package (as supplied by SPSS Inc, Marketing Departement, 444 North Michigan, Chicago, Illinois, 60611) was used for data entry and analysis.

Statistical analysis was carried out using non-parametric tests. Intra-group comparisons of subjective and objective data was analysed using the Friedman's T-test while inter-group comparisons of subjective and objective data was analysed using the Kruscal-Wallis H-test.

If the null hypothesis was rejected for either the Kruscal-Wallis H test or the Friedman's T test, then the multiple comparison Dunn Procedure was applied to the tests to determine between which groups and treatments respectively there was significant difference.

The Decision Rule and the Null Hypothesis

Kruscal-Wallis H Test

The null hypothesis (H₀) states that there is no difference between the groups A, B or C while the alternate hypothesis (H₁) states that there is a difference between the groups.

H₀ : there is no difference.
H₁ : there is a difference.

Decision Rule
For a two tailed test:
Reject H₀ if P<α.
Accept $H_0$ if $P\geq \alpha$.

$P$ was the observed level of significance ($\alpha = 0.05$)

**Friedman's T Test**

The null hypothesis ($H_0$) states that there is no improvement between the treatments while the alternate hypothesis ($H_1$) states that there is an improvement between the treatments.

$H_0$: There is no improvement between treatments.

$H_1$: There is an improvement between treatments.

**Decision Rule**

For a two tailed test:

Reject $H_0$ if $P < \alpha$.

Accept $H_1$ if $P \geq \alpha$.

$P$ was the observed level of significance ($\alpha = 0.05$)
CHAPTER 4

THE RESULTS

4.1 Introduction

This chapter covers the results obtained from the statistical analysis of the data collected from the following measurement criteria:

The Short Form McGill Pain Questionnaire
The Numerical Pain Rating Scale- 101
The Algometer readings
The Myofascial Diagnostic scale
Trigger point characteristics and demographic statistics are also tabulated.

4.2 Criteria governing the admissibility of data

Data for the study was collected from patients who fulfilled the inclusion and exclusion criteria and completed the full course of the programme. Subjective data from responses to the McGill pain questionnaire and NRS-101 were completed by the patient under the supervision of the researcher and the objective measures, that is, the algometer and myofascial diagnostic scale scores were collected only by the researcher.

4.3 The Hypotheses

The null hypothesis ($H_0$) was the same for groups $A$ (continuous), $B$ (pulsed) and $C$ (sham-ultrasound):
$H_0$: There is no difference between the intra-group data.
The alternate hypothesis \( (H_1) \) is the same for groups A, B and C and is described below:

\[ H_1: \text{There is a difference between the intra-group data.} \]

**A further null hypothesis and alternate hypothesis was required in order to integrate the data from the three groups.**

\[ H_0: \text{There is no difference between the inter-group data.} \]

\[ H_1: \text{There is a difference between the inter-group data.} \]

If the hypothesis \( (H_0) \) is rejected for the Friedman’s test (intra-group) or the Kruscall-Wallis-H test (inter group), then the multiple comparison procedure will have to be applied utilizing the Dunn Procedure to determine between which treatments and which groups respectively significant improvement occurred.

### 4.4 The analyzed data

#### 4.4.1 P-Value

The data was analyzed at the \( \alpha = 0.05 \) level and the decision rule was applied as follows:

- Reject the null hypothesis if the P-Value \( < \alpha \)
- Accept the null hypothesis if the P-Value \( \geq \alpha \)

In order to conclude that there is a statistically significant improvement at the \( \alpha = 0.05 \) level, the P-Value would have to be \( < 0.05 \).
4.5 Demographic Data

Table 1 gender distribution with a sample of 60 patients.

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>MALE</th>
<th>FEMALE</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>A(CONTINUOUS)</td>
<td>6</td>
<td>14</td>
<td>20</td>
</tr>
<tr>
<td>B (PULSED)</td>
<td>5</td>
<td>15</td>
<td>20</td>
</tr>
<tr>
<td>C (PLACEBO)</td>
<td>7</td>
<td>13</td>
<td>20</td>
</tr>
<tr>
<td>PERCENTAGE</td>
<td>30</td>
<td>70</td>
<td>100</td>
</tr>
</tbody>
</table>

The table reveals a female (70%) to male (30%) ratio of 2.33: 1 in the study, with a similar distribution of men and women in each group.

Table 2 Age distribution within a sample of 60 patients.

<table>
<thead>
<tr>
<th>GROUPS</th>
<th>18-23</th>
<th>24-30</th>
<th>31-35</th>
<th>TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>A(CONTINUOUS)</td>
<td>6</td>
<td>7</td>
<td>7</td>
<td>20</td>
</tr>
<tr>
<td>B (PULSED)</td>
<td>5</td>
<td>11</td>
<td>4</td>
<td>20</td>
</tr>
<tr>
<td>C (PLACEBO)</td>
<td>9</td>
<td>5</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>Percentage</td>
<td>33.3</td>
<td>38.33</td>
<td>28.33</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 2 reveals the age distribution of the participants in the study with the following mean group ages; Group A = 27.20, Group B = 26.70 and Group C = 25.90.
Table 3 Occupation distribution within the sample group

<table>
<thead>
<tr>
<th>OCCUPATION</th>
<th>GROUP A</th>
<th>GROUP B</th>
<th>GROUP C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Engineer</td>
<td>1*</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Clerical</td>
<td>2*</td>
<td>4*</td>
<td>6*</td>
</tr>
<tr>
<td>Student</td>
<td>5</td>
<td>6</td>
<td>3</td>
</tr>
<tr>
<td>Librarian</td>
<td>1*</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Auditor</td>
<td>1*</td>
<td>1*</td>
<td>-</td>
</tr>
<tr>
<td>Educator</td>
<td>2</td>
<td>-</td>
<td>2</td>
</tr>
<tr>
<td>Housewife</td>
<td>2</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Receptionist</td>
<td>3*</td>
<td>1*</td>
<td>3*</td>
</tr>
<tr>
<td>Consultant</td>
<td>1*</td>
<td>-</td>
<td>2*</td>
</tr>
<tr>
<td>Self-employed</td>
<td>2(1*)</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Chiropractor</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Beautician</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Technician</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Manager</td>
<td>-</td>
<td>1*</td>
<td>-</td>
</tr>
<tr>
<td>Driver</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>Artist(Graphic)</td>
<td>-</td>
<td>1*</td>
<td>-</td>
</tr>
<tr>
<td>Pharmacist</td>
<td>-</td>
<td>-</td>
<td>2*</td>
</tr>
<tr>
<td>Surveyor</td>
<td>-</td>
<td>-</td>
<td>1*</td>
</tr>
<tr>
<td>Journalist</td>
<td>-</td>
<td>-</td>
<td>1*</td>
</tr>
<tr>
<td>Unemployed</td>
<td>-</td>
<td>1</td>
<td>-</td>
</tr>
</tbody>
</table>

* Denotes participants that worked predominantly in an office (air-conditioned environment). This accounted for 33(55%) participants.

Table 3 revealed the occupational of participants, with a wide range of different occupations. There was a significant contribution of clerical workers (20%) and students (23.33%)
Table 4 Ethnic Group Distribution

<table>
<thead>
<tr>
<th>ETHNIC GROUP</th>
<th>GROUP A Continuous</th>
<th>GROUP B Pulsed</th>
<th>GROUP C Sham</th>
<th>PERCENTAGE</th>
</tr>
</thead>
<tbody>
<tr>
<td>BLACK</td>
<td>2</td>
<td>3</td>
<td>-</td>
<td>8.33</td>
</tr>
<tr>
<td>COLOURED</td>
<td>1</td>
<td>2</td>
<td>1</td>
<td>6.66</td>
</tr>
<tr>
<td>INDIAN</td>
<td>13</td>
<td>11</td>
<td>13</td>
<td>61.66</td>
</tr>
<tr>
<td>WHITE</td>
<td>4</td>
<td>4</td>
<td>6</td>
<td>23.33</td>
</tr>
<tr>
<td>TOTAL</td>
<td>20</td>
<td>20</td>
<td>20</td>
<td>100</td>
</tr>
</tbody>
</table>

The table reveals ethnic distribution of participants, with a significant number of Indians (61.66%).

4.6 Trigger point characteristics

Table 5 Trigger point distribution

<table>
<thead>
<tr>
<th>Group</th>
<th>Tp1-Right</th>
<th>Tp1-Left</th>
<th>Tp2-right</th>
<th>Tp2-Left</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>10</td>
<td>8</td>
<td>1</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>B</td>
<td>10</td>
<td>7</td>
<td>2</td>
<td>1</td>
<td>20</td>
</tr>
<tr>
<td>C</td>
<td>12</td>
<td>4</td>
<td>2</td>
<td>2</td>
<td>20</td>
</tr>
<tr>
<td>Percentage</td>
<td>53.33</td>
<td>31.66</td>
<td>8.33</td>
<td>6.66</td>
<td>100</td>
</tr>
</tbody>
</table>

Table 5 reveals data on trigger point distribution with a similar distribution of trigger points among all three groups. Tp1 accounted for 85% of the participants compared to the 15% by Tp2.
### Table 6 Duration of symptoms

<table>
<thead>
<tr>
<th>Group</th>
<th>Acute</th>
<th>Subacute</th>
<th>Chronic</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>12</td>
<td>1</td>
<td>7</td>
<td>20</td>
</tr>
<tr>
<td>B</td>
<td>10</td>
<td>4</td>
<td>6</td>
<td>20</td>
</tr>
<tr>
<td>C</td>
<td>12</td>
<td>1</td>
<td>7</td>
<td>20</td>
</tr>
<tr>
<td><strong>Percentage</strong></td>
<td><strong>56.66</strong></td>
<td><strong>10</strong></td>
<td><strong>33.3</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

The data reveals the duration of participants' symptoms. Acute (0-3 months) patients accounted for 56.66%, sub-acute (3-6 months) 10%, and 33.3% were chronic, with symptoms for more than 6 months.

### Table 7 Primary causes

<table>
<thead>
<tr>
<th>CAUSES</th>
<th>GROUP A Continuous</th>
<th>GROUP B Pulsed</th>
<th>GROUP C Sham</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Muscle Overload</td>
<td>7</td>
<td>5</td>
<td>5</td>
<td>28.33</td>
</tr>
<tr>
<td>Trauma</td>
<td>3</td>
<td>2</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Stress</td>
<td>3</td>
<td>3</td>
<td>3</td>
<td>15</td>
</tr>
<tr>
<td>Poor Posture</td>
<td>2</td>
<td>3</td>
<td>6</td>
<td>18.33</td>
</tr>
<tr>
<td>Environmental</td>
<td>2</td>
<td>5</td>
<td>-</td>
<td>11.66</td>
</tr>
<tr>
<td>Unknown</td>
<td>3</td>
<td>2</td>
<td>5</td>
<td>16.66</td>
</tr>
<tr>
<td><strong>TOTAL</strong></td>
<td><strong>20</strong></td>
<td><strong>20</strong></td>
<td><strong>20</strong></td>
<td><strong>100</strong></td>
</tr>
</tbody>
</table>

Table 7 reveals the primary causes of the trigger points. Muscle overload (28.33%) and poor posture (18.33%) were reported by patients as a significant cause of their trigger points.
4.7 Tables of Inter-group statistical results using the Kruskal-Wallis Test

4.7.1 Subjective Data

Table 8 Statistical results of the Short-Form McGill Pain Questionnaire for groups A, B, and C

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Group</th>
<th>N</th>
<th>Mean Rank</th>
<th>P-Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>20</td>
<td>28.00</td>
<td>.637</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>30.27</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>33.22</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>20</td>
<td>27.95</td>
<td>.005</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>23.15</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>40.40</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>A</td>
<td>20</td>
<td>25.33</td>
<td>.006</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>25.42</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>40.75</td>
<td></td>
</tr>
</tbody>
</table>

This table reflects that at treatment 1, the null hypothesis was accepted (P ≥ 0.05) indicating no difference between groups A (continuous), B (pulsed) and C (sham).

At treatment 2 and 4, the null hypothesis was rejected (P < 0.05) indicating statistically significant improvement between groups A, B and C.
Table 9 Statistical results of the Numerical Pain Rating Scale-101 for groups A, B, and C

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Group</th>
<th>N</th>
<th>Mean Rank</th>
<th>P-Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>20</td>
<td>28.52</td>
<td>.425</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>34.65</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>28.33</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>20</td>
<td>26.88</td>
<td>.040</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>26.38</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>38.25</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>A</td>
<td>20</td>
<td>28.83</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>21.67</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>41.00</td>
<td></td>
</tr>
</tbody>
</table>

This table reflects that at treatment 1, the null hypothesis was accepted (P ≥ 0.05) indicating no difference between groups A (continuous), B (pulsed) and C (sham).

At treatment 2 and 4, the null hypothesis was rejected (P < 0.05) indicating statistically significant improvement between groups A, B and C.
4.7.2 Objective data

Table 10 Statistical results of the Algometer readings for groups A, B, and C

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Group</th>
<th>N</th>
<th>Mean Rank</th>
<th>P-Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>20</td>
<td>32.63</td>
<td>.774</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>30.05</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>28.83</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>20</td>
<td>32.35</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>39.25</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>19.90</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>A</td>
<td>20</td>
<td>34.10</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>39.20</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>18.20</td>
<td></td>
</tr>
</tbody>
</table>

This table reflects that at treatment 1, the null hypothesis was accepted (P ≥ 0.05) indicating no difference between groups A (continuous), B (pulsed) and C (sham).

At treatment 2 and 4, the null hypothesis was rejected (P < 0.05) indicating statistically significant improvement between groups A, B and C.
Table 11 Statistical results of the Myofascial Diagnostic Scale Scores for groups A, B, and C

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Group</th>
<th>N</th>
<th>Mean Rank</th>
<th>P-Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>A</td>
<td>20</td>
<td>35.20</td>
<td>.291</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>27.63</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>28.67</td>
<td></td>
</tr>
<tr>
<td>2</td>
<td>A</td>
<td>20</td>
<td>29.88</td>
<td>.002</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>21.13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>40.50</td>
<td></td>
</tr>
<tr>
<td>4</td>
<td>A</td>
<td>20</td>
<td>25.33</td>
<td>.006</td>
</tr>
<tr>
<td></td>
<td>B</td>
<td>20</td>
<td>25.60</td>
<td></td>
</tr>
<tr>
<td></td>
<td>C</td>
<td>20</td>
<td>40.58</td>
<td></td>
</tr>
</tbody>
</table>

This table reflects that at treatment 1, the null hypothesis was accepted (P ≥ 0.05) indicating no difference between groups A (continuous), B (pulsed) and C (sham).

At treatment 2 and 4, the null hypothesis was rejected (P < 0.05) indicating statistically significant improvement between groups A, B and C.
4.8 Tables of Intra-group Analysis

4.8.1 Objective data

Table 12 Statistical results using Friedman’s test to analyse results obtained from Algometer readings at treatment, one, two and four.

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>N</th>
<th>Mean</th>
<th>Mean ranks</th>
<th>P-Value</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>A</td>
<td>1</td>
<td>20</td>
<td>1.73</td>
<td>1.30</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>2.52</td>
<td>1.83</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>4.24</td>
<td>2.88</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>1</td>
<td>20</td>
<td>1.73</td>
<td>1.08</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>2.83</td>
<td>2.00</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>4.82</td>
<td>2.92</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td>20</td>
<td>1.63</td>
<td>1.30</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>1.90</td>
<td>2.08</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>2.60</td>
<td>2.63</td>
<td></td>
</tr>
</tbody>
</table>

The data reveals that the null hypothesis was rejected (P < 0.05) for the Algometer readings, indicating a statistically significant improvement among all groups over the treatment period.
Table 13 Statistical results using Friedman’s test to analyse results obtained from Myofascial Diagnostic scale readings at treatment, one, two and four.

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatments</th>
<th>N</th>
<th>Mean</th>
<th>Mean ranks</th>
<th>P-Values</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>20</td>
<td>15.90</td>
<td>2.78</td>
<td>.000</td>
</tr>
<tr>
<td>A</td>
<td>1</td>
<td></td>
<td>13.25</td>
<td>2.10</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>7.60</td>
<td>1.13</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td></td>
<td>20</td>
<td></td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>1</td>
<td></td>
<td>15.80</td>
<td>2.83</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>11.95</td>
<td>2.08</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td></td>
<td>7.35</td>
<td>1.15</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td></td>
<td>15.55</td>
<td>2.55</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td></td>
<td>15.30</td>
<td>2.35</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td></td>
<td>10.80</td>
<td>1.10</td>
<td></td>
</tr>
</tbody>
</table>

The data reveals that the null hypothesis was rejected (P < 0.05) for the myofascial diagnostic scale readings, indicating a statistically significant improvement among all groups over the treatment period.
### 4.8.2 Subjective data

**Table 14 Statistical results using Friedman’s test to analyse results obtained from the Short Form McGill Pain Questionnaire readings at treatment, one, two and four.**

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>N</th>
<th>Mean</th>
<th>Mean ranks</th>
<th>P-Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1</td>
<td>20</td>
<td>37.45</td>
<td>2.80</td>
<td>1.92</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>27.56</td>
<td>1.92</td>
<td>1.27</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>16.33</td>
<td>1.27</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td>B</td>
<td>1</td>
<td>20</td>
<td>41.44</td>
<td>2.90</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>27.22</td>
<td>1.83</td>
<td>1.27</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>19.89</td>
<td>1.27</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td>20</td>
<td>45.89</td>
<td>2.28</td>
<td>.079</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>42.99</td>
<td>2.13</td>
<td>1.60</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>35.13</td>
<td>1.60</td>
<td></td>
</tr>
</tbody>
</table>

The data reveals that the null hypothesis was rejected (P < 0.05) for groups A (continuous) and B (pulsed), indicating a significant improvement over the treatment period. The null hypothesis was accepted (P ≥ 0.05) for group C (sham) indicating no improvement over the treatment period.
Table 15 Statistical results using Friedman’s test to analyse results obtained from the Numerical Pain Rating Scale-101 readings at treatment, one, two and four.

<table>
<thead>
<tr>
<th>Group</th>
<th>Treatment</th>
<th>N</th>
<th>Mean</th>
<th>Mean ranks</th>
<th>P-Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>1</td>
<td>20</td>
<td>55.8</td>
<td>2.88</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>40.9</td>
<td>1.85</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>30.0</td>
<td>1.27</td>
<td></td>
</tr>
<tr>
<td>B</td>
<td>1</td>
<td>20</td>
<td>59.6</td>
<td>2.90</td>
<td>.000</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>39.5</td>
<td>1.98</td>
<td>(&lt;.001)</td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>23.3</td>
<td>1.13</td>
<td></td>
</tr>
<tr>
<td>C</td>
<td>1</td>
<td>20</td>
<td>55.0</td>
<td>2.58</td>
<td>.001</td>
</tr>
<tr>
<td></td>
<td>2</td>
<td>20</td>
<td>50.2</td>
<td>1.85</td>
<td></td>
</tr>
<tr>
<td></td>
<td>4</td>
<td>20</td>
<td>44.9</td>
<td>1.58</td>
<td></td>
</tr>
</tbody>
</table>

The data reveals that the null hypothesis was rejected for the NRS-101 (P < 0.05), indicating a statistically significant improvement among all groups over the treatment period.
4.9 Multiple Comparison Tests

4.9.1 The Dunn Procedure for the Kruskal-Wallis H test.

If \( |R_J - R_{J^1}| \geq z_{1-\frac{\alpha}{k(k-1)}} \sqrt{\frac{N(N+1)}{12} \left[ \frac{1}{n_1} + \frac{1}{n_j} \right]} \), then the difference \( |R_J - R_{J^1}| \) are declared significant at the \( \alpha \) level.

In the above formula: \( k \) (no. of samples) = 3
\( N \) (no. of observations) = 60
\( Z = 1.96 \)

\( z_{1-\frac{\alpha}{k(k-1)}} \sqrt{\frac{N(N+1)}{12} \left[ \frac{1}{n_1} + \frac{1}{n_j} \right]} = 10.82 \)

thus if \( |R_J - R_{J^1}| \geq 10.82 \) then \( R_J - R_{J^1} \) is declared significant.

For the purpose of this study \( R_1 \) is the 1st treatment, \( R_2 \) is the 2nd treatment and \( R_4 \) is the 4th treatment.

4.9.1.1 Inter-group Analysis

Table 16 Dunn Procedure for Algometer Readings.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Rank Average</th>
<th>Difference</th>
<th>Rank Average</th>
<th>Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>34.10</td>
<td>5.00</td>
<td>39.20</td>
<td>B</td>
</tr>
<tr>
<td>B</td>
<td>18.20</td>
<td>20.90*</td>
<td>18.20</td>
<td>C</td>
</tr>
<tr>
<td>A</td>
<td>34.10</td>
<td>15.90*</td>
<td>18.20</td>
<td>C</td>
</tr>
</tbody>
</table>
The data reveals **statistically significant differences** (\(| R_j - R_j^1 | \geq 10.82\)) between groups B (pulsed) & C (sham) and A (continuous) & C, while no difference was observed between groups A and B.

### Table 17 Dunn Procedure for the Myofascial Diagnostis Scale Scores.

<table>
<thead>
<tr>
<th>Group</th>
<th>Rank Average</th>
<th>Difference</th>
<th>Rank Average</th>
<th>Group</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>25.33</td>
<td>0.27</td>
<td>25.60</td>
<td>B</td>
</tr>
<tr>
<td>B</td>
<td>25.60</td>
<td>14.98*</td>
<td>40.58</td>
<td>C</td>
</tr>
<tr>
<td>A</td>
<td>25.33</td>
<td>15.25*</td>
<td>40.58</td>
<td>C</td>
</tr>
</tbody>
</table>

The data reveals **statistically significant differences** (\(| R_j - R_j^1 | \geq 10.82\)) between groups B (pulsed) & C (sham) and A (continuous) & C, while no difference was observed between groups A and B.

### Table 18 Dunn Procedure for the NRS-101 Scores.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Rank Average</th>
<th>Difference</th>
<th>Rank Average</th>
<th>Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>28.83</td>
<td>7.16</td>
<td>21.67</td>
<td>B</td>
</tr>
<tr>
<td>B</td>
<td>21.67</td>
<td>19.33*</td>
<td>41.00</td>
<td>C</td>
</tr>
<tr>
<td>A</td>
<td>28.83</td>
<td>12.17*</td>
<td>40.75</td>
<td>C</td>
</tr>
</tbody>
</table>

The data reveals **statistically significant differences** (\(| R_j - R_j^1 | \geq 10.82\)) between groups B (pulsed) & C (sham) and A (continuous) & C, while no difference was observed groups A and B.
Table 19 Dunn Procedure for the Short-Form McGill Pain Questionnaire.

<table>
<thead>
<tr>
<th>Groups</th>
<th>Rank Average</th>
<th>Difference</th>
<th>Rank Average</th>
<th>Groups</th>
</tr>
</thead>
<tbody>
<tr>
<td>A</td>
<td>25.33</td>
<td>0.09</td>
<td>25.42</td>
<td>B</td>
</tr>
<tr>
<td>B</td>
<td>25.42</td>
<td>15.33*</td>
<td>40.75</td>
<td>C</td>
</tr>
<tr>
<td>A</td>
<td>25.33</td>
<td>15.42*</td>
<td>40.75</td>
<td>C</td>
</tr>
</tbody>
</table>

The data reveals statistically significant differences (\( |R_J - R_J^1| \geq 10.82 \)) between groups B (pulsed) & C (sham) and A (continuous) & C, while no difference was observed between groups A and B.

4.9.2 Friedman’s T Test coupled with the Dunn procedure.

Let \( R_J \) and \( R_J^1 \) be the \( j^{th} \) and \( j^{1th} \) treatment rank totals.

Let \( \alpha \) be the experimental wise error rate. Usually =0.10

If \( |R_J - R_J^1| \geq z\sqrt{bk(k+1)/6} \), then \( R_J \) and \( R_J^1 \) are declared significant.

In the above \( b= \) no. of blocks

\( k= \) no. of treatments at which data was recorded.

\( Z= \) value of the inverse normal distribution corresponding to \( 1-(\alpha/k(k+1)) \)

for this study, \( k=3, b=20, \) and \( \alpha=0.10. \)

i.e if the difference of the rank totals is \( \geq 13.41 \), then \( R_J \) and \( R_J^1 \) are declared significant.
For the purpose of this study $R_1$ is the $1^{st}$ treatment, $R_2$ is the $2^{nd}$ treatment and $R_4$ is the $4^{th}$ treatment.
4.9.2.1 Intra-Group Analysis

* STATISTICALLY SIGNIFICANT

Table 20 Dunn Procedure for Group A- Algometer Readings.

<table>
<thead>
<tr>
<th>RANK TOTAL</th>
<th>DIFFERENCE</th>
<th>RANK TOTAL</th>
</tr>
</thead>
<tbody>
<tr>
<td>R₁</td>
<td>26.00</td>
<td>36.60</td>
</tr>
<tr>
<td>R₂</td>
<td>36.60</td>
<td>57.60</td>
</tr>
<tr>
<td>R₁</td>
<td>28.80</td>
<td>57.60</td>
</tr>
</tbody>
</table>

The table reveals that $R₁ - R₂ = 10.6 < 13.41$, therefore between treatments 1 and 2 there is no improvement.

$R₂ - R₄ = 21 \geq 13.41$, therefore between treatments 2 and 4 there is statistically significant improvement.

$R₁ - R₄ = 32.60 \geq 13.41$, therefore between treatments 1 and 4 there is statistically significant improvement.

Table 21 Dunn Procedure for Group B- Algometer Readings.

<table>
<thead>
<tr>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>R₁</td>
<td>21.60</td>
<td>40.00</td>
</tr>
<tr>
<td>R₂</td>
<td>40.00</td>
<td>58.40</td>
</tr>
<tr>
<td>R₁</td>
<td>21.60</td>
<td>58.40</td>
</tr>
</tbody>
</table>
The table reveals that $R_1 - R_2 = 8.40 \geq 13.41$, therefore between treatments 1 and 2 there is statistically significant improvement.

$R_2 - R_4 = 18.41 \geq 13.41$, therefore between treatments 2 and 4 there is statistically significant improvement.

$R_1 - R_4 = 36.80 \geq 13.41$, therefore between treatments 1 and 4 there is statistically significant improvement.

**Table 22 Dunn Procedure for Group C- Algometer Readings.**

<table>
<thead>
<tr>
<th></th>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>$R_1$</td>
<td>26.00</td>
<td>15.60*</td>
<td>41.60</td>
<td>$R_2$</td>
</tr>
<tr>
<td>$R_2$</td>
<td>41.60</td>
<td>11.00</td>
<td>52.60</td>
<td>$R_4$</td>
</tr>
<tr>
<td>$R_1$</td>
<td>26.00</td>
<td>26.60*</td>
<td>52.60</td>
<td>$R_4$</td>
</tr>
</tbody>
</table>

The table reveals that $R_1 - R_2 = 5.60 \geq 13.41$, therefore between treatments 1 and 2 there is statistically significant improvement.

$R_2 - R_4 = 11.00 < 13.41$, therefore between treatments 2 and 4 there is no improvement.

$R_1 - R_4 = 26.60 \geq 13.41$, therefore between treatments 1 and 4 there is statistically significant improvement.
Table 23 Dunn Procedure for Group A- Myofascial Diagnostic scale Readings.

<table>
<thead>
<tr>
<th></th>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>( R_1 )</td>
<td>55.60</td>
<td>13.60*</td>
<td>42.00</td>
</tr>
<tr>
<td>( R_2 )</td>
<td>42.00</td>
<td>19.40*</td>
<td>22.6</td>
</tr>
<tr>
<td>( R_1 )</td>
<td>55.60</td>
<td>33.00*</td>
<td>22.60</td>
</tr>
</tbody>
</table>

The table reveals that \( R_1 - R_2 = 13.60 \geq 13.41 \), therefore between treatments 1 and 2 there is statistically significant improvement.

\( R_2 - R_4 = 19.40 \geq 13.41 \), therefore between treatments 2 and 4 there is statistically significant improvement.

\( R_1 - R_4 = 33.00 \geq 13.41 \), therefore between treatments 1 and 4 there is statistically significant improvement.

Table 24 Dunn Procedure for Group B- Myofascial Diagnostic scale Readings.

<table>
<thead>
<tr>
<th></th>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>( R_1 )</td>
<td>56.60</td>
<td>16.00*</td>
<td>40.60</td>
</tr>
<tr>
<td>( R_2 )</td>
<td>40.60</td>
<td>17.60*</td>
<td>23.00</td>
</tr>
<tr>
<td>( R_1 )</td>
<td>56.60</td>
<td>33.60*</td>
<td>23.00</td>
</tr>
</tbody>
</table>

The table reveals that \( R_1 - R_2 = 16.00 \geq 13.41 \), therefore between treatments 1 and 2 there is statistically significant improvement.
\[ R_2 - R_4 = 17.60 \geq 13.41, \text{ therefore between treatments 2 and 4 there is statistically significant improvement.} \]

\[ R_1 - R_4 = 33.60 \geq 13.41, \text{ therefore between treatments 1 and 4 there is statistically significant improvement.} \]

**Table 25 Dunn Procedure for Group C- Myofascial Diagnostic scale Readings.**

<table>
<thead>
<tr>
<th></th>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>( R_1 )</td>
<td>51.00</td>
<td>10.40</td>
<td>40.60</td>
<td>( R_2 )</td>
</tr>
<tr>
<td>( R_2 )</td>
<td>40.60</td>
<td>18.60*</td>
<td>22.00</td>
<td>( R_4 )</td>
</tr>
<tr>
<td>( R_1 )</td>
<td>51.00</td>
<td>29.00*</td>
<td>22.00</td>
<td>( R_4 )</td>
</tr>
</tbody>
</table>

The table reveals that \( R_1 - R_2 = 10.40 \leq 13.41 \), therefore between treatments 1 and 2 there is **no improvement**.

\[ R_2 - R_4 = 18.60 \geq 13.41, \text{ therefore between treatments 2 and 4 there is statistically significant improvement.} \]

\[ R_1 - R_4 = 29.00 \geq 13.41, \text{ therefore between treatments 1 and 4 there is statistically significant improvement.} \]
**Table 26 Dunn Procedure for Group A NRS-101 Readings.**

<table>
<thead>
<tr>
<th></th>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>R₁</td>
<td>57.60</td>
<td>20.60*</td>
<td>37.00</td>
<td>R₂</td>
</tr>
<tr>
<td>R₂</td>
<td>37.00</td>
<td>11.60</td>
<td>25.40</td>
<td>R₄</td>
</tr>
<tr>
<td>R₁</td>
<td>57.60</td>
<td>32.20*</td>
<td>25.40</td>
<td>R₄</td>
</tr>
</tbody>
</table>

The table reveals that $R₁ - R₂ = 20.60 \geq 13.41$, therefore between treatments 1 and 2 there is **statistically significant improvement**.

$R₂ - R₄ = 11.60 < 13.41$, therefore between treatments 2 and 4 there is **no improvement**.

$R₁ - R₄ = 32.20 \geq 13.41$, therefore between treatments 1 and 2 there is **statistically significant improvement**.
Table 27 Dunn Procedure for Group B- NRS-101 Readings.

<table>
<thead>
<tr>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>R₁</td>
<td>58.00</td>
<td>18.40*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>39.60</td>
</tr>
<tr>
<td>R₂</td>
<td>39.60</td>
<td>17.00*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>22.60</td>
</tr>
<tr>
<td>R₁</td>
<td>58.00</td>
<td>35.40*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>22.60</td>
</tr>
</tbody>
</table>

The table reveals that $R₁ - R₂ = 18.40 \geq 13.41$, therefore between treatments 1 and 2 there is **statistically significant improvement**.

$R₂ - R₄ = 17.00 \geq 13.41$, therefore between treatments 2 and 4 there is **statistically significant improvement**.

$R₁ - R₄ = 35.40 \geq 13.41$, therefore between treatments 1 and 4 there is **statistically significant improvement**.

Table 28 Dunn Procedure for Group C- NRS-101 Readings.

<table>
<thead>
<tr>
<th>Rank Totals</th>
<th>Difference</th>
<th>Rank Totals</th>
</tr>
</thead>
<tbody>
<tr>
<td>R₁</td>
<td>51.60</td>
<td>25.40*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>37.00</td>
</tr>
<tr>
<td>R₂</td>
<td>37.00</td>
<td>5.40</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31.60</td>
</tr>
<tr>
<td>R₁</td>
<td>51.60</td>
<td>20.00*</td>
</tr>
<tr>
<td></td>
<td></td>
<td>31.60</td>
</tr>
</tbody>
</table>

The table reveals that $R₁ - R₂ = 25.40 \geq 13.41$, therefore between treatments 1 and 2 there is **statistically significant improvement**.

$R₂ - R₄ = 5.40 \leq 13.41$, therefore between treatments 2 and 4 there is **no improvement**.
\( R_1 - R_4 = 20.00 \geq 13.41 \), therefore between treatments 1 and 4 there is 
\textit{statistically significant improvement}. 
5.1 Demographic Data

5.1.1 Gender distribution

The distribution of men and women (Table 1) among the three groups were similar, however women comprised 70% of the participants in the study. The significantly higher participation of women in this study is consistent with the results of myofascial pain syndrome studies conducted by Broome (1996) 83.3%, Miller (2000) 63.3%, van Aardenne (2002) 66.6% and Prithipal (2003) 68.33%. Severino and Moline (1990), reported a possible hormonal link to the severity of myofascial pain syndrome, as they found that women experienced more pain during the second week of their menstrual cycles. Other factors that need consideration when assessing the greater female participation, include the higher female to male ratio of the population in which the study was conducted together with the possibility that women may have shown a greater interest to advertisements and participation in the study.

5.1.2 Age distribution

The age range for the study was 18-35 with similar mean ages (Table 2) reported for each group. Patients were also evenly distributed across the age range with 33.3% in the 18-23-age range, 38.33% and 28.33% in the 24-30 and 31-35 ranges respectively. The age restriction of 18-35 ensured a young population of patients and minimized pain that may be caused by accompanying degenerative joint and disc disease. Esenyel et al (2000)
also utilized an age restriction of 18-35 for the same reasons. Miller (2000) reported an average age of 31 and 50% of the participants between the age of 20-30, Rowley (2001) reported an average age of 30.26 with 86.99% between the age of 17-37 and van Aardenne(2002) also reporting similar findings, with 53.3% between the ages of 20-35. These studies had been conducted at a tertiary institution with significant student participation (Miller (2000) 23.33%, Rowley (2001) 10% and van Aardenne (2002) 25%) and this may be responsible for a lower average age, when compared to Travell et al (1999) reporting the 30 –49 age group as having the highest prevalence of trigger points.

5.1.3 Occupation distribution

When considering subject's occupations and working environments, 55% worked in an office environment predominantly at a desk using a computer. The role of posture, ergonomics and constant exposure to an air-conditioned environment, play a vital role in development and perpetuation of myofascial trigger points. Working in an office environment also reduces vocational physical activity and as reported earlier, Sola et al (1981) discovered that sedentary workers were more susceptible to myofascial pain syndrome than labourers, implying a protective effect of daily vigorous activity.

5.1.4 Ethnic group distribution

When examining the ethnic group distribution (Table 4) in this and other studies, there seems to be no link between ethnicity and the development of myofascial pain syndrome and it presents little or no epidemiological value.
5.2 trigger point characteristics

5.2.1 Trigger point distribution

Eighty-five percent of patients presented with active Trapezius Tp1, which was found to be more prevalent than Tp2 in all three groups (Table 5). The greater prevalence of Tp1 compared to Tp2 in this study was consistent with Travell and Simons (1999), however in this study Tp1 and Tp2 occurred more commonly on the right (61.66%) than the left (38.33%). When considering the handedness of the sample group 88.33% were right handed, 8.33% were left handed and 3.33% were ambidextrous, this may indicate a correlation between the predominant use of one hand and the development of ipsilateral upper trapezius trigger points.

5.2.2 Duration of trigger point symptoms.

Thirty-three percent of the patients in this study had chronic symptoms (Table 6). Travel et al (1999) reports that acute single trigger points are simple and easy to diagnose and treat, while if they are allowed to become chronic, they are complicated, more painful, time-consuming and expensive to treat thus the significant number (33%) of chronic patients in this study may have influenced the outcomes or the investigation.
5.3 Subjective Data

5.3.1 Numerical pain rating scale – 101

This scale is used to monitor levels of pain perception and quality, experienced by patients. A reduction in the mean score indicates an improvement in the pain experience.

Inter-group comparison

At treatment one the null hypothesis was accepted indicating no differences between groups A (continuous), B (pulsed) and C (placebo). This indicated a homogenous distribution of patients with regard to their levels of pain perception.

At treatment two and four the null hypothesis was rejected indicating a statistically significant difference between groups A (continuous), B (pulsed) and C (placebo), as a result multiple comparison tests were performed utilizing the Dunn procedure. This revealed no statistically significant difference between the treatment groups A (continuous) and B (pulsed), while statistically significant differences were observed between the treatment groups (A and B) and the placebo controlled group (C).

Chettiar (2001) investigated the efficacy of action potential therapy in the treatment of myofascial pain syndrome using the same subjective and objective measures as this study. He also reported that data from the numerical pain rating scale-101 after the first treatment revealed no difference between the treatment and control groups, similarly, Chettiar (2001) reported significant differences between the treatment and control group after the fourth treatment.
Intra-group analysis

The null hypothesis was rejected for all three groups indicating statistically significant improvement among all three groups.

This necessitated multiply comparison tests using the Dunn procedure. In-group A (continuous) the data provided by the NRS-101 shows significant improvement between treatments one and two, and one and four, while no improvement between treatments two and four. In group B (pulsed) there was statistically significant improvement between all treatments, one to two, two to four, and one to four. Group C (placebo) showed significant improvement between treatments one and two, and one and four, while no improvement between treatments two and four.

Chettiar (2001) reported statistically significant improvement in the treatment group over the treatment period, however in contrast no significant improvement within the control group. The control group in this study received placebo action potential therapy, with the electrodes placed over the trigger point, without the emission of low frequency current. In contrast, the application of placebo ultrasound involves the rhythmic movement of the transducer head over the trigger point, with the muscle held in a slight stretch thus generating a therapeutic massage effect. This may have contributed to the statistically significant improvement observed within the control (sham ultrasound) group.
5.3.2 Short-form McGill pain questionnaire SF-MPQ

The data obtained with the SF-MPQ according to Melzack (1987) provides information regarding sensory, affective and overall intensity of pain. An improvement is noted by a decrease in the scores.

Inter-group comparisons

At treatment one, the null hypothesis was accepted indicating no differences between groups A (continuous), B (pulsed) and C (sham). This indicated a homogenous distribution of patients among the three groups.

At treatment two and four the null hypothesis was rejected indicating a statistically significant difference between groups A, B and C, as a result multiple comparison tests were performed utilizing the Dunn procedure. This revealed no statistically significant difference between the treatment groups A (continuous) and B (pulsed), while statistically significant differences were observed between the treatment groups (A and B) when compared to the placebo controlled group (C).

The inter-group statistical results were consistent between the two subjective measures and revealed that although no statistical difference could be determined between groups A and B, there was a significant difference between these two treatment groups and, the placebo controlled group C.

Similarly Chettiar (2001) reported no differences between the treatment and control groups at the first treatment, but also reported significant differences between the treatment and control group after the fourth visit. Van Aardenne (2002) also used the same subjective and objective measures
when investigating the efficacy of Magnesium Phosphate, as an adjunct to dry needling in the treatment of myofascial pain syndrome. She reported that data from the Short-form McGill pain Questionnaire revealed no difference between the treatment group (dry needling and Magnesium Phosphate) and the control group (only dry needling).

**Intra-group comparison**

The null hypothesis was rejected for groups A (continuous) and B (pulsed) indicating significant improvement between the first and fourth treatments.

The null hypothesis was accepted for group C (the placebo control) indicating no improvement over the treatment period.

In contrast Chettiar (2001) and Van Aardenne(2002) reported that data from the SF-MPQ revealed significant improvement for both the treatment and control groups.

### 5.4 Objective Data

#### 5.4.1 Algometer readings

The algometer was used to measure the amount of force that the patient could tolerate on the TrP. An increase in readings indicates an increase in pain threshold resulting from decreased TrP sensitivity.

**Inter group comparison**

The null hypothesis was rejected for data collected at treatment one indicating no difference between groups A (continuous), B (pulsed) and C
(pulsed) and again (P value .774) this reflected a homogenous and indiscriminate distribution of subjects between the three groups. Chettiar (2001) also reported a homogenous sample at treatment one with the rejection of the null hypothesis indicating no statistically significant difference between the treatment and control group.

At treatment two and four the null hypothesis was rejected indicating a statistically significant difference between groups A (continuous), B (pulsed) and C (placebo), as a result multiply comparison tests were performed utilizing the Dunn procedure. This revealed no statistically significant difference between the treatment groups A and B, while statistically significant differences were observed between the treatment groups (A and B) when compared to the placebo controlled group (C). Van Aardenne (2002) and Chettiar (2001) also reported significant differences between the treatment and control groups.

**Intra-group comparison**

The null hypothesis was rejected for all three groups indicating statistically significant improvement in all three groups.

When the Dunn Procedure was coupled with the Friedman s T test, group A showed no significant improvement between the first and second, but significant improvement was observed between the second and fourth and first and fourth treatments. Treatment group B, showed significant improvement between all treatments, one to two, two to four and one to four. The placebo controlled group showed improvement from treatments one to two and one to four but no improvement between two and four.
5.4.2 Myofascial Diagnostic Scale

This scale was used to show the extent to which patients were suffering from MPS and a decrease in scores would indicate an improvement.

Inter-group comparison

Consistent with the other measures the null hypothesis was again accepted at treatment one indicating that patients within the three groups were homogenously distributed with regards to the extent of their symptoms.

At treatment, two and four the null hypothesis was again rejected indicating a statistically significant difference between groups A (continuous), B (pulsed) and C (sham), as a result multiple comparison tests were performed utilizing the Dunn procedure. Consistent with all other inter-group analysis including subjective data, no statistically significant difference was observed between the treatment groups A (continuous) and B (pulsed), while statistically significant differences were observed between the treatment groups (A and B) when compared to the placebo controlled group(C).

Intra-group comparison

The null hypothesis was rejected for all three groups indicating statistically significant improvement among all three groups.

This necessitated multiply comparison tests using the Dunn procedure. Group A (continuous) and B (pulsed) statistically significant improvement between all treatments, one to two, two to four and one to four.
The placebo controlled, group C demonstrated no improvement between treatment one and two but significant improvement between treatments two and four and one and four.

Assessment of the statistical analysis revealed a consistency between the objective and subjective data. This consistency validates the sensitivity of the measures and negates the influence of bias due to patient mood, attitude to treatment and consistency in the researchers collection of objective data.

The Myofascial Diagnostic Scale provided data that was consistent with that of the validated and widely used pressure algometer.
5.5 Discussion

This study was different from other myofascial studies conducted at the Durban Institute of Technology, especially with regards to its design. A double-blinded placebo-controlled study is often difficult to implement in the investigation of myofascial pain syndrome. However, the strength of the study was increased by reducing patient and researcher bias and the addition of a placebo control group enhanced the value of the study.

During the course of the study four participants (6.25%), dropped out of the study for the following reasons:

- One developed measles and was excluded.
- Two patients did not complete the course of treatments within the prescribed period (these were students and the treatment period coincided with their holidays).
- One patient was not satisfied with only receiving ultrasound therapy and preferred treatment as a normal patient in the Chiropractic clinic.

The age restriction (18-35) minimized the influence of pain from degenerative joint and disc disease. The provision of information on environmental perpetuating factors to patients in the study enhanced the possible therapeutic influence of Ultrasound.

Evaluation of the data revealed that all three groups (treatment groups A and B, placebo controlled group C) showed a significant decrease in the levels of pain perception, overall pain intensity and extent to which the patients were suffering with myofascial pain syndrome together with an overall increase in pain thresholds.

Binder et al (1985) investigated the effectiveness of therapeutic ultrasound in the treatment of soft tissue lesions. They randomly assigned seventy-six
patients with lateral epicondylitis to either an ultrasound treatment group or a placebo group (sham ultrasound). Binder et al (1985) reported that the two groups showed no significant differences in the mean severity of any of the clinical variables at presentation with 29% of the placebo-controlled group showing satisfactory objective outcome. One of the factors that they attributed the improvement in the placebo group too, was the massage effect of the transducer head over the affected area during mock insonation. The significant improvement of the placebo controlled group in this study may be attributed to proprioceptive stimulation contributing to pain reduction via the Gate Control Theory as described by Melzack and Wall (1965), education on the influence of environmental factors (provision of a list of environmental perpetuating factors to each patient) and natural remission.

When the Dunn Procedure was applied to the Kruskal-Wallis H test, significant differences were observed between the treatment groups (A and B) and the placebo controlled, group C, accepting hypothesis one (1.3.1) and two (1.3.2) respectively.

Evaluation of the two waveforms revealed no statistical difference between Group A (continuous waveform) and Group B (pulsed waveform) and hypothesis three was rejected (1.3.3). However, there were varied responses to the different waveforms. Patients receiving the continuous waveform, showed significant increases in pain threshold only after the second treatment as compared to the immediate (after the first treatment) effect of pulsed Ultrasound. The benefit of significantly reduced levels of pain perception tapered after the second treatment in the continuous ultrasound group while the pulsed ultrasound group B, continued to show significant improvement with subsequent treatments.
When considering the two groups (A and B) over the treatment period, although no statistical difference was observed, the pulsed waveform (Group B) showed an immediate effect in decreasing trigger point sensitivity and gave an indication that further treatments would further decrease the levels of pain perception and increase pain thresholds when compared to the continuous waveform (Group A). Although both the continuous (Group A), as reported by Lehman et al (1972), and the pulsed (Group B) waveforms as reported by Hogan et el (1982) are responsible for an increase in blood-flow, essential to the treatment of myofascial trigger points, the immediate decrease in trigger point sensitivity and more sustained decrease in the levels of pain perception and thresholds observed in the Group B (pulsed) may be attributed to the observed changes in cell membrane permeability to ions such as Calcium due to acoustic streaming, as reported by Dyson(1987). Acoustic streaming as reported by Dyson (1987) is generated by the non-thermal effect of the pulsed wave-form of ultrasound. The more sustained improvement seen in the pulsed waveform group may be attributed to the therapeutically significant benefit of non-thermal ultrasound’s ability to stimulate protein synthesis, soft tissue repair and subsequent tissue regeneration as reported by Hogan et al (1982)

These therapeutic benefits of pulsed ultrasound are clearly of important therapeutic value in the recovery of tissue because of damage, especially due to chronic ischemia as seen in myofascial trigger points.
5.6 Limitations of the study.

The study acknowledged the duration of the patients symptoms but it did not incorporate this factor into the study design. Travel and Simons (1999) reported that chronic trigger points are more difficult to manage and require more time to respond to therapy. This may have influenced the data.

The use of an objective measure (Myofascial Diagnostic Scale) that has not been validated in a clinical trial is not desirable although it was evident in the study that the data it provided was consistent with that of the well validated widely used Algometer. The Scale was adequate for this study where only one trigger point was treated but will be limited when used to assess multiple trigger points.

Only trigger points in the upper trapezius were evaluated. Travel and Simons (1999) reports that all trigger points in the myotic unit needs to be evaluated and treated, disregard of other trigger points may cause treatment failure.

This study could not comment on the sustained benefits therapeutic ultrasound, as a follow-up evaluation was not incorporated into the study design.

This study did not take into consideration perpetuating factors other than environmental ones. This is an important factor in the prognosis of myofascial pain syndrome and deserves consideration in clinical studies.
Chapter Six

Conclusion and Recommendations

6.1 Conclusion

The purpose of this study was to evaluate the efficacy of the two forms of ultrasound (continuous and pulsed) when compared to a placebo controlled group receiving sham ultrasound (and to also determine the relative effectiveness of the two waveforms of ultrasound, continuous and pulsed).

This study has shown that both waveforms of Ultrasound are more effective than placebo ultrasound in the treatment of myofascial pain syndrome. When considering the relative effectiveness of the two waveforms no statistical significant difference was observed, although the therapeutic benefits of the pulsed waveform seem to be immediate and better sustained over the treatment period.

Esenyel et al (2000) recommended that therapeutic ultrasound should be offered to patients in the treatment of myofascial pain syndrome, especially to patients who want to avoid injections.
6.2 Recommendations

Further investigation is recommended into the possible benefit of proprioceptive stimulation in the management of myofascial pain syndrome as produced by the Ultrasound head over the trigger point.

Ultrasound needs to be further evaluated in a study of similar design (to the present one), but must also consider the duration of symptoms, treating other deeper trigger points, different frequencies, a follow-up evaluation, and greater consideration of perpetuating factors. Research on myofascial pain syndrome also needs to consider the effects of perpetuating factors, especially the psychological aspects relating to anxiety and depression. Little effort is committed to investigating and quantifying the clinical effects of these factors. The clinical value of investigating a single therapeutic tool as in this investigation is questionable; as it is evident, that myofascial pain syndrome is most effectively managed with a multidisciplinary approach.

The myofascial diagnostic scale produced similar data to the algometer in this study but needs to be investigated in a well-designed clinical trial.
REFERENCES


Hutchings, T.A. 1998. The treatment of myofascial syndrome using transcutaneous nerve stimulation: A comparison between two types of


APPENDIX 9

Title of research:
The treatment of myofascial pain syndrome using therapeutic ultrasound, on upper trapezius trigger points: A double-blinded placebo controlled study comparing the pulsed and continuous wave-forms of ultrasound.

Supervisor: Dr A. Docrat

Researcher: M.G. Pillay

The following is a list of environmental factors that needs consideration while on the research:

- Avoid working in an area directly under an air-conditioner duct. Either move your work station or cover the duct.

- Avoid sleeping with a fan directed at your back during the night. Rather turn the fan away.

- If, you use a telephone frequently or for prolonged periods, get a speakerphone or a headset.(never laterally flex the head to hold the phone between the shoulder and ear.)

- Avoid constriction of the upper trapezius by a thin, tight bra-strap by using a wider, non-elastic strap that is worn more laterally on the shoulders.