

THE TREATMENT OF MYOFASCIAL
SYNDROME USING TRANSCUTANEOUS
ELECTRICAL NERVE STIMULATION
(TENS): A COMPARISON BETWEEN
TWO
TYPES OF ELECTRODE PLACEMENTS

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*Dissertation submitted to the faculty of health services in
partial compliance with the requirements for the Master's
Degree in Technology: Chiropractic, at Technikon Natal.*

*I, Tracey Ann Hutchings, do hereby declare that this
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DATE OF SUBMISSION: 20 MAY 1998

20/5/1998

DEDICATION

I would like to dedicate this work to my parents, Ron and Wendy Hutchings. It has been a long road but we finally did it. Thank you for believing in me when I didn't believe in myself. Thank you for your constant love, understanding, patience and support. -
I couldn't have done it without you.

ACKNOWLEDGEMENTS

I would like to thank the following people for their assistance:

Dr R. Mathews, my supervisor. Thank you for your guidance and invaluable advice.

Mr Worku, for patiently assisting me with my statistical analysis.

The Technikon Natal Library staff, especially for your help in ordering research articles.

My good friend Judy Carney for your tireless encouragement over countless cups of coffee.

All the patients who participated in this study without whom this study would not have been possible.

Lastly my friend and saviour, Jesus Christ. I am nothing without Him, but in Him I can do all things.

ABSTRACT

Myofascial syndrome is a very common condition which is frequently encountered at Chiropractic clinics. It is also a very complex condition and as such is a very frustrating one to treat effectively. ~~TENS is recognised as~~ a clinically effective modality in the treatment of Myofascial syndrome, however guidelines with respect to the most effective electrode placements are lacking.

The aim of this investigation is to determine the relative effectiveness of placing electrodes over the active myofascial trigger point with placing them over the pain referral zone in the treatment of Myofascial syndrome using TENS and passive stretching, with reference to both subjective and objective clinical findings, in order to determine which of these methods is more effective.

Thirty subjects, each with complaints of muscular pain of varying degrees and duration, were recruited into this study by means of advertising, referrals and personal communication. The subjects had to be between the ages of 16 and 60 and live within a fifteen kilometre radius of Technikon Natal Chiropractic Clinic. There were no delimitations with respect to race, gender or other co-existing conditions. In order to be included in the study, the subject had to exhibit at least five of the eight diagnostic criteria for Myofascial syndrome and it had to be ensured that the use of TENS on that subject was not contra-indicated. The subjects were divided randomly into two experimental groups.

The relative effectiveness of the two treatments was measured in terms of subjective and objective clinical findings. Subjective findings were obtained from the short form McGill Pain Questionnaire and the Numerical Rating scale 101 which were used to assess the quality and quantity of the pain respectively and the Pain Disability Index, which was used to ~~assess the degree~~ of disability experienced by the subject. Objective findings were obtained using a pressure algometer which measures the subject's pressure threshold and gives an indication of the trigger point intensity.

Due to the small sample size of the study, data analysis involved the use of Non-Parametric tests. The Mann-Whitney unpaired test was used to compare the results between the two groups. Wilcoxon's Signed Rank test was used to compare the results within each group. Three methods were used to measure central tendency; the median, mode and mean. Other summary statistics obtained were standard error and coefficient of variation. The results obtained were presented visually in the form of bar graphs.

Group 1 had electrodes applied directly over the active myofascial trigger point (AMFTP). All of the subjects reported a significant improvement between the first and last treatment in terms of both subjective and objective clinical findings. There was no further significant improvement between the last treatment and the one month follow-up, except in terms of disability, which did continue to decrease.

Group 2 had electrodes placed over the pain referral zone. There was once again a significant improvement between the first and last treatment in terms of both subjective and objective clinical findings. In the case of group 2, the only significant improvement between the last treatment and the one month follow-up consultation was in terms of pain intensity, measured by means of the NRS 101.

There was no significant difference, in terms of subjective or objective clinical findings, between groups one and two. Placing the electrodes over the pain referral zone is therefore as effective as the traditional placement directly over the active myofascial trigger point and is therefore an effective alternative in the treatment of Myofascial syndrome. This may be of use in certain cases where the application of electrodes directly over the AMFTP is not practical or is contra-indicated.

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LIST OF ABBREVIATIONS

α - level of significance of the test

S - significant difference

NS - no significant difference

~~T11 - first treatment, Group 1~~

T21 - final treatment, Group 1

T31 - one month follow-up, Group 1

T12 - first treatment, Group 2

T22 - final treatment, Group 2

T32 - one month follow-up, Group 2

If $p \leq 0.025$ there is a significant difference

If $p > 0.025$ there is no significant difference

DEFINITION OF TERMS

Active Myofascial Trigger Point

A focus of hyperirritability in a muscle or its fascia that is symptomatic with respect to pain; it refers a pattern of pain at rest and/or on motion that is specific for that muscle. An active trigger point is always tender, prevents full lengthening of the muscle, weakens the muscle, usually refers pain on direct compression, mediates a local twitch response of muscle fibres when adequately stimulated and often produces specific referred autonomic phenomena, generally in its pain referral zone (Travell and Simons 1983 1:1)

Effectiveness

The ability to produce a specific result or to exert a specific measurable influence (Dorland's 1988)

Myofascial Syndrome

Pain and/or autonomic phenomena referred from active myofascial trigger points with associated dysfunction (Travell and Simons 1983 1:3)

Objective Clinical Findings

Findings that are perceptible to the external senses. (Dorland's 1988) In this study they refer to findings on full physical examination, localised digital palpation, algometer readings and the diagnostic criteria stipulated by Travell and Simons (1983 1:18-19)

Pain referral zone

The specific region of the body at a distance from a trigger point, where the phenomena (sensory, motor, autonomic) that it causes are observed. (Travell and Simons 1983 1:4)

Subjective Clinical Findings

Findings perceived only by the affected individual. (Dorland's 1988) In this study they refer to readings obtained from the McGill short form pain questionnaire, the Numerical Rating scale 101 and the Pain disability index.

Transcutaneous Electrical Nerve stimulation

The application of a pulsed rectangular wave current via surface electrodes on the patient's skin. (Forster and Palastanga:103)

CHAPTER ONE: INTRODUCTION

1.1. DEFINITIONS

1.1.1. Myofascial syndrome (MFS) describes a condition in which pain and/or autonomic phenomena are referred from active myofascial trigger points (AMFTPs) with associated dysfunction. (Travell and Simons 1983 1:3.)

1.1.2. An active myofascial trigger point (AMFTP) is a focus of hyperirritability in a muscle or its fascia that is symptomatic with respect to pain; it refers a pattern of pain at rest and/or on motion that is specific for the muscle. An AMFTP is always tender, prevents full lengthening of the muscle, weakens the muscle, usually refers pain on direct compression, mediates a local twitch response of muscle fibres when adequately stimulated and often produces specific referred autonomic phenomena generally in its pain reference zone. (Travell and Simons 1983 1:1.)

1.2. EXTENT

Myofascial syndrome is an extremely common cause of chronic musculoskeletal pain and disability. Simons (1988) found that by far the majority of patients (85 %) admitted to a certain chronic pain centre, suffered primarily from Myofascial syndrome. These findings strongly supported those of Fishbain et al. (1986) who conducted a study on 283 patients presenting

to a chronic pain clinic. Eighty five percent of these patients were also diagnosed with Myofascial syndrome.

The fact that it appears to be so neglected by the medical profession is possibly due to the confusion which has arisen ~~because of the number of different terms which are used to~~ describe the same condition. Diagnoses such as "Muscular Rheumatism", "Myalgia", "Myofasciitis" and "Fibrositis" are all synonymous with Myofascial syndrome. (Travell and Simons 1983:6-12.) This confusion of terms makes accurate studies of incidence and prevalence of this condition difficult.

1.3. TREATMENT AIM

Myofascial syndrome is a local muscular condition which develops as a result of acute or chronic muscle strain. This results in a domino-like effect of tissue damage, sustained intramuscular contraction, uncontrolled metabolism, ischaemia, accumulation of metabolites and eventually in contracture. Stimulation of nociceptors within the muscle results in the characteristic pain associated with Myofascial syndrome. (Gatterman 1990:291,293.) Treatment is aimed at disrupting these reverberating neural circuits which are responsible for the "memory" of pain. One way in which this can be accomplished is by hyperstimulation analgesia - one of the oldest methods of pain relief. By terminating the pain, function and abnormal neural activity can be normalized (Melzack 1981.)

To date, many techniques have been used in the treatment of Myofascial syndrome. These include amongst others; ultrasound, ischaemic compression, massage, dry needling, stretch and spray, stretch without spray, moist heat, drug therapy, biofeedback and transcutaneous electrical nerve stimulation (TENS) (Travell and Simons 1983 1:27,86-92.)

1.4. TREATMENT EFFECTIVENESS

Effectiveness is obviously of primary importance when choosing a treatment modality, however other factors such as cost, availability and patient suitability also need to be considered.

1.4.1. ISCHAEMIC COMPRESSION

Ischaemic compression is easy to perform by both the doctor and patient and requires no additional equipment. Needling has been shown to be a more effective method of treatment (Kraft 1968), although ischaemic compression is a valuable alternative in certain areas like the infraspinatus and sternalis muscles which are relatively thin and overlie bone (Travell and Simons 1983 1:87.)

1.4.2. NEEDLING

Needling of AMFTPs is a well known and very effective form of treatment, whether done by dry needling, or with the injection of saline or a local anaesthetic (Travell and Simons 1983 1:27.)

1.4.3. STRETCH AND SPRAY

Travell and Simons (1983 1:63) consider this technique to be more effective in terms of treatment time and patient comfort, than both needling and ischaemic compression. This view however is based on clinical experience only and not on controlled clinical trials.

1.4.4. OTHER TREATMENTS / COMPARISONS

A number of other effective treatments have also been described:

Massage has the greatest benefit in the case of latent trigger points, in other words in the absence of referred pain. Enthusiastic massaging of muscles containing AMFTPs may however aggravate the condition. Various massage techniques such as cross friction, stroking and kneading have been advocated. (Travell and Simons 1983 1:87,88.)

Three stretching techniques; passive stretching, rhythmic stabilization and Lewit's technique can be effective. Stretching is usually considered an essential adjunct to other methods such as needling or spray. However, in the case of newly activated or latent trigger points, stretching alone may bring immediate relief. (Travell and Simons ~~1983 1:89.~~)

Ultrasound, moist heat and drug therapy in response to symptoms such as pain, muscle tension, insomnia and depression are other alternative forms of treatment. (Travell and Simons 1983 1:89-92) Interferential therapy has also been advocated in the treatment of Myofascial syndrome. (Gatterman 1990:353) The use of a pen electrode facilitates the treatment as its small head allows the practitioner to apply the current directly over the AMFTP.

I was not able to find any studies comparing the effectiveness of any of these treatment modalities to TENS in the treatment of MFS.

1.4.5. T.E.N.S

Although the mechanism of action of TENS is not clearly understood (Gatterman 1990, 345); its success in the relief of pain caused by Myofascial syndrome has been demonstrated by, amongst others, Walsh et.al (1995). They did stress however, that these hypoalgesic effects are critically dependant on the combination of stimulation parameters and that further research

is required in this area.

Melzack (1981) explained the mechanism of TENS as a form of hyperstimulation analgesia similar to needling and hypothesised that TENS could therefore have the same therapeutic effect. By means of ~~clinical trials~~ he discovered that the application of TENS to trigger points or acupuncture points provided a powerful means of pain control. He did not compare the relative effectiveness of TENS and dry needling in the treatment of MFS, but did find that in the case of lower back pain, that although both procedures produced significant pain relief, neither procedure was statistically more effective.

TENS is an effective, cost efficient, safe and non-invasive treatment modality and should therefore be used in preference to more invasive and potentially harmful alternatives in the treatment of Myofascial syndrome. (Gatterman 1990: 346)

1.5. COMPLICATIONS

1.5.1. STRETCH AND SPRAY

Fluori-Methane is used in the vapocoolant stretch-and-spray technique. Complications may include freezing of the skin and resultant ulceration if applied for longer than six seconds; urticaria owing to cold allergy and possible toxicity under conditions of hypoxia (Travell and Simons 1983 1:67-70.) This technique, although effective, is also potentially harmful and

should only be performed by those with adequate training in this technique. It is therefore not ideally suited for home use.

1.5.2. NEEDLING

Needling is admittedly a very effective form of treatment (Lewitt 1979.) However this technique requires great precision and there is the risk of infection associated with any invasive technique (Brown 1987.) Needling of the trigger point may also result in complications such as fainting, convulsions, post treatment drowsiness, damage to underlying viscera and the development of a painful haematoma (Baldry 1989:53.)

1.5.3. OTHER FORMS OF TREATMENT

Although the use of ultrasound is relatively safe, complications such as burns, cavitation and hyperstimulation with subsequent aggravation of symptoms may occur if not administered correctly. Its use is contra-indicated in vascular conditions such as thrombophlebitis; over areas of acute sepsis; for six months following radiotherapy, and in the presence of tumours. The back and abdomen should be avoided in the case of pregnancy, as should the chest area if the patient has a cardiac pacemaker. The cervical ganglion and vagus nerve should also be avoided if the patient has a history of heart disease. (Forster and Palastanga 1985:175-176.)

The only possible danger associated with interferential therapy is electrode burns (Forster and Palastanga 1985:111.) The contraindications are the same as those listed below for T.E.N.S.

1.5.4. T.E.N.S

TENS is a relatively safe treatment modality. The only danger is electrical burns which may occur if a bare electrode touches the skin or if the electrodes are positioned too close together resulting in a skin current. (Forster and Palastanga 1985: 111.) Contra-indications are the use of TENS in patients with a cardiac pacemaker or cardiac arrhythmia. Electrodes should also not be placed over anaesthetic or broken skin; on either side of the cranium; across the front of the neck or in the vicinity of the carotid sinus; over the heart or eye; over open wounds, infection, thrombosis, active tuberculosis or malignancy (Gatterman 1990 :344,346.)

1.6. COST EFFECTIVENESS

1.6.1. STRETCH AND SPRAY / NEEDLING

Fluori-Methane is available locally under the trade name "Painfreeze" and is relatively inexpensive. Disposable acupuncture needles are sold for approximately R2 each. Discarding the needle after use is the safest method as it reduces the chance of introducing infection through the

acupuncture site and prevents the risk of contracting a blood borne disease such as AIDS or Hepatitis. The only additional expenses are cotton wool swabs and alcohol.

1.6.2. ELECTROTHERAPY: A COMPARISON

TENS units are cheaper than both ultrasound and interferential current and they are readily available. The latest prices available (April 1997) are as follows: IFC - R6620; Ultrasound - R5518; Combined IFC and Ultrasound R9995 and TENS - R574. These quotes are on the most basic models currently available.

Doctors in a small or newly established practice will therefore find a TENS unit far more affordable. As it is portable and battery operated, it is cost efficient and very convenient to use. Portable TENS units are easy to operate and can therefore be purchased and self administered by the patient. This is especially advantageous to those patients with a chronic pain syndrome who can then control their condition both effectively and cost efficiently.

1.7. ELECTRODE PLACEMENT WITH T.E.N.S.

The most effective electrode placement when using T.E.N.S. is an area of considerable debate and an important consideration, as the placement of the electrodes is one of the most important factors determining the success or failure of the treatment. The chosen site should be anatomically or physiologically

related to the pain source. In the case of Myofascial syndrome, the electrodes could therefore be placed directly over, or above and below the tender trigger point. In either of these positions they are anatomically related to the source of pain. Alternatively the operator could choose to place one electrode over the painful area and another between the painful area and the spinal cord, or at selected sites within the pain referral zone so that they are physiologically related to the source of pain. Generally, the skin below the selected site should have an intact sensory mechanism so that the electrical input can be readily directed into the central nervous system and the chosen area should also allow ease of application of the electrodes. Bony prominences and excessively hairy areas should therefore be avoided as it will be difficult to ensure good contact between the electrode and the underlying skin. Hair also interferes with the conduction of the current. (Gersh and Wolf 1985.)

There are therefore a number of potential electrode placements to choose from when using TENS. However clinical trials are needed to determine which of these options are more effective.

1.8. STUDY AIM AND OBJECTIVES

The aim of this investigation is to determine the relative effectiveness of placing electrodes over the AMFTP with placing them over the pain referral zone in the treatment of myofascial syndrome using TENS and passive stretching, with reference to

both subjective and objective clinical findings in order to determine which of these methods is more effective.

The objectives are as follows :

1) ~~the first objective is to compare the~~ effectiveness of placing electrodes over the AMFTP with that of placing them over the pain referral zone in the treatment of Myofascial syndrome using TENS and passive stretching in terms of subjective clinical findings.

2) the second objective is to compare the effectiveness of placing electrodes over the AMFTP with that of placing them over the pain referral zone in the treatment of Myofascial syndrome using TENS and passive stretching in terms of objective clinical findings.

3) the third objective is to correlate the data obtained during the study in order to determine which of these treatment methods is the more effective.

1.9. IMPORTANCE OF THE STUDY

Myofascial syndrome may result in a chronic pain syndrome if not diagnosed timeously and treated effectively. Chronic pain syndromes in turn may have a number of negative financial repercussions. (Travell and Simons 1983 1:6)

Myofascial syndrome is a difficult, complex and frustrating condition to treat. This is due to the numerous psychological, biomechanical and nutritional factors which may all play a role in its cause. It is therefore important that doctors are made aware of the most effective treatment modalities available to them.

CHAPTER TWO: LITERATURE REVIEW

2.1. INTRODUCTION

Travell and Simons (1983 1:3) defined Myofascial syndrome as ~~pain, dysfunction and,~~ or autonomic phenomena which are referred from active myofascial trigger points. A few years later in 1988, after conducting a number of studies on this condition, Simons concluded that it was the most common single finding in patients who presented to chronic pain treatment centres. It is also often a source of frustration to both the doctor and the patient. Laboratory tests, X-rays, physical and orthopaedic examination generally reveal negative results, consequently, the doctor is unable to find any conclusive objective findings and therefore often concludes that the complaint is psychogenic in origin, leading to frustration on the part of the patient. (Sandman 1981) Effective treatment of this condition is also very difficult as there are so many factors involved. Simons (1976) reviewed the possible causes associated with MFS and identified at least fifteen major causes.

Failure on the part of the doctor to diagnose and effectively treat this painful condition may result in a chronic pain syndrome, characterised by features such as depression, substance abuse, anger, hostility and physical disability. (King and Goddard 1994) There may also be negative economical repercussions, (Travell and Simons 1983 1:6) if this condition

results in time away from work, either directly as a result of the pain caused by the Myofascial syndrome, or indirectly due to the resultant chronic pain syndrome and the associated features as described above.

~~The following chapter will briefly summarise and critically~~ review the literature available on both Myofascial syndrome and the application of TENS in the treatment of this condition. I will discuss the causes of this condition as well as the postulated underlying mechanism involved. I will also discuss the diagnosis, treatment and monitoring of this condition in terms of both subjective and objective findings. The application of TENS will then be discussed and conclusions drawn from the available literature as to the effectiveness of this treatment modality in the treatment of Myofascial syndrome.

2.2. Causes of Myofascial syndrome

Emotional or physical stress is the initiating factor in the development of myofascial trigger points. The development of Myofascial syndrome in an individual however, is dependent on the genetics, personality, conditioning and physiological state of that individual. (Sola 1981.)

There are many factors which may precipitate or aggravate TPs. The most important factors were summarised and discussed by Simons (1976) He divided them into three main groups :-

1) Physical: The most common factor was chilling of the muscle. Other examples listed were acute or chronic ~~trauma to the~~ muscle, fatigue, prolonged immobility and poor posture.

2) Medical: TPs may follow an acute illness or be associated with chronic infection. Metabolic disturbances, nutritional inadequacies and psychological factors were also included.

3) Pathophysiological: Local hypoxia due to impaired circulation, hyperirritability of muscle receptors resulting in sustained contraction and a possible allergic or autoimmune response have all been suggested as possible factors.

It is very important that these factors be recognised and corrected in order to obtain lasting results. (Lewitt 1984)

The possible causes of MFS will now be discussed with specific reference to biomechanical factors, nutritional inadequacies, metabolic and endocrine inadequacies and psychological factors.

2.2.1. Biomechanical factors

These may be anatomical factors as in the case of skeletal assymetry or disproportion, or external factors such as

misfitting furniture or constricting pressure (Travell and Simons 1989 1:104-114.)

Leg length inequality is an example of structural asymmetry and a common cause of lower back pain. This asymmetry predisposes the patient to developing TPs which may in turn accentuate and prolong pain, both in the lower back and cervical region. (Sola 1981.) Travell and Simons (1983:104) believe that a leg length difference of as small as 0.5 cm may predispose the quadratus lumborum muscle to developing TPs and that TP involvement of this muscle is the most commonly overlooked source of lower back pain. The pelvis tilts down on the side of the short leg and as the origin of the quadratus lumborum muscle is the medial part of the iliac crest, (De franca et al. 1981) this places a constant strain on the muscle thereby predisposing it to the development of TPs.

Poor posture and misfitting furniture are also common perpetuating factors. The purpose of a good posture or correctly fitting furniture is to keep all the parts of the body in a position of least strain and maximal support. (Gatterman 1990:264) This includes placing the muscles in a resting position, which reduces the chance of them developing TPs.

Faulty posture is often seen in the self-conscious adolescent who rounds her shoulders to hide her developing breasts, the secretary who sits at her keyboard all day or cradling the

telephone receiver between her ear and shoulder and the diligent student who spends hours hunched over the desk. A correctly designed or fitting chair is one that does the work of the postural muscles by supporting the body, thereby allowing the muscles to relax with a minimal expenditure of energy. (Travell and Simons 1983:112.)

Constriction of a muscle also predisposes that muscle to developing trigger points. A heavy bag hung over the shoulder or bra straps supporting large breasts could compromise the upper fibres of the Trapezius muscle. Similarly, a tight collar and tie could effect the sternocleidomastoid muscle. (Travell and Simons 1983:114.) The prolonged constriction of the muscle would result in an area of local ischaemia and consequently the development of TPs.

2.2.2. Nutritional inadequacies

There are certain vitamins and minerals which, when deficient, perpetuate or predispose the patient to developing Myofascial syndrome. These are vitamins B1, B6, B12 and C, and the minerals calcium, potassium, magnesium and iron. (Travell and Simons 1983:114)

These water soluble vitamins function as coenzymes in certain metabolic reactions. Two of these reactions are glycolysis and the tricarboxylic acid cycle. Both of these reactions lead to the formation of ATP a primary source of energy which fuels

certain physiologic activities. (Conn 1987:165,324,344,393.)

During muscle contraction, cross bridges form between the actin and myosin filaments. These cross bridges are only released in the presence of ATP. Therefore, if insufficient ATP is produced this will result in a sustained intramuscular contraction i.e. TP formation. (Schneider 1994.) Deficiency of calcium, iron, or potassium interferes with normal muscle function. According to Travell and Simons (1983:141), a deficiency of these minerals tends to increase the irritability of existing TPs, however they do not offer an explanation for this. As such, they appear to be more of a perpetuating factor than a predisposing factor. Calcium initiates muscular contraction, (Guyton 1987:63,64) therefore a deficiency would not result in a sustained contraction as in the case of a vitamin deficiency.

2.2.3. Metabolic and Endocrine inadequacies

Any factor that adversely affects the intramuscular metabolism will perpetuate TPs.(Travell and Simons 1983:143)

Hypothyroidism is an endocrine disorder characterised by insufficient circulating thyroid hormones. As these hormones have a direct effect on the metabolic rate of the body, a deficiency results in a hypometabolic state. (Guyton 1987:576.) This affects the skeletal muscle too, thereby predisposing the muscle to developing TPs. (Travell and Simons 1989 1:146)

Hypoglycemia is defined as a blood glucose level of below 50mg/dl. (Krause 1992:552) As there is insufficient fuel (glucose) to serve as a substrate for the various metabolic activities, the intramuscular metabolism is impaired. TPs appear to be aggravated and perpetuated by any compromise of the intramuscular metabolism. (Travell and Simons 1989 1:143.) Hyperuricaemia (gout) is another metabolic disorder which aggravates and perpetuates TPs although the mechanism is unclear.(Travell and Simons 1983:148)

2.2.4. Psychological factors

In a condition like Myofascial syndrome, where there are so many variables involved, it is often tempting to explain the majority of cases in terms of psychogenic factors. Environmental stress at home or in the workplace, anxiety and depression are often blamed for the patient's condition. Although this may well be true in many cases, it must never be assumed to be primary. (Gatterman 1990: 286.) Sandman and Backstrom (1984) discussed the interaction of the body and mind and its importance in MFS. They explained that two aspects had to be considered; MFS as a response to stress and the psychogenic component of the stress itself. They also postulated a mechanism by which psychological stress may cause a change in the physiological function of the muscle and stressed the importance of addressing both of these aspects to effectively treat MFS.

If untreated (or ineffectively treated), Myofascial syndrome may develop into a chronic pain syndrome. (King and Goddard 1994) This is typically associated with depression ,various anxiety disorders and drug and/or alcohol dependence (Fishbain et al. 1986) Fishbain et al. (1986) conducted a study on 283 chronic pain patients with pain of at least two years duration. Each patient was evaluated independently by a neurosurgeon and a physiatrist over a three day period. The most frequent primary treatment diagnosis (85%) was Myofascial syndrome. This study was strengthened by the fact that there was over 95% agreement with regards to diagnosis, between the neurosurgeon and the physiatrist. I also found it interesting that analysis of the psychiatric diagnoses (according to DSM-III), showed, that although depression was common, psychogenic pain disorder was rare.

Although there are a number of literature sources available on Myofascial syndrome, it became apparent during my search, that the leading authorities on this subject are David Simons and Janet Travell. Together they compiled "The Trigger Point Manual" and wrote a number of journal articles, either together, or in collaboration with other researchers. As a result of this, the author of any journal article concerning Myofascial syndrome quotes extensively from their work. This lack of primary literature does mean that the evidence available tends to be unvaried and as such, is not of a very high quality, however, until proven otherwise, it remains the best source of information available.

2.3. Underlying mechanism in the development of myofascial trigger points

2.3.1. INTRODUCTION

Myofascial syndrome may have an abrupt or insidious onset, depending on the causative factors that are involved. Travell and Simons (1981) hypothesised that the development of AMFTPs could be divided into two stages; an initial dysfunctional stage and a subsequent dystrophic stage with demonstrable histological changes.

2.3.2. ANATOMY OF A MUSCLE FIBRE

The smallest independent unit of skeletal muscle is the muscle fibre. Large numbers of these fibres are grouped together in fascicles. Groups of fascicles, in turn, make up the muscle as we know it. (Krause 1986: 132)

Each muscle fibre is surrounded by a sarcolemma. The muscle fibre is composed of myofibrils which run the length of the muscle fibre and are surrounded by sarcoplasm (the cytoplasm of the muscle cell). The myofibril is in turn composed of myofilaments which consist of actin or myosin molecules. (Krause 1986 134,135.)

The myosin filament is composed of myosin molecules. Each

molecule consists of two free heads at one end and a tail at the other end. The tails form the body of the filament and the heads form the cross bridges for the sliding filament mechanism which will be explained shortly. These heads also act as an ATPase enzyme to convert ATP to ADP in order to release energy for the contraction process. (Guyton 1987:60-61.)

The actin filament is composed of actin, troponin and tropomyosin molecules. Each strand is composed of thirteen G - actin molecules each of which has an ADP molecule attached to them. These ADP molecules are the active sites which will react with the cross bridges of the myosin molecules during the sliding filament mechanism. During the resting phase, these active sites (ADP molecules) are covered by tropomyosin to prevent contraction from occurring. Troponin is attached to this tropomyosin molecule and is composed of three molecules; Troponin I, Troponin T and Troponin C. These molecules are attracted to actin, tropomyosin and Ca^{++} respectively. (Guyton 1987:61.)

This Troponin - Tropomyosin complex has an inhibitory effect on the sliding filament mechanism. When Ca^{++} is present, this inhibitory effect is itself inhibited and contraction takes place. (Guyton 1987: 57 - 61.)

Each myofibril is closely linked to a sarcoplasmic reticulum (SR), a network of sarcotubules which surrounds the myofibril and contains a high concentration of calcium ions (Ca^{++}).

Inward extensions of the sarcolemma also surround each myofibril. These extensions are known as T-tubules and they allow rapid transmission of nerve impulses to the myofibrils. (Krause 1986: 134,135.) This nerve impulse in turn passes to the SR causing it to release Ca^{++} into the sarcoplasm. This is thought to be due to the opening of calcium channels at the onset of the action potential although the actual mechanism is unknown. These calcium ions diffuse to the adjacent myofibrils where they bind to Troponin C and initiate muscle contraction. (Guyton 1987: 65.)

2.3.3. THE PHYSIOLOGY OF MUSCLE CONTRACTION

Muscle contraction is brought about by the sliding filament mechanism. During this process the actin filaments are pulled inward along the myosin filaments so that they overlap. This sliding movement is believed to be due to the mechanical, chemical or electrostatic forces that are generated by the interaction of the cross bridges of the myosin filaments with the actin filaments. It is postulated that each myosin head bends back and forth by attaching to active sites on the actin molecule, then breaking away to return to its original perpendicular position. In this way the actin filament is pulled towards the myosin filament in a step by step fashion known as the walk along theory of contraction. (Guyton 1987:60-62.)

The function of ATP is vital in bringing about muscle contraction. Before contraction begins, the ATP-ase activity of the myosin head converts ATP to ADP. At this stage the head is in a perpendicular position and the ADP molecule is attached to it. The resulting energy is store in the head to initiate the contraction process.

Following a nerve impulse, the Ca^{++} ions difuse over the myofibril and inhibit the troponin-tropomyosin complex by binding with Troponin - C. This in turn uncovers the active sites on the actin molecule and allows the myosin heads to bind with them. This bond causes a conformational change in the head of the myosin molecule, which tilts away from it's original perpendicular position and pulls the actin filament along with it. Once the head tilts, the ADP molecule is released freeing the site on the myosin head to which a new ATP molecule can then attach. This sequence is then repeated, gradually increasing the strength of the contraction. (Guyton 1987: 62-63.)

2.3.4. DEVELOPMENT OF AN AMFTP

Any explanation of the development of an AMFTP must be able to explain the clinical symptoms which characterise trigger points: a) a palpable band, b) spontaneous pain and hyperirritability (manifested as local tenderness), c) the local twitch response, d) a region of increased metabolism and/or decreased circulation and e) autonomic phenomena

(Gatterman 1990:294.)

a) A PALPABLE BAND

An explanation for the palpable hardness associated with TPs, ~~has been attempted by many authors over the years.~~ Possible physiological mechanisms included the accumulation of fibrous tissue, local edema, altered viscosity of the contractile elements, a ground substance infiltrate, local vascular engorgement and fatty infiltration.(Simons 1976) Increased tension due to increased neural activity is also unlikely since many muscles containing TPs are electrically silent at rest. (Kraft et al. 1968) The most likely mechanism (Simons 1976) seems to be that of sustained contracture or transient contraction of muscle fibres on a reflex basis and is explained as follows:

Trauma to a muscle may be acute or chronic. If the overload is sufficiently stressful, especially in the case of acute strain, there is some degree of tissue damage. This may involve the sarcoplasmic reticulum; blood vessels with the release of platelets; and connective tissue with the break down of mast cells. (Gatterman 1990:291.)

Disruption of the sarcoplasmic reticulum would result in the release of Ca^{++} ions. This in turn would initiate the sliding filament mechanism in that section of the muscle fibre. A palpable band would be produced if this happened to a group of

neighbouring muscle fibres. (Travell and Simons 1981.)

b) SPONTANEOUS PAIN AND HYPERIRRITABILITY

Pain originates from the over stimulation of sensitive receptors. (Sandman 1981) ~~Two types of~~ nociceptors; mechanoreceptors and chemoreceptors, are of particular interest in MFS. When firm pressure is applied to a TP (ischaemic compression), mechanoreceptors are stimulated and continue to discharge for several seconds. Similarly, chemoreceptors respond to chemical stimulation. (Sandman 1981) Muscle afferents transmit these stimuli by means of the type C nerve fibres. (King and Goddard 1994)

Disruption of small blood vessels results in the leakage of platelets which in turn leak serotonin. (Gatterman 1990:291) Serotonin stimulates and sensitizes the chemoreceptors in the muscle thereby lowering their threshold. (King and Goddard 1994)

Mast cells are found in loose connective tissue and along blood vessels. They contain heparin and histamine. (Krause 1986:56) Damage to these cells results in the release of these substances. (Gatterman 1990:291) Histamine, like serotonin, stimulates and sensitizes the chemosensitive pain receptors. However, these substances do not only stimulate the chemoreceptors, they also decrease the threshold of the mechanoreceptors and thermosensitive pain receptors (Guyton

1987: 378,379), thereby making the AMFTP hyperirritable and tender.

c) THE LOCAL TWITCH RESPONSE

~~The local twitch response (LTR) is a valuable diagnostic tool~~ associated with MFS as it is objective and measurable. This palpable, and frequently visible, local twitch of the muscle fibres within the palpable band, is elicited by snapping palpation of the band. (Hong 1994.)

The acidity of the accumulated metabolites and specific metabolites such as prostaglandins are thought to sensitize the group III and group IV nerve endings which act as muscle nociceptors. (Mense and Schmidt 1977) Some of these nociceptors also respond to stretch. (Kniffki et al. 1978) It is thought that mechanical stimulation of the TP, brought about by snapping palpation or needling would stimulate these sensitized receptors and initiate a spinal reflex pathway. Afferent impulses transmitted to the spinal cord cause appropriate motorneurons to fire resulting in a LTR of the taut band fibres. It is believed to result from the summation of motor unit potentials (Hong 1994) and is easily recorded as a transient discharge of motor unit activity. (Sandman 1981)

d) INCREASED METABOLISM AND/OR DECREASED BLOOD FLOW

The uncontrolled contractile activity results in an area of increased metabolism and an accumulation of metabolites. This uncontrolled contraction will continue unabated as long as there are free Ca^{++} ions and ATP available. The SR is unable to remove these free Ca^{++} ions because of the damage which it ~~has sustained. The~~ prolonged contraction eventually leads to fatigue of the muscle. (Gatterman 1990: 291)

Sustained contraction also impairs the local blood flow (Sandman et al. 1984) as the blood vessels will be compressed by the surrounding muscles. The intense local metabolic activity could also produce substances which further sensitize the pain receptors and result in a local reflex vasoconstriction to help control the excessive metabolic activity (Sandman et al. 1984), this would further enhance the local ischaemia.

e) AUTONOMIC PHENOMENA

TPs sometimes manifest secretory and vasomotor autonomic phenomena within their pain referral zone such as pallor and coldness, coryza, lacrimation, sweating and pilomotor activation. (Sandman 1981)

Hubbard and Berkoff (1993) conducted a clinical trial on 29 patients with MFS. They inserted a needle EMG into both latent and active TPs. Spontaneous EMG activity was recorded in all cases. They concluded that this activity was due to sympathetic

stimulation of intrafusal fibres, resulting in an involuntary muscle tension. This also helps to explain the autonomic phenomena such as sweating and localised vasoconstriction which are sometimes associated with AMFTPs. This was an interesting study as sympathetic innervation of intrafusal fibres had ~~traditionally been refuted. These findings are also in stark~~ contrast with those of Travell and Simons (1983 1:5-44), who concluded that TP showed no resting activity and that any action seen was either due to the insertion of the needle or from adjacent muscle fibres.

The fact that the trigger point persists after the excess calcium should theoretically have diffused away (probably within hours, certainly within a few days) can be explained by a second mechanism. Prolonged and sustained contraction results in a nearly total depletion of ATP. Without ATP the myosin heads can not detach from the actin filament, therefore the sarcomere (a sub-unit of overlapping actin and myosin molecules) remains in that shortened state. This ATP deficient contracture is surrounded by a region of increased metabolism. This may explain why the ATP can not be replenished and the TP persists. (Travell and Simons 1983 1:36-37.)

If untreated, these mechanisms described above may lead to a dystrophic phase with demonstrable histological changes. (Travell and Simons 1981) In 1976, Simons critically reviewed a large number of muscle biopsy studies. Findings ranged from

normal, to those showing histopathological changes such as an infiltration of connective tissue into and among the muscle fibres, fatty infiltration, proliferation of sarcolemmal nuclei and oedema. These findings were clearly conflicting and inconclusive and he concluded that further studies were needed in this area.

2.4. REFERRED PAIN

Referred pain refers to pain which is felt in a part other than the site of the lesion due to distortion in the sensory pathways (Sandoz 1985) In the case of MFS, the pain originates from the TP but is felt at a distance from its origin.

This pain referral zone rarely coincides with the entire distribution of a peripheral nerve or dermatomal segment. (Gatterman 1990:413.) An example of this is Trapezius TP1 - the most common TP in the body. It is found in the upper border of the Trapezius muscle, just above the superior angle of the scapula. It refers pain along the posterolateral aspect of the neck to the mastoid process, the back of the orbit and sometimes to the angle of the jaw, the occiput, the lower molars and the pinna. (Travell and Simons 1983 1:184.)

There are a number of hypotheses that attempt to explain referred pain patterns. The most widely accepted of these is the convergence projection theory. It suggests that continuous stimulation of nociceptors in the skeletal muscle causes nerve impulses to be conveyed via afferent nerve fibres to the dorsal

horn cells. This establishes a state of hyperexcitability. Cutaneous afferents also converge on these dorsal horn cells. A strong input from the skeletal muscle afferents is mistakenly interpreted as coming from the corresponding skin site and gives rise to pain which is felt to be in the latter tissue.

~~(Travell and Simons 1983 1:31.)~~

Pain at rest is the differentiating factor when diagnosing active or latent TPs. A latent TP is clinically silent with respect to pain but may cause restricted motion and weakness of the affected muscle. An active TP causes pain, however this pain is rarely located at the actual TP, instead it is usually experienced in the pain referral zone which is characteristic for that muscle. (Travell and Simons 1983 1:12,13.)

The difference between an active TP and a latent TP appears to be the degree of irritability that exists according to the level of afferent nerve stimulation. (Schneider 1994:8,9) As this irritability seems to be related to the sensitization of muscle afferents by substances such as histamine, serotonin and prostaglandins (Gatterman 1990:294), it follows then that it must be related to the amount of tissue damage that has taken place.

The amount of stress needed to convert a latent TP to an active TP depends on the degree of conditioning of the muscle. It also depends on the number and the severity of perpetuating factors. These were described in 2.2 of this chapter. Specific

situations that may activate a latent TP are; prolonged or sudden shortening of the muscle, chilling of the muscle (especially from air-conditioners), and during or following a viral illness. (Travell and Simons 1983 1:14.)

2.5. Diagnosing Myofascial syndrome

Travell and Simons (1983 1:18-19) listed the criteria necessary to diagnose an active myofascial trigger point, they are :-

- a) A history of a sudden onset following acute overload, or a gradual onset following chronic overload of the affected muscle.
- b) Characteristic pain referral patterns which are specific to individual muscles. These patterns do not appear to vary much between individuals. (Sandman 1981)
- c) Weakness and restriction in the stretch range of motion of the affected muscle.
- d) Presence of a taut palpable band in the affected muscle.
- e) Focal tenderness on digital pressure over this taut palpable band.
- f) Reproduction of the patient's pain during digital pressure or needling of the trigger point. The pain is characteristically deep and poorly localised. (Sola 1981)
- g) Elicitation of a local twitch response on snapping palpation or following dry needling of the trigger point.

- h) Elimination of the patient's symptoms by therapy directed specifically at the affected muscle.

They stated that the elicitation of a local twitch response, or reproduction of the patient's pain, are the two criteria which most strongly indicate the presence of ~~an active~~ myofascial trigger point. Sandman (1981) felt that sensitive palpation of the affected muscle was the doctor's primary objective diagnostic tool and that identification of the AMFTP and the subsequent pain referral pattern would establish a method of diagnosis.

2.6. Measurement of subjective and objective clinical findings associated with Myofascial syndrome

2.6.1. OBJECTIVE MEASUREMENTS

a) ALGOMETRY

The measurement of pain can be achieved objectively, albeit indirectly with an algometer. Algometry is a diagnostic technique used to measure pressure thresholds and it is a recognised diagnostic method used to quantitatively document the sensitivity of TPs. (Kruse et al. 1992.) The reliability of the algometer as an index of myofascial trigger point sensitivity was demonstrated by Reeves et al. (1986)

Algometry is obviously therefore a useful tool for documenting quantitatively the sensitivity of TPs and for monitoring the effectiveness of a chosen treatment protocol. Ohrbach (1990) did caution however, that AMFTPs are discrete pain sites and that patient feedback regarding the accuracy of tip placement is important to improve pressure pain threshold measurements.

b) BLOOD TESTS

Routine laboratory tests measuring ESR, blood count and serum muscle enzymes are all normal in patients with MFS. (Simons 1976) Although a number of biochemical studies have been conducted, the results are inconclusive. A few studies have shown a shift in the distribution ratio of Lactic dehydrogenase (LDH) - isoenzymes and although further studies are needed, this may prove to be a valuable diagnostic tool. (Simons 1976)

This enzyme is found in skeletal muscle and catalyzes the conversion of Pyruvic acid to Lactic acid during glycolysis. The distribution ratio is affected by the pH of the tissue, (Conn 1987:353,354) therefore any change in pH (which does occur with impaired metabolism) could affect the distribution ratio.

c) EMG STUDIES

Hubbard and Berkoff (1993) demonstrated spontaneous needle EMG activity of myofascial trigger points. However the needle

insertion has to be very precise as activity is only recorded from the 1-2 mm nidus of the TP. If performed correctly this method offers an objective method of diagnosis and evaluation. The discreteness of the AMFTP and the consequent difficulty in locating the precise point would explain why other ~~investigators have failed to record any~~ EMG activity from the TP. (Simons 1976)

Hong (1994) recently confirmed the findings of Kraft et al.(1968) that there was no EMG activity in the taut band. EMG activity was recorded however when a local twitch response was elicited. The recording of EMG activity from a muscle also depends on the sensitivity of the equipment used. Hubbard and Berkoff used high sensitivity EMG recording when they demonstrated the spontaneous needle EMG activity of TPs. (Hong 1994.)

d) THERMOGRAPHY

Thermography is a non-invasive imaging technique which records differences in body surface temperature. It has been proven to be both a sensitive and practical method for diagnosing MFS. (Diakow 1988). Thermographic images show an area of hyperthermia in the area of the AMFTP and usually also over the pain referral area.(Kruse et al. 1992) This is consistent with reports of vasodilation due to (as yet unexplained) autonomic feedback cycles associated with TPs. Kruse et al. (1992), in their controlled study of eleven patients with AMFTPs, showed

that compression of the TP resulted in a reduction of temperature both locally and throughout the pain referral zone. They demonstrated that these autonomic changes occurred independently of the sensory changes, as the pain was usually only experienced in the more proximal part of the pain referral zone, however the thermal changes occurred throughout the pain referral zone. These peripheral autonomic changes are not seen in Fibromyalgia (a chronic non inflammatory diffuse muscle disorder which presents similarly to MFS). The authors concluded their study by advocating thermography as an important tool for differentiating between MFS and Fibromyalgia, as both conditions show similar results with algometry and EMG studies.

2.6.2. SUBJECTIVE MEASUREMENTS

a) THE MCGILL SHORT FORM PAIN QUESTIONNAIRE:

This questionnaire provides a quantitative profile of three aspects of pain and is considered the leading pain measurement scale. (Melzack 1975)

b) NUMERICAL RATING SCALE 101:

This has been described as an effective method for estimating pain intensity. (Jensen et al. 1986)

c) PAIN DISABILITY INDEX:

The measurement of disability has become an important aspect of clinical trials and research. It can be used to monitor the patient's response to treatment as well as to assess his or her physical function. ~~(Yang et al. 1993.)~~ McDowell (1987) defined disability as the restriction in a person's ability to perform a function in a manner considered normal for a human being. The pain disability index assesses the degree of disability experienced by the patient due to a painful condition (Tait et al. 1987) such as Myofascial syndrome.

d) PSYCHOLOGICAL EVALUATION

Psychological factors have been identified as a viable component of Myofascial syndrome and these factors should be taken into consideration when assessing the patient. The case history should include questions relating to the patient's lifestyle, personality type and general sense of well being. Note should also be taken of the patient's posture and body language.

Two main aspects must be considered: psychological factors as a cause of the pain syndrome and the psychogenic component of the pain itself. Psychogenic factors may include depression,

tension, anxiety and a type-A personality. If a chronic pain syndrome is allowed to develop, the pain itself may become a source of stress and depression. (Sandman et al. 1984.)

e) PATIENT FEEDBACK

As the active myofascial trigger point is a discrete site, assistance in locating the most tender point through patient feedback improves validity. (Ohrbach 1990) The characteristic pain referral patterns elicited by digital compression of these tender points, have been documented extensively by Travell and Simons. (1983 v.1 and 1992 v.2)

The literature reviewed indicated that algometry was the method of choice for obtaining objective measurements of trigger point sensitivity. This is probably due to time and cost constraints, as well as availability and ease of application of the chosen instrument. From the literature available, it appears that there are no questionnaires that have been devised specifically for monitoring the pain and disability associated with Myofascial syndrome. The evidence does suggest however that the McGill short form questionnaire, the Numerical rating scale and the pain disability index are popular and reliable choices.

2.7. Treatment of Myofascial syndrome

2.7.1. INTRODUCTION

Treatment of active myofascial trigger points is directed towards the disruption of reverberating neural circuits (Melzack 1981) responsible for the "memory" of the pain and hence the self perpetuating nature of the condition.

2.7.2. PAIN: A POSSIBLE EXPLANATION

The gate control theory published by Melzack and Wall in 1965 opened the way to find new techniques which would be able to modulate pain. This theory proposed enhancing normal physiologic activities rather than disrupting them by using drugs or surgery. In this way pain would be controlled by enhancing normal physiologic inhibitory mechanisms. The theory is based on the idea that the substantia gelatinosa (SG) - a system of neurons in the dorsal horn of the spinal cord - has an inhibitory effect on the transmission of impulses from first to second order neurons. This creates a physiological gate which may be "opened" or "closed". The gate is "opened" by the small afferent nerve fibres which block this inhibitory effect, and "closed" by the large afferent nerve fibres which enhance the inhibitory effect of the SG. Therefore stimulation of the large fibres "closes" the gate to the small nociceptor fibres and blocks the perception of pain in the brain. Inhibitory signals from the brain also play an important role. (Melzack

and Wall 1965.)

The gate control theory remains just that - a theory, however, until another theory is formulated to improve on our understanding of the nature of pain, our understanding of pain relief will remain just as unclear. (Catterman 1990: 345.)

2.7.3. CHOICE OF TREATMENT MODALITY

There are a number of modalities which are effective in the treatment of MFS. These include ultrasound, ischaemic compression, massage, dry needling, stretch and spray, stretch without spray, moist heat, drug therapy, biofeedback and TENS. (Travell and Simons 1983 1:27,86-92.) These modalities have been discussed in terms of their relative effectiveness, possible complications and cost effectiveness in chapter 1. Each modality exerts its effect on a different aspect of the AMFTP and MFS as a whole.

Ischaemic compression, massage and needling all work directly on the trigger point. Ischaemic compression and massage produces a local stretch of the shortened sarcomeres, a pressure nerve block and changes in the perfusion of the skin which are likely to correspond to those taking place in the muscle. Release of pressure causes a reactive hyperaemia which flushes out the accumulated metabolites and sensitizing substances responsible for the self perpetuating cycle of the AMFTP. (Travell and Simons 1980.)

Needling is thought to mechanically disrupt the shortened contractile element or nerve endings and in so doing break the feedback loop responsible for sustaining the AMFTP. It may also cause the release of intracellular potassium by damaging the muscle fibres which, if in a high enough concentration, could in turn cause a depolarization block of nerve fibres. (Travell and Simons 1980.)

It is a well documented fact that dry needling often produces prolonged relief of myofascial pain (Melzack 1981) and it is regarded by many as the most effective form of treatment. (Simons 1976) Many authors have advocated injecting the muscle with saline or a local analgesic (Simons 1976), however Lewit (1979) in his study on the needle effect in the relief of myofascial pain, concluded that it was the actual insertion of the needle that relieved the condition and that the use of saline or a local analgesic was, in fact, unnecessary.

Moist heat reduces the tension of the underlying muscles and improves the blood supply which assists in dispersing the accumulated metabolites. Heat also appears to have a sedative effect on nerve conduction. (Forster and Palastanga 1985 :113.) Ultrasound has both a therapeutic thermal and micromassage effect, (Forster and Palastanga 1985 :172) therefore it combines the physiological effects of both heat and massage as described above.

Drug therapy may play a role in the treatment of MFS with respect to pain relief, muscle relaxation and anti-inflammatory action. It may also be useful in relieving associated conditions such as insomnia and depression. (Travell and Simons 1983 1:90) Melzack (1981) hypothesised that pain may be produced by "pattern generating mechanisms" in the transmission pathways between the cortex and the dorsal horn cells as a result of abnormal activity. Input to these pathways is received from multiple sources; the peripheral, autonomic and central nervous system. Therapy needs to modulate this input by using all possible techniques, singularly or in combination. Analgesics could for instance be administered along with TENS for a greater degree of pain control than either one alone.

Biofeedback does not affect the AMFTP directly but has been found effective in the treatment of stress and pain management. Other useful relaxation techniques are progressive relaxation, visual imagery, breath control and time management. (Sandman and Backstrom 1984)

Stretching, with or without spray, does not work on the trigger point directly but on the muscle/s containing the AMFTP. Stretching causes the muscle to gradually restore its full range of motion and normal function by separating the locked actin and myosin filaments and eliminating contraction. (Sandman and Backstrom 1984) It is possible to inactivate newly activated or only slightly irritable TPs with stretch alone. (Travell and Simons 1983 1:89) Stretching should therefore be

considered an essential component of any treatment protocol. Specific stretches must be chosen to stretch the affected muscle without causing further damage to the area.(Sandman 1981)

~~There is~~ plenty of evidence to support the use of passive stretching to increase muscle range of motion. It is also a very safe technique. Studies have shown that maintaining the stretch for as little as fifteen seconds is adequate to achieve the desired results. (Wilkinson 1992.)

Perpetuating and/or predisposing factors, as described in 2.2. of this chapter, must also always be considered and addressed in order to develop a truly effective and lasting treatment protocol. MFS has an excellent prognosis with proper diagnosis and treatment. (Sandman 1981)

2.7.4. THE USE OF TENS IN THE TREATMENT OF MYOFASCIAL SYNDROME

Melzack (1981) hypothesised that TENS (Transcutaneous Electrical Nerve Stimulation) could be used in the same way as acupuncture -for brief periods of time at moderate to high intensity - and to the same effect in the treatment of MFS. TENS selectively stimulates the large, fast A - beta fibres. These electrical impulses are transmitted to the spinal cord. Noxious impulses (originating from the AMFTP) are conducted via the small slow A - delta and C fibres. Both the large and small afferent nerve fibres converge on the substantia gelatinosa.

(Forster and Palastanga 1985 :100-102.) According to the gate control theory (Melzack 1981), these noxious impulses would be blocked at the spinal cord level by the input received from TENS via the large A beta afferent nerve fibres. A frequency of approximately 100 Hz appears to be most conducive in producing this effect. (~~Graff-Badford et al.~~ 1988)

TENS therefore does not inactivate the TP directly but works through the nervous system to control the pain associated with MFS. It would be logical to assume therefore, that the therapeutic effect of TENS would not be lasting. If this was the case, it would follow that the treatment of MFS with TENS would be a waste of time, as the actual cause of the pain was not being dealt with and the pain would therefore keep recurring. Melzack (1981) however explains the prolonged relief obtained by TENS in terms of two hypotheses:

- 1) There are reverberating neural circuits which underlie "memories" of the earlier injury (TP activation). These circuits are facilitated by low level inputs received from the pathological structures or processes associated with the AMFTP. These circuits are disrupted for long periods of time, or even permanently, by electrical stimulation as is the case with TENS.

- 2) When the pain the patient is so accustomed to is blocked, even for a short period of time, the patient resumes normal motor activity. The resulting normal proprioceptive input may

prevent the resumption of the abnormal reverberating neural circuits which underlie the pain.

The effectiveness of TENS in the relief of pain caused by Myofascial syndrome has been documented. (Walsh et al. 1995) ~~Other studies, such as the one designed by Graff-Radford et.al~~ (1988), have also been conducted using TENS for the treatment of this condition. These studies did not question the effectiveness of TENS as a choice of treatment, but rather, investigated the best stimulation parameters to use during the treatment. However, no discussion of the effectiveness of TENS for musculoskeletal pain would be complete without mention of the Clinical Practice Guideline for acute low back problems compiled by the Agency for Health Care Policy and Research (1994).

This twenty three member multidisciplinary board critically reviewed the literature available on the treatment of acute low back pain using, amongst others, TENS. Of the thirty four articles reviewed, only nine met their stringent criteria for consideration in the study. Based on these articles, the board concluded that there was insufficient evidence to support the use of TENS in the treatment of low back pain.

Although this study was undoubtedly a significant one and was based on strong research based evidence of a high quality and scientific nature, it was limited to acute low back pain as a result of spinal pathology only. These findings can therefore

not be extrapolated to encompass Myofascial syndrome, which is a completely separate entity. The only study involving acute low back pain and TENS that met the review criteria, involved TENS in the form of electroacupuncture with unstipulated parameters and can therefore not be compared to the form of TENS used in this particular study. ~~The other studies involving~~ TENS were considered inconclusive. Therefore, although this study was an important contribution to the effective treatment of acute low back pain, I feel that it bears no relevance as far as the treatment of Myofascial syndrome is concerned.

In a survey conducted by Lindsay et al.(1990) of 73 private physiotherapy practices in Australia, more clinics owned a TENS unit than any other electrical stimulator. Unfortunately, clinical trials to establish guidelines for the most effective usage of electrotherapy modalities is lacking. (Lindsay et al.1990) This leads to confusion and a lot of guess work and practitioners are unable to make full use of these modalities. This study will attempt to alleviate some of the confusion surrounding the use of TENS.

2.8. Application of TENS

2.8.1. Stimulation Parameters

Graff-Radford et al.(1988) conducted a controlled clinical trial using TENS in the treatment of Myofascial syndrome. They showed that the most significant pain reduction was achieved

using 100 Hz and 250 msec stimulation i.e. high frequency high intensity TENS.

These results were supported by the findings of Walsh et al.(1995) in a controlled study to investigate the most effective combination of stimulation parameters in the treatment of MFS. They found that the greatest hypoalgesic effect was found with a high frequency (110 Hz) and long pulse duration (200 msec).

The intensity of stimulation should be low enough to produce a comfortable, tingling, electrical paraesthesia. At this intensity the muscle is stimulated, but without the undesirable side effect of tetanic contraction. (Forster and Palastanga 1985:105)

Despite two decades of clinical usage, few controlled studies have been completed to investigate the relative effect of different combinations of TENS on pain relief. As the clinical effects of TENS are dependent on the correct combination of stimulation parameters - further research is required in this area. (Walsh et al. 1995.)

2.8.2. Treatment duration

There are no definite guidelines as to the most effective length of stimulation. Graff-Radford et al. (1988) chose a treatment duration of 10 min per treatment. Walsh et al. (1995)

applied TENS for 15 minutes per session, but they seemed to consider the combination of the stimulation parameters to be of greater importance than the treatment duration. Grimmer (1992) applied TENS for a 30 minute duration in her investigations on the effect of TENS on painful osteoarthritic ~~knees. All three studies~~ produced positive results, therefore, until definite guidelines are established, the treatment duration relies upon examiner discretion.

2.8.3. Electrode placement

The effectiveness of TENS for pain control is critically dependant on the correct electrode placement (Lindsay et al. 1990), however guidelines in this regard are vague. Two suggestions are placement directly over the trigger point or over the area of greatest pain intensity (the pain referral zone). (Forster and Palastanga 1985 : 105) Melzack (1975) demonstrated that TENS could be used effectively to reduce pain when applied directly over the TP. Graff-Radford et al. (1988) compared four modes of TENS. In each group the negative electrode was placed over the TP. There was a significant reduction in pain in three of the groups. As the electrode placement was constant, the difference in effectiveness was due to the different intensities applied in terms of amplitude, pulse width and frequency.

Walsh et al. (1995) conducted a peripheral nerve conduction study to investigate the hypoalgesic effect of TENS. For the purpose of this study, the electrodes were attached to the skin over the course of the nerve. Electrode placement was however a constant, the stimulation parameters were variable and the results were analyzed accordingly.

Robinson (1996) advocated placing the electrodes directly over the painful area in the treatment of soft tissue inflammatory conditions. In a controlled study of the effect of TENS on painful osteoarthritic knees, Grimmer (1992) achieved positive results by positioning the electrodes over the appropriate acupuncture points to maximise intrinsic opiate response. In this study once again however, the electrode placement was a constant. The variable was the choice of either Burst mode or High rate TENS.

There are therefore a number of choices of electrode placement. The author could not find any studies comparing the relative effectiveness of different electrode placements in the treatment of MFS nor any reference to the application of electrodes over the pain referral zone in the treatment of MFS.

Kruse et al. (1992) used thermography to demonstrate that sympathetic autonomic changes associated with MFS occur throughout the pain referral zone. Post ganglionic autonomic fibres are type C fibres, the same fibres that conduct most of the sensory information in peripheral nerves. (Guyton

1987:368). The fact that TENS indirectly modulates the transmission of impulses through these nerve fibres by stimulating the A beta nerve fibres was discussed in 2.7.4. There does therefore seem to be physiological evidence to support electrode placement over the pain referral zone, although any controlled studies to ~~that effect, could not be~~ found.

Graff-Radford et.al. (1988) suggested that TENS alone may be insufficient for the long term relief of pain caused by Myofascial syndrome because, although the subjects who participated in their study experienced a significant reduction in pain, there was no significant alteration in TP sensitivity. However in the light of the hypotheses proposed by Melzack (1981) and discussed in 2.7.4. ,this may not be true. Further studies are needed to evaluate the long term effects of TENS in MFS.

2.9. SUMMARY

From this discussion of the related literature it can be seen that Myofascial syndrome is an extremely common and problematic condition which affects a wide cross section of the population. It can also be concluded that TENS is a safe, non-invasive and cost effective modality which has long been recognised and widely used as an effective form of treatment for acute and chronic, superficial and deep pain syndromes (Ruud 1995). However there are still many questions which need to be answered before the optimal effectiveness can be achieved from this modality.(Robinson 1996)

This investigation will attempt to answer some of these questions by determining the relative effectiveness of placing electrodes over the AMFTP with placing them over the pain referral zone in the treatment of Myofascial syndrome, using TENS and passive stretching with reference to both subjective and objective clinical findings, in order to determine which of these methods is more effective.

CHAPTER 3: MATERIALS AND METHODS

3.1. OBJECTIVES OF THE STUDY

1) the first objective is to compare the effectiveness of placing ~~electrodes over the~~ AMFTP with that of placing them over the pain referral zone in the treatment of Myofascial syndrome using TENS and passive stretching in terms of subjective clinical findings.

2) the second objective is to compare the effectiveness of placing electrodes over the AMFTP with that of placing them over the pain referral zone in the treatment of Myofascial syndrome using TENS and passive stretching in terms of objective clinical findings.

3) the third objective is to correlate the data obtained during the study in order to determine which of these treatment methods is the more effective.

3.2. STUDY DESIGN AND PROTOCOL

3.2.1. SUBJECTS

Thirty subjects were needed to conduct the trial. In order to accomodate the possibility of non-compliant subjects or withdrawel of subjects from the study, forty subjects were initially solicited. Of the forty subjects recruited into this

trial; ten responded to advertisements read in the newspaper or heard over the radio, seventeen were referred for inclusion in the study either by other patients or colleagues and thirteen were approached as existing patients. None of the patients approached, declined the opportunity to participate in the study. ~~During the course of the study,~~ four of the subjects withdrew voluntarily and six were excluded due to failure to comply with the requirements of the study.

The subjects all lived or worked within a fifteen kilometer radius of the Technikon Natal Chiropractic day clinic. The average age of the subject was thirty six years. Sixty seven percent of participants were female, thirty three percent were male. Seventy seven percent were European, the remaining twenty three percent were Asian. Sixty three percent were employed, the remaining thirty seven percent were retired, unemployed or students.

Each subject complained of muscular pain of varying degrees and duration. This study only excluded those muscles to which the application of TENS would be contraindicated. The most common muscle involved was the Trapezius muscle (fifty percent); this was followed by Gluteus Medius (seventeen percent), Teres Major and Levator Scapulae (ten percent each), Infraspinatus (six percent), Rhomboids and Gluteus Maximus (approximately three percent each)

3.2.2. PATIENT EXAMINATION

A case history (Appendix 2) was taken from each subject, who was also given a full physical examination (Appendix 3). The patient was questioned as to where they experienced the pain and those muscles that could possibly account for that pain were then examined for the presence of AMFTPS. For example; if the subject complained of a temporal headache; the Trapezius, Sternocleido-mastoid, Temporalis and Suboccipital muscles would then be examined for the presence of AMFTPs. This is in accordance with the pain referral patterns set out by Travell and Simons in volumes one and two of the trigger point manual.

In order to be included in the study the subject had to exhibit at least five of the eight diagnostic criteria for Myofascial syndrome (Travell and Simons 1983 1: 18,19). A site of local tenderness was essential to the diagnosis. Reproduction of the pain by compression of the AMFTP is specific and strongly diagnostic. These two criteria, as well as the presence of a taut palpable band in the muscle, a local twitch response and restriction in the stretch range of motion of the affected muscle qualified the subject for inclusion in the study.

3.2.3. ENTRANCE REQUIREMENTS

- 1) Only patients between the ages of 16 and 60 years could be included.
- ~~2) There were no delimitations with respect to, race or gender;~~
or with respect to other co-existing conditions.
- 3) The onset of the pain may have been sudden or insidious and the condition may have been acute or chronic.
- 4) An AMFTP had to be identified which reproduced the patient's main pain complaint.
- 5) A taut palpable band had to be present which was tender when compressed. The pain referral pattern had to correspond to those characteristic pain patterns documented by Travell and Simons in volumes one and two of the trigger point manual.
- 6) Any muscle could be included except those to which the application of TENS would have been contra-indicated. That included those over the carotid sinus, larynx, pharynx and those on either side of the head (Temporalis) which would have resulted in the current running trans-cerebrally.
- 7) Patients fitted with a cardiac pacemaker or who suffered from cardiac arrhythmia had to be excluded.

8) The skin to which the electrodes were to be attached had to be inspected. Subjects with broken or anaesthetic skin were excluded.

Each subject who met the entrance requirements was asked to complete an informed consent form (Appendix 1).

3.2.4. GROUP SELECTION AND ELECTRODE PLACEMENT

The subjects were divided randomly into two treatment groups by blindly choosing a number from a container. Forty numbered pieces of paper were placed in a container. Twenty papers had the number "1" written on them and the remaining twenty had the number "2" written on them. The subjects were not told about the significance of the number they had chosen.

If the subject had more than one AMFTP the most active TP was selected for treatment. The most active TP was chosen objectively by means of algometer readings. The TP which produced the lowest algometer reading was selected.

Treatment group 1 was represented by the number "1". Subjects in group 1 would have the negative (black) electrode placed directly over the AMFTP in accordance with the method stipulated by Graff-Radford et al. (1988) The AMFTP was palpated and then straddled by two fingers to keep it in place. The negative electrode was then applied directly over this TP. The positive (red) electrode was applied alongside this

electrode at least 2 cm away to prevent a skin current and electrode burn.

Treatment group 2 was represented by the number "2". Subjects in group 2 would have electrodes applied at the most proximal and most distal point of the pain referral zone. If this involved applying the electrode over an anatomical site which would be contra-indicated, for example over the eye, then the electrode would be applied at the nearest possible site within the pain referral zone. The negative electrode was applied most proximally and the positive electrode was applied most distally. These basic principles of electrode placement applied regardless of the muscle being treated.

The most common AMFTP treated was TP1 of the Trapezius muscle. TP1 refers pain unilaterally along the posterolateral aspect of the neck from the upper border of the Trapezius muscle to the mastoid process. It may also extend to the temple, back of the orbit, angle of the jaw or occiput.

In this case the negative electrode was applied over the proximal border (the upper border of trapezius) and the positive electrode was applied over the distal border (the mastoid process, the temple, adjacent to the orbit, over the angle of the jaw or as close as possible to the occiput but below the hairline.)

The patient was given a brief explanation about Myofascial Syndrome and the mechanism of TENS. It was also explained that the patient would be required to attend a maximum of ten treatment sessions or as many as were required before they were asymptomatic. They were told that each treatment session would ~~last ten minutes and that they would be required to return for~~ a follow-up consultation approximately one month after the final treatment. The treatment duration was based on a study by Graff-Radford et al. (1988) during which the effect of TENS on myofascial pain and trigger point sensitivity was assessed. The author could not find any reference which recommended a definite number of treatment sessions and therefore decided on a maximum of ten treatments. A lower limit was not specified.

3.3. SUBJECTIVE AND OBJECTIVE MEASUREMENTS

Prior to the first treatment the subject was asked to complete a short form McGill Pain Questionnaire - Appendix 4 (Melzack 1987) and a Numerical rating scale 101 (NRS 101) - Appendix 5 (Jensen et al. 1986) to assess the quality and the quantity of the pain respectively.

The degree of disability experienced by the subject due to the existing Myofascial syndrome was assessed by asking the subject to complete a Pain Disability Index (PDI) - Appendix 6 (Tait et al. 1987.) These questionnaires were completed again once the patient was asymptomatic or at the end of ten treatments and again at the one month follow - up.

The short form McGill Pain Questionnaire assesses the quality of the patient's pain. The patient is asked to rate fifteen different descriptions of pain as either none, mild, moderate, or severe. To score this questionnaire, each response is given a numerical rating as follows:

None - 0

Mild - 1

Moderate - 2

Severe - 3

Each response was then added together and the sum divided by forty five. This figure was then expressed as a percentage.

The NRS 101 involves rating the severity (or quantity) of pain when it is at its least and when it is at its worst. This is done on a scale of 0% - 100%, with 0 representing no pain at all and 100 representing the worst pain imaginable. For statistical analysis, these percentages (worst and least) were used as is.

The Pain Disability Index (PDI) rates the amount of disability which the patient typically experiences (an average) in seven different categories of life activity. Each category is rated on a scale of 0 - 10, with 0 signifying no disability at all and 10 signifying that, that activity is prevented due to the pain. For statistical analysis a general disability score ranging from 0 to 70 is calculated by adding up the ratings of each category.

After identifying the AMFTP by digital palpation, the trigger point intensity was measured using a pressure algometer. (Reeves et al.1986) Algometer readings were taken before the first treatment, when the patient was asymptomatic, or at the end of ten treatments and again at the one month follow-up.

Algometry involves the measurement of ~~a patient's pressure~~ threshold. This threshold is that pressure which is first perceived as painful by the patient as increasing pressure is applied. (Travell and Simons 1992 2:12)

The algometer used in this study was a force dial manufactured by Wagner instruments :P.O. Box 1217, Greenwich, CT 06836 U.S.A.) Readings were taken as follows-:

The AMFTP was identified through palpation and vocal feedback from the patient to indicate the exact site which reproduced their clinical pain. This tender site was marked with a pen. The plunger of the algometer was placed over this mark with the shaft perpendicular to the skin.

The patient was instructed to indicate, through vocal feedback, when the sensation changed from pressure to pain, at which point the reading was taken.

The reading was measured in terms of kg/cm² and was recorded as such for statistical analysis.(Ohrbach 1990.)

3.4. TREATMENT

Each application of TENS lasted ten minutes. Carbon impregnated rubber electrodes with adhesive skin mounts were used and were placed either over the trigger point or over the pain referral zone depending on the treatment group concerned ~~as described~~ in 3.3. A dual channel Biomed Plus TENS unit was used in this study. It is manufactured by Biomedical Life systems, Incorporated. (1120 Sycamore Avenue, Suite F Vista, California 92083, U.S.A.) The settings chosen were based on the study by Graff-Radford et al.(1989), who concluded that they were the most effective settings for reducing the pain and TP sensitivity associated with MFPDS. The settings were as follows:

- 1) Wave form: modulated
- 2) Frequency: 100 Hz
- 3) Pulse width: 250 msec

The intensity was set to patient comfort below the threshold of muscular contraction. (Graff - Redford et al.1989)

Post-treatment stretching facilitates any procedure that attempts to inactivate an AMFTP, by reducing TP irritability and releasing the associated muscle tension. (Travell and Simons 1992 2:9) Passive stretch involves stretching the involved, relaxed muscle through its range of motion to the point of moderate pain (it should never be painful). In this

way the muscle is slowly restored to its full normal length by steadily and gradually increasing the force. Passive home stretches done on a regular basis are the key to maintaining this normal length and sustaining relief from the pain of Myofascial syndrome. (Travell and Simons 1992 1:66,89,96) None of the literature concerning the treatment of MFS appears to specify a time duration for passive stretching. For the purpose of this study, the involved muscle was stretched, immediately following the treatment, until it reached its greatest possible length whilst remaining within the patient's pain tolerance. The patient was also instructed to do the same stretch twice a day at home and to maintain that stretch for at least twenty seconds.

The patient was also questioned about possible causative factors such as poor posture, stress and overuse or chilling of the muscles and was advised accordingly. Other possible causative factors such as nutritional or endocrine problems were not addressed in this study. Patients were allowed to continue any analgesic or anti-inflammatory medication that they were taking for the condition but were asked to limit their intake as much as possible.

Each subject was scheduled for a series of ten treatments spread over a maximum period of 6 weeks. Each subject was treated for a maximum of ten sessions or until asymptomatic. The patient was considered asymptomatic when the TP no longer referred pain spontaneously (The TP had become latent). No

treatment was given to latent TPs. The patient was reassessed approximately one month after the final treatment.

3.5. DATA ANALYSIS

All the data was obtained from the NRS 101, algometer readings, the McGill short form questionnaire and the Pain Disability Index. The scoring of the questionnaires was discussed in 3.4.

Because of the small sample size, (15 subjects per treatment group) Non-parametric tests had to be utilised as follows:

3.5.1. To compare the results between groups 1 and 2, 12 Mann-Whitney Unpaired tests were used. The two groups were treated as being independent of each other. The purpose of these tests was to ascertain whether there was any significant difference between the two groups at the $\alpha = 0.05$ level of significance with respect to NRS 101, and the pain disability index.

Hypothesis testing and decision rule:

The null hypothesis H_0 states that there is no significant difference between the two groups with respect to the variable of interest. The alternative hypothesis states that there is a significant difference between the two groups.

$H_0 : p > 0.025$

$$H_1 : p \leq 0.025$$

$\alpha = 0.05$ = level of significance of the test.

Decision rule:

For a two tailed test,

Reject H_0 if $P > 0.025$

Accept H_1 if $P \leq 0.025$

P is the observed significance level of the test.

3.5.2. To compare the results within group 1, 12 Wilcoxon's Signed Rank tests were used. The purpose of these tests was to establish whether there was any significant improvement between the first (T1) and last (T2) treatment, the first treatment and the 1 month follow - up (T3) and between the last treatment and the 1 month follow - up. All tests were done at the $\alpha = 0.05$ level of significance.

Hypothesis testing and decision rule:

The null hypothesis states that there is no significant improvement between consultations T1 and T2, T1 and T3 and T2 and T3 within group 1 with respect to the variable of interest. The alternative hypothesis states the contrary of the null hypothesis.

H_0 : There is no significant improvement

H_1 : There is a significant improvement

$\alpha = 0.05$ = level of significance of test

Decision Rule:

For a two - tailed test,

Reject H_0 if $P > 0.025$

Accept H_1 if $P \leq 0.025$

P is the observed significance level of the test.

3.5.3. To compare the results within group 2, 12 Wilcoxon's Signed Rank tests were used. The purpose of these tests was to establish whether there was any significant improvement between the first (T1) and last (T2) treatment, the first treatment and the 1 month follow - up (T3) and between the last treatment and the 1 month follow - up. All tests were done at the $\alpha = 0.05$ level of significance.

Hypothesis testing and decision rule:

The null hypothesis states that there is no significant improvement between consultations T1 and T2, T1 and T3 and T2 and T3 within group 2 with respect to the variable of interest. The alternative hypothesis states the contrary of the null hypothesis.

H_0 : There is no significant improvement

H_1 : There is a significant improvement

$\alpha = 0.05$ = level of significance of test

Decision Rule:

For a two - tailed test,

Reject H_0 if $P > 0.025$

Accept H_1 if $P \leq 0.025$

P is the observed significance level of the test.

3.5.4. Three methods were used to measure central tendency; the median (middle value), mode (most often occurring value) and mean (average). Other summary statistics obtained were standard error and the coefficient of variation.

3.5.5. Bar graphs were used to present a visual summary of the results obtained from the Mann - Whitney and Wilcoxon's signed rank tests. These graphs were constructed using the package Quatro Pro.

3.5.6. The statistical package "statgraphics version 6 +" was used for data entry and analysis. This package is manufactured by Manugistics Inc. (2115 East Jefferson Street, Rockville, Maryland, 20852, U.S.A.)

The results are presented in the next chapter.

CHAPTER 4: RESULTS

The results of this study are divided into four different sections. Each section deals with a different form of measurement used in the study, these are as follows:

- 4.1. NRS 101
- 4.2. The McGill short form questionnaire
- 4.3. The Pain Disability Index
- 4.4. Algometer readings

Each section will determine whether there was a significant improvement within each group and whether there was a significant difference in the results between each group.

Group 1 had electrodes placed directly over the ATP

Group 2 had electrodes placed over the pain referral zone.

The following abbreviations will be used:

T11 - Treatment 1, Group 1

T21 - Final treatment, Group 1

T31 - 1 Month follow - up, Group 1

T12 - Treatment 1, Group 2

T22 - Final treatment, Group 2

T32 - 1 Month follow - up, Group 2

S - Significant Difference

NS - No Significant Difference

4.1. Comparison with respect to NRS 101

TABLE 1: Based on median readings. The lower the reading the more favourable the result.

	FIRST	FINAL	1 MONTH F/U
GROUP ONE	45	30	20
GROUP TWO	30	15	10

These results are presented visually in a bar graph (Appendix 7)

The summary statistics are as follows:

WITHIN GROUP 1:

MEAN: T11 = 47.2
T21 = 26.2
T31 = 18.2

MODE: T11 = 40
T21 = 30
T31 = 15

STANDARD ERROR:

T11 = 5.07
T21 = 4.84
T31 = 2.84

COEFFICIENT OF VARIATION:

T11 = 41.6
T21 = 71.5
T31 = 60.4

STANDARD DEVIATION:

T11 = 19.65
T21 = 18.73
T31 = 10.99

WITHIN GROUP 2:

T12 = 36.4
T22 = 17.7
T32 = 12.2

T12 = 30
T22 = 10
T32 = 0

T12 = 3.91
T22 = 3.87
T32 = 3.35

T12 = 41.6
T22 = 84.82
T32 = 106.3

T12 = 15.13
T22 = 14.98
T32 = 12.97

The results of the Wilcoxon Signed Rank tests are as follows:

WITHIN GROUP 1

T11 vs T21 : S

T11 vs T31 : S

T21 vs T31 : NS

WITHIN GROUP 2

T12 vs T22 :S

T12 vs T32 :S

T22 vs T32 :S

The result of the Mann - Whitney test between group 1 and 2 is as follows:

T11 vs T12 :NS

T21 vs T22 :NS

T31 VS T32 :NS

4.2. Comparison with respect to McGill short form questionnaire.

TABLE 2: Median readings (percentages). The lower the percentage, the more favourable the result.

	FIRST	FINAL	1 MONTH F/U
GROUP ONE	31	9	7
GROUP TWO	13	7	7

These results are presented visually in a bar graph (Appendix 8)

The summary statistics are as follows:

WITHIN GROUP 1

MEAN:T41 = 34.8

T51 = 13.4

T61 = 8.1

MODE:T41 = 22

T51 = 0

T61 = 7

WITHIN GROUP 2

T42 = 17.9

T52 = 10.1

T62 = 9.67

T42 = 13

T52 = 0

T62 = 0

STANDARD ERROR:

T41 = 5.9	T42 = 3.03
T51 = 3.46	T52 = 3.7
T61 = 3.1	T62 = 3.7

COEFFICIENT OF VARIATION:

T41 = 65.7	T42 = 65.7
T51 = 99.9	T52 = 141.1
T61 = 148.7	T62 = 148.2

STANDARD DEVIATION:

T41 = 22.86	T42 = 11.75
T51 = 13.39	T52 = 14.3
T61 = 11.9	T62 = 14.3

The results of the Wilcoxon Signed Rank tests are as follows:

WITHIN GROUP 1

T41 vs T51 : S
T41 vs T61 : S
T51 vs T61 : NS

WITHIN GROUP 2

T42 vs T52 : S
T42 vs T62 : S
T52 vs T62 : NS

The result of the Mann - Whitney test between group 1 and group 2 is as follows:

T41 vs T42 : NS
T51 vs T52 : NS
T61 vs T62 : NS

4.3. Comparison with respect to the Pain Disability Index

TABLE 3: Median readings. The lower the score, the more favourable the result.

	FIRST	FINAL	1 MONTH F/U
GROUP ONE	23	8	4
GROUP TWO	12	2	2

These results are presented visually in a bar graph (Appendix 9)

The summary statistics are as follows:

WITHIN GROUP 1

MEAN:T71 = 25.1

T81 = 9.13

T91 = 5.1

MODE:T71 = 23

T81 = 0

T91 = 0

WITHIN GROUP 2

T72 = 14.5

T82 = 5.13

T92 = 4.1

T72 = 6

T82 = 0

T92 = 0

STANDARD ERROR:

T71 = 3.47

T72 = 2.91

T81 = 2.26

T82 = 1.96

T91 = 1.36

T92 = 1.73

COEFFICIENT OF VARIATION:

T71 = 53.53

T72 = 77.6

T81 = 95.98

T82 = 147.79

T91 = 104.3

T92 = 164.3

STANDARD DEVIATION:

T71 = 13.45

T72 = 11.27

T81 = 8.77

T82 = 7.59

T91 = 5.28

T92 = 6.68

The results of the Wilcoxon Signed Rank tests are as follows:

WITHIN GROUP 1

WITHIN GROUP 2

T71 vs T81 : S

T72 vs T82 : S

T71 vs T91 : S

T72 vs T92 : S

T81 vs T91 : S

T82 vs T92 : NS

The result of the Mann - Whitney test between group 1 and group 2 is as follows:

T71 vs T72 : NS

T81 vs T82 : NS

T91 vs T92 : NS

4.4. Comparison with respect to algometer readings

TABLE 4: Median readings. The higher the reading, the more favourable the result.

	FIRST	FINAL	1 MONTH F/U
GROUP ONE	1.6	4.1	4
GROUP TWO	2	4.2	4.2

These results are presented visually in a bar graph (Appendix 10)

The summary statistics are as follows:

WITHIN GROUP 1

MEAN:T101 = 1.88

T111 = 3.63

T121 = 3.72

MODE:T101 = 1.6

T111 = 4.1

T121 = 5

WITHIN GROUP 2

T101 = 2.11

T112 = 4.16

T122 = 4.22

T102 = 1.4

T112 = 4.2

T122 = 4.2

STANDARD ERROR:

T101 = 0.2	T102 = 0.18
T111 = 0.31	T112 = 0.35
T121 = 0.31	T122 = 0.36

COEFFICIENT OF VARIATION:

T101 = 40.92	T102 = 32.42
T111 = 33.38	T112 = 32.66
T121 = 31.81	T122 = 33.48

STANDARD DEVIATION:

T101 = 0.77	T102 = 0.68
T111 = 1.21	T112 = 1.36
T121 = 1.18	T122 = 1.41

The results of the Wilcoxon Signed Rank tests are as follows:

WITHIN GROUP 1

T101 vs T111 : S
T101 vs T121 : S
T111 vs T121 : NS

WITHIN GROUP 2

T102 vs T112 : S
T102 vs T122 : S
T112 vs T121 : NS

The result of the Mann - Whitney test between group 1 and group 2 is as follows:

T101 vs T102 : NS

T111 vs T112 : NS

- T121 vs T122 : NS

CHAPTER FIVE: DISCUSSION

5.1. INTERPRETATION OF RESULTS

The results of this study, which were presented in chapter 4, will now be interpreted under the following three sub-headings:

5.1.1. Findings within group one

5.1.2. Findings within group two

5.1.3. Differences between groups one and two

5.1.1. FINDINGS WITHIN GROUP ONE

The patients in group one had electrodes applied directly over the ATP. All of these patients reported a significant improvement between the first and last treatment in terms of pain intensity (NRS 101), pain character (McGill) and disability (PDI). These results are summarised in Tables 1, 2 and 3 and depicted graphically in Appendices 7, 8 and 9 respectively. A significant improvement between the first and last treatment was also measured objectively by means of algometry. These measurements are summarised in Table 4 and depicted graphically in Appendix 10.

There was no significant improvement between the last treatment and the one month follow - up consultation in terms of pain intensity, pain character or pressure threshold (algometry), however further significant improvement was reported in terms of disability.

5.1.2. FINDINGS WITHIN GROUP TWO

The patients in group two had electrodes applied over the pain referral zone. Once again a significant improvement was reported between the first and last treatment in terms of pain intensity, pain character and disability. These results are summarised in Tables 1, 2 and 3 and depicted graphically in Appendices 7, 8 and 9 respectively. Similarly, a significant improvement was recorded objectively by means of algometry. These measurements are summarised in Table 4 and depicted graphically in Appendix 10.

Like group one, there was no further significant improvement between the last treatment and the one month follow - up consultation in terms of pain character or pressure threshold. The disability rating did not show any further improvement either. There was however further significant improvement in terms of pain intensity.

5.1.3. DIFFERENCES BETWEEN GROUPS ONE AND TWO

Although four outcome measures were used in this study, only two primary measures, pain severity and pain disability, were used to determine the differences between the groups. The reason for this was to avoid a possible type I ~~error, whereby~~ the possibility exists that a positive result may be found by chance only.

Comparison of the two groups showed that there was no significant difference in terms of pain severity and disability between the two groups after the first, last and follow-up treatment. These results are summarised in Tables 1 and 3 and depicted graphically in Appendix 7 and 9. Electrode placement over the pain referral zone is therefore as effective as placement directly over the AMFTP.

5.2. DISCUSSION

This study therefore supports the findings of Graff-Radford et al.(1988) and Walsh et al.(1995) who concluded that TENS was effective in the treatment of MFS. Both these authors however applied the electrodes directly over the AMFTP and concentrated on adjusting the stimulation parameters, rather than on the electrode placement itself. Despite the suggestion of electrode placement over the pain referral zone by a number of authors (Forster and Palastanga 1985:105), no studies could be found to support this theory. This study would therefore appear to

prove that electrode placement over the pain referral zone is as effective as traditional electrode placement directly over the AMFTP, there are however a number of limitations to this study which must be taken into account.

This trial ~~was conducted on thirty~~ subjects only, a relatively small sample size. Further trials, using a larger sample size and parametric analysis may find that TENS is not effective in both groups. The use of non-parametric statistics may also have decreased the chances of detecting changes due to intervention. The findings of this trial should therefore not be considered conclusive, but rather as a foundation used to plan larger more controlled studies.

The use of advertising for patient recruitment does leave the study vulnerable to the Hawthorne effect. A phenomenon whereby only those people with a high degree of interest respond for inclusion in the study. This may significantly effect the results. However in this study, only sixteen percent of the participants responded to advertising and would therefore not make a significant difference.

No symptom threshold was set. The subjects were treated for a maximum of ten treatments or until asymptomatic. There is therefore the possibility that natural history may have accounted for the subject's recovery. Analysis of the patient's records showed that the average number of treatments needed was 7. Sixty seven percent of the subjects recovered in less than

10 treatments and thirty percent of these were asymptomatic in five or less treatments. Therefore although the possibility of natural history accounting for the subject's recovery in any condition does exist, it seems to only be in a small percentage.

The treatment of Myofascial syndrome is difficult and complex due to the number of variables involved. These include amongst others, factors such as stress, nutritional inadequacies and biomechanical factors. This study should therefore have been controlled as possible. Possible improvements would be to exclude any subjects using medication for their condition, to restrict the study to only one specific muscle and to include a control group to provide an indication of what may happen if no treatment was given.

5.3. SPECULATION

Although all the patients in this study experienced a significant improvement in terms of pain and disability, it is difficult to ascertain to what extent TENS played a role in their recovery. ~~The placebo effect and the natural history of~~ MFS must also be taken into account.

Myofascial syndrome is an extremely difficult condition to conduct research on as there are so many variables involved. Stress, unaccustomed exercise, chilling of the muscle in an air-conditioned office or prolonged shortening of the affected muscle for example when driving long distances, are just a few factors which may cause the patient to regress despite the efforts of the researcher.

Patient compliance in terms of home exercises and keeping regular appointments are two further complications which are beyond the control of the researcher.

This study also did not take into account any nutritional inadequacies, metabolic or endocrine abnormalities, or focus in any great detail on any psychological abnormalities which may have prevented the patient's full recovery.

Without taking all these factors into account and conducting the study in a controlled environment, it is impossible to eliminate all possible variables.

Despite these problems, the general trend seems to indicate that TENS is an effective form of treatment for MFS and that placing the electrodes over the pain referral zone is an equally effective alternative to placing the electrodes directly over the ATP. The findings of this trial should not ~~however be considered conclusive, but rather as a foundation~~ used to plan larger more controlled studies.

CHAPTER SIX: CONCLUSIONS AND RECOMMENDATIONS

6.1. CONCLUSIONS

The aim of this investigation was to determine the relative effectiveness of ~~placing electrodes over the AMFTP~~ with placing them over the pain referral zone in the treatment of MFS using TENS and passive stretching, with reference to both subjective and objective clinical findings in order to determine which of these methods was more effective.

Analysis of the subjective findings within both groups revealed that the patients did experience a significant improvement in their symptoms. There was no significant difference in terms of subjective findings between the groups.

Analysis of the objective findings within both groups also revealed a significant improvement. There was no significant difference in terms of objective findings between the groups.

In comparing the results between the two treatment groups, there was no statistically significant difference between placing the electrodes directly over the AMFTP and placing them over the pain referral zone.

Placing the electrodes over the pain referral zone is therefore as effective as the traditional placement directly over the AMFTP and is, as such, an effective alternative therapeutic approach in the treatment of MFS.

6.2: RECOMMENDATIONS

This study should be repeated with a larger sample size to further validate the results.

Any study involving MFS should emphasise patient education to minimise the effect of predisposing or perpetuating factors. Patient compliance should also be stressed. This could prevent the lack of significant continued improvement and ensure long term effectiveness.

TENS can be used to effectively treat patients with MFS and is especially useful when treating patients who would prefer a non-invasive technique.

As TENS is a relatively cheap instrument and is easy to use, patients with chronic or recurrent conditions can purchase their own TENS unit and learn to manage their condition at home.

Although there is no significant difference in effectiveness between placing the electrodes directly over the AMFTP or over the pain referral zone, the practitioner may find that in certain situations, placement directly over the AMFTP is not practical. This may occur for example with TPs in the ~~suboccipital muscle where the patient's hair may get in the way~~ or if there is a skin lesion or loss of sensation in the vicinity of the TP making the application of electrodes in that area contra - indicated.

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-CONSENT FORM-

Welcome to this research programme.

I....., hereby understand that i will have to undergo a full Case History, Physical Examination and a Cervical Regional Examination conducted by the researcher. If clinically deemed necessary, X-rays will be taken of the Cervical Spine.

I also agree to abide by the rules and instructions, which will be explained to me by the researcher. If I do not abide to the rules and instructions, then I will be excluded from this study.

I therefore give my informed consent to be included into this research programme.

Thank you for your time and co-operation.

Signature :.....
(PATIENT)

:.....
(INTERN)

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC
CASE HISTORY

Patient: _____ Date: _____
file #: _____ X-Ray#: _____
Age: _____ Sex: _____ Occupation: _____
Intern: _____ Signature: _____

FOR CLINICIAN'S USE ONLY

Initial visit clinician: _____ Signature: _____

Case History:

Examination:

Previous:

Current:

X-Ray Studies:

Previous:

Current:

Clinical Path. lab:

Previous:

Current:

Case Status:

PTT: Conditional: Signed Off: Final Sign out:

Recommendations:

Intern's Case History

1. Source of History:
2. Chief Complaint: (patient's own words)

3. Present Illness:

- ▶ Location
- ▶ Onset
- ▶ Duration
- ▶ Frequency
- ▶ Pain (Character)
- ▶ Progression
- ▶ Aggravating Factors
- ▶ Relieving Factors
- ▶ Associated S & S
- ▶ Previous Occurrences
- ▶ Past Treatment and Outcome

4. Other Complaints:

5. Past Medical History:

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

6. Current health status and life-style:

- ▶ Allergies
- ▶ Immunizations
- ▶ Screening Tests
- ▶ Environmental Hazards (Home, School, Work)
- ▶ Safety Measures (seat belts, condoms)
- ▶ Exercise and Leisure
- ▶ Sleep Patterns
- ▶ Diet
- ▶ Current Medication
- ▶ Tobacco
- ▶ Alcohol
- ▶ Social Drugs

7. Immediate Family Medical History:

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

8. Psychosocial history:

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

9. Review of Systems:

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

TECHNIKON NATAL CHIROPRACTIC DAY CLINIC

PHYSICAL EXAMINATION

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

1. VITALS

Pulse rate:
Respiratory rate:
Blood pressure: R L
Temperature:
Height:
Weight:

2. GENERAL EXAMINATION

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

3. CARDIOVASCULAR EXAMINATION

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

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

Palpation: - Apex Beat (character + location):
 - Right or left ventricular heave:
 - Epigastric Pulsations:
 - Palpable P2:
 - Palpable A2:

Pulses:

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

Percussion: - borders of heart

Auscultation:

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

4. RESPIRATORY EXAMINATION

1) Is this patient in Respiratory Distress ?

Inspection

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

Palpation

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

Percussion

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

Auscultation

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

5. ABDOMINAL EXAMINATION

1) Is this patient in Liver Failure ?

Inspection

- Shape:
- Scars:
- Hernias:

Palpation

- Superficial:
- Deep = Organomegally:

- Percussion** - Rebound tenderness:
- Ascites:
- Masses:

Rectal Examination

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

External genitalia:
Hernias:
Masses:
Discharges:

Gait and Posture

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

G.C.S.:

- Eyes:
- Motor:
- Verbal:

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

[illegible]

- Pupillary light reflexes = Direct:
 = Consensual:
- Fundoscopy findings:
- III Ocular Muscles:
 Eye opening strength:
- IV Inferior and Medial movement of eye:
- V a. Sensory - Ophthalmic:
 - Maxillary:
 - Mandibular:
 b. Motor - Masseter:
 - Jaw lateral movement:
 c. Reflexes - Corneal reflex
 - Jaw jerk
- VI Lateral movement of eyes
- VII a. Motor - Raise eyebrows:
 - Frown:
 - Close eyes against resistance:
 - Show teeth:
 - Blow out cheeks:
 b. Taste - Anterior two-thirds of tongue:
- VIII General Hearing:
 Rinnes = L: R:
 Webers lateralisation:
 Vestibular function - Nystagmus:
 - Rombergs:
 - Wallenbergs:
 Otoscope examination:
- IX & Gag reflex:
- X Uvula deviation:
 Speech quality:
- XI Shoulder lift:
 S.C.M. strength:
- XII Inspection of tongue (deviation):

Motor System:

- a. Power
 - Shoulder = Abduction & Adduction:
 = Flexion & Extension:
 - Elbow = Flexion & Extension:
 - Wrist = Flexion & Extension:

- Forearm = Supination & Pronation:
 - Fingers = Extension (Interphalangeals & M.C.P's):
 - Thumb = Opposition:
 - Hip = Flexion & Extension:
 - = Adduction & Abduction:
 - Knee = Flexion & Extension:
 - Foot = Dorsiflexion & Plantar flexion:
 - = Inversion & Eversion:
 - = Toe (Plantarflexion & Dorsiflexion):
- b. Tone
- Shoulder:
 - Elbow:
 - Wrist:
 - Lower limb - Int. & Ext. rotation:
 - Knee clonus:
 - ankle clonus:
- c. Reflexes
- Biceps:
 - Triceps:
 - Supinator:
 - Knee:
 - Ankle:
 - Abdominal:
 - Plantar:

Sensory System:

- a. Dermatomes
- Light touch:
 - Crude touch:
 - Pain:
 - Temperature:
 - Two point discrimination:
- b. Joint position sense
- Finger:
 - Toe:
- c. Vibration:
- Big toe:
 - Tibial tuberosity:
 - ASIS:
 - Interphalangeal Joint:
 - Sternum:

Cerebellar function:

Obvious signs of cerebellar dysfunction:

- = Intention Tremor:
- = Nystagmus:
- = Truncal Ataxia:

Finger-nose test (Dysmetria):
Rapid alternating movements (Dysdiadochokinesia):
Heel-shin test:
Heel-toe gait:
Reflexes:
Signs of Parkinsons:

8. SPINAL EXAMINATION:(See Regional examination)

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

9. BREAST EXAMINATION:

Summon female chaperon.

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

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

APPENDIX 4

PATIENT NAME : -----

FILE # : ----- DATE : -----

	<u>NONE</u>	<u>MILD</u>	<u>MODERATE</u>	<u>SEVERE</u>
1 THROBBING	0) _____	1) _____	2) _____	3) _____
2 SHOOTING	0) _____	1) _____	2) _____	3) _____
3 STABBING	0) _____	1) _____	2) _____	3) _____
4 SHARP	0) _____	1) _____	2) _____	3) _____
5 CRAMPING	0) _____	1) _____	2) _____	3) _____
6 GNAWING	0) _____	1) _____	2) _____	3) _____
7 HOT-BURNING	0) _____	1) _____	2) _____	3) _____
8 ACHING	0) _____	1) _____	2) _____	3) _____
9 HEAVY	0) _____	1) _____	2) _____	3) _____
10 TENDER	0) _____	1) _____	2) _____	3) _____
11 SPLITTING	0) _____	1) _____	2) _____	3) _____
12 TIRING-EXHAUSTING	0) _____	1) _____	2) _____	3) _____
13 SICKENING	0) _____	1) _____	2) _____	3) _____
14 FEARFUL	0) _____	1) _____	2) _____	3) _____
15 PUNISHING-CRUEL	0) _____	1) _____	2) _____	3) _____

McGILL PAIN QUESTIONNAIRE

APPENDIX 5

NUMERICAL RATING SCALE - 101

Name:

Date:

Please indicate on the line below the number between 0 and 100 that best describes the pain of your major problem at this point, when it is at its worst. A zero (0) would mean "no pain at all" and a hundred (100) would mean "pain as bad as it could be." Please write only one number.

Please indicate on the line below the number between 0 and 100 that best describes the pain of your major problem at this point, when it is at its least. A zero (0) would mean "no pain at all" and a hundred (100) would mean "pain as bad as it could be". Please write only one number.

APPENDIX

PAIN DISABILITY INDEX

The rating scales below are designed to measure the degree to which several aspects of your life are presently disrupted by chronic pain. In other words, we would like to know how much your pain is preventing you from doing what you would normally do, or from doing it as well as you normally would. Respond to each category by indicating the *overall* impact of pain in your life, not just when the pain is at its worst.

For each of the seven categories of life activity listed, please circle the number on the scale which describes the level of disability you typically experience. A score of 0 means no disability at all, and a score of 10 signifies that all of the activities in which you would normally be involved have been totally disrupted or prevented by your pain.

1. *Family/Home Responsibilities.* This category refers to activities related to the home or family. It includes chores and duties performed around the house (eg, yard work) and errands or favors for other family members (eg, driving the children to school).

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

2. *Recreation.* This category includes hobbies, sports, and other similar leisure time activities.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

3. *Social Activity.* This category refers to activities which involve participation with friends and acquaintances other than family members. It includes parties, theater, concerts, dining out, and other social functions.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

4. *Occupation.* This category refers to activities that are a part of or directly related to one's job. This includes nonpaying jobs as well, such as that of a housewife or volunteer worker.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

5. *Sexual Behavior.* This category refers to the frequency and quality of one's sex life.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

6. *Self Care.* This category includes activities which involve personal maintenance and independent daily living (eg, taking a shower, driving, getting dressed, etc).

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

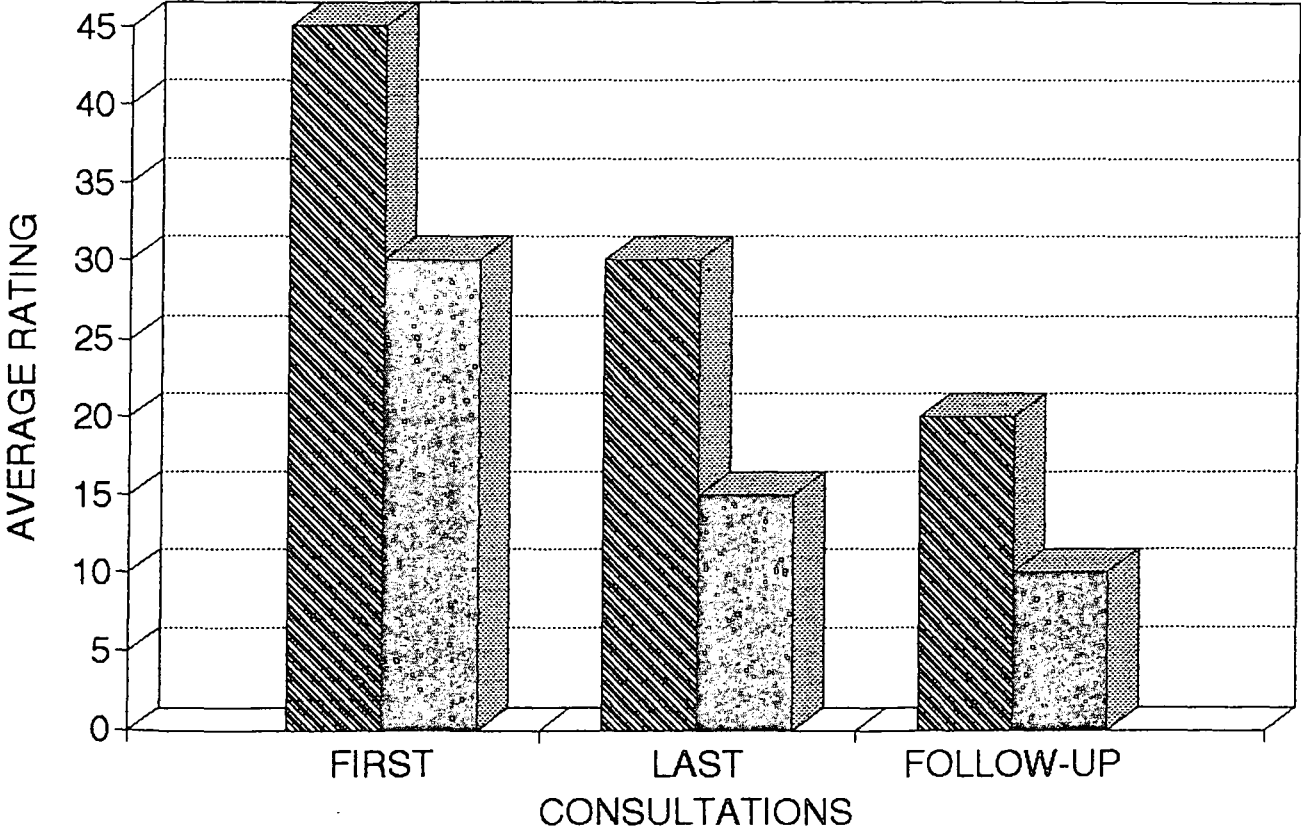
7. *Life-Support Activity.* This category refers to basic life-supporting behaviors such as eating, sleeping, and breathing.

0 1 2 3 4 5 6 7 8 9 10

no disability total disability

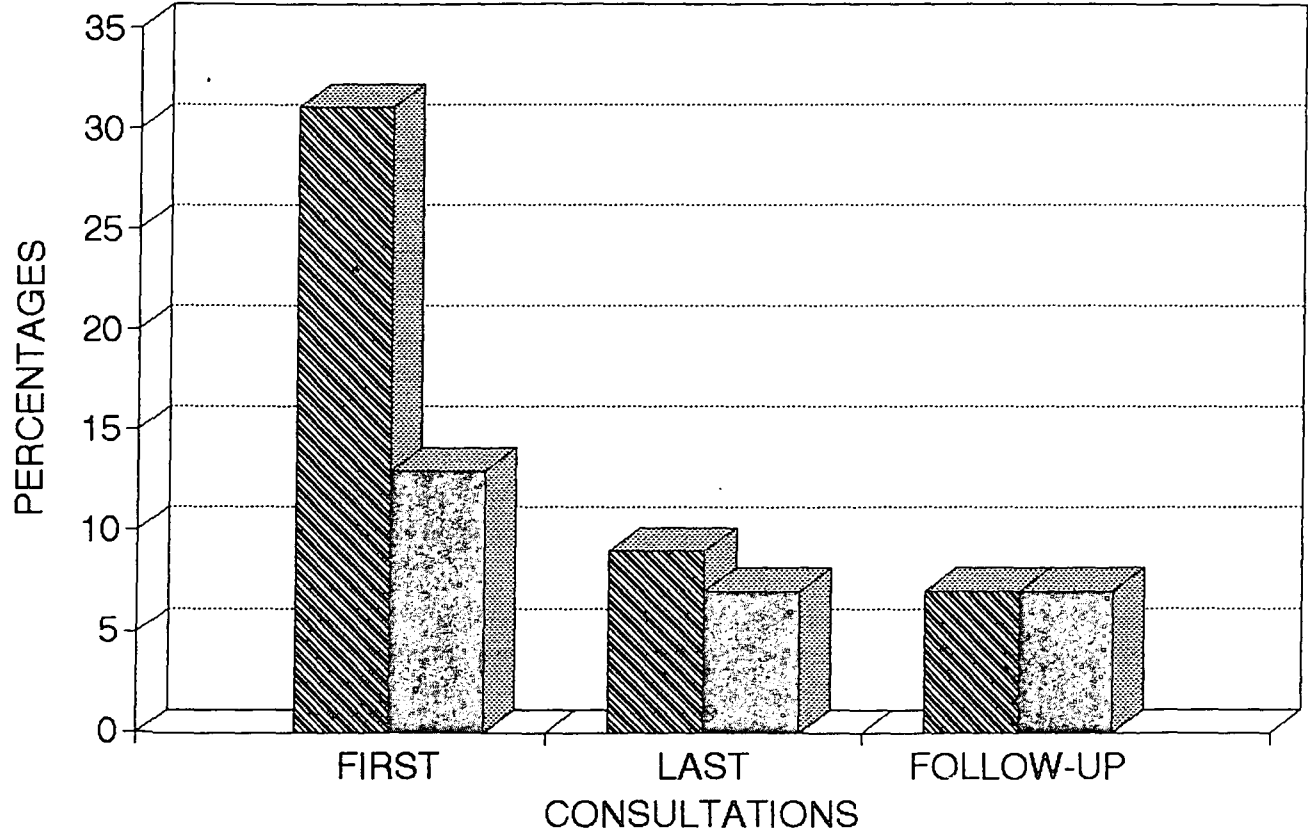


NRS 101
MEDIAN READINGS

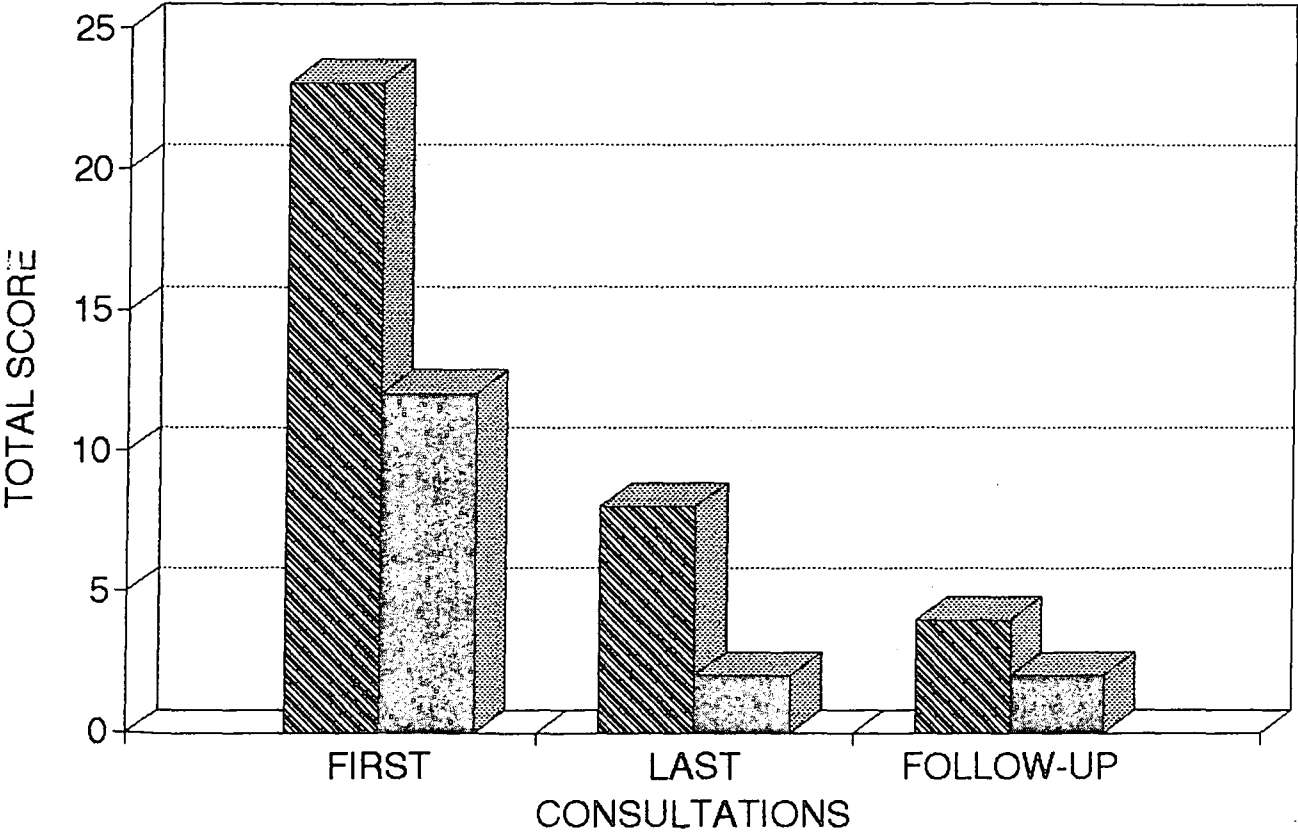


McGILL QUESTIONNAIRE

MEDIAN READINGS



PAIN DISABILITY INDEX
MEDIAN READINGS



ALGOMETER READINGS

MEDIAN READINGS

