Chiropractic management of Fibromyalgia Syndrome

A mini dissertation submitted to the

Faculty of Health sciences, Technikon Witwatersrand, Johannesburg
in partial fulfillment of the requirements for
the degree of Master in Technology
in the program Chiropractic by

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Date: 30/4/2004

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Date: 30/4/2004
DECLARATION

I, Werner Ferreira, declare that this dissertation is my own, unaided work. It is being submitted for a Master's Degree in Technology: Chiropractic at the Technikon Witwatersrand, Johannesburg. It has not been submitted before for any degree or examination at any other Technikon or University.

Werner Ferreira

April, 2004
ABSTRACT

The purpose of this study was to determine whether the combination of chiropractic care and adjunctive methods (including ultrasound and ischaemic compression) would reduce pain levels in fibromyalgia patients.

This was a simple randomised study and twenty four candidates, out of thirty recruited, participated in it. These candidates were divided into a control group and an experimental group. The experimental group consisted of thirteen candidates while the control group consisted of eleven candidates. The candidates in the control group were treated using only adjunctive methods, which included ultrasound and ischaemic compression. The candidates in the experimental group were treated using the above mentioned adjunctive methods in combination with spinal adjustments or mobilisations. Each candidate in both groups was treated twice a week over a five-week period. Thus, there were a total of ten visits per candidate. Each candidate received treatment every second day in order to allow the body adequate time to recover and detoxify between sessions.

On the first visit a full case history (Appendix F), lumbar spine regional (Appendix G), cervical spine regional (Appendix H) and physical examination (Appendix I) was completed for every candidate.

Each candidate was required to complete specific questionnaires at specific times during the course of the study. The candidate completed a Visual Analogue Pain Scale (Appendix C) at the beginning of each treatment, Neck Disability Index (Appendix D) and Oswestry Low Back Pain Questionnaire (Appendix E) at the beginning of treatment one, five and ten. As far as tender points were concerned, algometer readings were taken, using these points, on the first, fifth and tenth visit, respectively. All questionnaires and the algometer readings were completed before any treatment was administered.
The results indicated a decrease in reported pain levels and percentage disability expressed as a percentage) was achieved in the experimental group that received chiropractic adjustments and adjunctive methods (ultrasound and ischaemic compression). The results for the control group – that received adjunctive methods alone – also indicate a decrease in reported pain levels and percentage disability. These results, however, were found to be not statistically significant.

Thus in conclusion, it can be noted that chiropractic care in conjunction with adjunctive methods including ultrasound and ischaemic compression may be beneficial in the management of Fibromyalgia Syndrome patients, specifically in improving their reported pain levels and percentage disability.
DEDICATION

• To my Heavenly Father for bringing me where I am today.

• To my parents, Leon and Linda for giving me the opportunity to enhance my education, for standing by me and for supporting me all the way.

• To my darling wife, Karin, for being my pillar of strength through the years. I love you always.

• To my mentor and special friend, Dr. Johan Kotze. Thank you for opening my eyes to the beauty of the Chiropractic profession. Thank you for your ongoing support and commitment to me and most of all, thank you for believing in this project and me.

• A special thanks to Rustelle and Ruane Kotze for all your love and support. I love you guys!

• To Dr. B Losco for all your guidance and the long hours devoted to this project. I appreciate it.

• To the Middelburg practice and staff for your ongoing support.

• Finally a special thank you to the 24 candidates who participated and finished the therapy program. Without you this project would not have been a success.
ACKNOWLEDGEMENTS

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Thank you for fulfilling the task of supervisor and for all your advice and support.

Dr. B. Losco
Thank you for fulfilling the task of promoter and for all your advice and support.

Mr. N. de Villiers
Thank you for your input regarding the statistical aspect of this study.

To all the candidates who participated in the study:
Thank you for your willingness, patience and time.
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CHAPTER ONE

INTRODUCTION

Fibromyalgia Syndrome was first described by William Balfour, a surgeon at the University of Edinburgh, in the early eighteen hundreds. For many years it was known by different names, including chronic rheumatism, myalgia and fibrositis. For a while some physicians thought that the condition’s origin was primarily psychological. Fibromyalgia Syndrome is not a disease that has a known cause and well-understood mechanisms for producing symptoms. It is however, incorrect and a disservice to the patient, to label all soft tissue chronic pain conditions “Fibromyalgia”. Fibromyalgia is a specific, chronic nondegenerative, nonprogressive, noninflammatory, truly systematic pain condition (Starlanyl and Copeland, 2001:7-8).

1.1. PROBLEM STATEMENT

Although the American Medical Association (AMA) recognised Fibromyalgia Syndrome as a true physical illness in 1987, most physicians today still lack the training to diagnose and treat it. What the medical community now calls “Fibromyalgia” is not well categorised into specific subsets in the rigorous manner that other conditions such as diabetes or multiple sclerosis are classified and studied (Starlanyl and Copeland, 2001:7). Although not a crippling disorder, Fibromyalgia Syndrome does cause widespread disability, primarily due to misdiagnosis and inappropriate therapy. There are numerous theories about the etiological, pathophysiology and treatment of this common ailment. To add to the confusion even more, there is not even consensus on the criteria used to establish a diagnosis of Fibromyalgia Syndrome (Gatterman, 1990: 317).
1.2. **HYPOTHESIS**

The combination of chiropractic care in conjunction with adjunctive methods (to include ultrasound and ischaemic compression) could significantly reduce symptomatology in Fibromyalgia patients, especially pain levels, thereby improving their quality of life.

1.3. **PURPOSE OF THE STUDY**

The purpose of this study was to determine whether the combination of chiropractic care and adjunctive methods (including ultrasound and ischaemic compression) would reduce pain levels in Fibromyalgia patients.

1.4. **IMPORTANCE OF THE PROBLEM**

Chiropractic management addresses both peripheral mechanisms of pain transmission and cognitive or central mechanisms of pain control (endogenous pain modulating systems), which is crucial in treating chronic pain syndromes. Spinal manipulation affects the muscle spindle apparatus (through stimulation of mechanoreceptors, enhancing spinal gating mechanisms) and thereby reduces pain, increases range of motion and reduces myofascial hypertonicity (Blunt, Rajwani and Guerriero, 1997:394). The approach to Fibromyalgia Syndrome remains multidisciplinary, and therefore chiropractic care, as an effective and cost effective discipline, has an important role to play in the management thereof.
CHAPTER 2

LITERATURE REVIEW

2.1. INTRODUCTION

According to Bennett (1995:269-275), Fibromyalgia Syndrome (FMS) is the commonest cause of widespread pain, yet it may remain undiagnosed for a long time. The number of people suffering from FMS is unknown, but uncertainty and frequent misdiagnosis cause considerable havoc in the lives of many patients. The word "Fibromyalgia" is derived from the Greek "Algia", meaning pain, and "Myo" indicating muscle and the Latin "Fibro", meaning the connective tissue of tendons and ligaments. The word "syndrome" means a group of signs and symptoms that occur together and that characterise a particular abnormality. Fibromyalgia is not a new disease, but has been called by different names, including "chronic rheumatism" and "fibrositis". FMS was recognised by the American Medical Association (AMA) as a true illness and major cause of disability in 1987. However, most physicians today still lack the skills to diagnose and treat it effectively. FMS, although not curable at present, is very treatable with numerous treatments aimed at improving the health and quality of life of patients (Starlanyl and Copeland, 2001:5).

2.2. DEFINITION OF FIBROMYALGIA SYNDROME:

Fibromyalgia, or Fibromyalgia Syndrome, is characterised by widespread chronic musculoskeletal aching, pain, and stiffness with associated tenderness on palpation at multiple sites, referred to as tender points (Rachlin and Rachlin, 1994:3).
What it is:
- Fibromyalgia is a distinct clinical syndrome deserving of informed medical care and continued research to better understand chronic widespread pain (Russel, 1999:445-454).
- Fibromyalgia can be a source of substantial disability (Kaplan, et. al., 2000:785-789).

What it isn’t:
- Fibromyalgia is not a musculoskeletal disorder (Simms, 1998:346-350).
- Fibromyalgia is not progressive (Wolfe, et. al., 1997:1571-1579).
- Fibromyalgia is not the same as chronic myofascial pain (Gerwin, 1999:209-215).
- Fibromyalgia is not a homogenous condition (Starlanyl and Copeland, 2001:8).
- It is not the same as Chronic Fatigue Immune Deficiency Syndrome, but may be part of the same category of central nervous dysfunctions (Starlanyl and Copeland, 2001:8).
- Fibromyalgia is not an infectious condition (Starlanyl and Copeland, 2001:9).
- Fibromyalgia must not be categorised as a mental disorder (Starlanyl and Copeland, 2001:9).
- Fibromyalgia is not an autoimmune condition (Wittrup, et. al., 1999:273-277).

2.3. MYOFASCIA: WHAT IT IS, WHAT IT DOES AND WHAT YOU NEED TO KNOW:

Myofascial pain is the most common cause of musculoskeletal pain in medical practice (Bennett, 1995:269-275).

2.3.1 The importance of myofascia:

According to Greenman (Starlanyl, 2001:18), fascia can be separated into three layers; (1) the superficial fascia, (2) the deep fascia and (3) the sub serous fascia.

The superficial fascia is attached to the underside of the skin. Capillary channels, lymph vessels, as well as many nerves run through this layer. Healthy superficial fascia allows
fluent movement of the skin over the surface of the muscles. In FMS and Chronic Myofascial Pain, the superficial fascia is often stuck.

The deep fascia is a much tougher and denser material. Deep fascia is responsible for separating large sections e.g. the abdominal cavity. Deep fascia separates muscles from organs. The pericardium and pleura are all composed of specialised deep fascia.

Sub serous fascia, is the third layer of fascia. This loose tissue covers the internal organs and holds the network of blood and lymph vessels that keeps them moist. Sub serous myofascia therefore surrounds the blood vessels, lymph vessels and nerves. Pressure changes due to compression by tightening myofascia, can affect the cells that lie within the blood and lymph vessels and nerves.

The dural tube, which surrounds and protects the spinal cord and contains cerebrospinal fluid (CSF), is another form of fascia.

2.3.2 Ground substance:

The myofascia contains a material called ground substance that can change its form from liquid to solid and back again. In healthy individuals, the ground substance has a gelatinous consistency responsible for absorbing the forces generated by movement. It also functions as a shock absorber during trauma. When the ground substance changes from a liquid to a gel, and then into its more solid form, the myofascia tightens. The myofascia won’t reverse to its previously more liquid state without outside intervention. Important functions of the ground substance includes the transfer of nutrients from the parts of the body where they are broken down into usable materials, to the areas in the body where they will be used, as well as the removal of waste products from those areas of use. The ground substance is also responsible for maintaining the distance between connective tissue fibres, which prevents micro-adhesions from forming and keeps the tissue supple and elastic. If this critical distance is not maintained, the fibres become cross-linked by newly synthesised collagen cross-links, which, unlike healthy
linkages, are arranged haphazardly and are difficult to break apart. Once the ground substance has hardened it must be returned to its healthy, more fluid state and therefore, it isn't enough for the therapist to just break up the cross-linkages (Starlanyl and Copeland, 2001:19-20).

2.3.3 Muscles and metabolic wastes:

Cell products as well as wastes resulting from cellular processing must pass through the ground substance to reach the lymph and later be processed for removal. When the myofascia is "blocked" and stuck together informational substances or biochemical messengers can't function properly. Local nerve endings become irritated when waste and chemical toxins back up in the tightened muscle. These irritated nerves tell the brain to activate its arousal system to inform the body that something is going wrong. The ground substance becomes more solid because the fight and flight response is activated when the body mobilises to rid itself of irritation (Starlanyl and Copeland, 2001:20).

Fibrous myofascial adhesions can form anywhere along nerves and block healthy normal function. Myofascial trains develop as the fascia twists, turns and tightens, due to the stressors of life. These will restrict movement (Starlanyl and Copeland, 2001:20).

2.3.4 Trauma and immobilisation:

Relative immobility after a trauma results in the formation of micro-adhesions. These adhesions become progressively more fibrotic with increased time of immobilisation (Starlanyl and Copeland, 2001:21).

2.4. CLASSIFICATION OF FIBROMYALGIA SYNDROME:

According to Rachlin and Rachlin (1994:3), Fibromyalgia Syndrome may be classified as primary and concomitant. When a significant underlying or concomitant condition that
may contribute to pain is absent, the term primary fibromyalgia is used. When another condition such as rheumatoid arthritis (RA), osteoarthritis or hypothyroidism contributes to the pain or fatigue of fibromyalgia, it may be classified as concomitant. The term secondary fibromyalgia should not be used for concomitant fibromyalgia because satisfactory treatment of a concurrent disease such as RA or hypothyroidism does not significantly ameliorate fibromyalgia features e.g. pain, fatigue, and number of tender points.

The American College of Rheumatology (Wolf et. al., 1990:160-172), suggests the following criteria for the classification of fibromyalgia syndrome:

1) A history of widespread pain: Pain is considered widespread when all of the following are present: pain on the left and right side of the body; pain above and below the waists. Axial skeletal pain (cervical spine, anterior chest, thoracic spine, or lower back) must also be present. For the purpose of this definition, shoulder and buttock pain is considered as pain for each involved side, while low-back pain is considered lower segment pain.

2) Pain in eleven of eighteen tender points on digital palpation, performed with an approximate force of 4 Kg. For a tender point to be considered “positive” the subject must state that the palpation was painful and not just “tender”. Using spring gauge algometry can also assess tender points. Using the algometer, 4kg/cm² pressure is applied to each site. A site is considered tender when a pressure of less than 4kg/cm² induces an uncomfortable sensation, with the patient responding to the examiner that pain was experienced (Cox, 1999:252).
FIGURE 2.1 Location of nine bilateral tender point sites (Baldry, 2001:354).
According to Baldry (2001:355), the eighteen tender point sites include:

- **Occiput**: Bilateral, at the suboccipital muscle insertions.
- **Low cervical**: Bilateral, at the anterior aspects of the intertransverse spaces at C5-C7.
- **Trapezius**: Bilateral, at the midpoint of the upper border.
- **Supraspinatus**: Bilateral, at origins, above the scapula spine near the medial border.
- **Second rib**: Bilateral, at the second costochondral junctions, just lateral to the junctions on upper surfaces.
- **Lateral epicondyle**: Bilateral, 2 cm distal to the epicondyles.
- **Gluteal**: Bilateral, in upper quadrants of buttocks in anterior fold of muscle.
- **Greater trochanter**: Bilateral, posterior to the trochanteric prominence.
- **Knee**: Bilateral, at the medial fat pad proximal to the joint line.

For classification purposes, patients will be said to have fibromyalgia if both criteria (1) and (2) are satisfied. Widespread pain must have been present for at least 3 months. The presence of a second clinical disorder does not exclude the diagnosis of fibromyalgia (Baldry, 2001:355).

A definite diagnosis of FMS, according to the American College of Rheumatology criteria, should only be made when no other medical disease can explain the symptoms (Wolf *et al.*, 1990:162-172). Schneider and Brady (2001:530-531), identified large numbers of patients diagnosed with FMS who never had simple blood tests performed to rule out conditions such as anaemia or hypothyroidism. This inability to adequately examine and perform laboratory tests may lead to a falsely high rate of FMS diagnosis. Therefore, they proposed a new classification system outlined in Figure 2.2.
2.5. EPIDEMIOLOGY AND CLINICAL FEATURES OF FMS

2.5.1. Epidemiology:

FMS occurs most commonly among women in the 40- to 60-year-old age group; 85% to 90% of the patients being women (Bengtsson, et. al., 1986:340-347). Fibromyalgia has however, also been described among juveniles (Roizenblatt, et. al., 1997:579-585) and the elderly (Yunus, et. al., 1988:987-995).
2.5.2. Clinical features:

Symptoms:

According to Rachlin and Rachlin (1994:4), the most common and distinguishing symptoms of FMS are generalised pain, stiffness, fatigue and poor sleep.

Pain is the most common presenting symptom of FMS. The pain is usually present in all four limbs, as well as the upper or lower back. Very rarely will patients present with predominantly or exclusively one-sided pain. Although these patients may not fulfil the current American College of Rheumatology criteria for FMS, they should be diagnosed as having the disorder and treated as such if features of this illness e.g. fatigue, poor sleep, multiple tender points and paraesthesia are present. Pain is described as aches, soreness, hurting or burning. The “hurt all over” feeling described by two thirds of patients, has been found to be useful in differentiating FMS from other conditions (Wolfe, et. al., 1990:160-172). Common sites for pain or stiffness include the lower back, neck, shoulder regions, arms, hands, knees, hips, thighs, legs and feet (Yunus, et. al., 1989:62-71). The pain or stiffness is often aggravated by cold or humid weather, anxiety or stress, overuse or inactivity, poor sleep and noise (Prince, et. al., 2000:35-47).

Stiffness, normally worse in the morning and the evening, is present in most (85%) but not all patients.

Fatigue is common in FMS, with moderate to severe fatigue occurring in about 75% to 90% of patients (Wolfe, et. al., 1990:160-172). According to Rachlin and Rachlin (1994:6), it may be described as exhaustion, tiredness, lack of energy, fatigue and sometimes as a global feeling of general weakness. Very often fatigue may be the presenting feature in some patients. Fatigue, like pain, seems to be primarily of central origin similar to Chronic Fatigue Syndrome. Fatigue may be the result of poor sleep,
excessive physical activity, and physical deconditioning as well as psychological factors, pain, global severity and functional disability (Yunus, et. al., 2000:485-490).

Nonrestorative sleep is common in FMS (Goldenberg, 1987:2782-2787), with 75% of patients describing sleeping difficulties. Poor sleep may be indicated by difficulty in falling asleep, frequent awakening, light sleep and morning fatigue. Factors including restless leg syndrome and periodic limb movement disorder may contribute to disturbed sleep. Poor sleep may aggravate pain and may contribute to its pathophysiologic mechanisms (Yunus, et. al., 2000:485-490).

A swollen feeling and paraesthesia, predominantly present in the extremities, is present in half the patients. The mechanisms of paraesthesia and subjective swelling are unknown (Yunus, et. al., 1989:62-71). Other associated symptoms of FMS include: Headaches, Dysmenorrhoea, Irritable Bowel Syndrome, Restless Leg Syndrome, Sicca Symptoms, Raynauds' Phenomenon and Female Urethral Syndrome (Rachlin and Rachlin, 1994:5)

<table>
<thead>
<tr>
<th>Positive findings</th>
<th>Negative findings</th>
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<td>- Multiple tender points</td>
<td>- Absent joint swelling</td>
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<td>- Skin fold tenderness</td>
<td>- Normal range of motion of the joints</td>
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<tr>
<td>- Cutaneous hyperaemia</td>
<td>- Normal muscle strength</td>
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<td>- Reticular skin discoloration</td>
<td>- Normal sensory functions</td>
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<tr>
<td>- Diffuse puffiness of fingers (rare)</td>
<td>- Normal reflexes</td>
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TABLE 2.1. Physical signs of FMS (Rachlin and Rachlin, 1994:8)
**Laboratory tests in FMS**

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<tr>
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<td>- Sleep EEG studies</td>
<td>- Complete blood count</td>
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<td>- Neuroendocrine tests</td>
<td>- Erythrocyte sedimentation rate</td>
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<td>- Muscle enzymes</td>
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<td>- Thyroid function test</td>
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<td></td>
<td>- Rheumatoid factor</td>
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<td>- Antinuclear antibodies</td>
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<td>- Radiograph; bone scan</td>
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<td>- Electromyography</td>
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<td>- Muscle biopsy</td>
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**TABLE 2.2. Laboratory tests in FMS (Rachlin and Rachlin, 1994:9)**

Anti-nuclear antibodies (ANA) are present in about 10% of patients, similar to the frequency found in a healthy, normal control group with similar distribution of age, sex and race. Multiphase skeletal scintigraphy and electromyography were found to be normal in FMS (Bengtsson, et. al., 1989:1466-1469).

*Differential diagnosis and coexisting conditions:*

Myofascial pain syndrome: to be discussed in another section of this literature review.

Chronic Fatigue Syndrome: Most patients with FMS show increased amounts of substance P in their CSF. Levels of substance P in the cerebrospinal fluid of 15 patients with Chronic Fatigue Immune Deficiency Syndrome were within normal range (Starlanyi and Copeland, 2001:8).

Other conditions which may coexist or need to be differentiated from FMS include: Depression or anxiety, multiple bursitis or tendinitis, endocrine myopathies, occult
malignancies, connective tissue diseases, Rheumatoid arthritis, Systemic Lupus Erythmatosus, polymyalgia rheumatica and polymyositis.

2.6. BIOPATHOPHYSIOLOGIC MECHANISMS

It is clear that FMS is not a psychiatric disorder, but is based on neurochemical abnormalities that are generally different from those reported in psychiatric illnesses such as depression. FMS has a multifactorial aetiology, of which neuroendocrine aberration, particular central sensitisation or sensitivity, seems most important. Several other factors interact with these aberrations and further amplify pain; they include nonrestorative sleep, trauma, infection or inflammation, psychological distress, genetics, physical deconditioning and environmental factors, such as weather and noise (Yunus, et. al., 2001:351-377).

![Diagram showing the proposed model for biopathophysiologic mechanisms of FMS](image)

**FIGURE 2.3** A schematic representation of the proposed model for biopathophysiologic mechanisms of FMS (Rachlin and Rachlin, 1994:12).
2.6.1. The concept of central sensitisation:

Central sensitisation is defined as an exaggerated response of the central nervous system (CNS) to a peripheral stimulus that is normally painful or non-nociceptive, such as touch (allodynia), denoting hyperexcitability and hypersensitivity of the CNS neurons. Another characteristic of central sensitisation is the prolongation or persistence of pain.

A better understanding of central sensitisation has evolved in recent years through animal experiments (Dubner, et. al., 1992:96-103). A nociceptive stimulus, such as inflammation or direct nerve injury at the periphery, sends a bombardment of afferent impulses to the spinal cord dorsal horn via the C fibres, resulting in remarkable chemical, synaptic, molecular, and in some cases, anatomic changes in the neurons, both at the spinal and supraspinal levels. An increased synaptic efficacy allows “cross-talk” between neurons, modulating their response. Such a “cross-talk” between nociceptive and non-nociceptive neurons may explain the phenomenon of allodynia in FMS (Woolf, et. al., 1983:686-688).

Following a nociceptive stimulation at the periphery, substance P is released at the synapse in the dorsal horn and removes the magnesium block of N-methyl-D-aspartate (NMDA), allowing excitatory amino acids (EMM), e.g. Glutamate and Aspartate, to activate postsynaptic NMDA receptors. Such activation now permits several intramembranous and intracellular changes, e.g. alteration of cell membrane permeability, influx of calcium, excitation of secondary neurons and expression of c-fos (Bondy, et. al., 1999:433-439).

Wind-up phenomenon, as described in animal models, is defined by a progressively increased response of the secondary neurons following repeated and brief stimulus of the C fibres at the periphery, so that with each successive stimulus the response of these neurons increases and is stronger than the previous one. Wind-up is mediated by NMDA receptors inhibited by NMDA receptor antagonist (Woolf, et. al., 1991:293-299). In fact, NMDA may play an important role in the pathogenesis of FMS, considering that
ketamine, an NMDA receptor antagonist, has been shown in placebo-controlled studies to reduce muscular pain as well as temporal stimulation (Graven-Nielsen, et. al., 2000:483-491).

Although animal studies have clearly demonstrated a state of hyperexcitability in the CNS following a peripheral stimulus of inflammatory nature (central sensitisation), leading to central sensitivity, such source of peripheral nociception is not always obvious in FMS. Although a peripheral trauma (such as an automobile accident), inflammation (e.g. Rheumatoid arthritis), a degenerative disease of the joints, or possibly the mechanical stress of poor posture may provide a peripheral source of nociceptive stimulus in FMS, such a concept needs to be proven by appropriate studies (Wolfe, et. al., 1984:814-818).

2.6.2. Autonomic dysfunction:

Autonomic disturbance has long been suspected in FMS (Yunus, 1984:21-28). According to Rachlin and Rachlin (1994:16) the symptoms of FMS are too complex to be explained by an abnormality of a single system even if this system interacts with others. Although an autonomic dysfunction may contribute to some symptoms of FMS, these symptoms ensue from a host of complex interactions among many other factors, such as neurohormonal, environmental, psychological, and genetic, which may vary among several subgroups (Qiao, et. al., 1991:1383-1389).

2.6.3. Neurotransmitter or neurochemical dysfunctions:

Pain is transmitted by A-delta and C fibres; C fibres utilise substance P (SP) as an important neurotransmitter for pain transmission, although other neurotransmitters as well as excitatory amino acids are also involved (Besson, 1999:1610-1615). The neurotransmitters involved in pain inhibition include serotonin, norepinephrine, \( \gamma \)-aminobutyric acid (GABA), ekephalin, and other less studied neurochemicals (Coderre, et. al., 1993:259-285). An increased activity of the excitatory neurotransmitter SP or a
deficiency of the inhibitory ones such as serotonin may, therefore, cause amplified pain, as in FMS.

A significantly increased level of SP in the cerebrospinal fluid of FMS patients has been consistently demonstrated as compared with normal controls in several studies (Vaeroy, et. al., 1988:21-26). There was no correlation between cerebrospinal fluid SP and depression (Russell, et. al., 1994:1593-1601). A related finding is the fact that nerve growth factor, which is known to promote the growth of SP, has also been reported to be elevated in the cerebrospinal fluid (Gioveno, et. al., 1994:1564-1569).

Low levels of plasma or serum tryptophan (a precursor of serotonin) suggest serotonin deficiency in FMS, a decreased transportation of plasma tryptophan (an indicator of the brain entry of tryptophan), decreased serum serotonin, and a decreased level of cerebrospinal fluid 5-hydroxyindoleacetic acid (5HIAA, a metabolite of serotonin). Urinary 5HIAA levels have also been reported to be low in FMS (Kang, et. al., 1998:14-21). The role of peripheral serotonin in the pathogenesis of FMS was supported by two recent studies, recognising the algogenic property of serotonin at the peripheral tissues (Emberg, et. al., 1999:313-325; Emberg, et. al., 2000:31-39).

Significantly decreased levels of 3-methoxy-4-hydroxyphenethyene glycol and homovanillie acid in the cerebrospinal fluid of FMS patients, as compared with normal, pain-free controls, have also been reported (Ferraccioli, et. al., 1994:1332-1334).

2.6.4 Metabolic and endocrine dysfunction:

Several controlled studies have demonstrated hypothalamic-pituitary-adrenal (HPA) axis abnormalities in FMS, which include decreased 24-hour urinary free cortisol (Griep, et. al., 1998:1374-1381), loss of diurnal cortisol fluctuation and elevated evening cortisol levels (Croftord, et. al., 1994:1583-1592), and an exaggerated adrenocorticotropic hormone (ACTH) response to corticotropin-releasing hormone (CRH) with similar cortisol level between the FMS patients and controls (Griep, et. al., 1998:1374-1381), suggesting a relative hypocortisolaemia to elevated ACTH.
Growth hormone (GH) status has been found to be low in FMS in several studies. Insulin-like growth factor (IGF-1), which represents and integrated section of GH, is decreased as is the directly measured GH (Bennett, et. al., 1997:1384-1389).

The table below summarises the endocrine findings associated with FMS.

- Decreased 24-hour urine free cortisol
- Loss of normal diurnal fluctuations of cortisol
- Exaggerated ACTH response to both corticotropin releasing hormone and hypoglycaemia
- Low total basal plasma cortisol with normal basal free cortisol
- Delayed ACTH release after interleukin-6 administration
- Decreased response of thyroid hormones and thyroid stimulating hormone (TSH) to thyroid releasing hormone (TRH)
- Decreased insulin-like growth factor-1 (IGF-1)
- Decreased GH
- GH response to stimulation studies: both normal and increased in several studies using different agents for stimulation
- Normal nocturnal secretion of melatonin in 2 of 3 studies

TABLE 2.3 Endocrine findings in FMS (Rachlin and Rachlin, 1994:11).

2.6.5 Perpetuating and aggravating factors:

Perpetuating factors, according to Starlanyl and Copeland (2001:51), include those conditions or stressor responsible for causing a myofascial trigger point to remain in place, in spite of efforts to break it up. They may be behavioural, such as posture, mechanical, e.g. poorly fitting shoes, or biochemical, such as nutritional inadequacy. Some of these perpetuating factors are also aggravating and initiating factor.
Perpetuating factors:

Paradoxical breathing - This is one of the most common perpetuating factors of both FMS and chronic myofascial pain (CMP). This type of breathing occurs when the belly flattens during inhalation and expands during exhalation. It is important to monitor breathing throughout the day.

Adhesions - "Adhesions" means materials stuck together due to e.g. surgery, infection, endometriosis, etc. Some types of bodywork can be very effective in breaking up these adhesions regardless of their cause (Starlanyl and Copeland, 2001:52).

Environmental factors:

Starlanyl and Copeland (2001:53) have identified the following environmental factors:

1. Pollution
2. Allergic conditions, e.g. asthma and hay fever
3. Sensory changes, e.g. barometric and temperature pressure fluctuations, humidity and dampness.

Other perpetuating factors include:

- Ill-fitting, poorly designed furniture;
- Prolonged sitting;
- Immobility due to e.g. casts, prolonged bed rest, etc.
- Foot structure;
- Morton's neuroma
- Inappropriate care
- Infections and infestations, e.g. viral, bacterial, yeast or protozoal illness
- Lifestyle choices
  - Poor posture
  - Muscle abuse
  - Repetitive motion
  - Smoking and drinking alcohol
- Mechanical factors
  - Head forward posture
  - Body asymmetry
  - Short extremities
  - Ill-fitting shoes and socks
- Metabolic factors, e.g. vitamin and mineral inadequacy, insulin resistance, etc.
- Overwork
- Sexual or physical abuse
- Reactivate hypoglycaemia and insulin resistance
- Trauma
  - Whiplash
  - Traffic accidents
  - Surgery

**2.7 FLARE:**

Flare according to Dohrenbusch, *et al.*, (1997:334-341), refers to any overwhelming episode of symptom intensity that can develop gradually over time, or suddenly without warning.

Usually one or more activities or stressors trigger flares. Stressors could include a virus, a severe yeast infection, or a traffic accident. Other stressors include a major argument, a menstrual period, visitors, holidays, shift work, a very loud noise, an upper respiratory tract infection or allergic attack. Regardless of the type of stressor responsible for flare, it is very important that the patient identifies his or her common stressors and learns how to manage these stressors (Starlanyl and Copeland, 2001:153-154).

**2.8 FIBROFOG:**

Cognitive deficit may be one of the most frustrating and sometimes disabling symptoms of FMS (Starlanyl and Copeland; 2001:199).
Some FMS patients become very confused in large malls or even small crowds. This is due to the fact that they can’t process all the sensory input. It is believed that some of this is due to the brain having a higher metabolic rate than other tissues and, with FMS, there is insufficient delivery of oxygen, glucose and other substances that the brain cells need to function. When brain cells are thinking, their metabolic rate and the flow of blood to those cells increase. Some fog may be caused by inadequate thyroid hormone regulation of these processes (Lowe; 2000).

Some researchers have found that FMS causes slowed psychomotor speed for tasks that require sustained effort (Landro, et. al., 1997:297-306). People with fibrofog seem to sense things as patterns and series of patterns. Trying to establish a new pattern can cause a short circuit for example: You can reach for the light switch on the wall by the door, but it won’t be there. That’s because the light switch was in that location in an apartment you had thirty years ago. (Starlanyl and Copeland; 2001:200).

2.8.1 Fog formation

SPECT (Single Photon Emission Tomography) scans show that people with FMS have decreased blood flow in the right caudate nucleus of the brain as well as in the left and right thalami (Mountz, et. al.,1998:385-396). This decreased blood flow could be caused by neurotransmitter dysfunction, or by a problem in the glial cells.

Gliial cells are active in brain cell permeability (Heinemann, et.al.,2000:S185-S189). This permeability is important in allowing molecules like electrical ions to move in and out of the cells, thus affecting how well the cell functions. According to Silver, et.al.(1997:589-601), there is a bioelectric connection between glia, ion exchange, and cellular swelling in the brain. If too much fluid is allowed into the cell due to increased permeability, cellular contents come under increased pressure. It is believed that some fibrofog may be due to this form of water retention.
Other causes of fibrofog may include excessive histamine as well as excessive bodywork, i.e. massage, physiotherapy, chiropractic care, etc. (Starlanyl and Copeland; 2001:202).

2.9 PSYCHOLOGICAL FACTORS OF FMS:

Psychological factors are an important determinant of any pain - particularly chronic pain - irrespective of the cause, and FMS is no exception. Psychological distress perpetuates pain in a vicious circle. Chronic pain may cause psychological disturbance, which in turn may aggravate pain (Rachlin and Rachlin, 1994:19).

It is now clear that psychiatric diseases are not necessary for development of FMS. A good number of studies (but not all) have shown that the frequencies of psychiatric diseases, e.g. anxiety and depression, in FMS are similar to other chronic diseases, e.g. Rheumatoid arthritis (Ahles, et. al., 1991:1721-1726; Ahles, et. al., 1987:105-111; Clark, et. al., 1985:132-137; and Kirmayer, et. al., 1988:950-954).

Several studies utilised validated questionnaires for psychological symptoms and stress and found no difference in anxiety and depression between FMS patients and controls (Ahles, et. al., 1987:105-111; Clark, et. al., 1985:132-137; and Dailey, et. al., 1990:1380-1385). Uveges and colleagues (Uveges, et. al., 1990:1279-1283) found greater psychological distress, including depression, among FMS patients as compared to Rheumatoid arthritis patients, but daily stress was a significant covariate.

Virtually all studies have shown a greater degree of lifetime or daily stress in FMS as compared with normal controls as well as Rheumatoid arthritis (Ahles, et. al., 1984:1101-1106; Dailey, et. al., 1990:1380-1385; and Uveges, et. al., 1990:1279-1283). It seems that stress is an important factor in FMS, although a relationship between symptoms and stress has not been adequately addressed.
It has been suggested that FMS is a spectrum of depressive illnesses (Hudson, et. al., 1985:441-446; Hudson, et. al., 1989:15-22; and Hudson, et. al., 1990:552-564). Depression and FMS, however, are different diseases, based on psychological, biophysiological, and therapeutic studies (Yunus, et. al., 2001:351-377). As discussed earlier, the occurrence of psychiatric diagnoses in FMS is not greater than in other chronic diseases, e.g. Rheumatoid arthritis in a majority of studies. The HPA axis functions and sleep electro-encephalogram (EEG) findings are different in FMS than in depression (Lentjes, et. al., 1997:603-614). For example, the HPA axis is hyperactive in depression, with a blunted response of ACTH to CRH (Amsterdam, et. al., 1987:775-781). Also, 24-hour urinary cortisol was found to be significantly increased in depression, but not in FMS (Maes, et. al., 1998:328-335). The HPA functions in FMS are opposite of what has been found in depression. Moreover, the glucocorticoid receptors in the lymphocytes are normal in FMS (Lentjes, et. al., 1997:603-614), in contrast to low values found in depression (Whalley, et. al., 1986:859-861). Additionally, a fairly recent study found that patients with depression have significantly fewer tender points than those with FMS (Fassbender, et. al., 1997:76-79).

2.10 FIBROMYALGIA AND CHRONIC MYOFASCIAL PAIN:

Due to the fact that FMS and chronic myofascial pain (CMP) both share muscle pain as a symptom, many people with bilateral or widespread pain have been diagnosed with FMS, when in actual fact the pain is from CMP or some other source (Starlany and Copeland, 2001:32).

In the literature regarding FMS there are constant references to myofascial pain as a regional syndrome in contrast to FMS as the widespread syndrome. This is a particularly dangerous concept in chronic pain where myofascial pain is more likely to be generalised (Gerwin, 1999:209-215).

Individuals may have FMS and CMP as well as joint dysfunction and other perpetuating factors. Each condition requires separate attention and therefore it is important for
medical team members to understand the complex overlapping pain patterns existing in chronic pain patients (Starlanyl and Copeland, 2001:31).

Making a diagnosis of FMS and CMP can be complicated, because the symptoms of each person can vary in many ways. FMS is a chronic disease that can be controlled. CMP is a condition that is potentially curable, unless there is a fixed, uncorrectable underlying cause. The focus of treatment for both conditions is to restore more normal functioning with minimised pain (Gerwin, 1998:175-181).

2.10.1 The four causes of muscle pain:

Hans Kraus (Rachlin and Rachlin, 1994:205) described four causes of muscle pain: (1) trigger points, (2) muscle spasm, (3) muscle tension and (4) muscle deficiency.

(1) Muscle spasm: Muscle spasm refers to the involuntary contraction of muscle caused by chronic or acute trauma, excessive tension or organic disease (Skubick, 1998:12-15).

(2) Muscle tension: Kraus (Rachlin and Rachlin, 1994:206) defined muscle tension as "prolonged contraction of a muscle or muscle groups beyond functional or postural need".

(3) Muscle deficiency: When muscles are weak or stiff, they are considered deficient. Muscle deficiencies make one prone to injury and can themselves be a source of pain (Rachlin and Rachlin, 1994:206).

(4) Trigger points - Definition and types: According to Rachlin and Rachlin (1994:206), myofascial trigger points (TrPs) are small, circumscribed, hyperirritable foci in muscles and fascia, often found within a firm or taut band of skeletal muscle. Trigger points may also occur in ligaments, tendons, joint capsules, skin and periosteum (Rachlin and Rachlin, 1994:206). Hopwood (1994:227-237), classically defines a trigger point as the presence of discrete focal tenderness located in a palpable taut band of skeletal muscle, which produces both referred regional pain (zone of reference) and a local twitch response. A local twitch response is defined as a
transient, visible or palpable contraction or dimpling of the muscle and skin as the taut band of the trigger point contracts when pressure is applied. Trigger points help define myofascial pain syndrome.

The following table illustrates the different types of trigger points.

<table>
<thead>
<tr>
<th>ACTIVE</th>
<th>LATENT</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic</td>
<td>Asymptomatic</td>
</tr>
<tr>
<td>Tender and painful</td>
<td>May cause muscle shortening and weakness</td>
</tr>
<tr>
<td>Referred pain patterns</td>
<td>Tender</td>
</tr>
<tr>
<td>Local twitch response</td>
<td>Local twitch response</td>
</tr>
<tr>
<td>Autonomic phenomenon</td>
<td></td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Primary</th>
<th>Secondary</th>
<th>Satellite</th>
</tr>
</thead>
<tbody>
<tr>
<td>Develop independently and not as the result of trigger point activity elsewhere</td>
<td>Develop in neighbouring and antagonistic muscles as the result of stress and muscle spasm</td>
<td>Develop in the area of referred pain as the result of persistent resting motor unit activity</td>
</tr>
</tbody>
</table>

TABLE 2.4 Types of trigger points (Rachlin and Rachlin, 1994:207)

2.10.2 Tender points:

Tender points, according to Hopwood (1994:227-234) are associated with pain at the site of palpation only, are not associated with referred pain, and occur in the insertion zone of muscles, not in taut bands in the muscle belly. Patients with FMS have tender points by definition. Concomitantly, patients may also have trigger points with myofascial pain syndrome. Thus, these two pain syndromes may overlap in symptoms and be difficult to differentiate without a thorough examination by a skilled physician (Alvarez and Rockwell, 2002:1).

2.10.3 Trigger points vs. Tender points

The table below illustrates the differences between trigger and tender points.
<table>
<thead>
<tr>
<th>Trigger Points</th>
<th>Tender Points</th>
</tr>
</thead>
<tbody>
<tr>
<td>Local tenderness, taut band, local twitch response, jump sign</td>
<td>Local tenderness</td>
</tr>
<tr>
<td>Singular or multiple</td>
<td>Multiple</td>
</tr>
<tr>
<td>May occur in any skeletal muscle</td>
<td>Occur in specific locations that are symmetrically located</td>
</tr>
<tr>
<td>May cause a specific referred pain pattern</td>
<td>Do not cause referred pain, but often cause a total body increase in pain sensitivity</td>
</tr>
</tbody>
</table>

**TABLE 2.5 Trigger Points vs. Tender Points (Alvarez and Rockwell, 2002:2)**

### 2.10.4 Comparison of myofascial pain and fibromyalgia syndrome:

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Myofascial pain syndrome</th>
<th>Primary fibromyalgia syndrome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tenderness</td>
<td>Trigger points: belly and insertion</td>
<td>Multiple tender points</td>
</tr>
<tr>
<td>Pain</td>
<td>Referred pain</td>
<td>Generalised aching</td>
</tr>
<tr>
<td>Duration</td>
<td>Muscle specific - if untreated becomes chronic</td>
<td>Chronic - more than 3 months</td>
</tr>
<tr>
<td>Sex</td>
<td>Equal number of males and females</td>
<td>80% female</td>
</tr>
<tr>
<td>Prevalence</td>
<td>Common, 50% male and female</td>
<td>Uncommon, 4% primary, 11% secondary</td>
</tr>
<tr>
<td>Disturbed sleep pattern</td>
<td>Common secondary to discomfort due to position</td>
<td>Sleep disorder by definition greater than 80%</td>
</tr>
<tr>
<td>Treatment</td>
<td>Local muscle massage ice/heat, ultrasound, stretching exercises, ischaemic compression, spray and stretch, nutritional support.</td>
<td>Systemic light aerobic exercise, rest, decreased stress, psychologic support, nutritional support.</td>
</tr>
</tbody>
</table>

**TABLE 2.6 Comparison of Myofascial pain syndrome and Fibromyalgia syndrome (Gatterman, 1990:286).**
Other practical differences between CMP and FMS according to Starlnayl and Copeland (2001:32-33) includes the following:

- Restricted motion is not a part of FMS; Generalised fatigue is, but not the specific muscle weakness that is caused by trigger points.
- With CMP there is no pain; The areas of the muscle do not have trigger points or their referred patterns, unless FMS or something else is causing generalised pain.
- If the individual has FMS, no hard lumps, bumps and ropy bands will be found in the muscles. Those are part of the trigger points that characterise CMP.
- Generalised hypersensitivity to pain and/or allodynia (feeling pain from non-painful stimuli) is characteristic of FMS. If an individual has both symptoms, both conditions may be present.

2.11. MANAGEMENT OF FMS

Management of FMS is most effective when it is tailored to the individual patient. Principles of management according to Rachlin and Rachlin (1994:33), include the following:

2.11.1 Positive and empathetic attitude of the physician.
2.11.2 Firm diagnosis: According to Goldenberg (1999:777-785), most patients with FMS have had symptoms for 5-7 years before their consultation with a physician. Granges and associates (1994:523-529), suggest that earlier diagnosis and treatment may lead to better outcomes.
2.11.3 Diagnosis and management of comorbid conditions: Failure to recognise and treat concurrent disorders such as RA, Osteoarthritis, and spinal stenosis appropriately, may cause poor treatment outcomes (Creamer; 1999:622-637).
2.11.4 Patient education and reassurance: Patients should be reassured that although the disorder is a very painful one, it does not cause tissue damage, reduced life expectancy, deformity or cripling (Bennett; 1997:517-518).
2.11.5 Addressing aggravating factors.
2.11.6 Individualisation of management.
2.11.7 Improvement of sleep quality.
2.11.8 Gradual increase of physical activities.
2.11.9 Pharmacologic treatment:

Antidepressant agents:

Because of the fact that serotonin modulates both sleep and pain, serotonergic antidepressants have been evaluated in the management of FMS (Moldofsky; 1976:35-44). Most of these drugs increase the time spent in stage 4 sleep (Wilke; 1995:247-260). These drugs are widely used to provide analgesia in FMS and other chronic pain conditions (Godfrey; 1996:1047-1052).

Tricyclic antidepressants:

According to Leventhal (1999:850-858), amitriptyline is the most widely prescribed drug or pharmacologic agent for the treatment of FMS. Goldenberg and colleagues (1986:1371-1377) reported significant improvement in pain, sleep difficulties, morning fatigue, global assessment and tender point score in groups receiving amitriptyline. According to Creamer (1999:622-637), the recommended starting dose of 10mg at bedtime may be gradually increased to 50-70mg/day in 10mg increments on a weekly basis, if there is no response to the initial dosage and side effects are tolerable.

Another tricyclic antidepressant, cyclobenzaprine, has a similar chemical structure to amitriptyline (Barnes; 1980:221-224).

Selective Serotonin Re-uptake Inhibitors (SSRI):

Fluoxetine, a SSRI, has been evaluated in FMS with case reports reporting improvements in sleep disturbances and depression. Only one study found a positive effect on pain relief or TP score (Goldenberg; 1996:1852-1859).

Other antidepressant agents:
Other antidepressant agents include: trazadone (Wilke; 1996:247-260) as well as venlafaxine (Dryson; 2000:87).

- Hypnotics: Zopiclone and zolpidem, in double blind, placebo-controlled studies, were found to be helpful in improvement of subjective sleep complaints and daytime energy, but not effective in pain relief (Moldofsky, et al., 1996:529-533).

- Anti-inflammatory agents: Despite the fact that there is no evidence of tissue inflammation in FMS, 91% of FMS patients use anti-inflammatory agents (Wolfe, et al., 1997:1560-1570). FMS patients do not get considerable benefit from NSAID’S (Simms; 1994:917-934). Although NSAID’S may help concomitant arthritis or dysmenorrhea, gastrointestinal, renal or hepatic side effects should be considered before prescribing a NSAID in FMS (Rachlin and Rachlin; 1994:42).

- Analgesics: Paracetamol (Acetaminophen) is used frequently for pain control in FMS despite the fact that there is no controlled trial available regarding it’s efficacy (Rachlin and Rachlin; 1994:43).

Clinical experience have shown that a small dose of codeine,(15-30mg/day), is well tolerated on a long term basis, but it should only be used occasionally, such as during a flare-up (Yunus; 1996:1279-1285).

Tender Point Injections: A valuable adjunctive therapy in the management of FMS involves injections in the Tender Points with a mixture of 1% lidocaine and triamcinolone diacetate (Reddy, et al., 2000:7-18). Average duration of pain relief per injected site was 13 weeks. According to Rachlin and Rachlin (1994:44), following the injections, local ice should be applied for several hours, followed by 24-48 hours rest of the injected areas to minimise postinjection flare.
2.11.10 Nonpharmacologic interventions:

Nonpharmacologic interventions/treatments are important in FMS in addition to drug therapy (Berman and Swyers; 1999:487-492). Nonpharmacologic interventions include the following:

*Physical fitness training*

*Physical therapy* including stretching, strengthening, massage, mobilisation and physical energy modalities such as heat, ice, ultrasound, electrical stimulation and electromagnetic energy (Rachlin and Rachlin; 1994:48).

- **Ultrasound treatment of FMS patients:** In most FMS patients, myofascial trigger points respond well to ultrasound. This is especially true when ultrasound is used with stripping massage, moist heat and spinal adjusting (Simons and Simons; 1999). The main difference when using ultrasound in FMS patients, is that these patients may have to be treated with very low ultrasound intensity; perhaps fewer than 0,1 watts/cm². Constant feedback from the patient is essential (Lowe; 1995:7).

No other modalities appear to have the same set of soft tissue effects that ultrasound has (Lowe; 1989). The effects of ultrasound include the following:

- Ultrasound energy is converted into heat at tissue surface boundaries due to waves meeting intra-and intercellular resistance. The greatest effects of treatment occur where two unlike structures interface.
- The fine vibrations from the ultrasound exert a micromassage effect on the treated tissues.
- Ultrasound can alter the structure of scar tissue by breaking down the collagen fibrils with specific action on interstitial cement and by disengaging collagen cross bindings.
• Ultrasound has several chemical effects in tissues such as stimulating streaming of calcium ions from cells, increasing gaseous exchange and oxidation as well as liquefying some cellular gels.
• Ultrasound waves can induce the absorption of exudates and precipitates and reduce oedema.
• Ultrasound can inhibit impulse conduction in type C nerve fibres.
• Ultrasound waves may result in microdestruction of tissue deposits such as calcified haematomas and osseous proliferations.
• According to Jaskoviak and Schafer (1986), ultrasound waves may trigger enkephalin production, producing a mild sedative effect.

Ultrasound should preferably be administered in the continuous setting. The treatment protocol for Myofascial trigger points includes positioning the patient comfortably so that a passive stretch of the muscle to be treated is obtained. Fibromyalgia patients who experience discomfort from maintaining certain positions should be allowed to reposition themselves to remain as comfortable as possible (Lowe and Honeyman-Lowe; 1999:12-15).

During treatment, the ultrasound head, should be moved continually to prevent excess heat from accumulating on the face of the ultrasound head and in the skin. The ultrasound head should be moved slowly during treatment, about 1,25 to 2,5cm/second. Depth of penetration of the sound waves is approximately 5cm. When treating trigger points the usual intensity setting is 1,0 to 1,5watts/cm² (Jaskoviak and Schafer; 1986).

The intensity should always be adjusted to patient comfort (Hong,et.al., 1993:37-53).
Manual medicine techniques:

- Chiropractic manipulative therapy:

  - The chiropractic hypothesis: According to Gatterman, (1990:52), chiropractic therapy is based on the hypothesis that reversible joint lesions of the spine produce far-ranging effects on the human body. Chiropractors rely on spinal manipulation or adjustments as their primary therapeutic tool in reversing the subluxation complex. Modern chiropractic adheres to the idea that biomechanical dysfunction can have a profound effect, not only on the musculoskeletal system, but also on all other systems of the body. While the restoration and normalisation of joint function is the mechanism of chiropractic therapy, the ultimate goal is to promote homeostasis of the body.

  - The Chiropractic spinal adjustment/manipulation: Gatterman, (1990:405), defines the spinal adjustment as a specific form of direct articular manipulation utilising either long or short leverage techniques with specific contacts, and is characterised by a dynamic thrust of controlled velocity, direction and amplitude.

- The clinical anatomy of chiropractic adjusting according to Cramer (1995:17-51)

  Structures primarily affected during spinal adjusting:

  2. Interbody joints and discs.
  3. Spinal muscles

  Mechanisms of action of spinal adjusting:

  1. Movement of Menisci.
  2. Break-up of Adhesions.
  3. Stimulation of Mechanoreceptors within the Zygapophysial Joint Capsule and Related Reflex Responses.
4. Reflex Changes (including autonomic changes) secondary to stimulation of mechanoreceptors of the vertebral column and related ligaments and muscles.

5. Changes in intradiscal pressure.

6. Thoracolumbar Fascia and Spinal muscles.

1. Movement of Menisci:

Mooney and Robertson (1976:149-156) stated that manipulation might achieve its therapeutic effect by relieving the chronic reaction of the articular capsule and/or its lining synovial membrane to trauma. Such conditions would include catching of a synovial fold between the joint capsule and articular process and what some have described as the interaction of Zygopophyseal joint menisci.

Kos (1969:1088-1105) described the typical Z-joint meniscus as being attached to the capsule by loose connective tissue. More central to this attachment he identified synovial tissue and blood vessels, and the most central part of the meniscus was composed of dense connective tissue. Kos and Wolf (1972a:203-218, 1972b:8-9) felt that such menisci could become trapped within the Z-joint, resulting in traction forces being placed upon the pain sensitive articular capsule. They stated that a spinal adjustment has the effect of gapping the joint, thus allowing the meniscus to return to its normal position.

2. Break-up of Adhesions:

One of the most commonly described mechanisms of action of spinal adjusting is the alleviation of intra-articular adhesions that develop following hypomobility of the Z-joints (Janse, 1976:55; Triano, 1992:348). Mooney and Robertson (1976:149-156) believed these adhesions developed during the degenerative phase of progressive back pain. Several investigators have reported that spinal manipulation separates the articular surfaces of the Z-joints (Kos and Wolf, 1972a:203-218, 1972b:8-9; Cassidy and Kirkaldy-Willis, 1992:283-296). This “gapping” is the action that is thought to break up
adhesions. Elimination of adhesions allows the Z-joints to move, thus helping the motion segment (two adjacent vertebrae and the ligamentous structures connecting them) to re-establish a more normal state (Mooney and Robertson, 1976:149-156).

3. Stimulation of Mechanoreceptors within the Zygapophysial Joint Capsule and Reflex Responses:

Wyke (1985) states that there are three types of sensory receptors in the joint capsule of the Z-joints. Type I are very sensitive static and dynamic mechanoreceptors that fire continually to some extent even when the joint is not moving. Type II are less sensitive and fire only during movement and Type IV is slow conducting nociceptors. Wyke (1985) stated that Types I and II have a pain suppressive effect. He also stated that there is a "reflexogenic effect" initiated by Type I and II fibers that cause normalization of muscle activity on both sides of the vertebral column at the level of manipulation and several levels above and below the site of manipulation.

4. Thoracolumbar Fascia and Spinal muscles:

Some highly respected investigators (Peck et al, 1986:590-598) have proposed that a compartment type of syndrome may develop within the region located between the superficial and deep layers of the thoracolumbar fascia. Manipulation, soft tissue massage, and manual techniques to the lumbar spine may have a milking effect on the lumbar musculature within these facial layers. The deep back muscles, made up of the erector spinae and transversospinales groups, are frequently affected in lower back pain. Stretching of these structures during an adjustment or other forms of mechanical treatment, may help to alleviate increased muscle tension frequently identified with these muscles.

According to Haldeman,(1992:557-572), the following conditions are contra-indicated for spinal manipulative therapy:
- Inflammation and Infection
- Rheumatoid Arthritis and instability or acute inflammation.
- Ankylosing Spondylitis.
- Degeneration
- Degenerative Joint Disease (DJD).
- Neoplasm.
- Intoxication.
- Metabolic disturbances.
- Congenital malformations.
- Trauma.
- Psychogenic disturbances.

- The chiropractic fixation/subluxation complex: Two authoritative contemporary definitions of subluxation are given in the Foundation of Chiropractic Education and Monograph (1997):
  i. A motion segment in which alignment, movement integrity, and/or physiologic function is altered, although contact between the joint surfaces remains intact.
  ii. A complex of functional and/or structural and/or pathological changes that compromise neural integrity and may influence organ systems function and general health. A subluxation is evaluated, diagnosed and managed through the use of chiropractic procedures based on the best available rational and empirical evidence.

According to Gatterman, (1990:415), a subluxation is defined as the aberrant relationship between two articular structures, which may have functional or pathological sequelae, causing an alteration in the biomechanical and/or neurophysiological reflexes of these articular structures, their proximal structures, and/or body systems which may be directly or indirectly affected by them.

Gatterman, (1990:39), describes the pathophysiological components of the subluxation complex as encompassing the following two elements:
• Neuropathophysiology — subluxation causes irritation and/or compression of the neural components of the motion segment.

• Kinesiopathology — restriction of movement of the motion segments due to muscle hypertonicity, joint stabilisation, muscle spindle spasm cycle, joint sprain, muscle spasm and joint locking.

These pathophysiological elements may be corrected by manipulation.

Gatterman, (1990:622), defines a fixation as a dynamic fault, whereby an articulation has become temporarily immobilised in a position that it may normally occupy during any phase of physiological movement. It is the immobilisation of an articulation in a position of movement when the joint is at rest, or in a position of rest when the joint is moving through it’s range of motion.

• Chiropractic management of FMS: According to Pioro-Boisset, et.al.,1996:13-17, chiropractors are the most frequently consulted complementary medicine practitioners reported by patients with FMS. However, there are only two studies that report the efficacy of chiropractic management in FMS. An open-label study conducted by Hains and Hains (2000:228), evaluated the efficacy of chiropractic treatment that included ischaemic compression to tender points and spinal manipulation in 15 patients with FMS. Patients received a total of 30 treatments, 2-3 times weekly. Pain intensity, fatigue and sleep quality were measured by visual analogue scales at baseline, after 15 and 30 treatments, and one month after the end of the trail. The results of the study suggested that chiropractic care combining ischaemic compression and spinal manipulation, may help patients with FMS. A total of 60% of this sample reported a mean improvement of 77.1% in pain intensity in addition to a 63.5% improvement in the quality of sleep and a 74.8% improvement in fatigue level. This study suggests a potential role for chiropractic care in the management of fibromyalgia. Most subjects appear to have responded favourably to a course of 30 Chiropractic treatments including spinal manipulation and ischaemic compression therapy. Fifteen treatments seem to be an adequate cut-off point to
determine if a significant improvement in pain has occurred and if further care is warranted. Chiropractic care appears to provide benefits for at least 1 month after stopping therapy.

Blunt and associates, (1997:389-399), applied chiropractic management consisting of soft tissue massage, stretching, spinal manipulation, and patient education, which was administered 3-5 times a week for 4 weeks to 21 patients. At the end of the program, in the chiropractic group, clinical improvements were observed in the cervical and lumbar ranges of motion, straight leg raising, and self-reported pain severity. However, the no standardised treatment protocol and inadequate outcome measurements limit the conclusion of this study.

According to Blunt, et. al., (1997:390) the rationale for the use of chiropractic manipulation in the management of FMS includes the following:

- Inhibition of pain: Articular capsules of the spinal facet joints are densely populated with mechanoreceptors; thus increased proprioceptive input in the form of spinal mobility, tends to decrease the central transmission of pain from adjacent spinal structures.
- Relaxation of paraspinal muscles: manipulation will cause stretching of the apophyseal joint capsules, reflexly inhibiting facilitated motor neuron pools responsible for increased muscle tone.
- Breaking of articular adhesions: In chronic cases, there is a shortening of periarticular connective tissue, and intra-articular adhesions may form. Manipulation may stretch or break these adhesions.
- Increased range of motion: In acute and chronic spinal pain, joint movement is restricted and can be relieved by manipulation.

In summary, it seems reasonable to suggest that chiropractic management should be included in the treatment of the fibromyalgia patient. A short course of chiropractic treatment (4-8 weeks) may offer the fibromyalgia patient some pain relief, increased
range of motion in their cervical and lumbar spines and improvement in their overall level of flexibility. It is fair to say, then, that chiropractic management seems to improve function and not just pain, even in the chronic pain (fibromyalgia) patients. However, chiropractic care (solely) does not seem to offer a "break-through" or is not a panacea for the treatment of fibromyalgia. It is reasonable to offer the fibromyalgia patient a short course of comprehensive chiropractic care initially as an important component of their multidisciplinary treatment approach. The manual therapy (passive treatment) offered by chiropractors should facilitate the fibromyalgia patient’s progression into a functional restoration program. The fibromyalgia patient is commonly pain-focused and self-limiting in their behaviour. It is important for chiropractors to realize this and attempt to defocus them from their pain and attempt to improve their function. The fibromyalgia patient should be encouraged to reintegrate himself or her into a more productive lifestyle. They should also be encouraged to become more independent in their pain management. However, supportive chiropractic care may have a role in the long-term management of the patient (Blunt, et. al. 1997:396)

Other manual medicine techniques include: Electromyographic (EMG)-Biofeedback, acupuncture, hypnotherapy and Cognitive Behavioural Therapy (Rachlin and Rachlin; 1994:49)

2.12. Prognosis:

The prognosis is good when onset of symptoms is clearly defined, history of symptoms is short (less than one year) and the patient is young, with mild symptoms early in the illness. Factors related to poor prognosis include a long history of symptoms and patient inability to take responsibility for his or her well being (Nies, 1992:20-26).
CHAPTER THREE

METHODOLOGY

3.1.  INTRODUCTION

This chapter serves to describe and explain the way in which this project was constructed and carried out.

3.2.  STUDY DESIGN AND SELECTION CRITERIA

The study was conducted at the TWR Chiropractic Day Clinic and at the practice of Dr. Johan Kotze. Candidates for the study were recruited from Johannesburg and surrounding suburbs, and later in the study, from Middelburg and surrounding towns in Mpumalanga province. Apart from advertisements (Appendix B) in local newspapers, Fibromyalgia support groups in the above mentioned areas were also asked to participate in the study. The candidates had to be older than eighteen years, and had to be prediagnosed with Fibromyalgia Syndrome. Each candidate functioned as their own control to determine whether any change in pain occurred after each treatment.

Each candidate was required to sign a consent form (Appendix A), detailing that they had been fully informed about the treatment process and that they were willing to allow Chiropractic spinal adjustments or mobilisations as well as adjunctive methods, including ultrasound and ischaemic compression to be performed. They were also informed that they may withdraw from the study at any time should they so wish, and that all treatments were free of charge.

3.3.  TREATMENT PROTOCOL

Thirty candidates were recruited to participate in the study, but only twenty-four completed the study. The candidates were randomly assigned to one of the two groups
(a control and experimental group). The experimental group consisted of thirteen candidates, and the control group consisted of eleven candidates. The candidates in the control group were treated using only adjunctive methods, which included ultrasound and ischaemic compression. The candidates in the experimental group were treated using the above mentioned adjunctive methods in combination with spinal adjustments or mobilisations. Each candidate in both groups was treated twice a week over a five-week period. Thus, there were a total of ten visits per candidate. Each candidate received treatment every second day in order to allow the body adequate time to recover between sessions. Ultrasound was administered using the continuous setting. The treatment protocol involved positioning the patient comfortably, so that a slight passive stretch of the muscle to be treated, could be achieved. During treatment with continuous ultrasound, the ultrasound head should be moved continually to prevent excess heat from accumulating on the face of the ultrasound head and in the skin. The ultrasound head should be moved slowly during treatment, about 1.25-2.5 cm/second. The maximum depth of penetration of the sound waves is approximately 5 centimetres. When treating trigger points the usual intensity setting is 1.0-to-1.5 watts/cm² (Jaskowiak, Schafer, 1986). The size of the area to be treated is approximately 5 cm² in diameter. Attempting to treat too large an area will diminish the effectiveness of the treatment. The ultrasound should be applied to the trigger point for 4-5 minutes using a circular motion with the ultrasound head. The head should be moved at a speed of 1.25-to-2.5 cm/second. The intensity should always be adjusted to patient comfort. A warm, soothing effect for the patient is optimal. By the time the patient feels the soothing effect, the trigger point is usually desensitized. Following the ultrasound, the clinician should apply moist heat for 5-8 minutes followed by passive stretching of the muscle being treated. Finally the patient should be gently adjusted (Lowe and Honeyman-Lowe; 1999:12-15). The patients received full spine adjustments using diversified chiropractic techniques.

On the first visit a full case history (Appendix F), lumbar spine regional (Appendix G), cervical spine regional (Appendix H) and physical examination (Appendix I) was completed for every candidate.
Each candidate was required to complete specific questionnaires at specific times during the course of the study. The candidate completed a Visual Analogue Pain Scale (Appendix C) at the beginning of each treatment, Neck Disability Index (Appendix D) and Oswestry Low Back Pain Questionnaire (Appendix E) at the beginning of treatment one, five and ten. As far as tender points were concerned, algometer readings were taken, using these points, on the first, fifth and tenth visit, respectively. All questionnaires and the algometer readings were completed before any treatment was administered.

3.4. DATA COLLECTION AND INTERPRETATION

3.4.1. Subjective data collection

Candidates were required to complete questionnaires in order to collect subjective data i.e. Visual Analogue Pain Scale, Neck Disability Index, and the Oswestry Low Back Pain Questionnaire.

3.4.1.1. Visual Analogue Pain Scale

Pain is a subjective phenomenon and can only be measured by the patient who feels it. One method frequently used to express the severity of the pain is through the Visual Analogue Pain Scale. Huskinson (1982:768-769) has demonstrated the reliability and reproducibility of the Visual Analogue Pain Scale in evaluating the severity of chronic pain. The Visual Analogue Scale is a straight line, the ends of which are defined as the extreme limits of the sensation or response to be measured. One extreme of the line indicates no pain, the other extreme indicates severe pain. The patient is asked to place a “X” where they would rank their pain intensity.

The Visual Analogue Pain Scale provides the patient with a sensitive and reproducible method of expressing pain severity. Results have been shown to correlate with other
methods of measuring pain. This method is applicable to all patients regardless of language and can be used by children age five and older.

The demonstration of pain relief is an important measure of the outcome of treatment. The Visual Analogue Pain Scale is helpful in subjective demonstration of patient's overall response to treatment and can be utilised to demonstrate treatment outcomes (Conwell, 1991:102-103).

3.4.1.2. Oswestry Low Back Pain Questionnaire

The Oswestry Low Back Pain Questionnaire is a valid indicator of a patient's disability and avoids any interviewer bias by the questionnaire being administered by the patient.

The Oswestry Low Back Pain Questionnaire is divided into ten sections selected from a series of questions designed to assess limitations of various activities of daily living. The sections correlate closely with problems of patients with low back pain. Each section contains six statements. Each statement describes a greater degree of difficulty in that activity than the preceding statement. The statements are sentences that contain one idea and are simply worded. The patient marks the one statement in each section, which describes their limitations most accurately. Each section is scored on a scale of 0 - 5 with 5 representing the greatest disability. The six questions under each section are numbered 0 through 5 for the purposes of grading depending on which statement is answered whether the first statement, which is, graded 0 numerically in increasing to the last statement which is graded 5. The number for each section then is added with a total possible score of 50. Adding the numerical score, dividing this score by 50 and multiplying by 100 to get a percentage performs scoring. If one section is missed or is not felt to be applicable by the patient, the total number is divided by 5 less i.e. 45.
Interpretation of the disability score according to Conwell (1991:111-113)

- **0 to 20%: Minimal Disability:** This group can cope with most activities of daily living. Usually no treatment is indicated other than giving appropriate advice with regards to lifting, posture, physical fitness, diet, etc.

- **20 to 40%: Moderate Disability:** This group experiences more pain with sitting, lifting and standing. Travel and social life are more difficult and they may be off work. Personal care, sexual activity and sleeping are not grossly affected and the back condition can usually be managed by conservative treatment means.

- **40 to 60%: Severe Disability:** Pain remains the main problem in this group of patient’s but travel, personal care, social life, sexual activity and sleep are also affected. These patients require detailed investigation for possible psychological overlay.

- **60 to 80%: Crippled:** Back pain impinges on all aspects of these patient’s lives both at home and at work and positive intervention is required including surgical, nonsurgical and psychiatric consultation.

- **80 to 100%:** These patients are either bed bound or exaggerating their symptoms. This can be evaluated by careful observation of the patient during the medical examination. These patients require psychological/psychiatric consultation.

### 3.4.1.3. Neck Disability Index

This test is administered and graded similarly to the Oswestry Low Back Pain Questionnaire. There are ten sections to the questionnaire with each section having six statements. The patient is to mark one statement after each section, which most specifically describes their condition. The statements under each section are numbered zero to five. The numbers, which correlate to the statements, are added up giving the
total score. The first statement is graded zero, the second is graded one, and with the continuing statements being graded two, three, four and five. With ten sections and a total of five possible points for each section, the highest score could be 50. The examiner adds up the score, takes the score times two to get a percentage (Conwell, 1991:115).

The scoring which is suggestive of the patient's neck disability is as follows:

<table>
<thead>
<tr>
<th>Score</th>
<th>Disability</th>
<th>Percent disability</th>
</tr>
</thead>
<tbody>
<tr>
<td>0 – 4</td>
<td>No disability</td>
<td>0 – 8%</td>
</tr>
<tr>
<td>5 – 14</td>
<td>Mild disability</td>
<td>10 – 28%</td>
</tr>
<tr>
<td>15 – 24</td>
<td>Moderate disability</td>
<td>30 – 48%</td>
</tr>
<tr>
<td>25 – 34</td>
<td>Severe disability</td>
<td>50 – 68%</td>
</tr>
<tr>
<td>35 – 50</td>
<td>Complete disability</td>
<td>70 – 100%</td>
</tr>
</tbody>
</table>

3.4.2. Objective data collection

Objective data was collected using spring gauge algometry to assess tender points. Using the algometer, 4kg/cm² pressure is applied to each tender point site. A site is considered tender when a pressure of less than 4kg/cm² induces an uncomfortable sensation, with the patient responding to the examiner that pain was experienced (Cox, 1999:252). The algometer is useful for making a measurement of pain pressure threshold at a trigger point site so the initial tenderness can be compared to measurements following a therapeutic or experimental intervention. It is relatively objective, since the subject need not see the meter display, but the reading does depend on the subject's report of a subjective sensation. It is very useful for research studies and helpful in many clinical situations, but the user must be aware of three kinds of limitations when applying it to trigger points/tender points. First, the measurement, per se, indicates nothing about the source or cause of the tenderness being measured. The tenderness may be due to myofascial trigger points, to tender points of fibromyalgia, to bursitis, to severe spasm, etc. Therefore, by itself tenderness cannot
serve as a diagnostic criterion. The cause of the tenderness must be determined by other diagnostic observations. Second, the absolute value obtained at any one site can be strongly influenced by variations in the thickness and compliance of subcutaneous tissues from subject to subject and by inherent differences in the sensitivity of different muscles (Fischer, 1987:115-126). Third, the relatively high degree of skill required to use this instrument effectively, and the exquisite specificity of the location of the trigger point/ tender point being measured are generally underrated. The precise location of maximum tenderness of that trigger point/ tender point must first be established by palpation and with the subject’s cooperation. Since the tenderness of a nodule in a taught band is being measured, the foot plate must be centred over the point of maximum tenderness in the nodule, and pressure must be aimed precisely in the direction of maximum tenderness. The foot plate must remain in this position throughout the measurement. These difficulties can be at least partly ameliorated by averaging the lowest two of three readings if they are in reasonable agreement (Hong, et. al., 1996:61-79). Algometer measurements of the tender points were obtained considering the above mentioned guidelines, and recorded for statistical analysis. These measurements were taken at visit 1, 5, and 10 respectively.

3.5. STATISTICAL ANALYSIS

The data was analysed using the sample t-test and the Anova Test. The Anova test is a one-way analysis of variance to measure differences between two groups for measurements before and after treatment. This test was performed to ascertain statistical significance. The closer the value of P to zero, the less change of probability that the result was due to chance, therefore a P-value of less than 0.05 can be regarded as indicating a statistical significant difference between the experimental and control groups.
CHAPTER 4

RESEARCH RESULTS

4.1. The age and gender of all the participants

4.1.1. The average age of the experimental group

The age of the participants in the experimental group ranged from 24 to 62. The average age of the thirteen participants was 42.

![Bar chart showing the age distribution of patients 1-13.]

FIGURE 4.1. The age of the participants in the experimental group

4.1.2. The average age of the control group

The age of the participants in the control group ranged from 49 to 69. The average age of the eleven participants was 58.
4.1.3. Gender of the participants

In the experimental group there were 4 males and 9 females. The control group consisted of 3 males and 8 females.

4.2. Tender points

This section deals with the tender point readings of the participants (experimental and control group combined) that comprises the objective data collection:

The diagnosis of Fibromyalgia Syndrome, is the presence of pain in eleven of eighteen tender points. Tender points are assessed by using spring gauge algometry. For a tender point to be considered “positive” the subject must state that the palpation was painful and not just “tender”. A site is considered tender when a pressure of less than 4 Kg/cm² induces an uncomfortable sensation, with the participant responding to the examiner that pain was experienced (Cox, 1999:252).
4.2.1. Tender point: Suboccipital

FIGURE 4.3. Suboccipital tender point

4.2.2. Tender point: Anterior cervical

FIGURE 4.4. Anterior cervical tender point
4.2.3. Tender point: Trapezius

![Graph of Trapezius tenderness over visits]

FIGURE 4.5. Trapezius tender point

4.2.4. Tender point: Supraspinatus

![Graph of Supraspinatus tenderness over visits]

FIGURE 4.6. Supraspinatus tender point
4.2.5. Tender point: Second rib

![Graph showing algometer readings for the second rib.](image)

**FIGURE 4.7. Second rib tender point**

4.2.6. Tender point: Elbow

![Graph showing algometer readings for the elbow.](image)

**FIGURE 4.8. Elbow tender point**
4.2.7. Tender point: Gluteal

![Graph showing Allognet readings for left and right gluteal regions over visits.]

**FIGURE 4.9. Gluteal tender point**

4.2.8. Tender point: Greater trochanter

![Graph showing Allognet readings for left and right greater trochanter regions over visits.]

**FIGURE 4.10. Greater trochanter tender point**
4.2.9. Tender point: Knee

FIGURE 4.11. Knee tender point
4.3. Subjective data collection (Experimental and control group combined)

4.3.1. Visual Analogue Pain Scale

![Graph showing Visual Analogue Pain Scale](image)

**FIGURE 4.12** Visual Analogue Pain Scale
4.3.2. Oswestry Low Back Pain Questionnaire

![Oswestry Low Back Pain Questionnaire Chart]

**FIGURE 4.13.** Oswestry Low Back Pain Questionnaire
4.3.3. Neck Disability Index

FIGURE 4.14. Neck Disability Index
CHAPTER 5

DISCUSSION OF RESEARCH RESULTS

5.1. Tender points (Algometer measurements)

5.1.1. Suboccipital tender points

Algometer measurements for the suboccipital tender point in the experimental group gradually decreased over ten treatments. This clinical decrease in the algometer reading illustrates an increase in tenderness or pain over this specific tender point. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference ($P = 0.671$ left side; $P = 0.541$ right side; thus $P > 0.05$).

As far as algometer measurements for the control group are concerned: measurements increased at treatment 5 (pain or tenderness decreased) but showed a slight decrease at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference ($P = 0.646$ left side; $P = 0.784$ right side; thus $P > 0.05$).

When comparing algometer measurements for the control and experimental group: Measurements in the experimental group for treatment 1 and 5 was higher when compared to the measurements in the control group for the same treatments. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference. Algometer measurements for both groups are almost equal at treatment 10 (Refer to figure 4.3.).
5.1.2. Anterior cervical tender points

Algometer measurements for the anterior cervical tender point in the experimental group showed a slight increase at treatment 5. Measurements of the left anterior cervical tender point decreased at treatment 10, but remained the same on the right. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference ($P = 0.592$ left side; $P = 0.804$ right side; thus $P > 0.05$).

As far as algometer measurements for the control group are concerned: Measurements increased at treatment 5 and decreased slightly at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference ($P = 0.489$ left side; $P = 0.446$ right side; thus $P > 0.05$).

When comparing algometer measurements for the control and experimental groups: Measurements in the experimental group for treatment 1, 5 and 10 was slightly higher than that of the control group for the same treatments. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.4.).

5.1.3. Trapezius tender points

Algometer readings for the trapezius tender point in the experimental group showed a gradual decrease over 10 treatments. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference ($P = 0.691$ left side; $P = 0.743$ right side; thus $P > 0.05$).
As far as algometer measurements for the control group are concerned: Measurements increased at treatment 5 but showed a slight decrease at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.419 left side; P = 0.295 right side; thus P > 0.05).

When comparing algometer measurements for the control and experimental groups: Measurements in the experimental group for treatments 1 and 5 was higher than that of the control group for the same treatments. There is a statistically significant difference in the algometer measurements of the right trapezius tender point between the experimental and control group at treatment 1 (initial visit) P = 0.0248, thus P < 0.05). Algometer measurements for both groups were almost equal at treatment 10 (Refer to figure 4.5.).

5.1.4. Supraspinatus tender points

Algometer measurements for the supraspinatus tender point in the experimental group and control group increased at treatment 5 and decreased again at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference.

When comparing algometer measurements for the control and experimental groups: Measurements for treatment 1, 5 and 10 was higher in the experimental group than that of the control group for the same treatments. These differences in the mean values among the two groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.6.).
5.1.5. Second rib tender points

Algometer measurements for the second rib tender point in the experimental group and the control group increased at treatment 5 and decreased again at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference.

When comparing algometer measurements for the control and experimental groups: Measurements for treatment 1, 5 and 10 was higher in the experimental group than that of the control group for the same treatments. The difference in the mean values among the two groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.7.).

5.1.6. Elbow tender points

Algometer measurements for the elbow tender point in the experimental group showed an increase at treatment 5 and decrease at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.713 left side; P = 0.611 right side; thus P > 0.05).

As far as algometer measurements for the control group are concerned: Measurements increased gradually over the ten treatments. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.577 left side; P = 0.775 right side; thus P > 0.05).

When comparing algometer measurements for the experimental and control groups: Measurements in the experimental group was higher in treatment 1 and 5 than that of
the control group for the same treatments. Measurement in the control group at treatment 10 was higher than that of the experimental group at treatment 10. The difference in the mean values among the two groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.8.).

5.1.7. Gluteal tender points

Algometer measurements for the gluteal tender points in the experimental group showed a gradual increase over the ten treatments. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference ($P = 0.248$ left side; $P = 0.842$ right side; thus $P > 0.05$).

As far as algometer measurements for the control group are concerned: Measurements show a gradual increase at treatment 5 and a decrease at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference ($P = 0.477$ left side; $P = 0.428$ right side; thus $P > 0.05$).

When comparing algometer measurements for the control and experimental groups: Measurements in the experimental group for treatment 1 was lower on the left and higher on the right when compared to the control group for the same treatment. Measurements at treatment 5 were higher in the control group. Measurements at treatment 10 were higher in the experimental group. The difference in the mean values among the two groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.9.).
5.1.8. Greater trochanter tender points

Algometer measurements for the greater trochanter tender point in the experimental group and control group showed an increase at treatment 5 and a decrease at treatment 10. This difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference.

When comparing algometer measurements for the experimental and control groups: Measurements in the experimental group was higher for treatment 1, 5 and 10 when compared to the control group for the same treatments. The difference in the mean values among the two groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.10.).

5.1.9. Knee tender points

Algometer measurements for the knee tender point in the experimental group showed a gradual decrease over the 10 treatments. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.527 left side; P = 0.546 right side; thus P > 0.05).

As far as algometer measurements for the control group are concerned: Measurements increased slightly at treatment 5 and decreased slightly at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.949 left side; P = 0.816 right side; thus P > 0.05).

When comparing algometer measurements for the experimental and control groups: Measurements in the experimental group were higher when compared to the control
group for treatment 1 and 5. Measurements for both groups were equal at treatment 10. The difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.11.).

5.2. Visual Analogue Pain Scale

When considering the Visual Analogue Pain Scale, the pain rating in the experimental group, over ten treatments, showed a gradual decrease. This difference in the mean values among the treatments are greater than would be expected by chance; there is a statistically significant difference (P = 0.0148, thus P < 0.05).

As far as the pain rating for the control group is concerned: The pain rating decreased gradually up to treatment 6 after which it varied slightly. The difference in the mean values among the treatments are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (P = 0.525).

When comparing the pain rating of the control and experimental groups for specific treatments, a statistically significant difference was found at treatment 3, 5 and 6 (Refer to figure 4.12).

5.3. Neck Disability Index

The percentage disability decreased in both the experimental and control groups over ten treatments, but the difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.14).
5.4. Oswestry Low Back Pain Questionnaire

The percentage disability decreased in both the experimental and control groups over ten treatments, but the difference in the mean values among the groups are not great enough to exclude the possibility that the difference is due to random sampling variability; there is not a statistically significant difference (Refer to figure 4.13).
CHAPTER 6

CONCLUSION AND RECOMMENDATION

6.1. Recommendations

This study, or future studies may possibly be improved through the following recommendations:

- The treatments could be extended over a longer period of time to include at least thirty treatments involving spinal manipulation and ischaemic compression. This corresponds to a study done by Hains and Hains (2000:225-230).

- According to Hains and Hains (2000:225-230), fifteen treatments should be considered an adequate cutoff point to determine if a significant improvement in pain has occurred and if further care is warranted.

- Because of the fact that any form of bodywork (spinal manipulation, ischaemic compression, etc.) may leave the patient fatigued or bring about flu-like symptoms and low grade fever (Starlanyl and Copeland, 2001:98). Treatment sessions were limited to two sessions per week. It is suggested that treatment sessions per week be either increased on decreased depending on patients' tolerance, allowing the patient enough time to detoxify between treatments.

- Further investigation into this field of study is suggested using a larger sample size (minimum of 81 subjects per treatment group) according to Blunt, et. al., (1997:397).

- Due to the limited amount of research involving chiropractic and fibromyalgia, more research pertaining to the chiropractic approach to Fibromyalgia Syndrome specifically, is encouraged.
The approach to Fibromyalgia syndrome remains multi-disciplinary, and therefore studies involving other health disciplines such as Homoeopathy, in conjunction with chiropractic is suggested.

A further study could include an acupuncture or electroacupuncture group as Deluze, et. al., (1992:1249-1252) have found significant improvement among people with fibromyalgia using these modalities.

Working in closer conjunction with fibromyalgia support groups, Rheumatologists and other experts in the field of Fibromyalgia Syndrome with the purpose of building a good referral basis is highly recommended.

Certain patients who participated in this research program used non-steroidal anti-inflammatory drugs (NSAID's) and other pain medication during the course of treatment. This obviously has an effect on patients' pain levels and therefore inclusion criteria in future studies will have to be revised.

The clinical trial took 12 months to complete and 24 participants out of a recruited 32, completed the trail. The researcher considers the following as possible reasons:

- This study was started in Johannesburg at the TWR Day Clinic in Doornfontein. A considerable number of patients felt that this venue could not guarantee their safety, and therefore personal fears and prejudice prevented them from participating in the study. The study was then moved to Middelburg a couple of months later. Because of the fact that some patients had to travel distances in excess of 160 km twice a week, they withdrew from the research program.

- Patient compliance in some instances was really poor and whether this was due to the above mentioned factors or personal reasons is unknown. It has to be considered that fibromyalgia patients may feel worse after even the slightest amount
of bodywork and that the two sessions per week in some instances was too much for the patients to tolerate.

➢ Treatment was administered free of charge and this fact may lead to poor patient compliance as well.

The researcher hopes that the above mentioned recommendations will assist future researchers in the field of Fibromyalgia Syndrome.

6.2. Conclusion

The purpose of this study was to determine whether the combination of chiropractic care and adjunctive methods (including ultrasound and ischaemic compression) would reduce pain levels in fibromyalgia patients.

The results indicate a decrease in reported pain levels and percentage disability has been achieved in the experimental group that received chiropractic adjustments and adjunctive methods (ultrasound and ischaemic compression). The results for the control group – that only received adjunctive methods – also indicate a decrease in reported pain levels and percentage disability. These results, however, were found to be not statistically significant.

Thus in conclusion, it can be noted that chiropractic care in conjunction with adjunctive methods including ultrasound and ischaemic compression may be beneficial in the management of Fibromyalgia Syndrome patients, specifically in improving their reported pain levels and percentage disability.
REFERENCES


Amsterdam, J.D., Maislin, G., Winokur, A., et. al., (1987) Pituitary and adrenocortical responses to the ovine corticotropin releasing hormone in depressed patients and healthy volunteers, Arch Gen Psychiatry, Vol. 44, pp. 775-781


Cox, J.M. (1999) *Low Back Pain; Mechanism, Diagnosis and Treatment*, sixth edition, Williams & Wilkins, pp. 252


APPENDIX A: Subject information and Consent form

Dear patient

The purpose of this study is to determine the efficacy of Chiropractic care in conjunction with adjunctive methods in the management of Fibromyalgia Syndrome.

Although there is no cure for Fibromyalgia Syndrome, and loss of all symptoms is unusual, the treatment will be aimed at creating a better understanding of this condition and the management thereof.

You must be over the age of 18 years to be able to participate in this study. After you have been selected to participate in this study, you will be divided into one of two groups. Each group will receive a different combination of therapy. You will be required to attend two treatment sessions per week on alternate days at the TWR Chiropractic Day Clinic or at the practice of Dr. Johan Kotze in Middelburg, Mpumalanga.

Participation in this study is voluntary and you are free to refuse to participate or to withdraw your consent and discontinue participation at anytime. A signed copy of the consent form will be made available to you. I have fully explained the procedure and have answered all your questions you have had to the best of my abilities.

Date: ___________________________ Researcher: _______________________

I have been fully informed as to my rights and as to the procedure to be followed in this study and I understand that I am able to withdraw my consent at any time. I know that any questions, which I may have, will be answered.

Date: ___________________________ Participant: _______________________

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APPENDIX B: Advertisement

FIBROMYALGIA RESEARCH PROGRAM

ARE YOU OVER THE AGE OF 18 YEARS, AND HAVE BEEN DIAGNOSED WITH FIBROMYALGIA SYNDROME?

PARTICIPATE IN A FREE, SUPERVISED CHIROPRACTIC STUDY CONDUCTED AT THE PRACTICE OF DR. JOHAN KOTZÉ (14 PRES. KRUGER STREET, MIDDELBURG) IN ASSOCIATION WITH WITS TECHNIKON-SCHOOL OF CHIROPRACTIC.

FOR MORE INFORMATION CONTACT

WERNER FERREIRA

AT

073 301 9393 OR (013) 243 3141
APPENDIX C: Visual Analogue Pain Scale

Patient name: ________________ Today's date: ____________

How much pain have you had because of your condition in the past week?

Please mark on the line to indicate how severe your pain has been.

NO PAIN | Slight | Severe | PAIN AS BAD AS IT COULD BE

________ cm
APPENDIX D: Neck Disability Index

Name: _______________________________ Today's date: __________________

Date of birth: _______________________________ Examiner: __________________

How long have you had neck pain? _______ Years _______ Months _______ Weeks
How long have you had headaches? _______ Years _______ Months _______ Weeks

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

SECTION 1 - PAIN INTENSITY
☐ I have no pain at the moment.
☐ The pain is very mild at the moment.
☐ The pain is moderate at the moment.
☐ The pain is fairly severe at the moment.
☐ The pain is very severe at the moment.
☐ The pain is the worst imaginable at the moment.

SECTION 2 - PERSONAL CARE (washing, dressing, etc)
☐ I can look after myself normally without causing extra pain.
☐ I can look after myself normally but it causes extra pain.
☐ It is painful to look after myself and I am slow and careful.
☐ I need some help but manage most of my personal care.
☐ I need help every day in most aspects of self care.
☐ I do not get dressed, wash with difficulty and stay in bed.

SECTION 3 - LIFTING
☐ I can lift heavy weights without extra pain.
☐ I can lift heavy weights but it gives extra pain.
☐ Pain prevents me from lifting heavy objects off the floor, but I can manage if they are conveniently positioned, e.g. on a table.
☐ Pain prevents me from lifting heavy weights but I can manage light to medium weights if they are conveniently positioned.
☐ I can lift very light weights
☐ I cannot lift or carry anything at all.

SECTION 4 - READING
☐ I can read as much as I want with no pain in my neck.
☐ I can read as much as I want with slight pain in my neck.
☐ I can read as much as I want with moderate pain in my neck.
☐ I can hardly read at all because of moderate pain in my neck.
☐ I cannot read at all.

SECTION 5 - HEADACHES
☐ I have no headaches at all.
☐ I have slight headaches which come infrequently.
☐ I have moderate headaches which come infrequently.
☐ I have moderate headaches which come frequently.
☐ I have severe headaches which come frequently.
☐ I have headaches almost all the time.

SECTION 6 - CONCENTRATION
☐ I can concentrate fully when I want to with no difficulty.
☐ I can concentrate fully when I want to with slight difficulty.
☐ I have a fair degree of difficulty in concentrating when I want to.
☐ I have a lot of difficulty in concentrating when I want to.
☐ I have a great deal of difficulty in 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, but 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.
]

SECTION 8 – DRIVING
[
☐ I can drive without any neck pain.
☐ I can drive as long as I want with slight pain in my neck.
☐ I can drive as long as I want with moderate pain in neck.
☐ I cannot drive as long as I want 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 1 hr. 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 pain in my neck.
☐ 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 a 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.
]

COMMENTS:
APPENDIX E: The Oswestry Low Back Pain Questionnaire

Name: ________________________

Date of birth: ________________

Today's Date: ________________

Examiner: ________________

Please read carefully:
This questionnaire has been designed to give the doctor information as to how your low back pain has affected your ability to manage everyday life. Please answer every section, and mark in each section only the ONE BOX which applies to you. We realise that you may consider that two of the statements in any one section relate to you, but please just mark the one box which most closely describes your problem.

SECTION 1 - PAIN INTENSITY

☐ I can tolerate the pain I have without having to use pain killers.

☐ The pain is bad but I can manage without taking pain killers.

☐ Pain killers give complete relief from pain.

☐ Pain killers give moderate relief from pain.

☐ Pain killers give very little relief from pain.

☐ Pain killers have no effect on the pain and I do not use them.

SECTION 4 - WALKING

☐ Pain does not prevent me from walking any distance.

☐ Pain prevents me from walking more than 1 mile.

☐ Pain prevents me from walking more than ½ mile.

☐ Pain prevents me from walking more than ¼ mile.

☐ I can only walk using a stick or crutches.

☐ I am in bed most of the time and have to crawl to the toilet.

SECTION 2 - PERSONAL CARE

☐ I can look after myself normally without causing extra pain.

☐ I can look after myself normally but it causes extra pain.

☐ It is painful to look after myself and I am slow and careful.

☐ I need some help but can manage most of my personal care.

☐ I need help every day in most aspects of self care.

☐ I do not get dressed, wash with difficulty and stay in bed.

SECTION 5 - SITTING

☐ I can sit in any chair as long as I like.

☐ I can only sit in my favorite chair as long as I like.

☐ Pain prevents me from sitting more than 1 hour.

☐ Pain prevents me from sitting more than ¼ hour.

☐ Pain prevents me from sitting more than 10 minutes.

☐ Pain prevents me from sitting at all.

SECTION 3 - LIFTING

☐ I can lift heavy weights without extra pain.

☐ I can lift heavy weights but it gives me extra pain.

☐ Pain prevents me from lifting heavy weights off the floor, but I can manage if they are conveniently positioned (e.g. on a table).

☐ Pain prevents me from lifting heavy weights, but I can manage light to medium weights if they are conveniently positioned.

☐ I can lift only very light weights.

☐ I cannot lift or carry anything at all.

SECTION 6 - STANDING

☐ I can stand as long as I want without extra pain.

☐ I can stand as long as I want but it gives me extra pain.

☐ Pain prevents me from standing for more than 1 hour.

☐ Pain prevents me from standing more than ½ hour.

☐ Pain prevents me from standing for more than 10 minutes.

☐ Pain prevents me from standing at all.
SECTION 7 – SLEEPING
☐ Pain does not prevent me from sleeping well.
☐ I can sleep well only by using tablets.
☐ Even when I take tablets I have less than 6 hours sleep.
☐ Even when I take tablets I have less than 4 hours sleep.
☐ Even when I take tablets I have less than 2 hours sleep.
☐ Pain prevents me from sleeping at all.

SECTION 8 – SEX LIFE
☐ My sex life is normal and causes no extra pain.
☐ My sex life is normal but causes extra pain.
☐ My sex life is nearly normal but is very painful.
☐ My sex life is severely restricted by pain.
☐ My sex life is nearly absent because of pain.
☐ Pain prevents any sex life at all.

SECTION 9 – SOCIAL LIFE
☐ My social life is normal and gives me no extra pain.
☐ My social life is normal but increases the degree of pain.
☐ Pain has no significant effect on my social life apart from limiting my more energetic interests (e.g. dancing, etc.)
☐ Pain has restricted my social life and I do not go out as often.
☐ Pain has restricted my social life to my home.
☐ I have no social life because of pain.

SECTION 10 – TRAVELLING
☐ I can travel anywhere without extra pain.
☐ I can travel anywhere but it gives me extra pain.
☐ Pain is bad but I can manage journeys over 2 hours.
☐ Pain restricts me to journeys of less than 1 hour.
☐ Pain restricts me to short necessary journeys under 30 min.
☐ Pain restricts me from travelling except to the doctor or hospital.

OTHER COMMENTS:
APPENDIX F: Case History

TECHNIKON WITWATERSRAND
CHIROPRACTIC DAY CLINIC

CASE HISTORY

Date: ______________

Patient: ____________________________
File no: ______________

Age: ________ Sex: ________
Occupation: _______________________

Intern: ____________________________
Signature: _________________________

FOR CLINICIAN’S USE ONLY

Initial visit clinician: ______________
Signature: ______________

Case History: __________________________________________

Examination:

Previous: TWR
Other
Current: TWR
Other

X-ray Studies:

Previous: TWR
Other
Current: TWR
Other

Clinical path. lab:

Previous: TWR
Other
Current: TWR
Other

Case Status:

PTT: Conditional:
Signed off:
Final sign out:

Recommendations:
Intern's case history

1. Source of history:

2. Chief complaint: (Patient's own words)

3. Present illness:
   - Location
   - Onset
   - Duration
   - Frequency
   - Pain (character)
   - Progression
   - Aggravating factors
   - Relieving factors
   - Associated Sx's and Sg's
   - Previous occurrences
   - Past treatment and outcome

4. Other complaints:
5. Past history:
   
   General health status
   
   Childhood illness
   
   Adult illness
   
   Psychiatric illnesses
   
   Accidents / Injuries
   
   Surgery
   
   Hospitalisation

6. Current health status and lifestyle:
   
   Allergies
   
   Immunizations
   
   Screening tests
   
   Environmental hazards
   
   Safety measures
   
   Exercise and leisure
   
   Sleep pattern
   
   Diet
   
   Current medication
   
   Tobacco
Alcohol
Social drugs

7. Family history:

Immediate family:

Cause of death
DM
Heart disease
TB
HBP
Stroke
Kidney disease
CA
Arthritis
Anaemia
Headaches
Thyroid disease
Epilepsy
Mental illness
Alcoholism
Drug addiction
Other

8. Psychosocial history:

Home situation

Daily life

Important experiences

Religious beliefs
9. Review of symptoms:

   General
   Skin
   Head
   Eyes
   Ears
   Nose / sinuses
   Mouth / throat
   Neck
   Breasts
   Respiratory
   Cardiac
   Gastro-intestinal
   Urinary
   Genital
   Vascular
   Musculoskeletal
   Neurologic
   Haematologic
   Endocrine
   Psychiatric
APPENDIX G: Lumbar Spine Regional

TECHINKON WITWATERSRAND
CHIROPRACTIC DAY CLINIC

REGIONAL EXAMINATION
LUMBAR SPINE AND PELVIS

Date: ________________

Patient: ____________________________        File No: ________________

Clinician: ____________________________       Signature: ________________

Intern: _______________________________        Signature: ________________

A) STANDING
1. BODY TYPE
2. POSTURE
3. OBSERVATION:-

- Muscle Tone
- Bony + Soft Tissue Contours
- Skin
- Scars
- Discolouration
- Step deformity
4. SPECIAL TESTS

- Schober's Test
- Spinous percussion
- Treadmill
- Minor's sign
- Quick Test
- Trendelenburg Test

5. RANGE OF MOTION

- Forward flexion = 40–60°
- Extension = 20–35°
- L/R Rotation = 3–18°
- L/R Lat Flexion = 15–20°

/// = Painful limitation

/ = Pain free limitation
6. GAIT

- Rhythm, pendulousness
- On Toes (S1)
- On Heels (L4, 5)
- Half Squat on one leg (L2, 3, 4)
- Tandem Walking

7. MOTION PALPATION – Sacroiliac joint

B) SITTING

1. SPECIAL TESTS

- Tripod Test
- Kemp’s Test
- Valsalva Manoeuvre

2. MOTION PALPATION

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C) SUPINE

1. OBSERVATION
   - Hair, Skin, Nails
   - Fasciculations

2. PULSES
   - Femoral
   - Popliteal
   - Dorsalis Pedis
   - Posterior Tibial

3. MUSCLE CIRCUMFERENCE

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5. ABDOMINAL EXAMINATION

• Observation
• Abdominal Reflexes
• Auscultation Abdomen and Groin
• Palpation Abdomen and Groin

Comments: 

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7. SPECIAL TESTS

- SLR
- WLR
- Braggard's
- Bowstring
- Sciatic Notch Pressure
- Sign of the Buttock
- Bilateral SLR
- Patrick Faber
- Gaenslen's Test
- Gapping Test
- "Squish" Test
- Gluteus Maximus Stretch
- Thomas' Test
- Rectus Femoris Contracture Test
- Hip Medial Rotation
- Psosas Test

LATERAL RECUMBENT

- Sacroiliac Compression
- Ober's Test
- Femoral Nerve Stretch Test
- Myotomes:
  - Quadratus Lumborum Strength
  - Gluteus Medius Strength
PRONE

- Facet joint challenge
- Myofascial Trigger Points:
  - Quadratus Lumborum
  - Gluteus Medius
  - Gluteus Maximus
  - Piriformis
  - Tensor Fascia Lata
  - Hamstrings

- Skin rolling
- Erichsen's Test
- Sacroiliac Tenderness
- Pheasant's Test
- Gluteal Skyline
- Myotomes:
  - Gluteus Maximus strength

NON-ORGANIC SIGNS

- Pin-point pain
- Axial Compression
- Trunk Rotation
- Bum's Bench Test
- Flip Test
- Hoover's Test
- Ankle Dorsiflexion Test
- Pin-point pain
APPENDIX H: Cervical Spine Regional

TECHNikon Witwatersrand
CHIROPRACTIC DAY CLINIC

REGIONAL EXAMINATION
CERVICAL SPINE

Date: ___________

Patient: ___________________________ File No: ___________

Clinician: _______________________ Signature: ___________

Intern: ___________________________ Signature: ___________

OBSERVATION

- Posture
- Size
- Swellings
- Scars
- Discolouration
- Hairline
- Bony and soft tissue contours
- Shoulder level
- Muscle spasm
- Facial expression
**RANGE OF MOTION**

- Flexion = 45° - 90°
- Extension = 55° - 70°
- L/R Rotation = 70° - 90°
- L/R Lateral flexion = 20° - 45°

**PALPATION**

- Lymph nodes
- Trachea
- Thyroid gland
- Pulses / thrills
- Tenderness
- Muscle tone
- Active MF Trigger Points: SCM, Trapezius, Scaleni, Levator Scapulae, Posterior Cervical musculature
ORTHOPAEDIC EXAMINATION

1. Doorbell Sign
2. Max. Cervical Compression
3. Spurling's manoeuvre
4. Lateral Compression (Jackson's test)
5. Kemp's Test
6. Cervical Distraction
7. Shoulder abduction Test
8. Shoulder depression Test
9. Dizziness rotation Test
10. Lhermitte's Sign
11. O'Donoghue Manoeuvre
12. Brachial Plexus Tension
13. Carpal tunnel syndrome:
   - Tinel's Sign
   - Phalen's Test
14. TOS:
   - Halstead's test
   - Adson's test
   - Eden's (traction) test
   - Hyperabduction (Wright's) test – Pec Minor
   - Costoclavicular test

Remarks: ______________________________________________________
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102
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**COMMENTS:**

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APPENDIX I: Physical examination

Pertinent Physical

(NOTE: This form may only be used when you have completed 35 new patients)

Student Name: ___________________  Signature: __________
Doctor Name: ___________________  Signature: __________

Patient Information

Name: ___________________________  Occupation: __________
Age: ______________________________  Sex: __________________

Vitals:
Height: ___________________________  Weight: _______________
Pulse rate: _______________________  Respiratory rate: __________
Blood pressure: _________________  Temp: ______________

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