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FIBROMYALGIA: DIAGNOSIS AND MANAGEMENT

by

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Bachelor of Science in Physical Therapy
University of North Dakota, 1994

An Independent Study
Submitted to the Graduate Faculty of the
Department of Physical Therapy
School of Medicine
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in partial fulfillment of the requirements
for the degree of
Master of Physical Therapy

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This Independent Study, submitted by Janet Rasmusson McKinnon in partial fulfillment of the requirements for the Degree of Master of Physical Therapy from the University of North Dakota, has been read by the Faculty Preceptor, Advisor, and Chairperson of Physical Therapy under whom the work has been done and is hereby approved.

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PERMISSION

Title        Fibromyalgia: Diagnosis and Management

Department   Physical Therapy

Degree       Master of Physical Therapy

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ABSTRACT

Fibromyalgia is a nonarticular rheumatic disease of unknown origin. It is diagnosed by the presence of widespread pain of more than three months duration and tenderness at 11 or more of 18 tender point sites. Typical symptoms of this syndrome are generalized pain, stiffness, and chronic fatigue due to sleep disturbances. Other uncommon symptoms include headaches, anxiety, depression, irritable bowel syndrome, and numbness. These symptoms become worse with the presence of modulating factors such as weather, stress, fatigue, overexertion, or inactivity.

The purposes of this independent study are to examine the prevalence and demographics of this disorder, discuss the etiology and pathophysiology, list common symptoms, and present diagnostic criteria that have been proposed in the literature. In addition, suggestions will be provided on how to best manage this disorder.
CHAPTER I
INTRODUCTION

Fibromyalgia is a frequently occurring disorder in the outpatient physical therapy population. Fibromyalgia syndrome is defined as a form of nonarticular rheumatism with chronic, diffuse musculoskeletal aching. It is often associated with poor sleep, fatigue, and morning stiffness. These symptoms are aggravated by factors such as the weather, physical or mental stress, physical exertion, and poor quality sleep. Age is not a determining factor in the diagnosis of fibromyalgia.

Age at onset is difficult to determine because these patients have typically been seen in the clinic for a multitude of musculoskeletal disorders, including a number of rheumatic conditions. The onset of fibromyalgia may occur at any age, but the highest incidence is during middle age. The age of onset ranges from nine to 55 years of age with the most common age of onset occurring between 35 and 50 years of age.

Five types of onset are described in the literature. They include: (1) onset in childhood; (2) onset in adult life with a history of severe musculoskeletal complaints; (3) insidious onset in adult life; (4) onset following infection, surgery, or other event; and (5) onset following trauma. A few
patients will report the beginning of their symptoms after experiencing a traumatic event, but the majority of patients will not be able to recall any prior event that can readily be identified as the cause of their pain. No matter what the onset, the course of the syndrome continues in a chronic manner with little hope for remission.

Several studies show that fibromyalgia follows a chronic course. One study gave a four-year follow-up survey to patients with fibromyalgia. This study showed that 72.3% of patients reported an increase in pain, 15.7% reported no change in condition, and 9.6% reported improvement or no pain at the end of the four-year period. Another study surveyed 39 fibromyalgia patients annually, with initial contact on average of 1.3 years after diagnosis. Throughout three surveys, more than 60% of patients reported moderate to severe continuing symptoms. By the third survey, 97% still reported pain and 62% claimed moderate to severe morning tenderness. Only 3% experienced complete remission. The researchers concluded that fibromyalgia is a chronic disease with symptoms lasting at least three years after diagnosis with a very small chance for remission.

Similar results have been reported by Wolfe. At a follow-up evaluation at least a year after diagnosis, 90% of patients continued to complain of pain and 100% had chronic musculoskeletal pain.

Another study by Wolfe questioned 81 patients with fibromyalgia and revealed that 23% of patients had periods of two months or greater without
symptoms. The mean length of the disease for these patients was 12.7 years and the mean length of remission was 34 months (approximately 21% of the disease course). Therefore, the majority of the time spent in this disorder is in the active, painful state.

**Historical Review**

The history of how the diagnosis of fibromyalgia evolved is interesting because it spans hundreds of years. Researchers do not know exactly how long this syndrome has been afflicting humans. There are references to pain, exhaustion, and disturbed sleep, common features of fibromyalgia, as far back as in the Old Testament of The Holy Bible (Job 7:3-4, Job 30:16-17, Psalms 6:2-6).\(^\text{15}\) Despite an apparently long period in which symptoms have been reported, this condition was not fully recognized until the 1700s.

In the 1700s, German physicians divided rheumatism into two categories: articular and muscular. Articular rheumatism was associated with pain and stiffness in the joints and muscular rheumatism was described as a disease of the muscle associated with hardening or nodules. During approximately the same time frame, physicians in Great Britain began to characterize muscular rheumatism as a disorder of connective tissue rather than of muscle. In 1815, William Balfour was the first to postulate that the nodules located in muscle were caused by connective tissue inflammation.\(^\text{16}\) Sir William Gowers introduced the term fibrositis in 1904.\(^\text{17}\)
Gowers developed the term “fibrositis” based on his belief that inflammation was the primary etiological factor involved in this syndrome.\textsuperscript{17} He believed that sciatica was due to an inflammatory process involving the nerve. He rationalized that because lumbago (a type of muscular rheumatism) often caused sciatica, lumbago (and other types of muscular rheumatism) must be inflammatory as well. He thus called a variety of musculoskeletal conditions involving inflammation “fibrositis.” For years, fibrositis was used to describe a plethora of pain disorders and non-specific musculoskeletal aching.\textsuperscript{2}

Ralph Stockman,\textsuperscript{18} also in 1904, published a study stating that the painful nodules associated with chronic rheumatism may be caused by connective tissue hyperplasia or inflammation of the perimysium. He noted these nodules were most common in the fibrous ligaments of the joints, the musculotendinous junctions, and in the fibrous tissues of muscles, bones, or joints. He listed several factors capable of aggravating this condition, including cold and damp weather or muscle exertion. Because of Gowers’ and Stockman’s studies, inflammation was blamed as the cause of muscular rheumatism for years.

In the 1940s, researchers discovered there was no presence of inflammation in the connective tissue of patients with fibrositis.\textsuperscript{16,19} This made the term fibrositis a misnomer because “-itis” implies inflammation.\textsuperscript{20} Hench\textsuperscript{20} in 1976 proposed the term “fibromyalgia” to describe this syndrome. Fibromyalgia is currently the preferred name for widespread nonarticular pain and its use is endorsed by the American College of Rheumatology (ACR).\textsuperscript{11}
In the past, fibromyalgia was divided into two categories: primary and secondary/concomitant. Primary fibromyalgia was defined as that which occurred for unknown reasons and did not occur with any other musculoskeletal disorder. Secondary/concomitant fibromyalgia occurred in patients with other musculoskeletal or medical problems that mimicked the symptoms of fibromyalgia. These classes made the diagnosis of fibromyalgia confusing and subjective to the examiner. The position of the ACR is that primary and secondary/concomitant categories are not easily separated in a clinical setting. Therefore, the classifications of primary and secondary fibromyalgia should be abolished.

Prevalence and Demographics

The exact prevalence of fibromyalgia in the rheumatologic practice and the community is not known. Fibromyalgia is estimated to be the third most common rheumatic disorder after osteoarthritis (OA) and rheumatoid arthritis (RA). The greatest prevalence of fibromyalgia is found in rheumatology clinics, with a rate of approximately 15-20%. The prevalence of fibromyalgia is less in general medical clinics, with a rate of 3.7%. There are no studies regarding the prevalence of fibromyalgia in the community.

The demographics of this disorder are not specific except for gender. Research has shown that more than 70% of the patients with fibromyalgia are female. Studies have shown that the average patient with fibromyalgia has a slightly higher educational level and earns slightly more than the typical U.S.
citizen. Patients with fibromyalgia have a higher rate of marriage than the normal population.\textsuperscript{8,21} These data do not point out a specific demographic factor, or combination of factors, that could identify a patient with fibromyalgia from the general population. Therefore, it does not appear that demographics contribute to the etiology of this disorder.

The chapters that follow will describe common symptoms associated with this syndrome, present diagnostic criteria that have been proposed in the literature, and discuss theories related to etiology and pathophysiology. In addition, suggestions will be presented on how to help manage this disorder.
CHAPTER II

SYMPTOMATOLOGY AND DIAGNOSTIC CRITERIA

The most common symptoms associated with fibromyalgia include chronic fatigue due to sleep disturbances, stiffness, and generalized pain.\textsuperscript{5,6,8,11,21,23} Fatigue, severe enough to disturb ADLs, is a universal complaint.\textsuperscript{23} Sleep disturbances, such as intermittent sleep, early morning awakenings, and non-restorative sleep, were reported by 56\% to 90\% of patients.\textsuperscript{5,11,24} Poor sleep is believed to contribute to the stiffness associated with this syndrome. The general stiffness of fibromyalgia is typically most noticeable in the morning and does not decrease with activity.\textsuperscript{23} The pain associated with this syndrome is characterized by chronic, diffuse, musculoskeletal aching and soreness\textsuperscript{21} in the proximal musculature, such as the neck, shoulders, elbows, knees, hips, and back.\textsuperscript{23} The right side is usually more affected than the left.\textsuperscript{8}

Although the predominant symptoms of fibromyalgia are fatigue, stiffness, and pain, these are not the only symptoms capable of causing discomfort. The individual with fibromyalgia may also suffer from one or more of the following problems: recurrent headaches, irritable bowel syndrome, hypersensitivity to heat or cold, anxiety, depression, numbness in the hands or
feet, menstrual disturbances, subjective swelling, abdominal pain, and reticular skin discolorations.5,7,8,11,21,24,25

The symptoms of fibromyalgia apparently become worse with the presence of various modulating factors. These factors include cold and humid weather, weather changes, fatigue, overexertion, repetitive motion, emotional stress, caffeine, poor sleep, anxiety, air conditioning, and inactivity.7,11,14,16,26,27

The symptoms of fibromyalgia may become severe enough to create problems at the workplace and in the performance of activities of daily living (ADLs). A correlative relationship has been identified between patients' perceived status of their disorder and their ability to work. The work status of 176 patients with fibromyalgia were examined.22 Of the entire 176 patients in the study, 54% were employed, 30% had changed jobs due to the limitations of fibromyalgia, and 17% had stopped working due to the disease. Of those of working age (18-65 years old), 9.6% considered themselves disabled and 6.2% of this group received disability payments for musculoskeletal disorders. However, none of those were receiving disability compensation for the specific diagnosis of fibromyalgia. This study demonstrated that most patients with fibromyalgia are able to work even though it may be necessary to change their jobs in order to do so.

Patients with fibromyalgia are not as productive as their unaffected counterparts. Twenty-eight patients with fibromyalgia were evaluated and compared to 26 individuals with RA and 11 normal controls in the performance
of five standardized work tasks.\textsuperscript{22} It was found that fibromyalgia patients performed 58.6\% as much work as the normal controls and RA patients performed as much as 62.1\% of the controls. The ability to work was related to functional disability, psychological status, and pain. The strongest indicator of work performance was a self-assessed disability index.

In the same study, it was found that patients with fibromyalgia reported a significant time lost from work.\textsuperscript{22} Of those who were employed, the average time lost in six months was approximately five days. Fewer than nine percent lost more than seven days. Non-working patients reported that they were not able to perform ADLs an average of 12.6 days in a six-month period and 31\% were not able to function for more than seven days in the same time frame. The patient with fibromyalgia may expect to experience lost wages and compensation as a result of time lost from work.

In spite of the presence of characteristic symptoms and a history of inability to perform at work or complete ADLs, the diagnosis of fibromyalgia is difficult to make. One reason diagnosis is difficult is because fibromyalgia is poorly recognized by the medical profession.\textsuperscript{5} Several years may pass between the onset of symptoms and correct diagnosis.\textsuperscript{6} Patients will continue to see one health care professional after another in the hope of receiving a correct diagnosis. Failure to correctly make the diagnosis of fibromyalgia leads to considerable physical, mental, and financial drains for the patient. After time, patients may start to believe they are hysterical, hypochondriacal, or
malingering. In order for an effective treatment regime to begin, the correct diagnosis needs to be made. The correct diagnosis depends on an accurate history and physical exam of the patient.

The first step leading to the diagnosis of fibromyalgia is a detailed history from the patient and a physical examination. The patient's history will usually reveal a report of having received several medications and modalities without experiencing any noticeable changes in his or her condition. The most prominent findings of the physical examination are the lack of any specific objective information. The musculoskeletal examination reveals no swelling or tenderness, no soft tissue edema, no loss of range of motion, and no true muscle weakness. The laboratory reports are negative as well. There are no hematological, serological, histological, or radiological abnormalities. The patient appears to be in fair general health.

Overall, the diagnosis of fibromyalgia is based on the presence of chronic musculoskeletal pain and reproducible tender points. Tender points are the single most distinguishing feature of fibromyalgia. A tender point is a localized area where slight to moderate pressure creates a reproducible painful sensation. Tender points are usually located in symmetry at typical locations. The most common sites are at the tendino-osseous junctions and fat pads of the neck, shoulder, low back, and pelvic areas. The presence of tender points is a critical factor in the diagnosis of fibromyalgia.
Throughout the years, tender points have remained a common factor in the search for a proper diagnostic criteria for fibromyalgia. All studies agree that fibromyalgia patients have a decreased tolerance to pain over tender points, but studies differ in the number of tender points required for diagnosis.5,9,31 Smythe and Moldofsky32 in 1977 were the first to propose diagnostic criteria for fibromyalgia. The criteria for fibromyalgia have since been revised several times. The most current diagnostic criteria were developed by the Multicenter Criteria Committee headed by Wolfe et al11 and adopted by the ACR in 1990.

The current criteria were developed upon examination of 293 patients with fibromyalgia and 265 controls. The controls were age and sex matched with neck or low back pain syndromes, local tendinitis, trauma related pain syndrome, possible systemic lupus erythematosus (SLE), or possible RA. Using the data gathered from these subjects, fibromyalgia was defined as widespread pain (pain above and below the waist and on both sides of the body) with a duration of more than three months. In addition, tenderness at 11 or more of 18 tender point sites must be present. The tender points used for the examination are listed in Table 1.

The ACR criteria are regarded as the most sensitive, specific, and accurate to date. The criteria of widespread pain and tenderness at 11 of 18 tender points has a sensitivity, or true positive rate, of 88.5% and a specificity, or true negative rate, of 81.1%.11 This is better than any other previous
Occiput: at the suboccipital muscle insertions.

Low cervical: at the anterior aspects of the intertransverse spaces at C₅-C₇.

Trapezius: at the midpoint of the upper border.

Supraspinatus: at origin above scapular spine near the medial border.

Second rib: at the costochondral junctions.

Lateral epicondyle: 2 cm distal to the epicondyles.

Gluteal: in the upper outer quadrants.

Greater trochanter: posterior to trochanteric prominence.

Knee: at the medial fat pad proximal to the joint line.

(All the above points are bilateral.)
diagnostic criteria. Even though a consensus has been reached regarding the diagnostic criteria for fibromyalgia, the diagnosis may still be difficult to make or may be missed entirely.

Diagnosis is difficult as fibromyalgia can mimic a variety of conditions or illnesses. For this reason, it is necessary to perform a differential diagnosis. The following conditions need to be ruled out before a definitive diagnosis of fibromyalgia is made: early RA, early SLE, chronic fatigue syndrome, polymyalgia rheumatica, hypothyroidism, osteoarthritis, psychogenic rheumatism, early ankylosing spondylitis, or myofascial pain syndrome.5,30

Of all the similar differential diagnoses, myofascial pain syndrome and fibromyalgia are the most difficult to differentiate. These syndromes have several characteristics in common and may represent two extremes of the same disorder, even though they are regarded as two distinct clinical entities.16 Myofascial pain syndrome is defined by some as any regional soft tissue disorder and by others as a form of localized fibromyalgia; there currently is no consensus on the definition of myofascial pain syndrome.33 In its simplest form, myofascial pain syndrome consists of an acute onset of pain in one muscle following trauma to that muscle.16 Myofascial pain syndrome is generally more localized than fibromyalgia. However, it may involve multiple sites, cause systemic symptoms, become chronic, and eventually develop into fibromyalgia.14,25 In contrast, fibromyalgia is chronic, widespread, and systemic in nature from onset.14
The major distinguishing feature between these two syndromes is the presence of tender points and trigger points. As previously described, tender points are the major criteria for the diagnosis of fibromyalgia. Tender points are typically located at the attachment of muscle to bone or over fat, tendon, or aponeurosis. Pain referral is not characteristic of the tender points found in patients with fibromyalgia. Trigger points are characteristic of myofascial pain syndrome and are defined as painful areas of soft tissue and taut bands of muscle that produce radiating pain spontaneously or following pressure. Trigger points are located in the muscle belly and may also cause paresthesias or autonomic symptoms.

In summary, the most common symptoms of fibromyalgia are chronic fatigue due to sleep disturbances, stiffness, and generalized pain. Other less common symptoms include paresthesias, chronic headaches, and anxiety. Symptoms become worse with the presence of modulating factors such as changes in the weather, physical or mental stress, and poor sleep. The major distinguishing characteristic of fibromyalgia is the presence of tender points. The presence of tender points has led to the establishment of specific diagnostic criteria adopted by the ACR. The development of diagnostic criteria has made it possible to perform research on fibromyalgia which has led to the evolution of several theories related to its etiology.
CHAPTER III

ETIOLOGY AND PATHOPHYSIOLOGY

Early research by Gowers\textsuperscript{17} and Stockman\textsuperscript{18} implicated histological abnormalities in affected muscles as potential causes of fibromyalgia. However, these reports and others prior to 1980 cannot be correctly interpreted because they included a multitude of other conditions in their diagnostic criteria. With clearer diagnostic criteria, research has been easier to interpret, but still is inconclusive in regard to the etiology of this disorder.\textsuperscript{3} The current theories regarding the etiology and pathophysiology of fibromyalgia involve muscle abnormalities, pain perception, psychological factors, neurochemical imbalances, and sleep disturbances.

Muscle Abnormalities

It has been proposed that skeletal muscle is the cause of the disease-process associated with fibromyalgia. Changes in muscle metabolism, contraction and spasm, and the tissue itself have been studied. However, biochemical, histological, electromyographic, and radiographic research is inconclusive in its evidence regarding fibromyalgia.\textsuperscript{34-38}

It may be possible that muscle hypoxia contributes to the pain associated with fibromyalgia.\textsuperscript{34} Biopsy samples taken from painful tissue have shown
decreases in adenosine triphosphate, adenosine diphosphate, and phosphoryl creatine and increases in adenosine monophosphate and creatine.\textsuperscript{24} Chemical changes occurring during energy metabolism may be due to a defective synthesis or an increased rate of breakdown of the metabolites. While muscle hypoxia may be the factor leading to the altered metabolism, this has not been established. Regardless, the study shows real metabolic changes in painful muscles and supports the hypothesis that pain is of muscular origin.

Fibromyalgia pain of a muscular origin is supported by other researchers. One study\textsuperscript{35} observed that the muscle fibers of fibromyalgia patients are connected by reticular or elastic fibers which pull neighboring cells when an adjacent cell contracts. This creates a passive sideways stretch or pull which may cause pain or multiple contractions, leading to muscle exhaustion and pain. This study also concluded skeletal muscle is involved in fibromyalgia pain.

Electromyographic studies, however, do not implicate skeletal muscles as a pathophysiological factor. Needle electromyography (EMG) has been performed to determine if spontaneous activity or motor unit activity is present in patients with focal myofascial pain or fibromyalgia.\textsuperscript{36} Trigger points, tender points, and palpable muscle bands were examined. These sites were found to have normal EMG patterns at rest and with a voluntary muscle contraction. No electrodiagnostic evidence of denervation or spasm was found with focal myofascial pain or fibromyalgia. Full motor recruitment was present in all muscles tested. The authors concluded that palpable muscle changes were not
associated with increased electrical activity; something other than muscle contraction must be the cause of the symptoms. A similar study also found a lack of electromyographic evidence to prove that tender areas are in a state of spasm. EMG analysis of 16 patients found that all areas of so-called muscle spasm were electrically silent. These two studies contradict the pain-spasm-pain hypothesis and show that skeletal muscle is not affected in patients with fibromyalgia.

Magnetic resonance imaging also has failed to detect skeletal muscle abnormalities. Tender points within the trapezius and suboccipital musculature of patients with fibromyalgia were examined. No abnormalities, edema, or inflammation in the soft tissue or underlying bone were found. This study shows that skeletal muscle may not be the cause of the symptoms associated with fibromyalgia.

Pain Perception

It has been proposed that fibromyalgia is due to faulty pain perception. Pain is thought to be magnified in patients with fibromyalgia. The pain thresholds and pressure pain tolerances on the deltoid and tibia were compared in 46 females with fibromyalgia and 50 healthy controls. Subjects with fibromyalgia were found to have lower pain thresholds and lower pain tolerances than the control group. In addition, fibromyalgia patients typically describe their pain with more pain adjectives and describe more diffuse and burning pain than patients with RA.
Fibromyalgia has been referred to as a "pain amplification syndrome" with exaggerated tenderness at specific sites. Because the tender sites are not located exclusively in muscles, the pain must not be caused by any pathologic or pathophysiologic disorder of the muscles. Tender spots may be caused by any disorder of pain modulation. Factors such as poor sleep, cold exposure, and pain referred from deep structures are all capable of causing a general amplification of pain and tender spots.

Pain modulation impairments in the central nervous system (CNS) may alter pain perception. First, there may be a lack of inhibition of painful stimuli. Second, the disorders in pain modulation cause an increase in the sensitization of the muscle nociceptors. Increased sensitization begins with a disturbance in the muscular circulation. Microcirculation may be affected by factors such as incomplete relaxation between muscle contractions or decreased blood flow during work. The decrease in microcirculation causes sensitization of muscle nociceptors and results in pain at rest. Pain at rest can stimulate activation of the deep pain system causing peripheral sensitization. Persistent peripheral stimulation results in sensitization of pain neurons in the CNS. The sensitization makes it possible for low threshold stimuli to activate the pain system or allows spontaneous activity to occur in the nociceptive nerves.

Fibromyalgia may be caused by disorders in pain modulation. Individuals with fibromyalgia have been found to have decreased pain thresholds and pain tolerances as compared to normal controls. This finding has led to the belief
that disorders in pain modulation in the CNS may be the cause of fibromyalgia. The disorders in pain modulation causing hyperalgesia may occur as a result of abnormal levels of neurochemicals in the CNS.

**Neurochemical**

The role of a metabolic or neuroendocrine abnormality in the etiology of fibromyalgia has been implicated for the past 15 years. There may be an imbalance in the biochemical control of neural transmission in the brainstem, dorsal horn of the spinal cord, peripheral nerves, or autonomic system. Substances that have been proposed to cause symptoms are serotonin, tryptophan, and Substance P.

Serotonin is a neurotransmitter in the CNS needed for the regulation of deep, restorative sleep and the interpretation of painful stimuli. Serum levels of serotonin have been shown to be lower than normal in patients with fibromyalgia. Plasma levels of tryptophan, an amino acid precursor of serotonin, have also been found to be deficient in these patients. The low levels of serotonin and tryptophan may explain the sleep disturbances and hyperalgesia in patients with fibromyalgia.

The relationship of Substance P to the function of the nervous system has also been studied. Substance P is a neuropeptide thought to aid in the transmission of nociceptive information. Substance P dampens the discharges of sensory nerves when the level of serotonin is normal. If serotonin is deficient, it is unable to provide control over the nerves.
Theoretically, this results in an exaggerated response to normal stimuli. Serum levels of Substance P have been found to be elevated in the cerebral spinal fluid of patients with fibromyalgia. The combination of decreased serotonin and increased Substance P is believed to result in an exaggerated perception of normal sensory stimuli. It also suggests that pain and sleep disorders associated with fibromyalgia are both related to serotonin deficiency and occur at the same time, rather than one causing the other.

Abnormalities in the function of neurotransmitters, such as serotonin, tryptophan, and Substance P may cause many of the symptoms of fibromyalgia. Abnormal levels of these substances may contribute to the sleep disorders and increased sensitivity associated with this disorder. A better understanding of these neurochemicals will aid in the development of an effective treatment for this disorder.

**Psychological Factors**

The conflicting evidence regarding a pathophysiological explanation has led to the consideration of a psychological cause. Fibromyalgia has been considered to be a type of conversion or somatization disorder. However, there is no concrete evidence that fibromyalgia is caused by any psychiatric or psychological factors.

Studies have not found any relationship between psychological disorders and fibromyalgia. The National Institute of Mental Health’s Diagnostic Interview Schedule was given to 31 fibrositis patients, 14 RA patients, and four controls.
An association of depression with fibromyalgia was found, but it was not a causal relationship. The conclusion reached was that the majority of patients with fibromyalgia do not have psychopathology and are not likely to benefit from high doses of antidepressants or psychotherapy.\textsuperscript{40}

In addition, a similar study\textsuperscript{45} found no difference between patients with fibromyalgia, patients with RA, or controls with regard to frequency of lifetime diagnosis of any psychiatric disorder, such as major depression or anxiety-based disorders. The patients with fibromyalgia did not show a more significant history of depression than RA patients or controls.

From these studies, it appears that fibromyalgia is not caused by any psychological factor. If a patient does experience psychological problems, it is more likely that these problems occur as a result of fibromyalgia rather than cause it.\textsuperscript{19}

**Sleep Disturbances**

Abnormal sleep patterns seem to be the most commonly accepted etiology for fibromyalgia.\textsuperscript{15} The common theme of sleep disturbances was demonstrated in a survey in which 95\% of patients with fibromyalgia complained of waking tired, 100\% complained of waking with aching and stiffness, and 100\% complained of feeling tired throughout the day.\textsuperscript{13} Since there is almost universal occurrence of a sleep disturbance, it appears that sleep may be a substantial factor in the etiology of fibromyalgia. However, the role of sleep
disturbance as a causative factor is not entirely clear. Researchers do not know if it is a primary or secondary pathogenic factor.\textsuperscript{40}

Moldofsky and co-workers\textsuperscript{46} were the first to show that sleep disturbances are capable of producing fibrotic tender points in normal volunteers. Patients with fibrositis syndrome and healthy subjects undergoing Stage 4 sleep deprivation were studied. Both groups showed alpha rhythms in non-rapid-eye-movement (NREM) sleep. Alpha activity in NREM sleep normally accounts for about 20% of sleep; in fibromyalgia patients, this ratio may be as high as 60% to 80%.\textsuperscript{40} During the experiment, the healthy subjects undergoing sleep deprivation showed alpha rhythms in NREM sleep and they experienced temporary musculoskeletal symptoms, mood disturbances, morning stiffness, and increased point tenderness similar to those of the fibrositic patients.

An especially interesting finding of this study was that patients who were physically fit and maintained their level of exercise throughout the study experienced no symptoms or increased tenderness while deprived of sleep. This finding is significant because it indicates that exercise may help prevent this condition or it may help to control the symptoms if already present. This point will be discussed in further detail in the chapter on management.

These studies suggest the presence of an internal arousal system, triggered by trauma or disturbing emotional problems, causing alpha wave intrusion into normal delta wave Stage 4 sleep. This may increase fatigue, anxiety, and musculoskeletal aching. These factors may perpetuate the
disturbed sleep cycle. Moldofsky\textsuperscript{46} doubts that sleep disturbance is the primary cause of fibromyalgia, but it likely plays a role in perpetuating the symptoms. Therefore, successful treatment may lie in improving sleep physiology.

Throughout the years, many hypotheses have emerged regarding the etiology of fibromyalgia. Research results show that there is conflicting evidence regarding the etiology. It appears that there is no single etiological or pathophysiological factor that produces a causal relationship. Instead, several factors may interact to produce the symptoms of fibromyalgia. Further research is needed to determine the specific cause of fibromyalgia. Until this evidence is produced, the treatment of this disorder will continue to be a process of trial and error.
CHAPTER IV
MANAGEMENT

Voltaire describes the art of medicine as the ability to keep the patient entertained while the disease runs its inevitable course. This quote seems to perfectly describe the treatment effort applied to patients with fibromyalgia. The patients undergo physical, pharmacological, and sometimes psychological therapy in order to find a successful way to manage the symptoms of their disorder. Treatment has been found to be the most effective in patients who have been diagnosed correctly. The longer the symptoms have been present and the longer a person has been suffering from a state of chronic pain, the less the chance of response to treatment and recovery.

The goals of treatment are to provide patients with a means to manage their musculoskeletal pain, to decrease the severity of their symptoms, and to eliminate their dependence on medical services. Several options for the treatment of fibromyalgia exist. These include aerobic conditioning, acupuncture, transcutaneous nerve stimulation (TENS), biofeedback, and psychotherapy. Medications have also been attempted to control the symptoms. A typical program of treatment involves patient education, medication, physical therapy, and exercise.
Patient Education

Following diagnosis, the first step in the treatment of fibromyalgia is to educate and reassure the patient. It is helpful to include friends and family members in this process as their support is extremely important. The etiology and course of the disease should be thoroughly explained along with symptoms the patient might experience. This information could be included in an educational brochure.

Patients should be informed of the chronic nature of this syndrome and must understand that even though there is no cure for this syndrome, there are ways in which they can control their pain and increase physical functioning. The education process should stress the importance of active patient participation in the treatment program to discourage dependence on the therapist or on any passive modalities. The patient needs to be taught to avoid potential exacerbating factors, such as stress, anxiety, and overexertion.

Patients should recognize that changes in lifestyle and job may be necessary to decrease physical and mental stress. These changes should encompass body mechanics, posture, and ergonomics. Rest and relaxation are necessary at work and at home. Several short breaks should be taken throughout the day to change posture and decrease static muscle loading. Stretching exercises can be performed during the breaks. By making these changes, the individual with fibromyalgia is more likely to be able to perform his or her tasks at home or in the work place.
Medications

There is no single "miracle drug" capable of eliminating the symptoms of fibromyalgia. However, a group of drugs known as the tricyclic agents are capable of decreasing symptom severity.\textsuperscript{21} The success of the tricyclics is based on the premise that the etiology of fibromyalgia is caused by a sleep disturbance.\textsuperscript{46}

The tricyclics function primarily by increasing serotonin which helps to induce a deeper sleep and prevent early morning awakening.\textsuperscript{24,42} The patient benefits from increased quality and quantity of sleep.\textsuperscript{23} The tricyclics also have indirect muscle relaxant properties; they decrease motor neuron efferent activity at a supraspinal level.\textsuperscript{40} The most commonly used tricyclics are amitriptyline (Elavil) and cyclobenzaprine (Flexeril).\textsuperscript{16,23,24,40}

Amitriptyline and cyclobenzaprine have the basic structure of the tricyclic antidepressants and have a similar method of action.\textsuperscript{43} Amitriptyline has antidepressant properties in high doses, but in low doses it is effective at improving sleep, relaxing muscles, and producing analgesia.\textsuperscript{25} By improving sleep, it indirectly improves mood and decreases pain.\textsuperscript{42} Cyclobenzaprine is a tricyclic similar to amitriptyline, but has minimal antidepressant effects.\textsuperscript{49} It functions primarily as a muscle relaxant\textsuperscript{49} and is mildly effective at treating this disorder.\textsuperscript{23,49} Amitriptyline is usually preferred to cyclobenzaprine.

Non-steroidal anti-inflammatory drugs (NSAIDS) such as ibuprofen and naproxen are typically used to decrease inflammation and produce analgesia.
Since inflammation is not present in this disorder, NSAIDS are of limited value in fibromyalgia treatment.\textsuperscript{16,24,30} However, they may be helpful at decreasing the severity of pain at local areas and relieving chronic pain and stiffness.\textsuperscript{23,30}

A study comparing the effects of the tricyclics and NSAIDS found amitriptyline to be more effective than naproxen, but naproxen was more effective than placebo. The most effective therapy was the combination of both drugs, which gave an additive benefit.\textsuperscript{43} Similar enhancement by an NSAID was noted in a study comparing alprazolam and ibuprofen for the treatment of fibromyalgia.\textsuperscript{50} Alprazolam is a benzodiazepine derivative with anxiolytic and antidepressant effects. It has similar properties to amitriptyline and is better tolerated\textsuperscript{43} but more addictive.\textsuperscript{30} The greatest improvement in pain and in the patient rating of disease was with the ibuprofen and alprazolam group as compared to the ibuprofen and placebo, alprazolam and placebo, and double placebo groups.\textsuperscript{50}

Physical Therapy

Patients diagnosed with fibromyalgia are often referred to a physical therapist. A physical therapist is a health professional trained in the use of modalities, stretching and strengthening principles, cardiovascular exercises, and methods to restore muscle and joint mobility. The physical therapist will be able to help the patient establish a program of exercise that can be carried out independently.
When a patient is first seen in physical therapy, the initial goal is to decrease pain and relax tight muscles. This may be accomplished through the use of moist heat, high intensity galvanic stimulation, progressive relaxation training, or massage. The second goal is to teach the patient normal neuromuscular functioning. Instruction in posture and body mechanics will help to decrease stress placed on muscles. Gentle prolonged stretching will help the muscle return to normal resting length. EMG biofeedback is useful to teach the patient when muscles are contracting during postural movements.\(^\text{16}\)

Once a patient is seen for physical therapy, it is common for him or her to overutilize this resource. Modalities are often used at the patient’s request, despite inadequate studies to prove their benefit.\(^\text{30}\) Modalities, such as massage, acupuncture, ultrasound, and electrical stimulation, exhibit only a temporary effect at decreasing symptoms.\(^\text{30,31}\) The only documented evidence for the improvement of symptoms is with cardiovascular fitness training\(^\text{47,51}\) and EMG biofeedback.\(^\text{52}\) Controlled studies utilizing other modalities need to be done in order to justify their use.\(^\text{40}\) Because dependence and overuse of modalities eat up a large portion of the patients’ health care budget, they should only be used in the acute stage or in conjunction with an active exercise program.\(^\text{30}\)

**Exercise**

Exercise as a possible treatment or preventor of fibromyalgia was first proposed by Moldofsky\(^\text{46}\) in 1975. As previously discussed, this sleep
deprivation study demonstrated difficulty producing tender points in participants who were physically fit and were exercising at the time of the study. Therefore, it was hypothesized that physical fitness, especially cardiovascular training, may benefit patients with fibromyalgia.

Patients with fibromyalgia typically have decreased muscle tone and poor physical fitness. More than 80% of fibromyalgia patients scored below the average level of fitness as based on statistics from age and sex matched controls. It has been proposed that the chronic fatigue associated with this syndrome results in a sedentary lifestyle and a decrease in the fitness level of skeletal muscle. Consequently, the muscle is more susceptible to microtrauma which produces the pain associated with the disorder.

Aerobic exercise benefits the individual in the following ways. It may: (1) increase the resistance to microtrauma, (2) improve an individual's sense of control over his or her body, (3) improve circulation within muscle, and (4) improve mental attitudes. It is believed that cardiovascular training may decrease the symptoms of fibromyalgia through the activation of central or peripheral beta-endorphin systems or the release of adrenocorticotropic hormones (ACTH). During exercise, beta-endorphin is always co-released with adrenocorticotropic hormone (ACTH). Elevated beta-endorphin levels have been tied to changes in pain and affective ratings in long-distance runners. Release of ACTH and cortisol occurs in response to any stress; exercise greater than 60% of maximal oxygen consumption is considered to be
a stress. The release of beta-endorphin, cortisol, or ACTH with exercise is believed to cause the changes in pain ratings and mood experienced in people who exercise.\textsuperscript{54}

Several studies have proven that aerobic exercise is beneficial. A cardiovascular fitness training program was compared to a flexibility program in patients with fibromyalgia. Two studies by McCain and colleagues\textsuperscript{47,52} showed that a cardiovascular fitness program was superior to a flexibility program at decreasing the symptoms of fibromyalgia. Patients in the cardiovascular group reported the following changes after 20 weeks in the program: decreased pain ratings, decreased level of disease activity, improvement in psychological profiles, and improved cardiovascular fitness. These results were far more significant than the results of the flexibility group. Neither group, however, experienced a change in the percent of body area affected or improved sleep disturbances. These findings suggest both types of exercise may decrease the symptoms of fibromyalgia, but cardiovascular training produces better results. Other studies\textsuperscript{54,57} have demonstrated similar results attesting to the benefits of aerobic exercise, but results have not been as significant as McCain's.

A home program of exercise should be initiated early in the rehabilitation program to prevent dependence on passive modalities. The home program should include stretching, strengthening, and endurance exercises. To increase compliance, the exercises should be simple and enjoyable and should not necessitate major changes in lifestyle. The exercises should be demonstrated
to the patient and be provided in written form. The exercise prescription should include the number of repetitions, frequency, duration, contraindications, and instructions for progression for all types of exercise.

There are several guidelines that should be considered in the prescription of an exercise program. The exercise program should begin gently and in the pain-free range. Activity should be continually increased but kept within pain tolerance. Rest periods, if necessary, are important. Exercises that increase muscle tension, such as weight lifting, are not tolerated as well and should be introduced gradually.

In summary, the best treatment of fibromyalgia requires multiple strategies including education, behavior modification, modalities, physical conditioning, and a comprehensive approach to wellness. The patient should be an active participant in the treatment program. The most significant ways in which patients can help themselves are to: (1) become better educated in the cause of fibromyalgia, (2) remain active and try to gain an increased level of physical fitness, and (3) try to identify and eliminate exacerbating factors.
CHAPTER V
CONCLUSION

Fibromyalgia is defined as a form of soft tissue or muscular rheumatism primarily affecting muscles and their attachments to bones. Within the past two decades, this syndrome has been recognized by rheumatologists as the third most common rheumatic disorder after RA and OA. Fibromyalgia affects millions of people in all walks of life, but generally occurs most frequently in middle-aged females.

The exact cause of fibromyalgia is unknown. Several theories have been proposed to explain the etiology and pathophysiology of this syndrome including muscle abnormalities, abnormal pain perception, psychological factors, neurochemical imbalances, and sleep disturbances. Many different factors may trigger fibromyalgia and produce the pain, fatigue, and sleep disturbances associated with this syndrome. Triggering factors may include physical or emotional trauma, inactivity, overexertion, poor sleep, and weather changes.

The most common symptoms of fibromyalgia are sleep disturbances, stiffness, and generalized pain. Other symptoms that may be experienced include headaches, abdominal pain, irritable bowel syndrome, hypersensitivity to heat or cold, anxiety, depression, or reticular skin discolorations. Diagnostic
criteria proposed by the American College of Rheumatology include widespread pain (bilateral, axial, and above and below the waist) of more than three months' duration and tenderness to palpation at 11 or more of 18 tender points.

In general, the most effective treatment program is one in which the patient assumes an active role in the treatment program. Treatment is aimed at providing the patient with a way to manage his/her musculoskeletal pain, decrease severity of symptoms, and eliminate dependence on medical services.

With proper treatment, most people with fibromyalgia improve to some degree and are able to live with their disease. However, more research is needed to better understand the etiology of this syndrome. This research will allow the development of a more effective treatment plan and possibly even prevention of this disorder.

The current treatment program for fibromyalgia involves: (1) patient education, (2) medications to improve sleep and decrease pain, (3) physical therapy to normalize muscle tone and restore function, and (4) exercise to increase cardiovascular fitness and strengthen muscles. The exercise program should meet the needs of the patient and be designed specifically for each individual.
REFERENCES


