

THE RELATIVE EFFECTIVENESS OF  
CRYOTHERAPY AND MOIST HEAT IN THE  
TREATMENT OF MYOFASCIAL PAIN  
SYNDROMES

By

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requirements for the Master's Degree in Technology:  
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I, Martin Steenfelt Andersen, do hereby declare that this dissertation is  
representative of my own work.

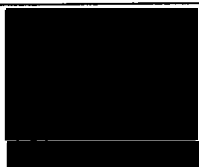
  
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## DEDICATIONS

This project represents the culmination of two years of 'hills and valleys'. This has been one of my biggest personal challenges with periods of triumph clashing with times of frustration. The final product is a testimony to the generosity and patience of certain individuals. I could not have achieved this goal without them.

To my Mom, Dad and brothers, you doubled my triumphs and halved my despairs, I thank you from the bottom of my heart. Your love and support carried me through many a strained period.

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The biggest thanks must go to the patients who gave up time and made the effort to help me complete this study. 'Knowledge is power' and without these many individuals, there would still be unanswered questions in my mind.

## ABSTRACT

Myofasciitis is a very common yet misunderstood problem. There are many treatments available yet there is no research to substantiate which of the many treatments available is the most effective (Travell and Simons 1983:6). The purpose of this study was to investigate the relative effectiveness of Cryotherapy versus moist heat in the treatment of myofasciitis of the shoulder girdle muscles.

Patients for this comparative, randomized clinical trial were obtained by consecutive sampling. Any patient between eighteen and fifty-five presenting to the Chiropractic Clinic at Technikon Natal with neck pain, upper back pain or shoulder pain was considered a potential candidate. Thirty patients underwent a screening process to assess their viability for the study. This screening procedure consisted of questions regarding the pattern of pain referral and of palpation of the relevant zones for muscle spasm, twitch responses, patient jump sign and/or referred pain. The thirty patients were randomly divided into two groups of fifteen. One group received cold and passive stretching and the other group received moist heat and passive stretching.

Each patient was treated five times within a three week period. Thereafter a follow-up appointment was scheduled one month after the final treatment to assess the long term effects of the treatments.

The subjective information was assessed using three questionnaires: (1) the CMCC Neck Disability Index, (2) the Numerical Pain Rating Scale-101 and (3) the Short Form McGill Pain Questionnaire. These three forms were used to subjectively assess various aspects of the patient's pain. Patients were required to fill these forms out at the first and



final treatments and again at the one month follow-up appointment.

The objective measurements were collected using the Algometer and the Cervical Range of Motion Goniometer. Readings were taken before and immediately after the first consultation, immediately after the final treatment and after a one month interval.

The information gathered at the relevant appointments was then statistically analyzed using the Wilcoxon Sign-ranked Test for intra-group analysis and the Mann-Whitney U Test for inter-group analysis. Summary statistics including the mean, standard deviation and standard error were obtained to support the results from the Wilcoxon's signed-rank test and the Mann-Whitney U test. Bar charts and tables were constructed to present the major findings from the Mann-Whitney and Wilcoxon signed ranked tests as a visual summary. The level of significance for all the tests was set at  $\alpha = 0.05$ .

According to the results of the Wilcoxon Sign-Rank Tests, within both groups, there was subjective and objective improvement during the treatment program. This improvement was maintained at a slightly lower level after one month. This supports hypothesis one and two which states that there will be improvement within each group. According to the Mann-Whitney U tests, there was no statistically significant difference between the two groups at any stage of the study which implies that both methods are equally as effective in the treatment of myofasciitis of the shoulder girdle muscles. Therefore one can reject the third hypothesis which states that there is a significant difference between the two groups.

This study suggests that both cold and passive stretching and moist heat and passive stretching are effective in treating myofascial trigger points. But this requires further research as

a control group was not used. In addition, it insinuates that both are equally as effective. It is the opinion of the author that cold and passive stretching is easier to use as it is more time efficient and less messy. Moist heat and passive stretching is recommended when treating older and frailer patients as it is a more gentle and soothing manner of treating myofasciitis.

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## DEFINITIONS

### 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, may be associated with referred autonomic phenomena, generally in its pain reference zone. (Travell and Simons 1983:1)

### FLAT PALPATION:

Examination by finger pressure that proceeds across the muscle at a right angle to their length, while compressing them against a firm underlying structure such as bone. (Travell and Simons 1983:2)

### ISCHEMIC COMPRESSION:

Also known as Acupressure, Myotherapy, Shiatsu, 'Thumb therapy'. The application of progressively stronger painful pressure on a trigger point for the purpose of eliminating the point's tenderness. (Travell and Simons 1983:2)

#### JUMP SIGN:

A general pain response of the patient who may cry out, and may withdraw in response to pressure applied on a trigger point. (Travell and Simons 1983:2)

#### LATENT MYOFASCIAL TRIGGER POINT:

A focus of hyperirritability in muscle or its fascia that is clinically quiescent with respect to spontaneous pain, it is painful only when palpated. (Travell and Simons 1983:2)

#### MYOFASCIITIS (ALSO MYOFASCIAL SYNDROME AND MYOFASCIAL PAIN SYNDROME):

Pain, tenderness, other referred phenomena, and the dysfunction attributed to myofascial trigger points. (Travell and Simons 1983:3)

#### PASSIVE RANGE OF MOTION:

The extent of movement (usually tested within a given plane) of an anatomical part at a joint when movement is produced by an outside force without voluntary assistance or resistance by the subject. (Travell and Simons 1983:3)

#### SUBJECTIVE CLINICAL FINDINGS:

Diagnostic procedures, as completed by the patient, that subjectively assess the condition of the same patient. This was achieved through the use of three questionnaires (CMCC Neck Disability Index, Numerical Pain Rating Scale-101 and the Short Form McGill Pain Questionnaire.

## REFERRED (TRIGGER POINT) PAIN:

Pain that arises in a trigger point, but is felt at a distance, often entirely remote from its source. The pattern of referred pain is reproducibly related to its site of origin. (Travell and Simons 1983:3)



# *CHAPTER ONE*

## *INTRODUCTION*

## CHAPTER 1: INTRODUCTION

The myofascial pain syndrome is one of the most common painful muscular dysfunctions found in patients (Hong et al. 1993). Myofascial trigger points are extremely common and become a distressing part of everyone's life at one time or another (Travell and Simons 1983:5).

In a study by Sola et al. (1954) 49.5% of 200 subjects were found to have one or more trigger points, and according to Goldenberg (1987) the estimated number of people in the United states suffering from myofasciitis falls between 3 and 6 million. Sola (1981) stated that the most common muscles affected by the myofascial pain syndrome were the trapezius, the levator scapulae and the infraspinatus. This was reinforced by Rubin (1981) who described myofasciitis of the trapezius as the most common cause of craniofacial referred pain.

Both Sandmann (1981) and Rosen (1993) agree that as a result of physicians failure to recognise and understand myofascial pain syndromes, and to correct the underlying dysfunctions, treatment protocols are often prolonged and unsuccessful. According to Rosen (1993), myofascial trigger points have been misdiagnosed as tendinitis, bursitis, entrapment neuropathy, overuse syndrome and even arthritis. Unrecognized and incorrectly treated myofascial pain syndromes are also a major cause of industrial lost time, resulting in applications for compensation totaling millions of dollars annually (Travell and Simons 1983 :6).

Adding to the uncertainty surrounding this syndrome is the fact that historically, many names have been used to describe the same condition. For example Myofascial Pain Syndrome has also been called muscular rheumatism, myalgia, myogelosis, interstitial myofibrositis, fibromyositis and myofasciitis to name a few (Auleciens 1995). There are, furthermore, no

definitive laboratory or imaging tests and it is, therefore, vital to become thoroughly acquainted with this enigma (Simons 1991).

The clinical characteristics of myofascial trigger points can be described as a localized pain associated with a tense, shortened muscle in which an exquisitely tender spot can be located. The tender spot lies in the taut band of muscle fibers, which is the cause of muscle tension and reduced range of motion, and refers pain in a specific pattern characteristic of that muscle. The patients referred pain may be reproduced by digital pressure on the tender point and may be associated with a localized twitch response (Simons 1991). Both Friction (1994) and Travell and Simons (1983 :16) describe an affected muscle as showing increased fatigability, stiffness, subjective weakness, pain on movement and slight restriction of range of motion which is unrelated to joint restriction.

Sola (1984) believes that muscle overload is the most common cause of trigger points but lists the following as also being significant: emotional and physical stress, disease, anxiety, fatigue and systemic diseases. Furthermore the postural muscles seem to be the most common muscles affected. A common upper trunk trigger point cluster would include the trapezius, levator scapulae and the infraspinatus while in the lower trunk active trigger points in the quadratus lumborum, the gluteus medius and the tensor fascia lata are typical.

Many authors claim that the treatments must include numerous adjunctive techniques to optimize the benefits. For example Rubin (1981) recommends correcting faulty body mechanics, systemic deficiencies and stress factors to avoid recurrent trigger point activity while Sandmann and Backstrom (1984) emphasize the importance of managing both the physiological and the psychological components of the problem. Both Lewitt and Simons

(1984) and Murphy (1989) mention putting the patient on a home exercise program to maintain any progress.

Treatment protocols are numerous and varied. The options chosen tend to be based on personal preference rather than on objective findings. According to Sola (1984) most of the treatments are aimed at disrupting, desensitizing or eliminating the pain-spasm-pain cycle which maintains the trigger point activity even after the initiating stress has resolved.

Sola and Williams (1955) described the process of wet-needling whereby an analgesic (procaine or lidocaine) or other substance (saline solution or sterile water) is injected into the trigger point. A similar technique is the process of dry needling described by Melzack (1981) whereby the trigger point is repeatedly penetrated by a needle, producing hyperstimulation analgesia. Garvey et al. (1989) preferred dry needling as there was no danger of a drug reaction or tissue necrosis, but Murphy (1989) reported more consistent success in treatment with wet needling. Lewitt and Simons (1984) described needling as being invasive, painful and not an option in patient autotherapy.

Murphy (1989) used various electrical modalities to treat trigger points which included high voltage stimulation, Transcutaneous electrical nerve stimulation, microamperage stimulation and ultrasound. Hong et al. (1993) felt that ischaemic compression was very effective in immediate decrease in pain. Travell and Simons (1983 : 87-88) listed three variations (digital pressure, kneading and vibratory) of ischaemic compression. This technique is painful and is limited by the patient's tolerance.

In a study by Lewitt and Simons (1984), stretching was used to relieve increased muscle tension associated with myofascial pain. They believed that stretching not only abolished trigger points in muscles, but also relieved painful ligaments and periosteum in the region of

attachment. Graff-Radford et al. (1987) believe that spray and stretch could reduce pain by 50% or more at the time of examination, while Travell and Simons (1983 : 63) felt that spray and stretch inactivates trigger points more quickly and with less patient discomfort than local injection and ischaemic compression. Furthermore Travell and Simons (1992 : 9) felt that the effects of vapocoolant spray could be obtained by stroking the affected area with ice. The pain threshold of a normal shoulder was assessed following treatment with ice and with heat and it was concluded that ice was more effective than heat in elevating the pain threshold, but it was acknowledged that an injury may react differently (Benson and Copp 1974). Gattermann (1990 : 534) wrote that the use of cold acts as a counterirritant to pain which facilitates a greater stretch. Hong et al. (1993) postulated that the use of heat may provide relief by resolving painful inflammatory reactions as a result of increasing the blood flow to the area but stated that it was unclear whether or not moist heat caused significant relief of the tightness in the taut bands of the muscle fibers.

Cryotherapy and moist heat, combined with passive stretching, are simple treatments that require no expensive machinery or equipment. The applications are not too time consuming and can be carried out with ease in the chiropractic office (Sandmann 1981). Of importance is the fact that the patient can be educated with respect to self treatment as demonstrated in a study of post-isometric relaxation by Lewitt and Simons (1984) where the best, lasting results in the relief of myofascial pain came from patients who practiced auto-therapy. Jones (1994) wrote that the pathomechanics regarding myofasciitis are not precisely defined and treatment methods are varied, while Sola and Williams (1955) felt there was a need to explore new therapeutic methods as responses to treatment were often unpredictable. Gatterman (1990 : 534) felt there was a lack of research regarding the use and physiological

mechanisms of Cryotherapy. In a study on the immediate effects of various treatment modalities on the pain threshold of an active myofascial trigger point, Hong et al. (1993) were unable to find any references in the literature regarding the measurement of pain threshold of trigger points in order to document the effectiveness of moist heat.

The purpose of this investigation was to compare the effectiveness of Cryotherapy and passive stretching versus moist heat and passive stretching in the treatment of myofascial trigger points. This was done in terms of the patients' objective and subjective clinical findings in order to determine which was the more effective treatment. The objective measurements included the use of the algometer and the Cervical Range of Motion (C.R.O.M.) goniometer while the subjective responses were analyzed using the Short Form McGill Pain questionnaire, the Numerical Pain Rating Scale-101 and the CMCC Neck Disability Index. These measurements could then be compared to statistically determine the more effective of the two treatments.

There appears to be a gap in the literature regarding the use of moist heat in conjunction with passive stretching and it is unclear whether Cryotherapy or moist heat, used in conjunction with passive stretching, is a more effective treatment protocol. Myofasciitis is such a common problem and successful clarification of the treatment protocols will contribute to the body of knowledge regarding this distressing condition. Practitioners will be able to chose the most effective means of treating the patient with the intention of quick, effective and long-lasting results with minimal discomfort to the patient.

# CHAPTER TWO

## REVIEW OF THE RELATED LITERATURE

## CHAPTER 2: REVIEW OF THE RELATED LITERATURE

### 2.1 INTRODUCTION

The high prevalence of pain originating from muscles is not surprising since collectively, the voluntary skeletal muscle constitutes the largest single tissue mass in the human body, accounting for more than 40% or more of the body weight (Gray 1973:8). The skeletal muscles are subject to wear and tear for the greater part of waking hours and one must expect them to be subjected frequently to disease (Good 1950). Bruce (1995) believes that despite the remarkable advances in modern health care, a void exists in the understanding, evaluation and management of day to day musculoskeletal aches and pains.

According to McClaflin (1994) the myofascial pain syndrome is a relatively unknown disease that evokes the image of vague general pain and dysfunction, while Auleciens (1995) believes that myofascial pain is one of the least understood yet most commonly encountered problems in an outpatient setting. Friction (1994) writes that myofascial pain is a regional muscle pain disorder characterized by local muscle tenderness and pain and is the most common cause of persistent regional pain such as back pain, shoulder pain, tension type headaches and facial pain.

As a result of clinician's failure to recognise and understand myofascial pain syndrome, and to correct the underlying cause, treatment prognosis is often prolonged or unsuccessful (Rosen 1993). This is a source of great frustration for patient and doctor as according to Sandmann (1981) the prognosis is excellent with proper diagnosis and management.



Travell and Simons have made much progress on the clarification of this condition. They too, however, admit that an effective treatment for this common and debilitating disorder is needed, as various techniques have been developed, all with varying degrees of effectiveness (Travell and Simons 1983:6).

## 2.2 PREVALENCE

According to Sola (1984), myofascial pain is probably the most common pain problem faced by physicians while Travell and Simons (1983 :5) believe that everyone will be affected at one time or another during their lives.

In a survey by Sola in 1954, 200 asymptomatic individuals were examined and 49,5% were found to have one or more trigger points. Furthermore, females were found to have a higher incidence than males, with 54% of 100 females testing positive. Finally 84,7% of the trigger points found occurred within four muscles, namely the trapezius, levator scapulae, infraspinatus and the scalenes. This could be explained by the fact that the stresses involved in the onset of the myofascial pain affect whole muscle groups and that the trigger points therefore tend to cluster.(Sola 1984).

According to Travell and Simons (1989) and Gerwin (1994), the upper trapezius is the most common cause of myofascial pain, while Sola (1984) and Bruce (1995) both believe that a common cluster of trigger points in the neck would include the trapezius, levator scapulae and the infraspinatus. The levator scapulae was found to be the most common cause of stiff necks (with severe restriction of neck rotation) although the upper trapezius, splenii and the sternocleidomastoid muscles were usually also affected (Travell and Simons 1989).

According to Sola (1984) and Gattermen (1990 :285) the muscles most commonly affected in the lower trunk are the quadratus lumborum, the gluteus medius and the tensor fascia lata. Sola (1981) believed that the reason the antigravity muscles are so commonly affected was that they are subjected to chronic stress and generally lack proper conditioning.

Trigger points are generally more common amongst sedentary individuals and are associated with chronic strain and stress. Laborers and athletes have a lower prevalence of trigger points and when present tend to result from overuse and injury rather than chronic stress. Sola (1984) and Bruce (1995) therefore concluded that exercise is of therapeutic value in the prevention of myofascial pain dysfunction. Travell and Simons (1983:6) believed that individuals in their mature years of maximum activity are most likely to suffer from active trigger points and with the advancing of years, with the associated reduced activity, the stiffness and restricted range of motion of the latent trigger points becomes more common. According to Travell and Simons (1983:5), Goldberg (1987) and Simons (1991) latent trigger points are far more common than active trigger points.

A problem to date is that most of the research conducted has involved clinical populations, therefore the prevalence of myofascial pain syndrome in the general population can only be estimated indirectly (Bruce 1995).

### 2.3 AETIOLOGY

Sola (1984) states that trigger points may assume two roles in the etiology of myofascial pain: a primary role as a translator of stress stimuli causing pain responses and a secondary role of pain intensification when pain becomes a component of the initiating stress.

A variety of body conditions exist which, when coupled with a triggering stress, will lead to trigger point activation. Body conditions include genetic factors, personality, physical conditioning and physiological state (previous injury and hormonal balance) while triggering stresses will include physical (disease/fatigue injury) and mental (fatigue/ anxiety) factors (Sola 1984). Rubin (1981) lists injuries, viral or bacterial infections, immobilization and psychogenic stress as significant etiological factors.

According to Murphy (1989), direct trauma to the muscle or indirect trauma such as a whiplash may disrupt the muscle tissue leading to inflammation and the subsequent formation of trigger points. Sandmann (1981) supports this view stating that organic pathology occurs at the point of sustained contracture, along with a decrease in blood supply and accumulation of metabolites. Gatterman (1990 :285) believes that repeated microtrauma is a significant factor while Yunus et al.(1987) state that the stress may be either acute or chronic. Sola (1984) believes that the initiating stress may be soft tissue trauma or contusions that cause pain out of proportion to the initial injury.

Fricton (1994) believes that trigger points often arise out of muscles that have been weakened through immobilization (e.g. use of cervical collars or extended bedrest). Rosen (1994) believes that the primary cause of trigger points is an imbalance between forces of an agonist and antagonist muscles, as this imbalance leads to overload and subsequent tissue breakdown. Fricton (1994) supports this writing that abnormal functional patterns contribute to abnormal proprioceptive input and sustained muscle contraction in an attempt to correct the poor postural relationship and to compensate for the altered neuromuscular function. Static loading of the muscles produces overload fatigue and microtrauma to the muscle fibers (Gatterman 1990 :285), a sentiment shared by McClaflin (1994). Gay et al. (1994) state that a muscle in myofascial dysfunction is not in a

state of constant or advanced fatigue but rather shows accelerated fatigue in response to normal functioning.

Sandmann and Backstrom (1984) believe that a patient's psychological profile is an important factor and list the following as examples of etiological factors: stress, depression, tension, anxiety, type A behavior patterns, irrational belief systems and learned helplessness. Friction (1994) agrees and includes maladaptive behavior such as pain verbalization, poor sleeping and dietary habits, lack of exercise, poor posture, bruxism and medication dependencies as being significant.

#### 2.4 PATHOPHYSIOLOGY

According to Sola (1984), the exact mechanism of trigger points has not been defined, but trigger points can be considered to be weak points within a muscle or its fascia that are particularly sensitive to stress-induced change. Auleciems (1995) states that trigger points tend to occur in muscles that frequently withstand sustained activity, such as the muscles in the back, neck and lower extremities.

Yunus (1988) believes that acute or chronic muscle stress is the primary initiating factor in the pathogenesis of trigger points. Gattermann (1990 :291), Friction (1994), Gerwin (1994) and Auleciems (1995) propose the following hypothesis to explain the initiating cycle behind the development of trigger points:

In any acute or chronic stress to a muscle there will be a degree of tissue damage involving disruption of the sarcoplasmic reticulum. The damage releases stored calcium into the injury site and impairs the ability of the sarcoplasmic reticulum to remove that calcium.

Furthermore, the injury may result in the disruption of small blood vessels, releasing platelets and leading thereafter to the release of serotonin. Any damage to the

connective tissue may cause the break down of mast cells, releasing histamine. These substances have the effect of sensitizing the nerve endings within the site of trauma causing pain.

The released calcium interacts with the available Adenosine Triphosphate (ATP) causing a prolonged contraction of the sarcomere leading to fatigue. It is this contraction which leads to the shortening and tensing of the muscle fibers which causes the characteristic taut palpable band found in myofascial trigger points.

The uncontrolled metabolic activity has the following effects:

1. It depletes the available ATP stores from which energy is generated to recock the contractile mechanism within the actin-myosin complex.
2. Various waste products such as lactic acid and prostaglandins may be produced causing an inflammatory reaction to occur. These substances are also capable of sensitizing the nerve endings.
3. A reflex, localized vasoconstriction results to control this metabolic activity. This reaction is mediated by autonomic nerves activated by trigger point sensory input to the central nervous system.

The vasoconstriction has the effect of reducing blood flow within the tissues causing a localized area of ischaemia and pain. The characteristic myofascial pain may outlast the initiating event, setting up a self generating cycle that is perpetuated through lack of proper treatment, sustained muscle tension, distorted muscle posture, pain reinforcing behavior and failure to reduce other contributory factors such as inactivity or sleep disturbance.

Sandmann and Backstrom (1984) believe that stress is also implicated in causing trigger points. They propose the following hypothesis:

Stress causes increased production of serotonin, epinephrine, norepinephrine, acetylcholine and dopamine which increases the excitability of nerve cells. The increased neurological input causes the individual to contract the muscles for long periods of time causing a decrease in blood flow in the area. This action decreases metabolism in the area and allows calcium, lactic acid and hyaluronic acid to accumulate. The cycle then continues as the authors have described above.

According to Sandmann (1981), normal function of the muscle tissue may be restored by stretching all the locked actin and myosin filaments far apart enough to eliminate contraction. Enough ATP will then accumulate to restore normal sarcoplasmic activity and allows the decreased circulation to slowly remove the build-up of metabolites.

## 2.5 PERPETUATING FACTORS

Friction (1994) states that a muscle is more predisposed to developing problems if it is held in a sustained contraction, either within or out of the normal position. According to Rosen (1994), the most important cause of recurrent pain and dysfunction is probably incomplete or inadequate rehabilitation of a previously painful process which has presumably resolved without the patient having achieved a normal range of motion or normal strength. Both Sola (1984) and Auleciems (1995) agree that the treatment of perpetuating factors is essential in treating the pain and improving the prognosis as the event that activates the trigger points is usually quite different from the factors that perpetuate them.

Rosen (1994) draws a distinction between tissue pain and tissue dysfunction writing that tissue dysfunction can exist in a dormant phase without interfering with normal activities. It is only when the individual stresses the tissues

beyond their normal requirements that this 'silent' perpetuating factor acts and clinical pain results.

Rubin (1981) lists injuries, viral or bacterial infections, immobilization, psychogenic stress and other environmental factors as significant perpetuating factors. Fricton (1994) implicates a unilateral short leg, a small hemipelvis, non-compensated scoliosis, occlusal abnormalities and poor position of the head and tongue as significant, while Sola (1984) stresses the individual's genetics, personality, conditioning and physiological state.

Rosen (1993), Graff-Radford *et al.* (1987) and Fricton (1994) are all in agreement over the perpetuating factors but the most comprehensive discussion comes from Simons and Simons (1989) and is as follows:

Perpetuating factors may be either Systemic or Mechanical.

Under the systemic factors are the following:

1. Nutritional inadequacies: This includes the B-complex vitamins (particularly B1, B6, B12 and Folic acid) and low serum levels of potassium, calcium, zinc, copper, iron and trace minerals.
2. Metabolic and endocrine dysfunction: This includes gout, anemia, low electrolyte levels and hypoglycemia. These factors increase muscle irritability and aggravate the symptoms caused by trigger points.
3. Chronic infection and infestation: e.g. bacterial and viral infections and parasitic infestations. Bacterial infections, like an abscessed tooth, infected sinus or chronic urinary tract infection are implicated, while viral infections, like colds and flu have been found to markedly increase the irritability of trigger points. Parasites include fishworms, giardiasis and amebiasis.
4. Post traumatic hyperirritability syndrome: These patients have suffered trauma severe enough to

disrupt the sensory pathways to the central nervous system and experience pain that is triggered off by normally inconsequential sensory stimuli.

5. Psychological stress: An undiagnosed and untreated chronic myofascial pain syndrome impacts severely on a patient's social and private lives and serves only to perpetuate the problem.

Under the mechanical factors are the following:

1. Anatomical variations: including a short leg, a hemipelvis, short upper arms or a long second metatarsal bone.
2. Seated postural stress: including chairs that are too high, or chairs with inadequate back support or arm rests.
3. Standing postural stress: standing in a head-forward posture can be improved by shifting the center of gravity over the balls of the feet and emphasizing the lumbar lordosis. A slanted walking or running surface is commonly implicated in causing trigger points.
4. Vocational stress: work that requires sustained shoulder elevation, arm abduction or forearm supination and a strong grasp are common culprits.

## 2.6 NEUROPHYSIOLOGY

There are two different kinds of cutaneous pain. The 'first pain' is conducted rapidly to the consciousness, is well localized and of short duration. This pain is mediated by the A-delta fibers, the thin myelinated fibers found in cutaneous tissues. These fibers respond to thermal and mechanical stimulation. The second pain is conducted more slowly, is not localized, is longer lasting and may continue long after the painful stimulus has ceased. This pain is mediated by the thin, unmyelinated group C fibers. The second nociceptors are present in the skin and in the muscle and may be responsible for the dull aching quality of trigger point pain. (Frampton 1985; Gerwin 1994.)



The injection of a noxious substance, like Bradykinin, into the skeletal muscle sensitizes the nociceptors to mechanical rather than thermal stimulation. The mechanical threshold of the nociceptors is lowered into the innocuous range leading to activation of the pain transmission pathways by normally non-pain producing stimulation (Gerwin 1994). This could explain why trigger points are hypersensitive and display pain continuously in the zone of reference (Fricton 1994).

Central to the concept of trigger points is the extraordinary tenderness and ability to produce pain at a distant site. The trigger point is a hypersensitive area in the tissue which has an expanded receptive field that includes distal areas that are usually distal to the primary trigger point area of pain. (Gerwin 1994).

According to Simon (1991) five mechanisms have been hypothesized to explain referred pain within man. These mechanisms are as follows:

1) Convergence Projection : A spinal nerve in the cord will receive input from different areas including internal organs and nociceptors coming from the skin. The brain has no way of distinguishing whether the input comes from the somatic structures or from the visceral organs. With trigger points, the pain is initiated by muscle nociceptors but is referred to a wider area served by other somatic receptors that converge on the same tract.

2) Peripheral branching of primary afferent nociceptors: Referred pain may occur when branches of the same nerve supply different areas of the body. In this model the brain misinterprets the messages originating from nerve endings of one part of the body as coming from the nerve branches supplying the other part of the body.

3) Convergence facilitation: Here excessive nerve activity originating within a trigger point facilitates a

spinothalamic cell that also receives low grade neural activity from other somatic areas. Normally this background activity is not sufficient to excite the spinothalamic tract. With the trigger point however, the threshold is lowered and stimulation that is normally innocuous, is enough to trigger off pain perception.

4) Sympathetic nervous system activity: Sympathetic nerves may cause pain by releasing substances that sensitize primary nerve endings in the region of perceived referred pain

5) Convergence or image projection at the supraspinal level: It is probable that as pain pathways converge peripherally, they may converge at the thalamic or cortical level. Therefore stimulation at one area may be perceived as coming from another area.

According to Gerwin (1994) the mechanism for the development of referred pain is similar in different tissues such as skin, muscle, viscera and nerves and patterns of pain may involve only one organ or mixed organs (e.g. skin and/or viscera and/or muscle).

According to Sandmann and Backstrom (1984), the pain cycle in trigger points can be broken by providing a distracting stimulus that inhibits the reflex pathways that perpetuate the cycle activity via the spinal cord and higher brain centres. Melzack (1981) calls this 'hyperstimulation analgesia.'

## 2.7 DIAGNOSIS OF MYOFASCIAL PAIN DYSFUNCTION SYNDROME

According to Sandmann (1981), physical examination, laboratory evaluation and radiological studies are generally negative, while orthopedic testing and range of motion may be completed without significant increase in the patient's

discomfort. One can therefore appreciate the importance of clinical pain in the diagnosis of the trigger points.

Yunus (1987) and Simons (1991) believe that none of the modern laboratory or imaging methods have been successful in establishing a diagnostic gold standard for any of the many points of view from which the patients are examined. In terms of laboratory testing Friction (1994) states that blood and urine samples are generally normal unless the abnormality is caused by a concomitant disorder. According to Travell and Simons (1989) and Auleciens (1995), certain laboratory tests may be useful in identifying positive perpetuating factors. Auleciens (1995) listed serum vitamin levels, blood chemistry panels, complete blood counts, sediment rates and thyroid studies as being useful in this regard.

Medication also appears to have little affect in treating trigger points. McClafflin (1994) and Gerwin (1994) both agree there is little evidence to support the presence of inflammation in myofascial pain syndrome and therefore non-steroidal anti-inflammatory drugs may only temporarily relieve pain.

Gerwin (1994) states that no consistent anatomical changes have been identified using light microscopy, electron microscopy or histochemistry. This is disputed by Rosen (1993) who stated that electron microscopy has shown muscle fibers bunching up within the taut band region.

Sandmann (1981) states that electromyographic (EMG) studies may show EMG activity to be absent within the tense muscle fibers which contain the active trigger point and that eliciting a twitch response by snapping palpation can record a transient burst of motor unit activity. In a study by Gay et al. (1994) on the characteristics of muscle fatigue in patients with myofascial pain-dysfunction syndrome, the EMG signal characteristics for normal adults and patients with trigger points were compared.

The following results were recorded:

1. In the patient group the mean endurance times were significantly shorter.
2. Three patients recorded no EMG activity within the tight muscle bands.

Simons (1991) believes that the localized twitch response is valuable as a totally objective confirmation sign that is pathognomonic of a myofascial trigger point.

The patients pain and distribution of pain on palpatory examination and goniometric testing for limit of movement or restriction of range of motion are diagnostic guidelines according to Sandmann (1981), while Travell and Simons (1989) believe reproduction of the patient's pain is the most valuable confirmatory evidence of an active trigger point.

Fischer (1987) advocates the use of pressure threshold measurements with the instrument known as the algometer to objectively identify the tender areas and is quoted as writing "The method has been proven to be useful in diagnosing tender spots and trigger points and their clinical management, particularly in the assessment of treatment results." Bendtsen et al. (1994) have developed an instrument known as the "Palpometer" which consists of a thin pressure sensitive plastic device which is attached to the palpating finger, with a scale recording the pressure applied to the device. Bendtsen et al. (1994) tested and found the Palpometer to have both inter and intra-examiner reliability.

Many authors differ in their requirements for diagnosis of myofascial pain syndrome but the most comprehensive clinical criteria are described by Simons (1991). According to Simons (1991), the findings should include five major criteria and at least one of three minor criteria. These findings are as follows:

## Major Criteria

Regional pain complaint.

1. Pain complaint or altered sensation in the expected distribution of referred pain from a myofascial trigger point.
2. Taut palpable band in an accessible muscle.
3. Exquisite spot tenderness at one point along the length of the taut band.
4. Some degree of restricted range of motion, when measurable.

## Minor Criteria

1. Reproduction of clinical pain complaint, or altered sensation, by pressure on the tender spot.
2. Elicitation of a local twitch response by transverse snapping palpation at the tender spot, or by needle insertion into the tender spot in the taut band.
3. Pain alleviated by elongation (stretching) of the muscle or by injecting the tender spot.

## 2.8 EXAMINATION

Travell and Simons (1989) state that the examination of the patient begins during the case history with any postural deviations, body asymmetries and disproportions being noted. Furthermore, they emphasize testing range of motion of an area e.g. in the neck as any active or latent trigger points associated with the taut, palpable bands will restrict the movement of that area. With the presence of taut palpable bands, Simons (1991) recommends palpating for any discomfort at the musculo-tendinous junction which seems to develop in response to sustain tension from the taut band.

Sola (1984) advocates the examination of the muscle in a relaxed and a partially stretched position as the painful area might be quite large and one needs to find the spot of

maximum tenderness. Travell and Simons (1989) caution against the use of excessive force during any manual testing as any maximum effort may overload irritable trigger points, severely increasing the pain levels. They stipulate that the muscle must be stretched to the verge of, but short of, discomfort which places the taut band under tension while relaxing the adjacent, uninvolved fibers.

According to Simons (1991) the patient must be supported in a relaxed manner with the muscle on moderate, non-painful stretch. Murphy (1989) describes three methods of examining the muscle fibers. The first method, direct digital palpation, involves walking the fingers across the involved area applying pressure and noting any tender areas. The second method, flat palpation involves rubbing the tissue against an underlying structure, searching for a taut band of muscle or fascia that may contain the trigger points. The third method, known as pincer palpation involves grasping the muscle between the thumb and forefinger and applying digital pressure to the muscle in a rolling action.

Sola (1984) states that pressure applied to the hypersensitive area should reproduce or accentuate the pain with the muscle feeling rope-like, indurated or tight. Murphy (1989) writes that one or more of three responses should occur on encountering a trigger point:

1. There will always be point tenderness in the area being examined and the patient may experience referred pain to a secondary area.
2. There may be a local twitch response or a transient contraction of the muscle that can be felt by the examiner.
3. The patient may wince, cry out or withdraw from the examiner in response to digital pressure on the taut flat muscle surfaces.

## 2.9 CLINICAL CHARACTERISTICS OF MYOFASCIAL TRIGGER POINTS

Travell and Simons (1983 :12) define an active myofascial trigger point as "a hyperirritable locus within a taut band of skeletal muscle, located within the muscular tissue and/or its associated fascia. The spot is painful on compression and can evoke characteristic referred pain and autonomic phenomena."

Authors such as Travell and Simons (1989), Murphy (1989) and Friction (1994), all support a variety of signs and symptoms, but the most comprehensive account comes from Rosen (1993) and is as follows:

1. Local manifestations- taut band, trigger point, local twitch response, localized weakness and restricted range of motion.
2. Remote manifestations- referred sensory (pain and tenderness) and referred motor (sensitization and spread of dysfunction) function.
3. Autonomic manifestations- local vasoconstriction and/or vasodilatation with referred vasoconstriction.
4. Epiphenomenon- nerve, tendon or blood vessel entrapment, reconditioning and psychosocial.

These factors are described in more detail below.

Rosen (1993) and Auleciems (1995) both agree that the trigger point (TP) can exist in a variety of forms. The TP's can be active, latent, secondary or satellite. Latent trigger points result in muscle stiffness, weakness, limited range of motion and dysfunction without persistent pain. Travell and Simons (1983 :4) describe the secondary and satellite TP's as follows:

A secondary trigger point becomes active when the muscle in which it is found is overloaded as a synergist acting for, or an antagonist countering the tautness of the muscle containing the primary trigger point. A satellite TP becomes active when the muscle in which it is found falls within the zone of reference of another trigger point.

Simons (1991) emphasizes the importance of the localized twitch response as an objective sign that is pathognomonic of a TP. Friction (1994) states that this twitch response can be elicited by placing the muscle in passive, moderate stretch and snapping the band containing the TP briskly, with firm pressure from a palpating finger moving across the muscle band at its most tender spot.

Travell and Simons (1989) emphasize that TP's do not cause neurological problems unless there is compression of a nerve by the taut band, causing motor and/or sensory loss. Gerwin (1994) supports this, stating that weakness occurs without atrophy and hypothesizes that this results from reflex inhibition of the anterior horn cell due to painful sensory input.

Travell and Simons (1989) believe that reproduction of the patient's pain on palpation is the most valuable confirmatory evidence of an active TP. The referred pain is described as a localized, subcutaneous ache with slightly blurred edges. TP's in the limbs tend to refer pain distally while certain muscles refer pain to the immediate vicinity (e.g. deltoid, gluteus maximus). The infraspinatus and the tensor fascia lata refer pain into the adjacent joints, giving the impression of an arthritis (Simons 1991). Sola (1984) stated that TP's may refer hypoesthesia or anesthesia instead of pain.

According to Rosen (1993), the tendency of TP's to spread, causing dysfunction in remote tissues can be attributed to overlapping referred motor and sensory pathways, concurrent



deconditioning and incomplete rehabilitation- alone or in combination. Fricton (1994) states that the affected muscles usually display increased fatigability, stiffness, subjective weakness and restricted range of motion that is unrelated to joint restriction.

Simons (1991) lists the following autonomic phenomena associated with TP's:

1. They tend to cause increased temperature changes, a view shared by Fischer (1986) who stated that heat conduction from an inflammatory focus causes a temperature increase over the TP site. Rosen (1993) was uncertain whether the temperature increase resulted from decreased removal of metabolic wastes or from chemical changes occurring at the site of the TP.
2. TP's in parts of the trapezius refer pilomotor activity down the arm, while the sternocleidomastoid muscle refers tearing of the eye and coryza homolaterally.
3. Finally constant pressure on an upper trapezius TP induced a reduction in the pulsations of the temporal artery bilaterally while the pain lasts.

According to Sandmann and Backstrom (1984) and Fricton (1994), patients have reported symptoms such as frustration, anxiety, depression and anger in chronic cases, with maladaptive behaviors such as pain verbalization, poor sleep and dietary habits, lack of exercise, poor posture, bruxism and medical dependencies with prolonged pain.

## 2.10 TREATMENT

Management of myofascial trigger points can be a difficult exercise. Many authors recommend different techniques, but choices appear to be based on personal preference rather than clinical evidence. All the treatments appear to be aimed at disrupting the reverberating neural circuits responsible for the self perpetuation of the pain-spasm-pain cycle. In

addition, attention must be paid to the perpetuating factors as they play a significant role in the success of a treatment.

Sola (1984) lists the following methods as being useful: applications of heat or cold, electrical stimulation, injection with a local anaesthetic or saline, or stimulation with a needle alone. Murphy (1989) favors the use of electrotherapeutic devices such as high voltage stimulation, T.E.N.S, microamperage stimulation and ultrasound while Travell and Simons (1989) advocate the use of: spray and stretch techniques, needle therapy, ischaemic compression and ultrasound. One can certainly appreciate that many options are available and there is uncertainty over which is the most effective.

#### 2.10.1 Stress management

Sandmann and Backstrom (1984) believe that the gains made in the treatment of the physiological body may be maintained by treating the psychological component as well. They advocate the use of progressive relaxation, autohypnosis, visual imagery, breathing control, biofeedback and time management in stress and pain management.

#### 2.10.2 Chiropractic treatment

Gatterman (1990:286, 296-297) states that chiropractic therapy is one of the most effective measures in the treatment of myofascial pain syndrome. Chiropractic care along with stretching, light aerobic exercise, adequate rest and relaxation, and changes in attitude and lifestyle can bring much relief to sufferers of myofascial pain.

#### 2.10.3 Ischaemic compression

The term 'ischaemic compression' was first coined by Travell and Simons (1983 :86) and referred to the initial blanching, followed by reactive hyperemia of the skin over a trigger point after manual pressure. Pressure is applied with

a thumb, finger, knuckle, or elbow depending on the size, depth and thickness of the muscle (Gatterman 1990 :296). The relaxed muscle is stretched to the verge of discomfort and pressure is sustained for up to as much as a minute with the pressure on the trigger point increasing slowly as the pain abates (Travell and Simons 1983:87). According to Sandmann (1981) the initial pressure should be firm rather than painful, with a gradual increase in pressure as excessive discomfort will cause the patient to tense up. Travell and Simons (1983:87) recommend following the treatment with the application of hot packs and active range of motion of the area.

#### 2.10.4 Needling

Needle techniques employ one of the oldest methods of pain relief, using hyperstimulation analgesia to disrupt abnormal neural activity (Melzack 1981). Sola (1984) lists three variations of this technique: injection with a local anaesthetic, a saline solution (wet needling) or stimulation with an acupuncture needle (dry needling). The procedure is the same for all three and is described by Travell and Simons(1983:84) as follows :

The trigger point is located by palpation and the needle is inserted 1-2cm away and directed towards the trigger point such that the needle approaches at an angle of about 30 degrees to the skin. A fanning technique is used, whereby the needle is repeatedly withdrawn out of the trigger point and redirected to penetrate a new part, ensuring maximum coverage of the area.

In wet needling, a local anaesthetic (lidocaine or procaine) is injected into the area (Travell and Simons 1983:75). Sola (1981) and Garvey (1989) have reported the best success with dry needling while Murphy (1989) and Rosen (1994) support wet needling. Broome (1996 :92) found both dry needling and wet needling (saline injection) equally effective, but preferred dry needling saying it caused less

patient discomfort. In a clinical trial injection of sterile water was found to be substantially more painful than the use of saline injections and demonstrated no better clinical outcome (Wreje and Brorsson 1995). According to Sola (1981), a portion of the benefit results from needle stimulation regardless of any substance injected. As can be appreciated, there are large differences in opinion regarding effective treatment protocols.

#### 2.10.5 Stretch and Spray

According to Travell and Simons (1983 :63-65), the spray and stretch technique inactivates myofascial trigger points more quickly and with less patient discomfort than local injection and ischaemic compression. This technique is especially useful when many muscles in an area are involved and the trigger points are interacting strongly.

There are two commercially available vapocoolant sprays.

1. Ethyl chloride which is too cold and is a rapidly acting anaesthetic which is potentially flammable and explosive.
2. Fluori-methane is a safer and more popular option and consists of 85% trichloromonofluoromethane and 15% dichlorodifluoromethane. This solution compound is nonflammable, chemically stable, non-toxic, non-explosive and does not irritate the skin. Travell and Simons (1992:9) felt that the effects of the vapocoolant spray could be as effectively obtained by stroking the affected area with plastic covered ice. The plastic prevents the skin from becoming damp which reduces the rate of change of the skin temperature produced by the ice (Travell and Simons 1983:69).

The procedure followed for the spray and stretch technique is as follows:

The patient is positioned comfortably with the region bare. The range of motion of the area is assessed for any restriction and as a comparison for post-treatment improvement. One end of the muscle is anchored so pressure can be applied at the other end to passively stretch the muscle. If spraying, the vapocoolant is directed at an angle of 30 degrees and is swept over the skin parallel to muscle fibers. The first sweep of the spray or ice is applied, at 10cm/sec, before any stretch is applied. The ice is applied in one direction only, first over the entire length of the muscle, in the direction of the referred pain, and then over the referred pain pattern. The spray and stretch steps can be repeated until full muscle length is obtained but any part of the muscle should be covered only three times before rewarming the muscle. Too much ice or spray may cool the underlying muscle and irritate the trigger points. (Travell and Simons 1983:65).

It is important to realize that in this technique, **stretch is the action and spray is the distraction**, as persistent gentle stretching is more likely to inactivate trigger points than spray without stretch. To fully inactivate TP's the muscle must be extended to its full length, but stretching by itself causes pain and reflex muscle spasm. The vapocoolant acts as a distracting agent on the central pain receptor areas bombarding with such a barrage of cold impulses that the pain impulses are obliterated. The flood of new stimulus acts as if the sensory input from the skin turns off the central nervous system feedback mechanism, breaking the reflex arc that sustains the TP activity and relaxing the muscles. The cold enables the muscle to be stretched to its full length without initiating the reflex muscle spasm (Travell and Simons 1983:64, 72, 73 ). One can appreciate that cold is a vital factor in this combination therapy but according to Hong et al. (1993) no references were found in the literature

documenting the effectiveness of moist heat and passive stretching.

#### 2.10.6 Stretching

Liebensen (1988) believes that psychological stress, nutritional inadequacies and metabolic problems are main causes of increased muscle tension which then causes neural facilitation, abnormal movement patterns and joint dysfunction. The primary goal of successful rehabilitation of the myofascial problems is not pain relief, but rather restoration of a normal range of motion to the tissue. Failure to achieve these goals results in residual dysfunction in the tissue setting the stage for recurrent disability and pain. (Rosen 1993) The muscle fibers affected by the trigger points are in spasm. This spasm must be resolved by stretching for the muscles to return to their normal length. Sandmann (1981) and Travell and Simons (1983 :35) both believe that stretching the muscle elongates the myosin heads from the reactive portion of the actin filaments eliminating the contraction and stopping the runaway metabolism.

Although Travell and Simons (1983:10) advocate the use of passive stretching in the spray and stretch technique, an effective alternative is available. Lewitt and Simons (1984) describe a technique known as Post-isometric relaxation. Here the muscle is placed in a stretched position short of pain and resistance. The examiner provides an isometric resistance against the patient's hypertonic muscle contraction for 5-10 seconds. After the contraction the patient is instructed to breathe out and relax during which any slack in the muscle is taken up. Without releasing the new position, the first two steps are repeated until full range of motion is acquired. In a clinical trial by Lewit and Simons (1984), using this technique, 94% of 244 patients experienced immediate relief while 63% experienced lasting relief from myofascial pain. Travell and Simons (1992:9)

support this technique describing it as "particularly effective".

One can appreciate the importance of stretching in the treatment of trigger points. Both Rubin (1981) and Murphy (1989) emphasise the importance of a home stretching program to maintain the improvement. Lewitt and Simons (1984) stated that patients put on a home therapy stretching program were more likely to gain lasting relief.

#### 2.10.7 Cryotherapy

According to Gattermann (1990:334) despite the long history of the therapeutic use of ice, the physiological effects are still obscure and despite a lack of research, Cryotherapy can have tremendous clinical value. According to Bierman (1955), Cryotherapy has been used with some success in the treatment of cancer, psychological disorders, shock, hyperpyrexia, cardiac surgery, acute epididymitis, hemorrhage and infections while Ellis (1961) used Cryotherapy for treating lumbago, acute wryneck, fibrositis, renal colic, dysmenorrhoea and a fractured rib. However, most importantly, Mennell (1975) stated that the use of cold can produce dramatic relief of pain arising from muscle spasm from irritated trigger points and of the pain of the myofascial component of visceral disease. Mennell (1975) emphasizes that the use of cold in the treatment of myofascial trigger points is to promote the restoration of the normal resting length of the muscle by substitution of sensory input and not by cooling of the muscle itself.

On application of cold to an area the initial reaction is one of vasoconstriction in an attempt to preserve heat. At 15° C and lower, 'the Lewis hunting reaction is noted' whereby alternating periods of vasodilatation are interspersed with periods of vasoconstriction while the body attempts to find and maintain a certain temperature. This prevents hypothermia, frostbite and tissue damage by shunting warm blood to the superficial areas (Gatterman 1990:334).

Forster and Palastanga (1985:200-202) have described the effects of cold as follows:

1. Cooling reduces the rate of nerve conduction. (This was confirmed in two separate trials on the effects of temperature on the discharges of muscle spindles by Ottoson (1965) and Mense (1978).)
2. Cooling reduces pain. The cold stimulation can be considered noxious and may result in the release of B-endorphins or enkephalin into the posterior horn resulting in temporary relief of pain. Mennell (1975) hypothesizes that the sensory receptors transmit more rapidly in the large nerve fibers than the muscle spindles and arrive at the dorsal horn before the pain impulses. During the refractory period in which the pain impulses are blocked the muscle spasm is relieved and the muscle can relax.
3. Reduction of spasm. The skin stimulus produced by the cold must have an affect on the general level of excitation or inhibition in the region of the anterior horn cells of the cord. Mennell (1975) states that the counterirritant application of cold plus muscle stretch reverses the pain mechanism of muscle spasm.
4. Reduction of oedema. During the alternating periods of vasoconstriction and vasodilatation, capillary blood flow is affected. Therefore an effect is produced which reduces swelling i.e. excess fluid is returned to the systemic circulation and more nutrients and repair substances are brought into the area. Furthermore the increased circulation could carry away chemical substances which are stimulating nociceptors and producing pain.
5. Reduction of spasticity. The mechanism here is supposedly the same as for muscle spasm (as described above).

According to Olson and Stravino (1972), the application of cold is very beneficial in acute trauma as vasoconstriction,



decreased tissue metabolism and blocked release of histamine (which is responsible for vasodilatation and exudate formation) are the immediate effects.

One can appreciate the value of cold in the treatment of myofascial pain syndrome but it is interesting to note that Gatterman (1990:334) after 18 years, still agrees with Olson and Stravino (1972) that there is a strong need for research in the field of Cryotherapy.

#### 2.10.8 Heat

According to Gatterman (1990:331), heat is one of the oldest and most widely used forms of physical modalities, with natural forms of heat (sun, sand and thermal waters) being used for as long as history can be traced. Shriber (1975:11) divides sources of heat into three categories i.e. (1) Chemical action, (2) Electrical currents, and (3) Mechanical work.

Examples of chemical action include the burning or oxidation of wood, gas or oil. All conductors of electricity generate heat by providing a resistance to the current flowing through them. Similarly, high frequency currents generate heat from resistance by the tissues through which they flow. Finally mechanical energy, in the form of friction, compression or percussion converts kinetic energy into heat energy.

Shriber (1975:12) states that the derivation of heat can be divided into three sources:

- 1) Conductive e.g. hot water bottles, paraffin baths, whirlpool baths and hot water baths.
- 2) Convective e.g. poultices, fomentations and electric blankets.
- 3) Radiation sources e.g. heat lamps, infrared units, diathermy and ultrasound units.

Forster and Palastanga (1985:112) believe that the physiological effects of heat include increased metabolic activity, increased blood flow, increased collagen extensibility and stimulation of nerve endings, producing a sedative effect. Both Forster and Palastanga (1985:113) and Gatterman (1990:331) agree that the effect of heat on nerve conduction needs to be investigated.

According to Gatterman (1990:331) the effects of heat are as follows:

1. Analgesia- this is probably due to stimulation of nerve endings, causing a counterirritant effect and a decrease in the transmission of pain impulses.
2. Reduction of muscle spasm- this is probably due to stimulation of the secondary nerve endings which decreases muscle tone. In addition the vasodilatation causes a removal of waste products from the muscle spasm area.
3. Oedema reduction- vasodilatation increases capillary flow and the return of blood and lymph to the general circulation. However in acute cases the heat may increase the trauma to the area aggravating the oedema.
4. Collagen relaxes- muscle, tendon and capsular ligaments increase their ability to stretch.
5. Chronic inflammation decreases due to increased nutrient level and white blood cells being carried to the area.

It is interesting to note that both Shriber (1975:14) and Forster and Palastanga (1985:113) advocate heat for relaxation of muscle spasm and of pain, factors which are essential in the treatment of trigger points, yet no research has yet been conducted on moist heat and passive stretching.

Travell and Simons (1983:71) state that moist heat is more effective than dry heat possibly due to a more effective absorption of the heat. Travell and Simons (1983:90) believe that the moist heat relaxes the underlying muscles thereby

diminishing the tension on the trigger points and reducing referred pain. Furthermore, Travell and Simons (1983:90) recommend combining moist heat and stretch exercises but do not state whether heat or cold in combination with stretching is more effective.

Olson and Stravino (1972) state that controversy has long existed over the relative therapeutic value of heat versus cold and recommend strongly a careful comparison of the effects of heat and cold in the treatment of patients with various neurologic and musculoskeletal conditions. According to Benson and Copp (1974), heat and cold were both significantly effective in raising the pain threshold of normal shoulders with ice being more effective than heat. This effect was temporary and after 30 minutes there was no effect on the pain threshold. In a study by Landen (1967) ice massage and hot packs were found to be equally effective in the symptomatic relief of lower back pain. In the group treated by ice, 64% of 59 patients reported some degree of improvement following initial treatment and 88% reported decreased pain at the time of discharge. In the group treated with heat, 64% of 58 patients reported improvement following the initial treatment while 85% reported decreased pain at time of discharge.

It would appear that there is much uncertainty over the relative effectiveness of heat and cold. Thus far, moist heat appears to have been used primarily as a post-treatment adjunct to soothe any residual pain following needling (Mennell 1975). Heat has been recommended in combination with stretching by Travell and Simons (1983:1990) but stretching and spray appears to be the treatment of choice. Research is vital to fill this gap in the literature to ascertain the relative effectiveness of cold and moist heat in the treatment of myofascial pain syndromes.

## 2.11 MUSCLE OVERVIEW

### 2.11.1 THE TRAPEZIUS (THE COAT HANGER)

According to Travell and Simons (1989) and Gerwin (1994), the trapezius is the most common muscle affected by trigger points. The trapezius consists of an upper, middle and lower portion. There are six trigger points found within the trapezius referring pain and a seventh trigger point referring autonomic phenomena. This muscle extends from the occiput to T12 and extends laterally to the clavicle in front, the acromion and to the spine of the scapula. The upper trapezius elevates the shoulders and rotates the glenoid fossa so the shoulder joint faces upwards. The lower portion assists in this endeavor and the middle portion strongly retracts the scapulae.

Trigger point 1: refers pain unilaterally upward along the posterolateral aspect of the neck to the mastoid process. If the trigger point is very active, pain may refer to the side of the head, centering in the temple and back of the orbit and occasionally to the angle of the jaw. This trigger point is sometimes associated with symptoms of dizziness and vertigo. The patient will usually complain of severe posterolateral neck pain, often with an ipsilateral temporal head pain. The patient is examined while lying supine, with the free border of the trapezius lifted off the underlying supraspinatus muscle in a pincer style grip. The trigger point is located within the anterior aspect of the muscle tissue, lateral to the angle of the neck. The muscle is rolled between the thumb and index finger, assessing for tight bands. The needle is directed upwards and remains within the muscle mass between the fingers. This eliminates the possibility of puncturing the lungs.

Trigger point 2: refers pain posteriorly to the referral pattern of TP1, up to the base of the occiput. The trigger point is located posterior and caudal to the free border of the upper trapezius. For patients with firm tissues, flat palpation is the most effective. Otherwise pincer palpation

of the deeper tissues posterior to TP1 can be used. To needle the trigger point, the patient lies on the opposite side, with the needle directed upwards away from the lungs.

Trigger point 3: refers pain severely to the high cervical region of the paraspinal muscles, to the adjacent mastoid area and to the acromion. This trigger point lies in the lateral margin of the lowest fibers where the muscle crosses the medial border of the scapula. This trigger point is identified by flat palpation of the tissues. To needle this trigger point, the patient lies on the uninvolved side with the tissues on mild stretch. This is achieved by swinging the arm forward and up. Penetration of the intercostal space is avoided by aiming the needle towards an underlying rib.

Trigger point 4: refers a steady burning pain downward along, and medial to the vertebral border of the scapula. TP4 is found high in the trapezius over the medial end of the infraspinatus and is identified by flat palpation just below the spine of the scapula. When needling, the patient is positioned as in TP3 above, with the needle aligned to the lateral fibers of the muscle and directed towards the ipsilateral shoulder.

Trigger point 5: refers superficial, burning pain medially between the spinous process of C7 and TP5. The trigger point is located by flat palpation of the superficial, horizontal fibers of the middle fibers, about 1cm medial to the scapular attachment of the levator scapula. To needle the trigger point, the patient lies on the uninvolved side with the arm placed between the legs to stabilize the scapula. The trigger point is quite superficial, so the trigger point is directed at a tangent to the skin.

Trigger point 6: refers an aching pain to the top of the shoulder, or to the acromion. The trigger point is found by flat palpation of the lateral fibers of the middle trapezius over the lateral end of the supraspinatus muscle near the

acromion. The patient is positioned as for TP5 when examining and when needling.

Trigger point 7: This infrequent trigger point refers 'gooseflesh' to the lateral aspect of the homolateral arm and sometimes to the thigh. The TP7 lies in the most superficial fibers of the middle trapezius as they cross the levator scapulae muscle. This trigger point refers the 'gooseflesh' when stimulated by flat palpation of the fibers and when needled. The patient is positioned as in TP6 above. (Travell and Simons 1983:183-201, Rubin 1981 and Gatterman 1990: 307-309.)

#### 2.11.2 LEVATOR SCAPULAE (STIFF NECK)

Travell and Simons (1989) believe that the levator scapulae is the most common cause of stiff, restricted neck movement while Sola (1955) found that the levator scapulae was second only to the trapezius in frequency of latent trigger points, with 20% of the sample testing positive.

According to Travell and Simons (1983:334-342), Travell and Simons (1989) and Gattermann (1990:306-307), the fibers of the levator scapulae attach above to the transverse process of the first four cervical vertebrae and below to the vertebral border of the scapula, between the root of its spine and the superior angle. The levator scapulae helps with rotation of the neck, assists with shrugging of the shoulders and when the scapula is anchored, rotates the glenoid fossa downwards. This assists with adduction of the upper limb.

The trigger points are activated by sustained contraction of the muscle in an elevated position. This is compounded by use of a fatigued muscle and exposure to cold. The muscle is susceptible to trigger point activation from postural stress, activity stress and to infections. Patient examination will reveal severe restriction of neck rotation.

There are two trigger points found in this muscle which refer pain to the angle of the neck, with a spillover zone running along the vertebral border of the scapula and to the posterior shoulder. The caudal trigger point may refer pain to the inferior angle of the scapula. The primary trigger point is located deep within the angle of the neck and a secondary trigger point is found 1.3cm superior to the angle of the scapula.

To locate the primary trigger point, the patient sits in a chair with the arms supported by armrests to relax the levator scapula and trapezius. The examiners fingers push the trapezius backwards to uncover and straddle the levator scapula. The patient rotates the head to the opposite side to bring the tissue up against the palpating finger. The position is the same for the secondary trigger point with flat palpation applied 1.3cm above the superior scapula angle.

When needling both the trigger points, the patient lies on the opposite side with the head supported by a pillow. For the upper trigger point, the needle is directed superiorly towards the transverse processes of the cervical vertebrae. For the lower trigger point the patient adopts a 'round shouldered' position to put the muscle tissue on partial stretch. The needle is then directed superiorly and tangential towards the angle of the neck. (Travell and Simons 1983 :334-342, Travell and Simons 1989 and Gattermann 1990:306-307.)

### 2.11.3 INFRASPINATUS (SHOULDER JOINT PAIN)

According to Travell and Simons (1983:377-386) and Gattermann (1990:315-317) the infraspinatus attaches medially to the infraspinatus fossa and laterally to the greater tuberosity of the humerus. This muscle is used in external rotation of the arm at the shoulder and for stabilizing the head of the humerus within the glenoid fossa during movement of the arm.

There are three trigger points found within this muscle. The patient sits in a chair and the hand and arm are brought across the front of the chest. The most common trigger point is found by flat palpation, just below the scapula spine, adjacent to the vertebral border. The 2nd trigger point is also found by flat palpation at the same level, at the midpoint of the scapula spine. A third and infrequent trigger point is found more caudally, next to the midpoint of the vertebral border of the scapula.

The first two trigger points refer pain deep within the shoulder joint, down the anterolateral aspect of the arm, to the lateral forearm, to the radial aspect of the hand and occasionally to the fingers. The 3rd trigger point refers pain to the adjacent interscapular muscles.

The trigger points are activated by repetitive movements like reaching back and up. The patient will demonstrate restricted internal and external rotation of the arm at the shoulder. To needle the trigger points, the patient lies on the opposite side, the arm adducted to 90° and the elbow resting on a pillow. The trigger points are trapped between two fingers and the needle is directed in towards the scapula fossa. Certain portions of the scapula are very thin and one must be sensitive to the resistance offered by the bone at that depth to prevent the possibility of causing a pneumothorax (Travell and Simons 1983:377-386 and Gattermann 1990:315-317).

## 2.12 SUMMARY

According to Travell and Simons (1983:63), stretch and spray is the 'workhorse' of myofascial therapy inactivating trigger points more quickly and with less patient discomfort than any other technique. There is overwhelming evidence to support its effectiveness. There does not appear to be a study comparing the relative effectiveness of heat and passive stretching versus cold and passive stretching. Furthermore there appears to be confusion over the benefits



of heat versus cold, despite the fact that both therapies are age-old.

The aim of this dissertation will be to compare the combination of cold and passive stretching versus moist heat and passive stretching in the treatment of myofascial trigger points, in an attempt to find out which is the more effective treatment, not only as a primary therapy, but also as an adjunctive therapy for this common and distressing condition.

# *CHAPTER THREE*

## *MATERIALS AND METHODS*

## CHAPTER 3: MATERIALS AND METHODS

### 3.1 INTRODUCTION

This study was designed as a comparative, randomized clinical trial. The objective was to compare two treatment groups (the combination of Cryotherapy and passive stretching versus the combination of moist heat and passive stretching) to assess for intra-group improvement. On conclusion of the treatment protocols, an inter-group statistical analysis was performed to determine whether one treatment protocol was more effective than the other. The more effective treatment group could then be used as either the primary treatment for myofasciitis or it could be used as an adjunct to other myofascial treatment options.

### 3.2 THE SUBJECTS

Patients were obtained by means of consecutive sampling, using advertisements posted around Technikon Campus and in local newspapers. No restrictions were placed on a patient's sex, racial group, income bracket or area of residence.

Any patient presenting to the clinic with neck pain, upper back pain or shoulder pain was considered a potential candidate for the study. These patients were then briefly screened and further investigations took place only if the researcher deemed the patient suitable for the study. The screening procedure involved questioning the patient on the pattern of pain referral, palpation of the relevant zones for muscle spasm, twitch response, patient jump sign and/or referred pain.

### 3.3 INCLUSION AND EXCLUSION CRITERIA OF PATIENTS

- 1) Patients had to be between the ages of eighteen and fifty-five.
- 2) Only patients diagnosed by the researcher as having active trigger points of the shoulder girdle muscles (trapezius, levator scapulae and infraspinatus muscles) were considered.
- 3) Any patient suffering from a local or systemic pathology would not be eligible for the study.
- 4) Patients were not allowed to take any analgesics, nor receive any other manual therapy for the duration of their participation. Manual therapy included chiropractic adjustments, any electrotherapies, any other myofascial treatments or soft tissue therapies.
- 5) The patient's condition had to comply with all eight criteria for the diagnosis of active trigger points of the trapezius, levator scapulae and infraspinatus as described by Travell and Simons (1990 :18-19):
  - Either a history of rapid onset during, or shortly following acute overload stress, or a history of gradual onset with chronic overload of the affected muscle.
  - A pattern of pain referred from the trigger point that is characteristic for that muscle in which it is located.
  - Weakness of the affected muscle with associated restriction in its stretch range of motion.
  - A taut palpable band in the affected muscle.
  - Intense focal tenderness of the taut band to applied pressure.
  - A local twitch response produced by needling or snapping palpation of the trigger point.
  - Reproduction of the characteristic pain patterns by needling or palpating the trigger point.
  - Elimination of the clinical presentation by specific trigger point therapy.

- 6) Any patient for whom Cryotherapy was contraindicated, as described by Gatterman (1990 :336), was excluded from the study:
- Weakened individuals such as geriatrics, infants, cachexics;
  - Individuals with psychological aversion to cold;
  - Hypersensitive individuals, secondary to:
    - a) histamine release.
    - b) cold haemolysins and agglutinins.
    - c) cryoglobulins.(Symptoms of hypersensitivity may include urticaria, erythema, itching, Raynaud's Phenomenon, ulceration, chills, gastrointestinal disturbances.)
  - Circulatory disturbances (e.g. Raynaud's disease, thrombangitis obliterans, peripheral vascular disease, high blood pressure, atherosclerosis, severe varicose veins, myocardial weakness.);
  - Some rheumatoid conditions, in which cooling may increase joint stiffness;
  - Hypothermic individuals.
- 7) Likewise the following list, as described by Gatterman (1990 :332), was used to exclude any patient for whom moist heat therapy was contraindicated:
- Insensitivity to heat (e.g. diabetics, postsurgical areas, drug induced insensitivity, neurological deficit);
  - Heat sensitivity (e.g. multiple sclerosis);
  - Severe circulatory disturbances (e.g. peripheral vascular disease, arteriosclerosis, cardiac patients, varicose veins, aneurysm, hypertension, diabetes mellitus);
  - Tendency to hemorrhage (e.g. hemophiliac);
  - Malignancy;
  - Advanced weakness;
  - Over a gravid uterus;
  - Active tuberculosis;
  - An encapsulated swelling in which rupture could cause damage;
  - An unreliable patient;
  - Severe edema.

Patients had to comply with all of the inclusion criteria in order to be accepted into the research program.

### 3.4 THE SAMPLE GROUP

A sample of thirty patients was randomly divided into two groups of fifteen according to the process of randomization as described by Scott-Dawkins (1995). Fifteen labels were inscribed with the letter 'I' representing the treatment group in which Cryotherapy and passive stretching was used. Likewise, fifteen labels were inscribed with the letter 'W' representing the treatment group in which moist heat and passive stretching was used. The thirty slips of paper were then folded such that the letters were obscured and were then put into a hat. After the hat had been sufficiently agitated, the labels were drawn one at a time. The sequence of letters drawn was then recorded next to a list numbering one to thirty (appendix A). In such a fashion the point in the list at which a patient joined the study would allocate the specific treatment for that patient. There was no patient blinding involved in this study as each patient was informed of the two treatments and to which group they had been allocated.

The treatment schedule used was similar in execution to that used by Jones (1995) and Broome (1996). Patients who passed the initial screening test and inclusion criteria underwent a detailed case history (appendix B), physical examination (appendix C) and cervical spine regional examination (appendix D). If, after this consultation the patient was still deemed acceptable, a series of five treatments within a three week period were booked. A follow-up appointment was then scheduled for one month after the fifth treatment.

### 3.5 INTERVENTIONS

Group A received ice and passive stretching as their treatment while group B received moist heat and passive stretching. The patients in group A were required to sit in a

comfortable position to permit voluntary relaxation. It was essential for the shoulder girdle region to be exposed during the treatment. Polystyrene cups in which water had been frozen were cut down to expose the ice. As recommended by Travell and Simons (1992 :9) the cup was then covered in plastic, which kept the patient dry and maintained a high rate of change in skin temperature, produced while stroking with the ice. Trigger points in the different muscles were dealt with separately as the stretching technique for each muscle was different. The treatment was then performed according to the following sequence :

- The edge of the ice was stroked in one direction, in parallel sweeps, two to three times, across the muscle without any stretch.
- The sweeps of ice then covered the pain referral zone specific to that muscle in the shoulder girdle in which secondary trigger points are commonly found.
- A gentle passive stretch was then applied to the muscle while the ice was being used.
- The stretch was taken to and maintained at the point of resistance, well within the patient tolerance level.
- The action of the ice covered the muscle at a rate of 10cm/sec.
- The sweeps were repeated in a rhythm of a few seconds on and a few seconds off until all the skin of the muscle and pain referral zone had been covered. The ten minute duration of the treatment was consistent with that advocated by Gatterman (1990 : 336)
- To complete the treatment the patient was required to actively flex/extend, laterally flex and rotate the neck and/or actively move the shoulder through horizontal adduction/abduction and external/internal rotation as recommended by Travell and Simons (1983 :65).

Group B were required to lie prone with the shoulder girdle region exposed. Water which had been boiled in a kettle was poured into a 20 liter bucket. Several towels were dipped into the hot water and the excess water drained off. The

towels were then folded in half and rolled up tightly in an attempt to maintain the heat. A hot towel was placed over the area of interest. As the towel cooled a new section was unrolled until a new towel was required. In this fashion a constant high level of heat was delivered to the muscle tissue. The duration of the treatment was approximately 20 minutes as advocated by Gatterman (1990: 332) and Hong et al. (1995). In the last five minutes of the treatment the patient was asked to sit up so the passive stretching could be done. The positions for the passive stretches were identical to those used in group A.

### 3.6 MEASUREMENTS

#### 3.6.1 Subjective Measures

At the initial consultation as previously stated, the case history, physical examination and cervical spine regional examination were completed. The active trigger points in each muscle were recorded on the Algometer form (appendix F), making their relocation easier and more accurate for subsequent treatments. This form served as a record to indicate which side and which trigger points in the trapezius, levator scapulae and infraspinatus muscles were active for the duration of the treatment program.

At the first consultation each patient was required to fill out a patient consent form (appendix E) granting the researcher permission to use them in the study. In addition this ensured that each patient was given a full description of the study and their role therein. The patient was obliged to fill out the short form McGill Pain Questionnaire (appendix G), the Numerical Pain Rating scale-101 (appendix H) and the CMCC Neck Disability Index (appendix I). These three forms subjectively assessed various aspects of the patient's pain.



At consultations two, three and four, no objective or subjective measurements were done. Only the allocated research treatment was performed on the patient.

At treatment five and at the one month follow-up, the objective and subjective measurements were repeated so that any improvement during the treatment and the duration of that improvement could be assessed.

The short form of the McGill Pain Questionnaire used in this study was described by Melzack (1987) as a means of subjectively providing information regarding the sensory, affective and evaluative dimensions of the patient's pain. The short-form McGill-pain questionnaire (SFMPQ) was developed to be used where detailed information regarding pain is required quickly. This questionnaire is divided into two sections. The first section consists of eleven adjectives describing the sensory dimensions of pain. The second section consists of four adjectives representing the affective dimension of pain and was not used in this study. That aspect of pain was covered in the completion of the Neck disability index form. A score of 0 to 3 was given for each adjective depending on whether the pain was ranked as 'none', 'mild', 'moderate' or 'severe' respectively. The option 'none' carried a nil score while 'severe' carried a score of three.

The Numerical Pain Rating Scale-101 (NPRS-101) was chosen because of the ease with which it can be administered and scored. Jensen et al. (1986) established its validity and reliability when providing subjective information about the levels of pain perceived by the patient. It was used to monitor the patient's progress with a decrease in pain intensity indicating improvement. The patient was asked to mark off a point on a 10 cm line, between 0 and 100, when the pain was at its worst. Likewise this was repeated on a second identical line when the pain was at its least with '0' indicating no pain and '100' indicating the most severe pain. The scores were calculated by measuring the distance from

'0' to the patient's mark. The two values from the 'worst pain' and the 'least pain' were added together, divided by two and finally expressed as a percentage of 100.

The CMCC Neck Disability Index (CMCC-NDI) was developed by Vernon and Mior (1991) and was chosen for this study for the subjective information it provided regarding the extent to which a patient's lifestyle was affected by the pain experienced. This index was adapted from the Oswestry low back pain Index (Fairbanks *et al.* 1980) and in a study of the NDI's reliability and validity by Vernon and Mior (1991) was found to demonstrate a high degree of test-retest reliability and internal consistency. The CMCC-NDI was developed because until 1991 no index had been available which dealt specifically with the cervical region. The CMCC-NDI consists of ten sections dealing with different aspects of a patients lifestyle. Each section had six options, with the first scoring '0' and the next five increasing progressively by a value of one, to a maximum of '5'. All the scores were added together and expressed as a percentage of the maximum score (50).

### 3.6.2 Objective Measurements

An objective assessment of changes in the patient's condition during the treatment and after one month was required for this study. To this end two instruments, the algometer and the goniometer, were used.

Algometer readings were carried out on the active trigger points recorded during the initial consultation while the range of motion of the neck was measured using the goniometer. These two instruments gave an objective assessment of the patient. Immediately after the allocated treatment had been carried out the algometer and goniometer readings were repeated so the immediate reaction to treatment could be objectively assessed. This occurred after the first treatment only.

The algometer and the goniometer were again used after the fifth treatment and at the one month follow-up appointment.

According to Fischer (1987) the reliability of the algometer as a tool for the diagnosis of tender spots and trigger points, as well as assessment of treatment results has been documented. Fischer (1987) states that changes in the patient's pressure threshold under standard clinical conditions can be regarded as reliable data. One can view the algometer as an instrument measuring pain sensitivity caused by pressure, therefore the more active a trigger point is, the more sensitive it should be. In the case of active myofascial trigger points, an increase in pressure tolerated after a treatment would indicate some improvement.

The algometer model used in this study was the FDK20 force-dial made by Wagner Instruments (P.O. BOX 1217, Greenwich, CT, 06836, U.S.A. Tel.: 203 869 9861) and supplied by Activator Methods Inc.

The algometer was used as follows:

- The dial on the gauge was set at zero.
- The 1cm rubber disc was placed on the point of maximum tenderness already located and documented at the first consultation.
- The patient was told to express the point at which pain is first perceived.
- The pressure was gradually increased at a rate of 1kg/sec as recommended by Fischer (1986).
- The pressure ceased as soon as the patient indicated discomfort either verbally or by withdrawing.
- The reading on the dial was then recorded on the Algometer sheet (appendix F).
- This procedure was then repeated for all the trigger points found within the specified muscles.

According to Rosen (1993) tissue function associated with myofascial pain can manifest as tissue tightness, tissue weakness, restricted range of motion of muscles and joints.

Disorders of the cervical-spine often alter the normal range of motion of the neck and response to therapeutic intervention can be documented clinically by measuring changes in neck movement (Youdas et al. 1991). The Cervical Range of Motion instrument (CROM) was found to demonstrate high inter-examiner and intra-examiner reliability in a study by Youdas et al. (1991). For this reason, the CROM was chosen to objectively assess the cervical range of motion.

The CROM model used in this study was the Performance Attainment Associates Model (appendix J) The procedure for the use of the CROM was as follows:

- The patient was made to sit in a chair with their thoracic spine maintaining contact with the back-rest of the chair and the lumbar-spine filling the gap between the seat and the back-rest. The feet were positioned flat on the floor and the arms rested freely at the sides.
- The plastic frame was mounted over the subject's nose bridge and ears and secured in place with Velcro straps.
- The three orthogonally arranged dials attached to the frame were checked to insure they were set at zero.
- The flexion, extension and lateral flexion movements were assessed by gravity dependent goniometers while rotation movements were assessed with a compass goniometer in conjunction with a magnetic yoke.
- To measure flexion the patient was told to make a 'double chin' and then flex further until full movement was attained.
- To measure extension the patient was instructed to 'nod the head back as far as the head will go'.
- To measure lateral flexion the patient was instructed to tip the left and right ears towards the left and right shoulders respectively. The patient was told not to turn their head and a hand was placed on the other shoulder to prevent any elevation.

- For rotation, the magnetic yoke was placed around the patient's neck and the appropriate dial set to zero. The patient was instructed to rotate the head as far to the left and right as possible without moving the shoulders.
- All measurements were recorded on the goniometer sheet (Appendix K).

### 3.7 STATISTICAL PROCEDURES

The sample size was small (30) therefore non-parametric tests were used to do the analysis. Parametric tests such as the two-sample unpaired t-test could not be used as the sample size per group was small.

#### 3.7.1 PROCEDURE 1: Wilcoxon sign rank test

The Wilcoxon Sign Ranked Test was used to find out whether there was any statistically significant change **within** group 1 and **within** group 2 before and after treatment 1, between treatment 1 and treatment 5, between treatment 5 and the follow-up appointment and finally between treatment 1 and the follow up appointment i.e.

Tx1(before)	vs. Tx1(after)
Tx1	vs. Tx5
Tx5	vs. FU
Tx1	vs. FU

#### Hypothesis testing and the decision rule:

The null hypothesis ( $H^0$ ) stated that there was no significant improvement during treatment 1, between treatments 1 and 5, between treatment 5 and the follow-up appointment and finally between treatment 1 and the follow-up appointment. The alternative hypothesis ( $H_1$ ) stated that there would be a significant difference between the treatment intervals stated above.

$H^0$ : there was no significant difference

$H_1$ : there was a significant difference

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

For a two-tailed test,

Reject  $H^0$  if  $P \leq \alpha/2 = 0.025$

Accept  $H^0$  if  $P > \alpha/2 = 0.025$

P was the observed significance level

### 3.7.2. PROCEDURE 2: Mann-Whitney Unpaired tests

This test was used to make comparisons between the two experimental groups. The two groups were treated as being independent of one another. The purpose was to find out whether there was a significant difference between the two groups at the  $\alpha/2 = 0.025$  level of significance with respect to the goniometric readings (forward flexion, extension, right and left rotation, right and left lateral flexion), Numerical Pain Rating Scale-101 (the average of the worst and least pain), the Short Form McGill pain Questionnaire, the CMCC-Neck Disability Index and finally the algometer readings. 37 Mann-Whitney Unpaired tests were used to compare groups one and two.

#### Hypothesis testing and the decision rule:

The null hypothesis ( $H^0$ ) stated that there was no significant difference between the two groups with respect to the variable of interest. The alternative hypothesis ( $H_1$ ) stated that there was a significant difference between the two groups.

$H^0: \mu_1 = \mu_2$

$H_1: \mu_1$  and  $\mu_2$  were significantly different from each other.

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

For a two-tailed test,

Reject  $H^0$  if  $P \leq \alpha/2 = 0.025$

Accept  $H^0$  if  $P > \alpha/2 = 0.025$

Note: P was the observed significance level

### 3.7.3. PROCEDURE 3: Summary statistics

Summary statistics including the mean, standard deviation and standard error were obtained to support the results from the Wilcoxon's signed-rank test and the Mann-Whitney U test.

If the two statistical tests calculated any significant difference between the 2 groups, then the mean was used to identify the superior group. The reliability of the mean was then measured using the standard deviation which measures the spread of the data around the mean. The bigger the value, the bigger the spread of the values and hence the less reliable the data. The standard error was used to measure the reliability of the mean used in the statistical tests.

As the Mann-Whitney U test and the Wilcoxon Signed-rank test used the median within the calculations, the mean was used to complement the results, increasing the reliability of the statistical analysis.

### 3.7.4. PROCEDURE 4: Diagrammatic representation of the data

Barcharts and tables will be constructed to present the major findings of the study as a visual summary. These barcharts and tables were able to give a summary of the results obtained from the Mann-Whitney and Wilcoxon signed ranked tests. The Barcharts will be made using the software package Microsoft EXCEL 97 SR-1 supplied by MICROSOFT CORPORATION. The tables will be constructed using Microsoft Word 97 SR-1 Version 6.0 also supplied by MICROSOFT CORPORATION. Furthermore the demographic data obtained from the patient's files will be displayed using pie-charts and tables, again using Microsoft EXCEL 97 SR-1.

The statistical package STATGRAPHICS PLUS VERSION 6+, supplied by MANUGISTICS INC. (2115 East Jefferson Street, Rockville, Maryland, 20852, USA) will be used for data entry and analysis.



# *CHAPTER* *FOUR*

## *RESULTS*

## CHAPTER 4: RESULTS

### 4.1 INTRODUCTION

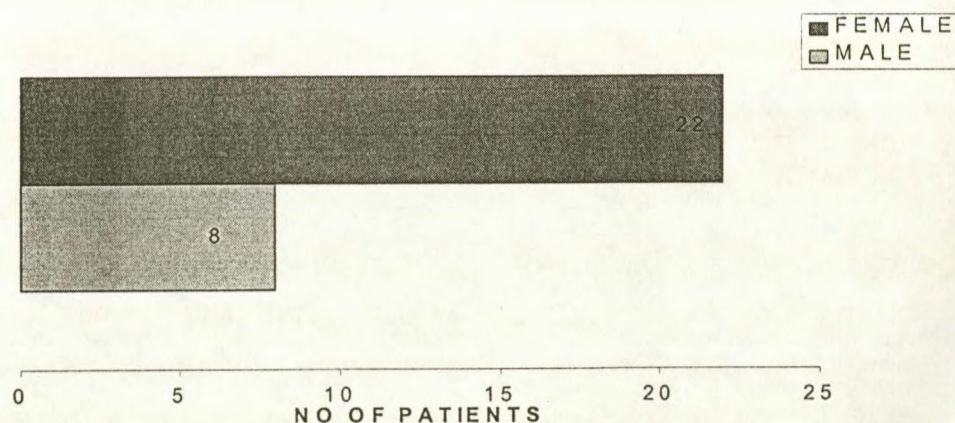
This chapter will present the results obtained from the clinical trial. The first set of data represents the Demographic data obtained from the patient's files.

The second set of data represents the statistical analysis of the results. As the sample group size was thirty patients, non-parametric hypothesis testing was used. The results from the statistical analysis are tabulated to display the mean, the standard deviation, the standard error and the probability value. The P-value is compared to the level of significance, which is set at  $\alpha = 0.05$ , for all the tests.

The objective findings to be analyzed included the algometer and the goniometer readings of flexion, extension, left and right lateral flexion and finally left and right rotation. The subjective findings to be statistically analyzed included the CMCC- Neck Disability Index, the Numerical Pain Rating Scale-101 and the Short Form McGill Pain Questionnaire.

### 4.2 DEMOGRAPHIC DATA

Figure1: The ratio of males to females within the sample was 8:22.





**Table 1:** The age distribution and gender distribution within the sample group

	GROUP 1 (ICE)	GROUP 2 (WARM)	TOTAL
<b><u>Age distribution:</u></b>			
Age range:	21-29	18-50	18-50
Average age:	23.60	28.53	26.07
<b><u>Gender distribution:</u></b>			
Female	9	13	22
Male	6	2	8

**Figure 2:** The location of the trigger points within the trapezius (trap), the levator scapulae (lev sca) and the infraspinatus (infrasp) of the cold group.

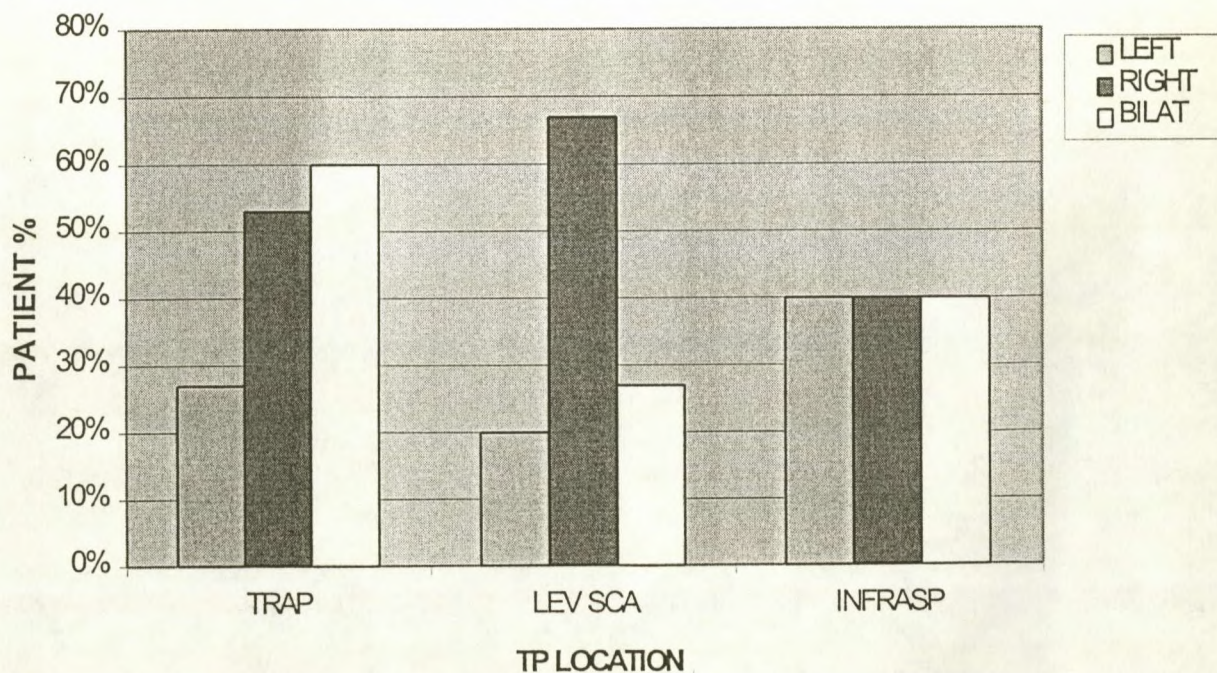




Figure 3: The location of the trigger points within the trapezius (trap), the levator scapulae (lev sca) and the infraspinatus (infrasp) of the warm group.

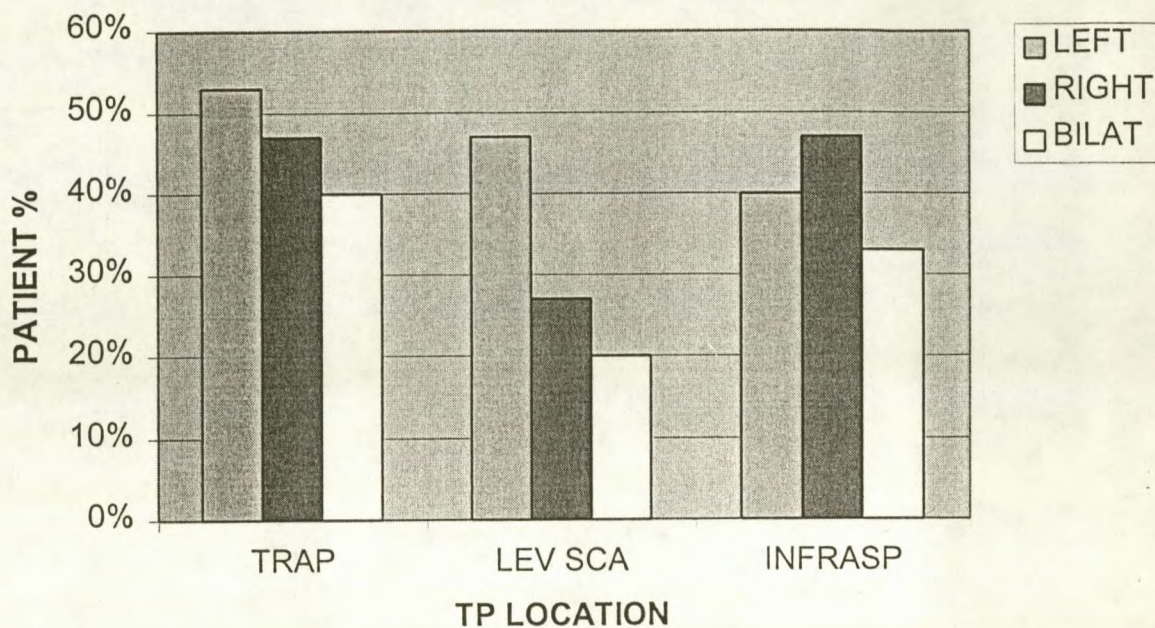


Figure 4: The presence of specific trigger points within the trapezius (trap), the levator scapulae (lev sca) and the infraspinatus (infrasp) of the cold group.

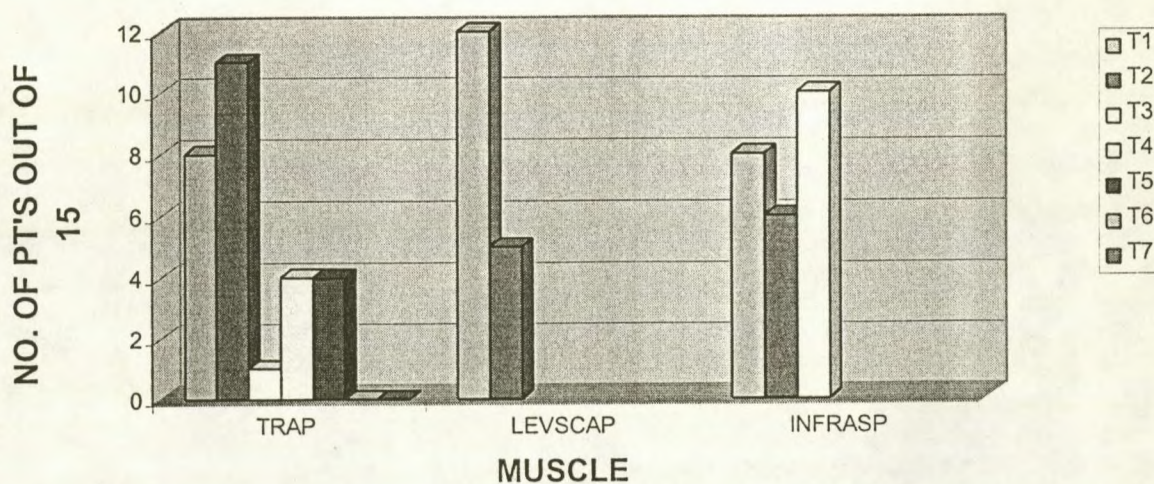
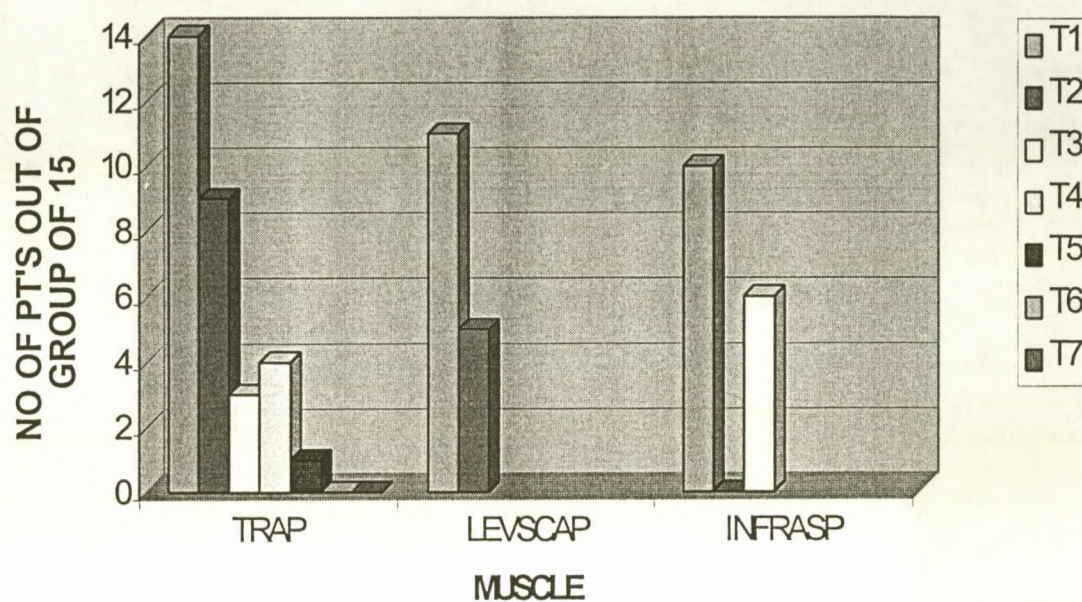




Figure 5: The presence of specific trigger points within the trapezius (trap), the levator scapulae (lev sca) and the infraspinatus (infrasp) of the warm group.



## 4.3 THE STATISTICAL ANALYSIS

### 4.3.1 Abbreviations

S.D. = Standard deviation

S.E. = Standard error

P-value = The observed significance level of the test

$H^0$  = The null hypothesis

$H_1$  = The alternate hypothesis.

$\alpha$  = The level of significance of the test

CMCC-NDI = CMCC Neck Disability Index  
Questionnaire

NPRS-101 = The Numerical Pain Rating scale-101  
Questionnaire

SFMGPQ = The short form McGill Pain Questionnaire

TX 1:before = Before treatment number one

TX 1:after = After treatment one

TX 5 = Treatment five

#### 4.4 NON-PARAMETRIC HYPOTHESIS TESTING

##### 4.4.1 INTRA-GROUP ANALYSIS: Wilcoxon sign-ranked tests

##### 4.4.1.1 Analysis of objective findings of GROUP 1 (cold + passive stretching)

Table 2: The results of the Wilcoxon's signed-rank test comparing the algometer and all goniometer readings before and after the first treatment for GROUP 1.

GROUP 1 : COLD + PASSIVE STRETCHING							
TREATMENT 1: before				TREATMENT 1 : after			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
<b>GONIOMETER</b>							
Flexion	74.53	13.63	3.52	0.2672	74.93	11.00	2.84
Extension	66.27	12.51	3.23	0.5791	67.13	10.34	2.67
Left lateral flexion	42.6	6.00	1.55	<b>0.0098</b>	46.80	6.62	1.71
Right lateral flexion	41.20	5.75	1.48	0.0433	44.40	4.73	1.22
Left rotation	68.40	7.05	1.82	0.0613	72.13	6.78	1.75
Right rotation	67.47	7.03	1.81	0.0433	70.53	8.57	2.21
<b>ALGOMETER</b>	2.02	0.54	0.14	<b>0.0098</b>	2.16	0.46	0.12

For the Goniometer readings of flexion, extension, right lateral flexion, left rotation and right rotation the null hypothesis is accepted and one can conclude that at the 95% confidence level, there was no significant objective improvement immediately after the first treatment.

For the Goniometer reading of left lateral flexion and for the algometer reading, the null hypothesis is rejected which indicates that at a 95% confidence level one can conclude that there was significant objective improvement immediately after the first treatment.

The standard deviation shows the spread of the data around the mean value. In all instances above, in both treatments, the S.D. values were similar enough to render the two sets of data reliable and comparable.

Table 3: The results of Wilcoxon's signed-rank test comparing the algometer and all goniometer readings between the first treatment and fifth treatment for GROUP 1

GROUP 1 : COLD + PASSIVE STRETCHING							
TREATMENT 1: before				TREATMENT 5: after			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
<b>GONIOMETER</b>							
Flexion	74.53	13.63	3.52	0.1490	77.33	8.37	2.16
Extension	66.27	12.51	3.23	0.1824	71.20	8.97	2.32
Left lateral flexion	42.60	6.00	1.55	0.0265	46.00	5.81	1.50
Right lateral flexion	41.20	5.75	1.48	0.0093	45.20	5.75	1.48
Left rotation	68.40	7.05	1.82	0.0613	73.20	8.38	2.16
Right rotation	67.47	7.03	1.81	0.0005	73.33	7.47	1.93
<b>ALGOMETER</b>	2.02	0.54	0.14	0.0005	2.46	0.46	0.12

For the Goniometer readings of flexion, extension, left lateral flexion and left rotation the null hypothesis is accepted and one can conclude that at the 95% confidence level, there was no significant objective improvement between the first and the fifth treatments.

For the Goniometer reading of right lateral flexion and right rotation, and for the Algometer reading, the null hypothesis is rejected which indicates that at a 95% confidence level one can conclude that there was significant objective improvement between the first and the fifth treatments.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.



Table 4: The results of the Wilcoxon's signed-rank test comparing the algometer and all goniometer readings between the first treatment and the one month follow-up appointment for GROUP 1

GROUP 1 : COLD + PASSIVE STRETCHING							
TREATMENT 1 :before				FOLLOW-UP APPOINTMENT			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
<b>GONIOMETER</b>							
Flexion	74.53	13.63	3.52	0.7728	74.40	7.90	2.04
Extension	66.27	12.51	3.23	0.0961	69.73	11.88	3.07
Left lateral flexion	42.60	6.00	1.55	0.7728	43.87	4.50	1.16
Right lateral flexion	41.20	5.75	1.48	0.5050	43.53	5.79	1.50
Left rotation	68.40	7.05	1.82	0.1814	71.87	7.72	1.99
Right rotation	67.47	7.03	1.81	<b>0.0161</b>	72.07	7.06	1.82
<b>ALGOMETER</b>	2.02	0.54	0.14	<b>0.0032</b>	2.57	0.68	0.18

For the Goniometer readings of flexion, extension, left and right lateral flexion and left rotation the null hypothesis is accepted and one can conclude that at the 95% confidence level, there was no significant objective improvement between the first treatment and the one month follow-up appointment

For the Goniometer reading of right rotation and for the Algometer reading, the null hypothesis is rejected which indicates that at a 95% confidence level one can conclude that there was significant objective improvement between the first treatment and the one-month follow-up appointment.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

Table 5: The results of the Wilcoxon's signed-rank test comparing the algometer and all goniometer readings between the fifth treatment and the one month follow-up appointment for GROUP 1

GROUP 1 : COLD + PASSIVE STRETCHING							
TREATMENT 5 : after				FOLLOW-UP APPOINTMENT			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
<b>GONIOMETER</b>							
Flexion	77.33	8.37	2.16	0.2672	74.40	7.90	2.04
Extension	71.20	8.97	2.32	0.5790	69.73	11.88	3.07
Left lateral flexion	46.00	5.81	1.50	0.2672	43.87	4.50	1.16
Right lateral flexion	45.20	5.75	1.48	0.1489	43.53	5.79	1.50
Left rotation	73.20	8.38	2.16	0.1814	71.87	7.72	1.99
Right rotation	73.33	7.47	1.93	0.1138	72.07	7.06	1.82
<b>ALGOMETER</b>	2.46	0.46	0.12	0.7728	2.57	0.68	0.18

For the Goniometer readings of flexion, extension, left and right lateral flexion, left and right rotation and the algometer reading, the null hypothesis is accepted. One can conclude that at the 95% confidence level, there was no significant objective improvement between the fifth treatment and the one month follow-up appointment.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

#### 4.4.1.2. Analysis of subjective findings of GROUP 1 (cold + passive stretching)

Table 6: The results of the Wilcoxon's signed-rank test comparing the CMCC neck disability index (CMCC-NDI), the Numerical Pain rating scale-101 (NPRS-101) and the Short form McGill pain questionnaire (SFMGPQ) between the first and the fifth treatment.

GROUP 1 : COLD + PASSIVE STRETCHING							
	TREATMENT 1				TREATMENT 5		
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
CMCC-NDI	21.47	8.63	2.23	0.0003	8.67	5.89	1.52
NPRS-101	41.30	7.37	1.90	0.0003	21.27	11.77	3.04
SFMGPQ	26.10	13.69	3.54	0.0019	9.61	6.90	1.78

For the CMCC-NDI, NPRS-101 and the SFMGPQ the null hypothesis is rejected and one can conclude that at a 95% confidence level, there was significant subjective improvement between treatments one and five.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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Table 7: The results of the Wilcoxon's signed-rank test comparing the CMCC neck disability index (CMCC-NDI), the Numerical Pain rating scale-101 (NPRS-101) and the Short form McGill pain questionnaire (SFMGPQ) between the first treatment and the one month follow-up appointment.

GROUP 1 : COLD + PASSIVE STRETCHING							
	TREATMENT 1				FOLLOW-UP APPOINTMENT		
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
CMCC-NDI	21.47	8.63	2.23	0.0005	7.07	6.63	1.71
NPRS-101	41.30	7.37	1.90	0.0032	20.33	15.84	4.09
SFMGPQ	26.10	13.69	3.54	0.0019	7.85	7.60	1.96

For the CMCC-NDI, NPRS-101 and the SFMGPQ the null hypothesis is rejected and one can conclude that at a 95% confidence level, there was significant subjective improvement between treatment one and the one month follow-up appointment.

The standard deviations of the CMCC-NDI and the SFMGPQ showed that the spread of the data was similar enough to render the two sets of data reliable and comparable.

There was a slight difference between the standard deviations of the NPRS-101 (Tx1: 7.37 vs. FU: 15.84) with the follow-up appointment data showing greater spread around the mean. This data is therefore more unreliable.

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Table 8: The results of the Wilcoxon's signed-rank test comparing the CMCC neck disability index (CMCC-NDI), the Numerical Pain rating scale-101 (NPRS-101) and the Short form McGill pain questionnaire (SFMGPQ) between the fifth treatment and the one month follow-up appointment.

GROUP 1 : COLD + PASSIVE STRETCHING							
	TREATMENT 5				FOLLOW-UP APPOINTMENT		
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
CMCC-NDI	8.67	5.89	1.52	0.4227	7.07	6.63	1.71
NPRS-101	21.27	11.77	3.04	0.6056	20.33	15.84	4.09
SFMGPQ	9.61	6.90	1.78	0.4227	7.85	7.60	1.96

For the CMCC-NDI and the NPRS-101 the null hypothesis is accepted and one can conclude that, at a 95% confidence level, there was no significant subjective improvement between the fifth treatment and the one-month follow-up appointment.

For the SFMGPQ the null hypothesis is rejected and one can conclude that, at a 95% confidence level, there was significant subjective improvement between the fifth treatment and the one-month follow-up appointment.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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#### 4.4.1.3 Analysis of objective findings of group 2 (heat + passive stretching)

Table 9: The results of the Wilcoxon's signed-rank test comparing the algometer and all goniometer readings before and after the first treatment for GROUP 2

GROUP 2 : HEAT + PASSIVE STRETCHING							
TREATMENT 1 :before				TREATMENT 1 :AFTER			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
<b>GONIOMETER</b>							
Flexion	70.67	11.87	3.07	0.5049	72.80	9.94	2.27
Extension	66.93	5.99	1.55	0.2672	67.73	7.52	1.94
Left lateral flexion	41.47	4.69	1.21	<b>0.0032</b>	47.87	5.78	1.49
Right lateral flexion	41.40	7.27	1.88	<b>0.0093</b>	44.87	4.82	1.24
Left rotation	64.93	8.00	2.06	<b>0.0008</b>	70.67	6.13	1.58
Right rotation	68.27	7.59	1.96	0.4226	69.8	5.53	1.43
<b>ALGOMETER</b>	1.98	0.44	0.11	<b>0.0003</b>	2.174	0.49	0.13

For the Goniometer readings of flexion, extension and right rotation the null hypothesis is accepted and one can conclude that at the 95% confidence level, there was no significant objective improvement between the first and fifth treatments.

For the goniometer readings of left and right lateral flexion and left rotation and for the algometer reading, the null hypothesis is rejected which indicates that at a 95% confidence level one can conclude that there was significant objective improvement between the first and fifth treatments.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

Table 10: The results of the Wilcoxon's signed-rank test comparing the algometer and all goniometer readings between the first treatment and fifth treatment for GROUP 2

GROUP 2 : HEAT + PASSIVE STRETCHING							
TREATMENT 1 :before				TREATMENT 5 : after			
Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.	
<b>GONIOMETER</b>							
Flexion	70.67	11.87	3.07	0.5790	73.60	10.70	2.76
Extension	66.93	5.99	1.55	0.0265	72.67	4.45	1.15
Left lateral flexion	41.47	4.69	1.21	0.0019	47.20	5.00	1.29
Right lateral flexion	41.40	7.27	1.88	0.0160	44.87	4.82	1.25
Left rotation	64.93	8.00	2.06	0.0014	73.47	4.87	1.26
Right rotation	68.27	7.59	1.96	0.0014	72.68	4.26	1.10
<b>ALGOMETER</b>	1.98	0.44	0.11	0.0019	2.32	0.47	0.12

For the Goniometer readings of flexion and extension the null hypothesis is accepted and one can conclude that at the 95% confidence level, there was no significant objective improvement between the first and fifth treatments.

For the goniometer readings of left and right lateral flexion, left and right rotation and for the algometer reading, the null hypothesis is rejected which indicates that at a 95% confidence level one can conclude that there was significant objective improvement between the first and fifth treatments.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

Table 11: The results of the Wilcoxon's signed-rank test comparing the algometer and all goniometer readings between the first treatment and the follow-up appointment for GROUP 2

GROUP 2 : HEAT + PASSIVE STRETCHING							
TREATMENT 1 : before				FOLLOW-UP APPOINTMENT			
Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.	
<b>GONIOMETER</b>							
Flexion	70.67	11.87	3.07	0.2278	73.67	10.53	2.72
Extension	66.93	5.99	1.55	0.2278	70.00	7.37	1.90
Left lateral flexion	41.47	4.69	1.21	0.2673	44.67	6.75	1.74
Right lateral flexion	41.40	7.27	1.88	0.2737	42.00	7.01	1.81
Left rotation	64.93	8.00	2.06	0.0704	70.80	6.27	1.62
Right rotation	68.27	7.59	1.96	0.2278	70.93	7.70	1.99
<b>ALGOMETER</b>	1.98	0.44	0.11	0.0389	2.24	0.53	0.14

For the Goniometer readings of flexion, extension, left and right lateral flexion, left and right rotation, and for the algometer reading, the null hypothesis is accepted and one can conclude that at the 95% confidence level, there was no significant objective improvement between the first treatment and the one month follow-up.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.



Table 12: The results of the Wilcoxon's signed-rank test comparing the algometer and all goniometer readings between the fifth treatment and the one month follow-up appointment for GROUP 2

GROUP 2 : HEAT + PASSIVE STRETCHING							
TREATMENT 5 : after				FOLLOW-UP APPOINTMENT			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
<b>GONIOMETER</b>							
Flexion	73.60	10.70	2.76	0.7892	73.67	10.53	2.72
Extension	72.67	4.45	1.15	0.2278	70.00	7.37	1.90
Left lateral flexion	47.20	5.00	1.29	0.2673	44.67	6.75	1.74
Right lateral flexion	44.87	4.82	1.25	0.7237	42.00	7.01	1.81
Left rotation	73.47	4.87	1.26	0.0700	70.80	6.27	1.62
Right rotation	72.68	4.26	1.10	0.2278	70.93	7.70	1.99
<b>ALGOMETER</b>	2.32	0.47	0.12	0.0613	2.24	0.53	0.14

For the Goniometer readings of flexion, extension, left and right lateral flexion, left and right rotation, and for the algometer reading, the null hypothesis is accepted. One can conclude that at the 95% confidence level, there was no significant objective improvement between the fifth treatment and the one month follow-up.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

4.1.1.4 Analyses of **subjective** findings of GROUP 2 (heat and passive stretching).

Table 13: The results of the Wilcoxon's signed-rank test comparing the CMCC neck disability index (CMCC-NDI), the Numerical Pain rating scale-101 (NPRS-101) and the Short form McGill pain questionnaire (SFMGPQ) between the first and the fifth treatment.

GROUP 1 : HEAT + PASSIVE STRETCHING							
	TREATMENT 1				TREATMENT 5		
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
CMCC-NDI	22.80	10.52	2.71	0.0033	11.07	7.55	1.95
NPRS-101	46.33	12.61	3.26	0.0003	25.33	16.89	4.36
SFMGPQ	30.67	15.76	4.07	0.0389	13.06	9.20	2.38

For the CMCC-NDI, the NPRS-101 and the SFMGPQ the null hypothesis is rejected and one can conclude that, at a 95% confidence level, there was significant subjective improvement between the first and the fifth treatment.

The standard deviations of the CMCC-NDI and the NPRS-101 showed that the spread of the data was similar enough to render the two sets of data reliable and comparable.

There was a slight difference between the standard deviations of the SFMGPQ (Tx1: 15.76 vs. Tx5: 9.20) with the treatment 1 data showing greater spread around the mean. This data is therefore more unreliable.

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Table 14: The results of the Wilcoxon's signed-rank test comparing the CMCC neck disability index (CMCC-NDI), the Numerical Pain rating scale-101 (NPRS-101) and the Short form McGill pain questionnaire (SFMGPQ) between the first treatment and the follow-up appointment

GROUP 1 : HEAT + PASSIVE STRETCHING							
TREATMENT 1				FOLLOW-UP APPOINTMENT			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
CMCC-NDI	22.80	10.52	2.71	0.0019	11.07	7.43	1.92
NPRS-101	46.33	12.61	3.26	0.0019	26.56	14.10	3.64
SFMGPQ	30.67	15.76	4.07	0.0019	14.22	11.69	3.02

For the CMCC-NDI, NPRS-101 and the SFMGPQ, the null hypothesis is rejected and one can conclude that, at a 95% confidence level, there was significant subjective improvement between the first treatment and the one-month follow-up appointment.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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Table 15: The results of the Wilcoxon's signed-rank test comparing the CMCC neck disability index (CMCC-NDI), the Numerical Pain rating scale-101 (NPRS-101) and the Short form McGill pain questionnaire (SFMGPQ) between the fifth treatment and the one month follow-up appointment.

GROUP 1 : HEAT + PASSIVE STRETCHING							
TREATMENT 5				FOLLOW-UP APPOINTMENT			
	Mean	S.D.	S.E.	P-value	Mean	S.D.	S.E.
CMCC-NDI	11.07	7.55	1.95	0.7518	11.07	7.43	1.92
NPRS-101	25.33	16.89	4.36	1.0000	26.56	14.10	3.64
SFMGPQ	13.06	9.20	2.38	0.5790	14.22	11.69	3.02

For the CMCC-NDI, NPRS-101 and the SFMGPQ, the null hypothesis is accepted and one can conclude that, at a 95% confidence level, there was no significant subjective improvement between the fifth treatment and the one-month follow-up appointment.

In all instances above in both treatments, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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#### 4.4.2 INTERGROUP ANALYSIS: Mann-Whitney Unpaired two tailed tests

This test was used to evaluate whether there was a difference in effectiveness of either of the two experimental groups.

##### 4.4.2.1 Analysis of objective findings:

Table 16: The results of the Mann-Whitney U test comparing the algometer readings of groups 1 and 2 before and after the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	Mean	S.D.	S.E.
TX1:BEFORE	2.02	0.54	0.14	0.9009	1.98	0.44	0.12
TX1:AFTER	2.16	0.46	0.12	0.8845	2.17	0.49	0.13
TX 5	2.46	0.46	0.12	0.3506	2.32	0.47	0.12
FOLLOW-UP APPOINTMENT	2.57	0.68	0.18	0.1912	2.24	0.53	0.14

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group 1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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Table 17: The results of the Mann-Whitney U test comparing the goniometer readings of flexion of groups 1 and 2 before and after the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	Mean	S.D.	S.E.
TX1:BEFORE	74.53	13.64	3.52	0.6318	70.67	11.87	3.07
TX1:AFTER	74.93	11.00	2.84	0.8998	72.80	9.93	2.57
TX 5	77.33	8.37	2.16	0.4165	73.60	10.70	2.76
FOLLOW-UP APPOINTMENT	74.40	7.90	2.04	0.7431	73.67	10.53	2.72

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group 1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean were similar enough to render the two sets of data reliable and comparable.

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Table 18: The results of the Mann-Whitney U test comparing the goniometer readings of extension of groups 1 and 2 before and after the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	Mean	S.D.	S.E.
TX1:BEFORE	66.27	12.51.	3.23	0.8328	66.93	5.99	1.55
TX1:AFTER	67.13	10.34	2.67	0.9834	67.73	7.52	1.94
TX 5	71.20	8.97	2.32	1.0000	72.67	4.45	1.15
FOLLOW-UP APPOINTMENT	69.73	11.88	3.07	0.9668	70.00	7.37	1.90

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group1 and group 2.

The standard deviations of extension within the two groups for TX1: after, TX5 and the Follow-up appointment showed that the spread of the data was similar enough to render the two sets of data reliable and comparable.

There was a slight difference between the standard deviations of the 2 groups at 'TX1: before'. The TX1: before data from group 1 showed greater spread around the mean. This data is therefore more unreliable than that of group 2.

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Table 19: The results of the Mann-Whitney U test comparing the goniometer readings of left lateral flexion of groups 1 and 2 before and after the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	mean	S.D.	S.E.
TX1:BEFORE	42.60	5.99	1.54	0.6545	41.47	4.69	1.21
TX1:AFTER	46.80	6.62	1.71	0.7376	47.87	5.78	1.49
TX 5	46.00	5.80	1.50	0.8504	47.20	5.00	1.29
FOLLOW-UP APPOINTMENT	43.87	4.50	1.16	0.7356	44.67	6.75	1.74

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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Table 20: The results of the Mann-Whitney U test comparing the goniometer readings of right lateral flexion of groups 1 and 2 before and after the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	mean	S.D.	S.E.
TX1:BEFORE	41.20	5.75.	1.48	0.4148	41.40	7.27	1.88
TX1:AFTER	44.40	4.73	1.22	0.8343	44.87	4.82	1.25
TX 5	45.20	5.75	1.48	0.9665	45.60	4.48	1.16
FOLLOW-UP APPOINTMENT	43.53	5.79	1.50	0.3844	42.00	7.01	1.81

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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Table 21: The results of the Mann-Whitney U test comparing the goniometer readings of left rotation of groups 1 and 2 before and after the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	mean	S.D.	S.E.
TX1:BEFORE	68.40	7.05.	1.82	0.1329	64.93	7.99	2.06
TX1:AFTER	72.13	6.78	1.75	0.3806	70.67	6.13	1.58
TX 5	73.20	8.38	2.16	0.7061	73.47	4.87	1.26
FOLLOW-UP APPOINTMENT	71.87	7.72	1.99	0.5442	70.87	6.27	1.62

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group 1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean were similar enough to render the two sets of data reliable and comparable.

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Table 22: The results of the Mann-Whitney U test comparing the goniometer readings of right rotation of groups 1 and 2 before and after the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	Mean	S.D.	S.E.
TX1:BEFORE	67.47	7.03.	1.81	0.8834	68.27	7.59	1.96
TX1:AFTER	70.53	8.57	2.21	0.3167	69.80	5.53	1.43
TX 5	73.33	7.47	1.93	0.3914	72.80	4.26	1.10
FOLLOW-UP APPOINTMENT	72.07	7.06	1.82	0.3910	70.93	7.70	1.99

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group 1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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#### 4.4.2.2 Analysis of subjective findings

Table 23: The results of the Mann-Whitney U test comparing the CMCC Neck Disability Index values of groups 1 and 2 at the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	Mean	S.D.	S.E.
TREATMENT 1	21.47	8.63	2.23	0.6174	22.80	10.52	2.72
TREATMENT 5	8.67	5.89	1.52	0.3911	11.07	7.55	1.95
FOLLOW-UP APPOINTMENT	7.07	6.63	1.71	0.1425	11.07	7.44	1.92

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group 1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

Table 24: The results of the Mann-Whitney U test comparing the Numerical Pain rating scale-101 values of groups 1 and 2 at the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	Mean	S.D.	S.E.
TREATMENT 1	41.30	7.37	1.90	0.1299	46.33	12.61	3.26
TREATMENT 5	21.27	11.77	3.04	0.6040	25.33	16.89	4.36
FOLLOW-UP APPOINTMENT	20.33	15.84	4.09	0.2626	26.57	14.10	3.64

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group 1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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Table 25: The results of the Mann-Whitney U test comparing the short form McGill pain questionnaire values of groups 1 and 2 at the first consultation, at the fifth consultation and at the follow-up appointment.

	GROUP 1 (COLD)				GROUP 2 (WARM)		
	Mean	S.D.	S.E.	p-value	Mean	S.D.	S.E.
TREATMENT 1	26.18	13.69	3.54	0.3614	30.67	15.76	4.07
TREATMENT 5	9.61	6.90	1.78	0.4427	13.06	9.20	2.38
FOLLOW-UP APPOINTMENT	7.85	7.60	1.96	0.1005	14.22	11.69	3.02

The null hypothesis is accepted, which indicates that at  $\alpha = 0.05$  level of significance there is no statistically significant difference between group 1 and group 2.

In all instances above in both groups, the S.D. values, showing the spread of the data around the mean, were similar enough to render the two sets of data reliable and comparable.

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# *CHAPTER* *FIVE*

## *DISCUSSION*

## CHAPTER 5: DISCUSSION

### 5.1 INTRODUCTION

In this chapter the results reported in chapter 4 will be discussed.

Any statistically significant information will be highlighted and examined for reliability. Furthermore, graphs will be used to highlight and clarify relevant information.

### 5.2 RANGES OF MOTION

The goniometer was used to measure the planes of motion within the cervical spine. These were as follows: flexion, extension, left and right lateral flexion and finally left and right rotation. Any muscle spasm within the muscles should result in decreased range of motion. Any improvement with a treatment protocol would be signified by increased readings of the goniometer.

#### 5.2.1 Discussion of the Intragroup analysis

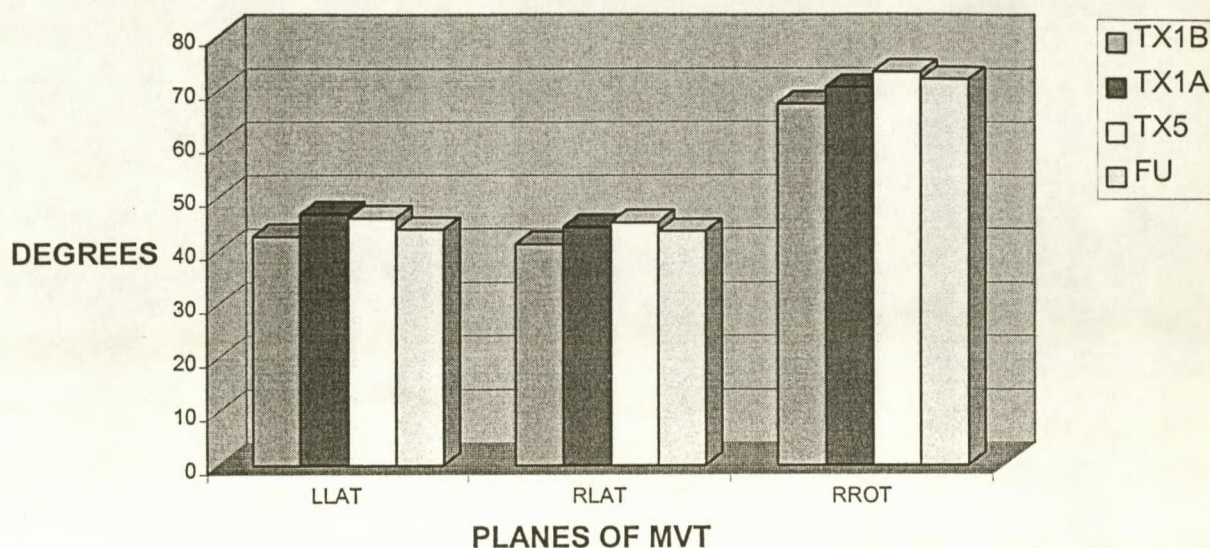
Immediate improvement was measured by using the goniometer before and after the first treatment. Improvement during the treatment was measured by taking readings before the first treatment and after the final treatment. A measurement was taken comparing the end of the final treatment with the follow-up appointment. This was done to clarify whether or not the expected improvement was temporary or of longer duration. Finally, overall change was measured by comparing the first treatment (before) with the follow-up appointment.



Within Group 1, the only movements to show statistically significance difference were left and right lateral flexion and right rotation (see figure 6)

The general *clinical* trend for Group 1 (cold + passive stretching) was to show immediate improvement in all planes of movement. This improvement continued through to the end of the five treatments. Thereafter there was a general decrease in readings. This was anticipated as the patient did return to their environments with all the daily stresses that initiated the trigger points. Although continuation of the stretches used during the treatments was recommended to each patient, to maintain the improvement, most patients did not follow this advice. This finding supports the study by Lewitt and Simons (1984) whereby the best, lasting results were found in patients *who used the stretches thereafter*. Of interest is the fact that only flexion showed no mean improvement after a one-month interval when compared to the baseline measurements (Table 4). All the other movements showed mean improvement over the initial readings prior to the first treatment. As the mean difference was only  $0.13^{\circ}$  (Tx1-74.53 to F.U-74.40) and the follow-up flexion data was found to be more reliable (S.D. 7.90 and S.E. 2.04) than the initial data (S.D. 13.63 and S.E. 3.52) this finding was probably co-incidental. It must be emphasized that the general trends were clinical observations rather than statistical evidence.

FIGURE 6 MEAN GONIOMETER VALUES

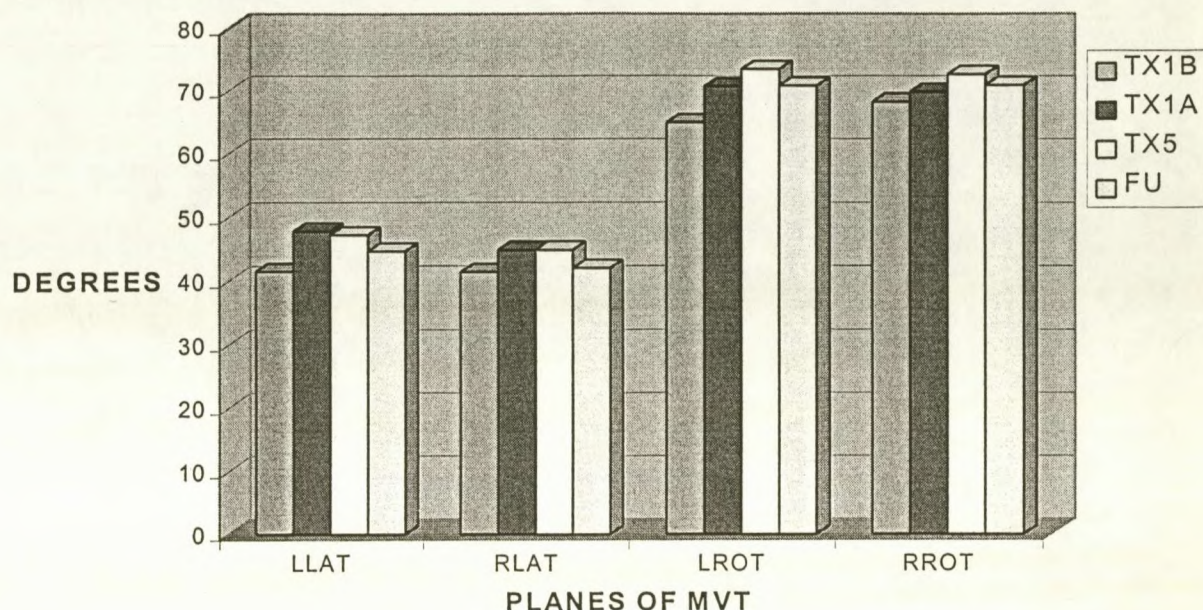


Within Group 2 (warm + passive stretching) the trends were similar to Group 1 with immediate improvement continuing and peaking at the end of treatment five, followed by the expected slight decline at the follow-up appointment. This time, however, all final readings showed improvement on the initial readings. Again these were clinical observations.

Within Group 2, the ranges of motion that showed statistically significant difference were lateral flexion bilaterally and rotation bilaterally. (See figure 7)



FIGURE 7 : MEAN GONIOMETER VALUES



Within Group 1, only left lateral flexion showed statistically significant immediate improvement (Table 2). During the course of the treatment, between treatment one and treatment five, right lateral flexion and right rotation were now statistically significant (Table 3). Of interest is the fact that left lateral flexion was very close to being significant with the  $p\text{-value}=0.0265$  (Reject  $H^0$  if  $P \leq \alpha/2 = 0.025$ ). This was consistent with the trend shown in the immediate improvement category. There was no statistically significant improvement between treatment 5 and the follow-up (Table 5). Between treatment one and the follow-up, right rotation showed statistically significant improvement (Table 4). Explanations will be hypothesized shortly.

Within Group 2, statistically significant immediate improvement was shown in left and right lateral flexion and in right rotation (Table 9). Statistically significant improvement over the treatment program was shown in lateral flexion and

rotation bilaterally (Table 10). Between treatment one and the follow-up appointment (Table 11) and between treatment five and the follow-up appointment (Table 12) there was no significant improvement. Explanations will be hypothesized in the following paragraphs.

According to Travell and Simons (1983: 183), the primary symptoms of trigger points within the trapezius muscle are pain rather than weakness and restriction of motion. Furthermore the goniometer was used to measure range of motion within the neck rather than the shoulder. Therefore, changes from the treatment within the trapezius and the infraspinatus may not be picked up by the goniometer.

One would expect the goniometer to reflect the improvement primarily from the levator scapulae. According to Travell and Simons (1983: 334), the primary symptoms include restriction of rotation to the same side and to a lesser extent extension of the neck. In figure 4.2.3, 65% of Group 1 had trigger points on the right (right rotation restricted) and 35% had them bilaterally (rotation restricted bilaterally). This would explain the improvement in right rotation between treatment one and five and between treatment one and the follow-up appointment. According to Magee (1992 : 40), rotation and lateral flexion always occur together in the cervical spine as a result of the shape of the facet joints (coronally oblique). Therefore one could hypothesize that any limitation within the rotation would also affect the mobility of the lateral flexion to the same side. This could explain the improvement of right lateral flexion between treatments one and five. The improvement before and after treatment one of left lateral flexion could be attributed to the percentage of patients who had trigger points bilaterally (35%).

Within Group 2, of the fifteen patients 45% had levator scapulae trigger points on the left, 25% on the right and 18% bilaterally. (Figure 3) This could account for the significant

immediate improvement within left and right lateral flexion and left rotation (Table 9). Furthermore this could account for the improvement between treatment one and five of rotation and lateral flexion bilaterally. It must be emphasized that these hypotheses are speculation rather than supported by hard statistical evidence.

Unfortunately no studies could be found to which these results could be compared.

Some difficulties were encountered in the use of the goniometer. Firstly the 2° scale made it difficult to measure small variations in the cervical range of motion. Secondly, in measuring rotation a compass was used. While every attempt was made to stabilize the patient's shoulders during movement, some rotation of the middle and upper thoracic spine may have occurred thereby, slightly distorting the readings. This could be overcome by having the patient sit flush up against a hard back chair.

#### 5.2.2 Discussion of the Intergroup analysis.

There was no statistically significant difference between the two groups with regards to mean gains in range of motion at any stage in the treatment program. Comparing the groups prior to the first treatment established a baseline to ensure that the two groups were comparable in nature. There was a slight difference in flexion with Group 1 having a mean of 74.53° as compared to Group 2 with a mean of 70.67° (Table 17). However Group 2 showed slightly better reliability with a standard deviation of 11.87 as compared to 13.64. It must be pointed out that these two values are close so the difference is small. In addition the mean values became reasonably comparable by the follow-up appointment (Group 1 mean: 74.40° vs. Group 2 mean: 73.67°). The only other baseline value to differ was left rotation (Table 21). Group 1 had a mean value of 68.40° and Group 2 had a mean value of 64.93°.

Again, however the values became similar by the fifth treatment (Group 1 mean: 73.20° vs. Group 2 mean: 73.47°). It must be recalled that neither flexion nor left rotation showed significant statistical difference when using the Mann Whitney U test.

Both groups showed improvement throughout the treatment program followed by the slight decrease in readings by the one month follow-up appointment.

One can therefore accept the null hypothesis which states that there is no significant difference between the groups with regards to the two treatments.

According to Magee (1992: 40), the maximum movement for the six planes of motion are as follows:

Flexion :	80°-90°
Extension:	70°
Rotation:	70°-90°
Lateral flexion:	20°-45°

This study supports the values of flexion and rotation, but contradicts lateral flexion and extension. Group 1 had a peak mean extension value of 71.20° (Table 18) while Group 2 had a peak mean extension value of 72.67° (Table 18). Likewise for lateral flexion, Group 1 had a peak mean value of 46.80° (Table 19) while Group 2 had a peak mean value of 47.87° (Table 19).

However, for this information to have any significance a larger sample group would be required.

### 5.3 ALGOMETER

The algometer was used to measure the amount of force that the patient could tolerate on the trigger points. Improvement would be signified by a decrease in the sensitivity of the



trigger point and an ensuing increase in the amount of pressure the patient would allow.

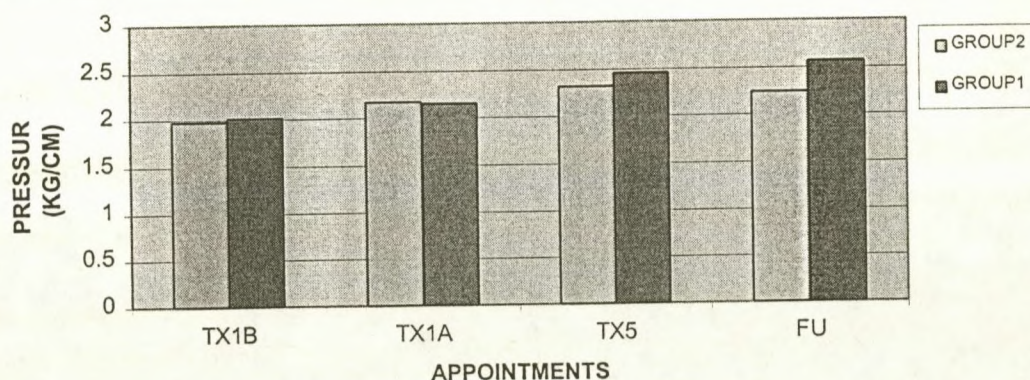
### 5.3.1 Discussion of the Intragroup analysis

The various values for the Algometer, for both groups, over the study period can be seen in figure 8 below.

Group 1 showed statistically significant improvement immediately after the first treatment (Table 2), over the whole treatment program (Table 3) and after the one month follow-up appointment (Table 4). Improvement was not expected between treatment five and the follow-up as patient's were again returning to their daily environment with all the associated stresses.

Group 2 showed statistically significant improvement immediately after the first treatment (Table 9) and over the whole treatment program (Table 10)

FIGURE 8 MEAN ALGOMETER VALUES



According to Fischer (1986), immediate effect is expressed by a difference in pressure threshold measurement before and after a therapeutic session, while long term effects may be assessed by repeated measurements. The results of this study confirm this, with the increase in pressure tolerated by the patient signifying improvement.

### 5.3.2 Discussion of the Intergroup analysis

There were no statistically significant differences between the 2 groups at any point during the study. (Table 16) Both groups showed improvement until the end of the fifth treatment. Thereafter Group 2 showed a decrease while Group 1 showed a continued increase after one month. This finding was unexpected and tended to contradict the usual pattern of a slight decrease after the one month follow-up appointment. For Group 1 the standard deviation at the one month follow-up was 0.68. This showed the greatest deviation from the mean and it is possible that the arithmetic mean was distorted by some 'fringe' values. These unusual values would have the effect of skewing the data and causing unexpected results.

The null hypothesis, which states that there is no statistically significant difference between the algometer readings of the two groups is therefore accepted.

The algometer used had two scales of measurement. The pressure could be measured in pounds per  $\text{cm}^2$  or kilograms per  $\text{cm}^2$ . It is advised that an operator should be certain which scale is being used from the first treatment, so that two different scales are not used when measuring the amount of pressure that can be tolerated.

### 5.4 CMCC NECK DISABILITY INDEX

This form was used to measure the patient's perception of the disability related to the myofascial pain. The patient was required to fill out the form at the first, fifth and follow-up appointment. Improvement would be signified by a decrease in the final percentage value.



#### 5.4.1 Discussion of the Intragroup analysis

There was a statistically significant improvement between the first and fifth treatment and the first treatment and the follow-up appointment of both groups. This indicates that the patient perceived improvement during the course of the five treatment program and that the patient believed the improvement lasted into the following month. For both groups, there was no significant decrease in the patient's disability perception between the fifth treatment and the one month follow-up. Of interest is the fact that in Group 1 the mean value after one month was lower than the mean value at the fifth treatment appointment ( Follow-up: 7.07. vs. Treatment 5: 8.67; tables 6 and 7). This indicates that the patients felt less disability after one month than they did after the final treatment, with regard to the myofascial pain. Within Group 2 the mean values at the fifth treatment and the follow-up appointment were identical (11.07; Table 23).

#### 5.4.2 Discussion of the Intergroup analysis

There was no statistically significant difference between the two groups in terms of mean improvement at any point in the study (Table 23). Therefore, one can accept the null hypothesis which states that there was no difference between the two groups in terms of the patient's perceived disability resulting from the myofascial problem.

The CMCC Neck Disability Index was a quick, efficient and 'user-friendly' form. The patients had no difficulty understanding what was required of them. This form is recommended for future projects of this nature.

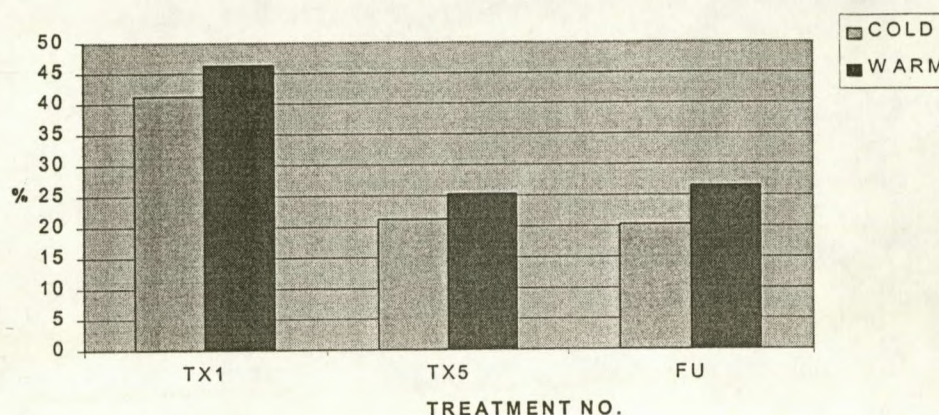
## 5.5 NUMERICAL PAIN RATING SCALE-101

The Numerical Pain Rating Scale-101 (NPRS-101) was chosen because of the ease with which it can be administered and scored. Jensen et al. (1986) established its validity and reliability when providing subjective information about the levels of pain perceived by the patient. It was used to monitor the patient's progress with a decrease in pain intensity indicating improvement. The mean values of both groups at the relevant consultations have been tabulated in figure 9 (See page 99).

### 5.5.1 Discussion of the Intragroup analysis

Within both groups there was a statistically significant decrease in pain between treatment one and treatment five (Tables 6 and 13) and between treatment one and the follow-up appointment (Tables 7 and 14). Once again, in neither group was there a statistically significant decrease in pain between the fifth treatment and the follow-up appointment. This could be attributed to the patients returning to the normal stresses within their daily lives and also to the poor patient compliance when continuing the stretches after the treatment program was completed.

**Figure 9:** The mean percentage of pain perception of group 1 and group 2 at treatment one, five and the follow-up.



Within Group 1 the mean decrease in pain continued after one month. This differed from Group 2 where the mean pain measurement peaked at the fifth treatment and then decreased slightly at the follow-up appointment.

One can therefore conclude that, clinically, the patients felt they had improved during the treatments and that improvement was maintained until the follow-up period.

#### 5.5.2 Discussion of the Intergroup analysis

According to the statistical tests there was no significant difference between the two groups at any point within the study. However attention must be drawn to Figure 9. One can see that there was a consistent difference between the 2 groups. Within Group 1 the mean values were less by at least 4.06% when compared to Group 2 at any point within the study. This could be explained by looking at Table 1. One can see that the average age for Group 1 was 23.60 as compared to Group 2 being 28.53.

The random allocation may have resulted in inadequate comparability with Group 1 having a greater percentage of

younger patients. Trigger points are generally more common amongst sedentary individuals and are associated with chronic strain and stress (Sola 1984; Bruce 1995). The older patients in Group 2 may have been more chronic and slower to respond to the treatments thereby increasing these subjective values. Furthermore Travell and Simons (1983: 6) believe that individuals in their mature years of maximum activity are most likely to suffer from active trigger points and with the advancing of years, with the associated reduced activity, the stiffness and restricted range of motion of the latent trigger points becomes more common. The younger patients in Group 1, responding more quickly to the treatment, would then give the impression that Group 1 had a more effective treatment.

The easy manner of using the Numerical Pain Rating Scale-101 makes it an ideal tool for research of this kind.

## 5.6 SHORT FORM MCGILL PAIN QUESTIONNAIRE

The short form of the McGill Pain Questionnaire used in this study was described by Melzack (1987) as a means of subjectively providing information regarding the sensory, affective and evaluative dimensions of the patient's pain. Improvement would be indicated by a decrease in the values as the program progressed.

### 5.6.1 Discussion of the Intragroup analysis

Statistically one can see that within Group 1, the patients believed there was a reduction in pain over the treatment program and over the whole study. As has been the trend there was no further *statistically significant improvement* from the fifth treatment into the follow-up appointment although the mean value did drop further (Table 8).

Within Group 2 there was no initial improvement between treatments one and five. This was unusual but the p-value was fairly close to the significant level of 0.025 ( $p=0.0389$ ). The standard deviation of the first treatment was high (S.D.=15.76 Table 13) indicating that there was a large spread of the data around the median. Likewise the standard error was also high (S.E.= 4.07 Table 13) indicating that the mean showed signs of unreliability. These could explain the break in the trend. There was statistically significant improvement between treatment 1 and the follow-up appointment which did not continue between the fifth treatment and the follow-up appointment. In this instance there was a slight worsening (increase) of the mean values between treatment five and the follow-up as has been the trend.

#### 5.6.2 Discussion of the Intergroup analysis

Like the Numerical Pain Rating Scale-101 there was no significant difference between the two groups at any point within the study. Once again there was a discrepancy in the data. According to Table 25, the mean values of Group 2 were consistently higher than the mean values of Group 1 indicating that the two groups were not totally comparable. This could again be explained by the slight age discrepancy between the two groups.

Generally the 2 objective instruments were easy to use and efficient. Aside from the problems already mentioned there were no other difficulties. Likewise the three subjective parameters were easy to use and are recommended for future studies of this nature.



# *CHAPTER SIX*

## *CONCLUSIONS AND RECOMMENDATIONS*

## CHAPTER 6: CONCLUSIONS AND RECOMMENDATIONS

From the results it would appear that there is no statistically significant difference between the two treatment groups in the treatment of myofasciitis of the shoulder girdle muscles. Both groups tended to increase their ranges of motion and the amount of pressure tolerated before the sensation of pain. This improvement tended to be maintained into the one month follow-up appointment, but at a lower level than the level at the fifth treatment. Therefore, convenience should be a factor when choosing one of the treatment options.

In order for the findings to be validated, a larger sample size is required. A sample size of thirty subjects enables one to make inferences but one cannot really draw conclusions. It must also be pointed out that a control group was not used in this study. Without a control group, it is possible that neither groups are effective. The efficacy of both groups needs to be established and it is therefore recommended that both groups be compared separately to a control group in order to assess their relative effectiveness.

In a busy practice time will always be an important consideration. Ice and passive stretching takes approximately 10 minutes as advocated by Gatterman (1990: 65). Moist heat and passive stretching takes approximately 20 minutes as advocated by Hong *et al.* (1995). Group 1 is therefore more time efficient. In addition the ice and passive stretching could be done at the same time. In Group 2 the practitioner had little to do while waiting for the heat aspect to finish before starting the stretches. Finally the cold and passive stretching required less preparation and was less disordered than the moist heat and passive stretching. It is therefore the opinion of this author that Group 1 was *easier* to treat.

It is the opinion of the author that the moist heat and passive stretching was a more pleasant and relaxing treatment than the

initial shock experienced with the cold and passive stretching. Although the patients in Group 1 felt refreshed afterwards, the initial contact was a little startling. It is recommended that more frail patients might enjoy the moist heat and passive stretching for the comfort factor.

Subjectively, the patients in Group 1 generally had smaller mean values at all times within the study. In addition the standard deviations within Group 1 tended to have smaller values indicating a smaller spread of data around the mean. This implied that the data within Group 1 was more reliable. (Tables 23-25) One can therefore surmise that the patients within Group 1 tended to show better overall subjective improvement.

It is recommended that a future study establish the importance of passive stretching within the treatment to find out the level of influence that stretching has within these treatment protocols. In addition, a future study could investigate the effectiveness of different stretching techniques, e.g. passive stretching versus post-isometric stretching, to find out which technique is the most effective.

Patient compliance was a problem with regards to post treatment stretching. This could be overcome by asking the patients to keep a diary describing trends thereby emphasizing the importance. A study into the long term effectiveness of post treatment stretching in the treatment of myofasciitis is recommended.

A future study is recommended to investigate the relative effectiveness of using ice as opposed to ethyl chloride in the treatment of myofasciitis.

In future studies of this nature, certain factors do need to be taken into account. These include the effect of gender, age and patient characteristics in the treatment on myofasciitis. The



experience and reliability of the examiner and the accuracy of measurement parameters do need to be considered. It is recommended that the issue of patient blinding, a control group and patient compliance also be addressed.

Considering the results within the study, it is the authors opinion that both methods of treating myofasciitis are as effective, although cold and passive stretching is easier and more time effective. A larger sample size is essential to validate these findings and in future studies of this nature.

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## APPENDICES

### APPENDIX A

TABLE DISPLAYING THE SEQUENCE OF RANDOM TREATMENT ALLOCATION

PATIENT NUMBER 1	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 2	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 3	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 4	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 5	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 6	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 7	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 8	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 9	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 10	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 11	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 12	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 13	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 14	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 15	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 16	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 17	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 18	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 19	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 20	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 21	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 22	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 23	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 24	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 25	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 26	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 27	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 28	<i>HEAT AND PASSIVE STRETCHING</i>
PATIENT NUMBER 29	<b>ICE AND PASSIVE STRETCHING</b>
PATIENT NUMBER 30	<i>HEAT AND PASSIVE STRETCHING</i>

APPENDIX B

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:  
Case history:

Signature:

Examination:

Previous: TN  
Other

Current: TN  
Other

X-ray studies:

Previous: TN  
Other

Current: TN  
Other

Clinical path. Lab.

Previous TN  
Other

Current: TN  
Other

Case status:

PTT: Conditional  
out:

Signed off:

Final sign

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 history :

General health status

Childhood illnesses

Adult illnesses

Psychiatric illnesses

Accidents and injuries

Surgery

Hospitalizations

6. Current health status and lifestyle :

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 drug

7. Family history :

Immediate family:

Age

Health

Causes of death

DM

Heart disease

TB

HBP

Stroke

Kidney disease

CA

Arthritis

Anemia

Headache

Thyroid disease

Epilepsy

Mental illness

Alcoholism

Drug addiction

Other

8. Psychosocial history:  
Home situation  
Daily life  
Important experiences  
Religious beliefs

9. Review of systems :

General

Skin

Head

Eyes

Ears

Nose/sinuses

Mouth/throat

Neck

Breasts

Respiratory

Cardiac

Gastro-intestinal

Urinary

Genital

Vascular

Musculoskeletal

Neurologic

Haematologic

Endocrine

Psychiatric

APPENDIX C

TECHNIKON NATAL CHIROPRACTIC CLINIC

PHYSICAL EXAMINATION

Underline abnormal findings in RED and elaborate on back of relevant page, if necessary.  
Mark "NAD" if normal.

Patient: \_\_\_\_\_ File # \_\_\_\_\_

                    Last name First name

Clinician: \_\_\_\_\_ Signature: \_\_\_\_\_

Intern: \_\_\_\_\_ Signature: \_\_\_\_\_

Date: \_\_\_\_\_

Height : \_\_\_\_\_ Weight: \_\_\_\_\_ Temp: \_\_\_\_\_

Rates:      Heart: \_\_\_\_\_ Pulse \_\_\_\_\_ Respiration: \_\_\_\_\_

Blood Pressure:      Arms:              L    /    R    /

                    Legs:              L    /    R    /

General appearance:



## STANDING EXAMINATION.

Minor's sign

Skin changes

Posture

Erect

Adam's

Ranges of motion:

T/L spine: Flexion: 90° (Fingers to the floor)

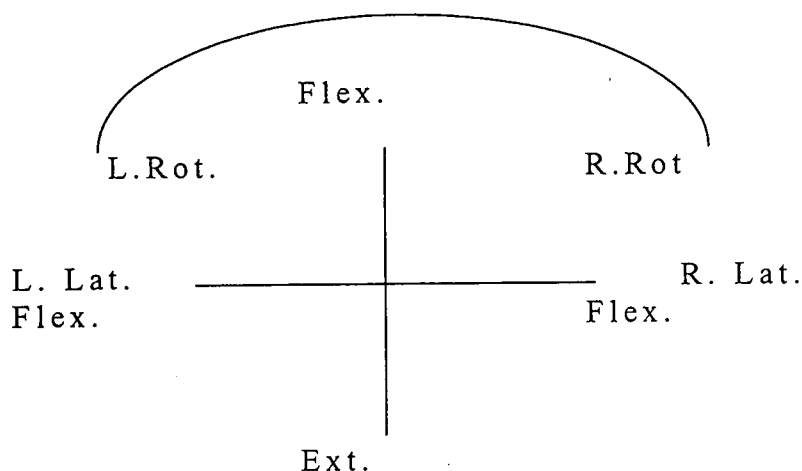
Extension: 50°

R.lat.flex: 30° (Fingers down leg)

L.lat.flex: 30° (Fingers down leg)

Rot. To R: 35°

Rot. To L: 35°



/ = pain-free limitation; // = painful limitation.

Romberg's sign.

Pronator drift.

Trendelenberg's sign.

Gait.

Rhythm

Balance

Pendulousness

On toes

On heels

Tandem

Half squat.

Scapular winging.

Muscle tone.

Spasticity/Rigidity.

Shoulder:

Skin

Symmetry

ROM - Glenohumeral  
Scapulo-thoracic  
Acromioclavicular  
Elbow  
Wrist

Chest measurement:

Inspiration

Expiration

Visual acuity

Breast examination:

Inspection:

Skin

Size

Nipple

Contour

Arms overhead

Hands against hips

Leaning forward

Palpation:

Axillary lymph nodes

SEATED EXAMINATION.

Spinal posture:

Head:

Scalp

Skull

Face

Skin

Eyes:

Conjunctiva

Sclera

Eyebrows

Lacrimal glands

Nasolacrimal duct

Alignment

Corneal reflex

Ocular movement

L  
III IV VI

R  
III IV VI

Visual fields

Accommodation

Iris

Pupils  
Red reflex  
Optic disc  
Vessels  
General background  
Macula  
Lens

Ears:

Auricle  
Ear canal  
Drum  
Auditory acuity  
Weber test  
Rinne test

Nose:

External  
Internal  
    Septum  
    Turbinates  
    Olfaction

Sinuses (frontal & maxillary):

Tenderness  
Transillumination

Mouth and pharynx:

Lips  
Buccal mucosa  
Gums and teeth  
Roof  
Tongue  
    Inspection  
    Movement  
    Taste  
    Palpation

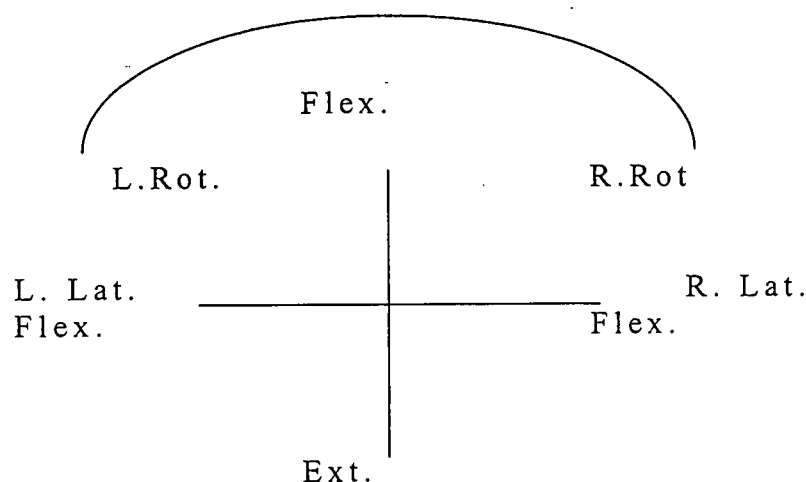
Pharynx  
    Inspection  
    CN X

Neck

Posture  
Size  
Swelling  
Scars  
Discoloration  
Hair line

ROM:

Flexion:	45°	chin to larynx
		Chin to sternum
Extension:	55°	forehead parallel to floor
L.lat.flex:	40°	
R.lat.flex:	40°	
L.Rot:	70°	
R.Rot:	70°	



Lymph nodes  
 Trachea  
 Thyroid  
 Carotid arteries (thrills, bruit)  
 CN V  
 CN VII  
 CNVIII (nystagmus)  
 CN IX  
 CN XI  
 TMJ

Inspection  
     ROM  
     Deviation  
 Palpation  
     Crepitus  
     Tenderness

Neurological:

Dermatomes

C5

C6

C7

C8

T1

Tendon reflexes

Biceps

Triceps

Brachioradialis

Muscle strength

C5

C6

C7

C8

T1

Co-ordination:

Point-to-point

Dysdiadochokinesia

Thorax:

Chest:

Inspection:

Skin

Shape

Respiratory distress

Rhythm (respiratory)

Depth

Effort

Intercostal/supraclavicular retraction

Palpation:

Tenderness

masses

respiratory expansion

tactile fremitus

Percussion

Lungs (posterior)

Diaphragmatic excursion

Kidney punch

Auscultation

Breath sounds

Vesicular

Bronchial

Adventitious sounds

Crackles (rales)

Wheezes (rhonchi)

Voice sounds

Broncophony

Whispered pectoriloquy  
egophony

Cardiovascular:

Auscultation (aortic murmurs)

Allen's test

#### SUPINE EXAMINATION

JVP

PMI

Auscultatory heart (l.lat.recumbent)

Respiratory excursion

Percussion chest (anterior)

Breast palpation

The abdomen:

Inspection:

Skin

Umbilicus

Contour

Peristalsis

Pulsations

Hernias (umbilical/inscisional)

Auscultation:

Bowel sounds

Bruit

Percussion

General

Liver

Spleen

Palpation:

Superficial reflexes

Cough

Light

Rebound tenderness

Deep

Liver

Spleen

Kidneys

Aorta

Intra-/retro abdominal wall mass

Shifting dullness

Fluid wave

Acute abdomen:

Where pain began and now

Cough

Tenderness

- Guarding/ rigidity
- Rebound tenderness
- Rovsing's sign
- Psoas
- Obturator sign
- Cutaneous hyperaesthesia
- Rectal examination
- Murphy's sign

Male genitalia and hernias.

Inspection:

- Skin
- Prepuce
- Glans
- Meatus
- Nits/lice
- Scrotum
- Inguinal /femoral bulges

Palpation:

- Penis (tenderness/induration)
- Testes
- Epididymis
- Inguinal canal
- Femoral canal
- Cremasteric reflex

Auscultation:

- Scrotal mass

Peripheral vasculature:

Inspection:

- Skin
- Nail beds
- Pigmentation
- Hair loss

Palpation:

- Pulses- radial, brachial, femoral, popliteal, post. Tibial, dorsalis pedis
- Lymph nodes- epitrochlear, femoral (horizontal & vertical)
- Temperature (feet and legs)
- Manual compression test
- Retrograde filling (Trendelenburg) test
- Arterial insufficiency test

Musculoskeletal:

ROM

Hip	Flex	90° /120°
	Ext.	15°
	Abd	45°
	Add	30°
	Int. rot	40°
	Ext. rot	45°
Knee		
	Flex	130°
	Ext.	0° /15°
Ankle		
	Plantarflex	45°
	Dorsiflex	20°
	Inversion	30°
	Eversion	20°

Leg length

Neurological:

Dermatomes

L1  
L2  
L3  
L4  
L5  
S1

Muscle strength

Hip flexion  
Knee extension  
Ankle dorsiflexion  
Plantar flexion

Tendon reflexes

Patellar  
Achilles  
Plantar reflex

Rectal examination:

Inspection

Sacroccocygeal and perianal areas

Palpation

Sphincter tone  
Tenderness  
Induration  
Nodules  
Prostate  
Seminal vesicles



Mental status

Appearance and behavior:

- Level of consciousness
- Posture and motor behavior
- Dress, grooming, personal hygiene
- Facial expression
- Affect

Speech and language:

- Quantity
- Rate
- Volume
- Fluency
- Aphasia

Mood

- Thought processes (logical, relevant, organized)
- Orientation (time, place, person)
- Recent memory; remote memory
- New learning ability

Higher cognitive functions:

- Information and vocabulary (general and specialized knowledge)

Abstract thinking.

APPENDIX D  
TECHNIKON NATAL CHIROPRACTIC DAY CLINIC.  
REGIONAL EXAMINATION - CERVICAL  
EXAMINATION

PATIENT: \_\_\_\_\_  
FILE#: \_\_\_\_\_ DATE: \_\_\_\_\_  
INTERN/RESIDENT: \_\_\_\_\_  
SUPERVISING CLINICIAN: \_\_\_\_\_

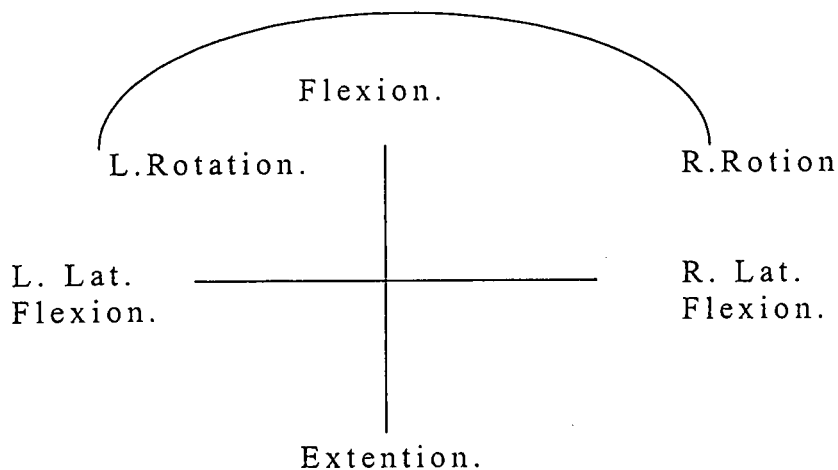
OBSERVATION:

Posture	Shoulder position:
Swellings	Left =
Scars	Right =
Discoloration	Muscle spasm
Hair line	Facial expression
Bony and soft tissue contours	

RANGE OF MOTION

Flexion =	45°
Extension =	70°
L/R rotation =	70°
L/R lateral flexion =	45°

Key: / painless limitation  
// painful limitation



Palpation :

Lymph nodes  
Trachea  
Thyroid gland

## ORTHOPAEDIC EXAMINATION

Tenderness

Active trigger points:

SCM

Scaleni

Levator scapulae

Posterior cervical musculature

Doorbell sign

Kemps sign

Halstead's test

Wright's(hyperabduction)test

Shoulder abduction test

Shoulder depression test

Dizziness rotation test

Brachial plexus tension test

Cervical compression

Lateral compression

Adson's test

Edens traction test

Lhermitte's test

Remarks: \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

## NEUROLOGICAL EXAMINATION:

<u>Dermatomes</u>			<u>Myotomes</u>			<u>Reflexes</u>		
Left		right	Left		Right	Left		right
C2			C1			C5		
C3			C2			C6		
C4			C3			C7		
C5			C4					
C6			C5					
C7			C6					
C8			C7					
T1			C8					
			T1					

<i>VASCULAR</i>	<i>LEFT</i>	<i>RIGHT</i>
BLOOD PRESSURE		
CAROTIDS		
SUBCLAVIAN ARTERIES		
WALLENBERG'S TEST		

COMMENTS: \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_  
 \_\_\_\_\_

MOTION PALPATION:

Jt. Play		Left						Right					Jt. Play	
P/A	Lat.	Fle	Ext.	LF	AR	PR		Fle	Ext.	LF	AR	PR	P/A	Lat.
							C0							
							C1							
							C2							
							C3							
							C4							
							C5							
							C6							
							C7							
							T1							
							T2							
							T3							
							T4							

APPENDIX E

**INFORMED CONSENT FORM**

(To be completed by the patient/subject) Delete whichever is not applicable.

TITLE OF THE RESEARCH PROJECT \_\_\_\_\_  
\_\_\_\_\_

NAME OF SUPERVISOR \_\_\_\_\_

NAME OF RESEARCH STUDENT \_\_\_\_\_  
\_\_\_\_\_

PLEASE CIRCLE THE APPROPRIATE ANSWER

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

PATIENT/SUBJECT NAME _____	Signature	_____
(In block letters)		
PATIENT/ GUARDIAN NAME _____	Signature	_____
(In block letters)		
WITNESS NAME _____	Signature	_____
(In block letters)		
RESEARCH STUDENT NAME _____	Signature	_____
(In block letters)		

**APPENDIX F**

PATIENT NAME \_\_\_\_\_

FILE NO. \_\_\_\_\_

DATE \_\_\_\_\_

MUSCLE	ALGOMETER READINGS						
TRAPEZIUS	TP1	TP2	TP3	TP4	TP5	TP6	TP7
TX 1:BEFORE							
TX 1: AFTER							
TX 5							
FOLLOW-UP							

MUSCLE	ALGOMETER READINGS	
LEVATOR SCAPULAE	TP1	TP2
TX 1: BEFORE		
TX 1: AFTER		
TX 5		
FOLLOW-UP		

MUSCLE	ALGOMETER READINGS		
INFRASPINATUS	TP1	TP2	TP3
TX 1 : BEFORE			
TX 1 : AFTER			
TX 5			
FOLLOW-UP			

## APPENDIX I

### CMCC NECK DISABILITY INDEX

PATIENT NAME: \_\_\_\_\_

FILE NO: \_\_\_\_\_

DATE \_\_\_\_\_

This questionnaire has been designed to give the doctor information as to show how your neck pain has affected your ability to manage in every day life. Please answer every section and tick only ONE bracket in each section that applies to you. We realize you may consider that two of the statements in any one section relate to you, but please mark just the box which most closely describes your problem.

<u>Section 1-Pain Intensity</u>	<u>Score (0-5)</u>
<input type="checkbox"/> I have no pain at the moment.	
<input type="checkbox"/> The pain is very mild at the moment.	
<input type="checkbox"/> The pain is moderate at the moment.	
<input type="checkbox"/> The pain is fairly severe at the moment.	
<input type="checkbox"/> The pain is very severe at the moment.	
<input type="checkbox"/> The pain is the worst imaginable at the moment.	
<u>Section 2 – Personal Care</u> (washing, dressing etc)	
<input type="checkbox"/> I can look after myself normally without causing extra pain.	
<input type="checkbox"/> I can look after myself but it causes extra pain.	
<input type="checkbox"/> It is painful to look after myself and I am slow and careful.	
<input type="checkbox"/> I need some help but manage most of my personal care.	
<input type="checkbox"/> I need help every day in most aspects of self-care.	
<input type="checkbox"/> I do not get dressed; I wash with difficulty and stay in bed.	
<u>Section 3 – Lifting</u>	
<input type="checkbox"/> I can lift heavy weights without extra pain.	
<input type="checkbox"/> I can lift heavy objects but it causes me extra pain.	
<input type="checkbox"/> Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned, for example on a table.	
<input type="checkbox"/> Pain prevents me from lifting heavy objects, but I can manage medium weights if they are conveniently positioned.	
<input type="checkbox"/> I can lift very light weights.	
<input type="checkbox"/> I cannot lift or carry anything at all.	
<u>Section 4: Reading</u>	
<input type="checkbox"/> I can read as much as I want to with no pain in my neck.	
<input type="checkbox"/> I can read as much as I like with slight pain in my neck.	
<input type="checkbox"/> I can read as much as I want with moderate pain in my neck.	
<input type="checkbox"/> I can't read as much as I want because of moderate pain in the neck.	
<input type="checkbox"/> I can hardly read at all because of severe pain in my neck.	
<input type="checkbox"/> I cannot read at all.	
<u>Section 5: Headaches</u>	
<input type="checkbox"/> I have no headaches at all.	
<input type="checkbox"/> I have slight headaches, which come infrequently.	
<input type="checkbox"/> I have moderate headaches, which come infrequently.	
<input type="checkbox"/> I have moderate headaches which come frequently	
<input type="checkbox"/> I have severe headaches, which come frequently.	
<input type="checkbox"/> I have headaches almost all the time.	

APPENDIX H

NUMERICAL PAIN RATING SCALE-101

PATIENT NAME: \_\_\_\_\_

FILE NO: \_\_\_\_\_

DATE: \_\_\_\_\_

Please indicate on the line below the point between 0 and 100 that best describes the pain you experience when the pain is at its WORST.

0 \_\_\_\_\_ 100

Please indicate on the line below the point between 0 and 100 that best describes the pain you experience when the pain is at its LEAST.

0 \_\_\_\_\_ 100



APPENDIX G

SHORT-FORM McGill PAIN QUESTIONNAIRE (SFMPQ)  
R.MELZACK

PATIENT NAME: \_\_\_\_\_

FILE NO: \_\_\_\_\_

DATE: \_\_\_\_\_

SECTION A: SENSORY DIMENSION OF PAIN

	NONE	MILD	MODERATE	SEVERE
THROBBING	0) ----	1) ----	2) ----	3) ----
SHOOTING	0) ----	1) ----	2) ----	3) ----
STABBING	0) ----	1) ----	2) ----	3) ----
SHARP	0) ----	1) ----	2) ----	3) ----
CRAMPING	0) ----	1) ----	2) ----	3) ----
GNAWING	0) ----	1) ----	2) ----	3) ----
HOT-BURNING	0) ----	1) ----	2) ----	3) ----
ACHING	0) ----	1) ----	2) ----	3) ----
HEAVY	0) ----	1) ----	2) ----	3) ----
TENDER	0) ----	1) ----	2) ----	3) ----
SPLITTING	0) ----	1) ----	2) ----	3) ----

SECTION B: AFFECTIVE DIMENSION OF PAIN

TIRING-EXHAUSTING  
SICKENING  
FEARFUL  
PUNISHING-CRUEL

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Section 6-Concentration

Score (0-5)

- ☐ I can concentrate fully when I want to with no difficulty.
- ☐ I can concentrate fully when I want to with some difficulty.
- ☐ I have a fair degree of difficulty in concentrating when I want to.
- ☐ I have a lot of difficulty concentrating when I want to.
- ☐ I have a great deal of difficulty concentrating when I want to.
- ☐ I cannot concentrate at all.

Section 7 -Work

- ☐ I can do as much work as I want to.
- ☐ I can only do my usual work, no more.
- ☐ I can do most of my usual work, but no more.
- ☐ I cannot do my usual work.
- ☐ I can hardly do any work at all.
- ☐ I cannot do any work at all.

Section 8- Driving

- ☐ I can drive my car without any neck pain.
- ☐ I can drive my car as long as I want to with slight pain in my neck.
- ☐ I can drive my car as long as I want to with moderate pain in my neck.
- ☐ I can't drive my car as long as I want to because of moderate pain in my neck.
- ☐ I can hardly drive at all because of severe pain in my neck.
- ☐ I can't drive my car at all.

Section 9- Sleeping

- ☐ I have no trouble sleeping.
- ☐ My sleep is slightly disturbed (less than 1hr sleepless).
- ☐ My sleep is mildly disturbed (1-2 hrs. sleepless).
- ☐ My sleep is moderately disturbed (2-3 hrs. sleepless).
- ☐ My sleep is greatly disturbed (3-5 hrs. sleepless).
- ☐ My sleep is completely disturbed (5-7 hrs. sleepless).

Section 10: Recreation

- ☐ I am able to engage in all my recreation activities with no neck pain.
- ☐ I am able to engage in all my recreation activities, with some neck pain.
- ☐ I am able to engage in most, but not all of my usual recreation activities because of pain in my neck.
- ☐ I am able to engage in few of my usual recreation activities because of pain in my neck.
- ☐ I can hardly do any recreation activities because of pain in my neck.
- ☐ I can't do any recreation activities at all.

**APPENDIX J**

PATIENT NAME \_\_\_\_\_

FILE NO. \_\_\_\_\_

DATE \_\_\_\_\_

<b>APPOINTMENT</b>	<b>CERVICAL GONIOMETER READINGS</b>					
	<b><i>FLEX</i></b>	<b><i>EXT</i></b>	<b><i>L.LAT/FL.</i></b>	<b><i>R.LAT/FL.</i></b>	<b><i>L. ROT.</i></b>	<b><i>R. ROT</i></b>
TX 1: BEFORE						
TX1: AFTER						
TX 5						
FOLLOW-UP						