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Tender Points/Fibromyalgia vs. Trigger Points/Myofascial Pain Syndrome: A Need for Clarity in Terminology and Differential Diagnosis

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ABSTRACT

Objective: This study reviews the clinical distinctions between fibromyalgia (FM) and myofascial pain syndrome (MPS), which represent two separate and distinct soft-tissue syndromes. The major aim of this article is to clarify the terminology associated with these syndromes and clearly define the parameters of differential diagnosis and treatment.

Data Sources: Pertinent articles in the chiropractic and medical literature are reviewed with an emphasis on the literature published from 1985–1994.

Study Selection: Studies were selected that emphasized differential diagnosis of FM and MPS, as well as individual articles on either FM or MPS.

Data Synthesis: The literature on fibromyalgia and myofascial pain syndromes has grown considerably since 1985. It is now clear that there are several important differences between FM and MPS. The most important criteria for differential diagnosis are the presence of *tender points (TePs)* and widespread, nonspecific, soft tissue pain in FM, compared with regional and characteristic referred pain patterns with discrete muscular *trigger points (TrPs)* and taut bands of skeletal muscle in MPS. The etiology of TePs is still unknown and it is uncertain which specific soft tissues are tender in FM patients. Myofascial TrPs are found within a taut band of skeletal muscle and have a characteristic "nodular" texture upon palpation. TrPs are thought to develop after trauma, overuse or prolonged spasm of muscles. Local treatment applied to TePs is ineffective, yet specific treatment of TrPs is often dramatically effective.

Conclusion: FM and MPS are two different clinical conditions that require different treatment plans. FM is a systemic disease process, apparently caused by dysfunction of the limbic system and/or neuroendocrine axis. It often requires a multidisciplinary treatment approach including psychotherapy, low dose antidepressant medication and a moderate exercise program. MPS is a condition that arises from the referred pain and muscle dysfunction caused by TrPs, which often respond to manual treatment methods such as ischemic compression and various specific stretching techniques. Both of these conditions are seen routinely in chiropractic offices; therefore, it is important for field practitioners to understand these distinctions. (J Manipulative Physiol Ther 1995; 18:398–406).

Key Indexing Terms: Fibromyalgia, Myofascial Pain Syndrome, Myofascitis, Fibrositis, Chiropractic, Trigger Point, Tender Point.

INTRODUCTION

There is a growing interest within the chiropractic profession toward the management of soft-tissue disorders. Postgraduate courses are being taught on topics such as sports injuries, trigger point therapy, muscular stretching techniques and soft-tissue rehabilitation.

Putting aside the "routine" soft-tissue injuries, such as ligament sprains, tendinitis and cartilage injuries, the literature supports the notion that there are two predominant soft-tissue syndromes seen in clinical practice: fibromyalgia (FM) and myofascial pain syndrome (MPS). Unfortunately, there seems to be confusion about the distinction between these two conditions. Only a few well-written reviews have been published in the chiropractic literature regarding the differential diagnosis of FM and MPS (1–3).

Much of the confusion comes from literature published before 1980. In the older literature, authors often took the liberty of interchanging the use of terms such as fibrositis, nonarticular rheumatism, myalgia, myofibrosis, etc. Many authors erroneously mixed the terms tender point and trigger point and commonly failed to distinguish between syndromes with widespread aching pain (FM) and those with regional, specific, referred pain patterns (MPS). The term "fibrositis" was abundantly used to describe anything from widespread nonarticular pain to specific muscle "hardenings" and probably was erroneously used to describe MPS patients and FM patients.

Rheumatology researchers were especially interested in discovering an etiology for an unknown syndrome characterized by chronic aches and pain in multiple locations throughout the musculoskeletal system. It was initially hypothesized that the soft tissues of these patients were inflamed by some type of systemic disease process, similar in nature to rheumatoid arthritis: hence the term "fibrositis." However, soft-tissue biopsy studies failed to uncover any histological evidence of inflammation and therefore the term "fibrositis" began to seem like a misnomer.

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Table 1. *Criteria for the diagnosis of fibromyalgia*[a]

For a patient to be diagnosed with Fibromyalgia, he/she must fulfill the two criteria listed below. In addition, there must be no signs or symptoms indicative of another systemic condition, such as rheumatoid arthritis, gout, hypothyroidism, lupus, etc. The widespread pain must have been present for at least three months before diagnosis.

1. **History of widespread pain for at least 3 months.**
   Definition: Pain is considered widespread when:
   (a) Pain is on both sides of the body;
   (b) Pain is above and below the waist;
   (c) Axial skeletal pain is present (neck, chest, thoracic or low back).
2. **Pain in 11 of 18 tender point sites on digital palpation.**
   Definition: Pain upon digital palpation of approximately 4 kg of pressure. A tender point must be painful to palpation, not just "tender'. Of the following 18 tender point sites, 11 must be painful:
   Occiput: at the suboccipital muscle insertions.
   Low cervical: at the anterior aspects of the intertransverse spaces C5–C7.
   Trapezius: at the midpoint of the upper border.
   Supraspinatus: above spine of scapula near medial border.
   Second Rib: upper lateral aspects of the 2nd costochondral junction.
   Lateral Epicondyle: 2 cm distal to the epicondyles.
   Gluteal: in upper outer quadrants of buttocks in anterior fold of muscle.
   Greater Trochanter: posterior to the trochanteric prominence.
   Knee: at the medial fat pad proximal to the joint line.

[a] Adapted from Wolfe et al. (5).

Table 2. *Diagnostic criteria for myofascial pain syndrome*[a]

To diagnose a MPS, *all* five major criteria should be present, and at least *one* of the three minor criteria.

Major criteria:

1. Regional pain complaint.
2. Pain pattern follows a known distribution of muscular referred pain.
3. Palpable taut band (in accessible muscles).
4. Exquisite focal tenderness at one point or nodule within taut band.
5. Some restricted range of motion or slight muscle weakness (when measurable).

Minor criteria:

1. Manual pressure on TrP nodule reproduces chief pain complaint.
2. Snapping palpation of the taut band at the TrP elicits a Local Twitch Response.
3. Pain is diminished or eliminated by muscular treatment, e.g., therapeutic stretch, ischemic compression or needle injection of the TrP.

[a] Adapted from Simons et al. (4).

In 1981, the term "fibromyalgia" was introduced as the definitive term for this clinical syndrome of widespread soft-tissue pain, replacing the outdated term "fibrositis." This new term is more clinically accurate because the syndrome is an *-algia* (hypersensitivity phenomenon) and not an *-itis* (or inflammatory phenomenon). Simons published an excellent review of the terminology associated with the muscle pain literature and outlined the changes in these terms since 1975 (4).

The 1980s was a decade marked by a flurry of research activity in rheumatology circles regarding widespread nonarticular pain. Several large consensus conferences were held that culminated in a 1990 position paper that provided for a clear definition and diagnostic criteria for the diagnosis of fibromyalgia (Table 1) (5). In 1983, Travell and Simons published the first volume of their classic work *Myofascial Pain and Dysfunction: The Trigger Point Manual* (6). This text clearly outlined the diagnostic criteria for MPS (Table 2), established a definition for myofascial trigger points and provided detailed descriptions of the characteristic referred pain patterns associated with specific muscles. The second volume of Travell and Simons' texts was released in 1992 and further added to the growing literature on myofascial pain syndromes (7). In 1993, the first issue of The Journal of Musculoskeletal Pain was published by Haworth Press in Binghamton, NY. This multidisciplinary journal was devoted exclusively to research articles on fibromyalgia and myofascial pain syndrome.

There are two basic differences between FM and MPS. First, FM is characterized by the presence of *Tender Points (TePs)*, which are defined as discrete areas of soft tissue that are painful to about 4 kg of palpatory pressure. In contrast, the *Trigger Points (TrPs)* that are found in MPS are defined as hyperirritable spots located within a taut band of skeletal muscle that are painful upon compression and give rise to characteristic referred pain and autonomic phenomena. The differences between TePs and TrPs will be discussed at greater length elsewhere in this article; however, for now it is important to realize that the abbreviation "TP" is being abandoned in the literature, because it fails to differentiate between trigger and tender points.

Secondly, FM is a systemic condition of unknown etiology, in which the patient is "sore all over." By definition, fibromyalgia has widespread, bilateral pain all over the body, including the torso, upper extremities and lower extremities. On the other hand, MPS is characterized by a regionalized pain syndrome that is usually unilateral and does not usually affect both upper and lower extremities simultaneously. Treatment applied to the TePs of FM patients gives little or no relief of symptoms, whereas specific myofascial techniques applied to TrPs will result in dramatic and often immediate relief of pain in MPS patients.

The next two sections of this article will briefly review the diagnostic criteria associated with FM and MPS (Table 3).

DISCUSSION

Fibromyalgia

FM is not a disease, but rather a *syndrome*. It is a clinical condition based on a common set of characteristic symptoms. As previously discussed, FM is characterized by two major symptoms: (a) widespread pain for greater than 3 months and (b) the presence of a defined number of tender points (TePs). Table 1 provides the currently established definition and criteria for making a diagnosis of FM. Prevalence of FM is about 10 to 20 times greater in women than in men; no reason is yet known for this disparity.

Typically, the FM patient presents with a primary symptom of bilateral, widespread musculoskeletal pain and stiffness that at first might seem to be rheumatoid arthritis or another systemic arthritic condition. Yet there is no true joint swelling or

Table 3. *Differential diagnosis of FM and MPS*

Symptom	Fibromyalgia	Myofascial Pain Syndrome
Pain pattern	Bilateral and widespread	Regional: specific referred pain patterns
Morning fatigue	Yes	No
Sleep disorder	Yes; strong correlation with FM	Sometimes; secondary to pain and discomfort of MPS
Soft tissue findings	Tender point	Trigger point
Palpable changes	None	Distinct "nodularity" over TrP; Palpable, taut "ropy" bands with associated local twitch response
Female: Male ratio	10–20:1	1:1
History/presentation	Chronic, widespread pain; morning fatigue, stiffness and pain with no known cause	History of acute or chronic muscle strain or injury; regionalized pain
Treatment approach	Treatment is systemic: • Low dose anti-depressants • Aerobic exercise • Psychotherapy • Manipulation may be helpful via CNS relaxation response or other neural effects	Treatment is specific and local: • Ischemic compression • Therapeutic stretches • Needle injection/physiotherapy • Manipulation for associated joint dysfunction/ subluxation
Muscle metabolism	Impaired *systemically* (?)	Impaired *locally*

edema, no radiographic evidence of joint destruction or degeneration and no laboratory confirmation of an elevated sedimentation rate, uric acid or rheumatoid factor. Also, 90% of patients state that they experience symptoms of fatigue, nonrestorative sleep and general stiffness upon rising in the morning.

During physical examination, the patient will be found to be extremely sensitive to manual pressure applied over soft tissues in many areas of the body. By using a pressure threshold meter or algometer, one can determine the presence of tender points (TePs) at the locations noted in Table 1 (8).

Historically, there was much debate and discussion among researchers as to how many TePs were necessary to make a diagnosis of FM. Even the anatomical locations or sites of the TePs themselves were hotly debated. During the course of the 1980s, when FM research was still evolving, several anatomical TeP "maps" and "count" criteria were proposed for the diagnosis of FM (9), which varied from 3–5 TePs out of a total of 40 locations to 12 TePs out of a total of 14 locations. The anatomical sites or locations of TePs were chosen because they represent common sites of pain and swelling in patients with systemic arthritic conditions. TePs do not conform to any specific muscle, tendon or ligament.

To gain a greater appreciation of the earlier fibromyalgia literature and the widely divergent positions held by the major researchers, one should obtain the proceedings of the 1985 World Symposium on Fibrositis/Fibromyalgia (10) and 1990 Advances in Pain Research and Therapy, Volume 17 (11). Both of these references are compilations of numerous articles that provide the reader with a wealth of knowledge regarding the evolutionary process that culminated in the establishment of FM as a new diagnostic entity. The present TeP criteria, 11 out of 18 sites, was established by a consensus process through the American College of Rheumatology and was published in 1990 (5).

Although there are only two major criteria for establishing a diagnosis of FM, widespread pain of three months duration and a minimum number of TePs, there are several other clinical symptoms associated with FM. Most FM patients have an associated sleep disorder in which they experience a disruption in REM sleep. Moldofsy studied the EEG activity of FM patients during sleep and found anomalous EEG patterns that correlated with FM patients and their measurements of morning pain and stiffness (12).

Interestingly enough, low doses of tricyclic antidepressant medications such as amitriptyline and cyclobenzaprine (Flexeril) have a significantly positive effect by reducing the number of TePs and relieving the morning stiffness of FM patients (13–15). These drugs are known to alter EEG activity and induce deeper low-wave sleep. Other studies have concluded that nonsteroidal anti-inflammatory drugs and systemic corticosteroids have no effect on FM symptoms (16. 17), which further supports the contention that FM is not an inflammatory condition.

The fact that antidepressant medications cause such a pronounced reduction of the morning pain and stiffness of FM patients led to the hypothesis that FM might be a disorder of pain modulation or muscle metabolism, secondary to psychological stress. The "stress hypothesis" also gained some ground when it was shown that FM patients, as a group, showed higher statistical levels of anxiety and depression when compared with normal control groups (18). An issue of great debate at present is whether anxiety and depression are etiological *causes* of FM or merely concomitant *symptoms*.

FM patients often experience many other symptoms that might be attributed to increased sympathetic nervous system activity, such as Raynaud's phenomenon, which is noted in 20–40% of FM patients. There are several other functional symptoms that are highly associated with FM, including irritable bowel syndrome, irritable or "nervous" bladder, headaches, atypical numbness and tingling sensations. Again, these associated symptoms have led many researchers to hypothesize that increased emotional stress may play a significant role in

the genesis of FM through increased limbic system or reticular activating system activity.

In 1992, the Second World Congress on Myofascial Pain and Fibromyalgia was held in Copenhagen, Denmark and the proceedings were published as a special double volume issue of the Journal of Musculoskeletal Pain (19). This conference concluded with a position paper and consensus document known as the Copenhagen Declaration, which summarizes the current position on FM by stating that:

> Fibromyalgia is a painful, nonarticular condition predominantly involving muscles; it is the commonest cause of chronic, widespread musculoskeletal pain. It is typically associated with persistent fatigue, nonrefreshing sleep and generalized stiffness. Women are affected some 10 to 20 times more often than men. Fibromyalgia is often part of a wider syndrome encompassing: headaches, irritable bowel syndrome, irritable bladder, dysmenorrhea, cold sensitivity, Raynaud's phenomenon, restless legs, atypical patterns of numbness and tingling, exercise intolerance and complaints of weakness. A varying proportion [20–50%] of fibromyalgia patients experience significant depression or anxiety which may contribute to the severity of symptoms or result from having chronic pain. Most fibromyalgia patients experience both diurnal and seasonal variations of symptoms. Typically, symptoms are worse during periods of cold damp weather, at the beginning and end of the day and during periods of emotional stress (19).

Myofascial Pain Syndromes

In 1952, Dr. Janet Travell gave a presentation at a pain research symposium that would later be published as a textbook chapter entitled *Pain Mechanisms in Connective Tissues* (20). She discussed her clinical observations and research findings that indicated the existence of predictable patterns of referred pain from muscles that were experimentally injected with noxious hypertonic saline solution.

She went on to propose the term "Trigger Area" to describe the clinical phenomenon in which one area of soft tissue was stimulated and another remote area of skin or soft tissue was affected. Travell suggested that myofascial tissues were somatic "organs," similar to visceral organs, in that each had its own individual and reproducible pattern of referred pain and that central nervous system reflex pathways were involved in the process. She found empirically that needle injection of procaine into the trigger areas could often eliminate pain immediately.

In 1957, Dr. Raymond Nimmo published his first article, titled *Receptors, Effectors and Tonus—A New Approach*, in which he proposed his Receptor-Tonus Theory (21). Nimmo suggested that the soft tissues of the body, especially the muscles, were the source of most patients' pain. He described his clinical observation that when certain areas of muscle were pressed, pain was elicited in areas far from his palpation. He also found empirically that deep manual pressure applied to these "sore spots" had a dramatic therapeutic effect by rapidly eliminating the pain.

Both Travell and Nimmo were simultaneously describing the same clinical phenomenon, myofascial pain syndrome (MPS). With MPS, the patient experiences a distinct pattern of regionalized referred pain from a specific muscle or group of muscles. In more chronic or complex cases, the patient may present with an overlap of several referred pain patterns that give the diagnostic illusion of widespread pain. The patient with an *acute* MPS will not generally describe his/her pain as "widespread." However, patients with a *chronic* case of MPS may have multiple TrPs and, hence, pain in multiple locations ("widespread"), which may be mistakenly diagnosed as FM.

Clearly, MPS is not a systemic condition like FM. Patients with an MPS present with a history of acute or chronic muscle strain and a characteristic pain pattern referred from a specific muscle. The pain of MPS is often provoked whenever the patient uses the affected muscle or joints controlled by that muscle. The criteria for making the diagnosis of MPS are listed in Table 1, as discussed earlier.

MPS is characterized by the presence of trigger points (TrPs) that palpate as "knots" or "nodules" that are found within taut bands; these bands have a distinct palpable texture commonly described as "ropy." In most cases, when a TrP is firmly pressed with moderate palpation, it will trigger referred pain and/or autonomic phenomena in a distant location. In addition, vigorous palpation of the TrP or the taut band will cause a local twitch response (LTR) within the muscle fibers of the taut band.

Travell and Simons have revolutionized the field of MPS with the publication of the two volumes of *Myofascial Pain and Dysfunction: The Trigger Point Manual*, in which they provide detailed descriptions and drawings of the characteristic referred pain patterns for all skeletal muscles in the human body. Knowledge of these referred pain patterns and the ability to perform a palpatory examination of muscle tissue for TrPs is essential to proper diagnosis of MPS. Although chiropractors typically excel in palpation, the ability to precisely locate TrPs and taut bands is a special psychomotor skill that requires training and repetition, much like learning the art of spinal manipulation/adjusting.

Typically, a patient with MPS presents to the clinician with a history of pain that may at first seem poorly localized, but is usually limited to one region of the body, e.g., shoulder and upper extremity, head and neck, low back/buttock, etc. However, if carefully queried, the patient will usually point to an area that he/she feels is the painful "source." The pain may be related to a traumatic injury in which the muscles were damaged either by strain or overload. Chronic symptoms may alternately be related to microtrauma from repetitive strain, poor posture and other latent causes of chronic muscular tension.

The formation of TrPs usually relates to some form of damage to muscle cells, either from *gross trauma* such as a strain injury or from *microtrauma* such as repetitive muscular tension. TrPs are often found in muscles that lie within the

scleratogenous referred pain zones of inflamed joints, myofascial referred pain zones of other TrPs, or even within the dermatomal referred pain zone of an inflamed nerve root. For example, it is common to find TrPs in the gluteal and piriformis muscles of patients with sciatic radiculopathy. There is some connection between TrPs and the central nervous system that is still poorly understood.

Although the presence of TrPs, taut bands and a specific referred pain pattern are essential to the diagnosis of MPS, several other examination findings are also worthy of mention. Patients with MPS will experience pain or stiffness when the affected muscles are stretched or strongly contracted in the shortened position. Often the affected muscles are physiologically inhibited and may seem "weak" when the patient attempts maximal contraction against resistance. One will also observe a reduced range of motion of the joints moved by the affected muscles, because the tense muscle fibers of the taut band(s) will not allow for a complete stretch of the muscle.

As with FM, MPS patients show no diagnostic imaging or laboratory findings indicative of a systemic disease process. However, TrPs are often found in the muscles surrounding joints afflicted with rheumatoid or degenerative arthritis; therefore, MPS is often found in association with systemic arthritic conditions. It is also important to note that TrPs are very commonly found in association with joint dysfunction/subluxation, especially in the muscles that are the prime movers of those dysfunctional joints.

Lewit has proposed that most musculoskeletal pain syndromes are caused by a combination of articular and muscular dysfunction; therefore, both osseous and soft tissue manipulative techniques are necessary for proper treatment (22). Bogduk and Simons recently showed a high correlation between the referred pain patterns from TrPs in the neck muscles and the scleratogenous patterns from the cervical zygapophyseal joints (23). They also conclude that, in clinical practice, joint and muscle dysfunction often go hand in hand and that it is important for the clinician to be able to differentiate between primary joint pain/dysfunction and primary muscle pain/dysfunction (TrP).

This relationship between joint and muscle dysfunction has been well known to the chiropractic profession in the form of segmental "taut and tender fibers" around the region of the vertebral subluxation/fixation. Vernon et al. showed that cervical manipulation caused a significant increase in the local paraspinal soft tissue pain threshold level, as documented by algometry (24). This study is important because it documents the common clinical finding that spinal manipulation can cause reflex relaxation of paraspinal muscles.

Trigger Points vs. Tender Points

It is extremely important to understand that the terms trigger point (TrP) and tender point (TeP) refer to two separate and distinct anatomical entities.

To review, a TeP is defined as a discrete area of soft tissue that is painful to digital pressure at an approximate force of 4 kg, which is about the amount of pressure it takes to blanch a fingernail. There is no definitive agreement at this time as to what specific soft tissue(s) are affected by the TeP or the mechanism by which they become tender. Many TePs may actually be TrPs, because all TrPs have the tenderness of TePs. It is also interesting to note that the 18 predetermined TeP sites are found directly over muscles known to be sources of common TrPs, confounding the issue even further.

It is known that FM patients are more "tender" everywhere, not just at the 18 TeP sites; this hypersensitivity may be thought of as a peripheral sign of some central disturbance within the nervous or neuroendocrine systems. This theory is substantiated by studies of cerebrospinal fluid that have detected elevated levels of substance P and decreased levels of serotonin (25) and serum studies that have detected lower levels of somatomedin-C in FM patients (26). Somatomedin-C has profound effects on muscle metabolism and is produced in response to the amount of circulating growth hormone. Almost all growth hormone is produced during deep stage-4 sleep, at which time serotonin levels are also replenished.

Extremely low doses of tricyclic antidepressants such as amitriptyline, given at bedtime, have the well-documented effect of reducing the number of TePs in FM, increasing the levels of brain stem serotonin and improving the quality of deep sleep. These data suggest that the sleep disorder associated with FM somehow alters the sensitivity of peripheral soft tissues to subthreshold stimuli and results in the appearance of TePs.

TePs do not exhibit any hard or nodular texture; neither do they have any distinctive texture that distinguishes them from surrounding soft tissues. EMG studies show no increased electrical activity anywhere within the region of the TeP. There is no associated taut band of muscle tissue and because TePs seem to be the manifestation of a systemic condition, local treatment of the TePs themselves has no therapeutic effect. However, if the examiner has mistaken a TrP for a TeP, there will be a "nodule" and local treatment will be effective, but only because a TrP (not a TeP) was treated inadvertently.

On the other hand, local treatment of TrPs by ischemic compression, spray and stretch technique, postisometric relaxation and/or injection often gives immediate relief of both the local pain over the TrP itself and the referred pain and autonomic phenomena associated with the TrP. In contrast with FM and TePs, there is no oral medication that is known to significantly reduce the number of TrPs in MPS patients.

A TrP is characterized by a very distinctive "nodular" texture upon palpation and is associated with a specific referred pain pattern when stimulated with sufficient pressure. A TrP is found within a taut, ropy band of skeletal muscle during cross-fiber palpation of a muscle belly and will elicit an LTR, in which fibers of the taut band will rapidly twitch whenever snapping palpation is applied over the TrP. The LTR and taut bands are not found in association with palpation of TePs.

The TrP nodule itself is thought to be a region of localized muscular contracture, in which a subset of muscle fibers are locked by failure of the actin/myosin heads to release. Simons has developed an "energy crisis" hypothesis, in which he

theorizes that the "lumpy" TrP nodule is actually a focal area of contracted sarcomeres, maintained by localized ischemia and metabolic deficiency (27). This model was developed to explain previous CMG studies that found no increased electrical activity from within the muscle fibers of the TrP itself.

However, a recent study by Hubbard and Berkoff found spontaneous spikes of increased electromyography (EMG) activity when a fine needle electrode was placed precisely within the TrP nidus (28). This increased EMG activity would continue as long as the needle electrode remained within the TrP nidus, but decreased immediately if the needle was moved even a few millimeters away from the TrP. The authors hypothesized that TrPs were maintained by some type of reflex loop with the central nervous system and that the electrodes were detecting EMG activity of the intrafusal fibers of muscle spindles.

Previous studies have detected increased EMG activity only in taut bands, not from TrPs; therefore, this study is very important and may have profound implications for future research into the TrP phenomenon. The authors explain their findings by stating that there may be a new, previously unrecognized *sympathetic* motor pathway to the intrafusal fibers of the muscle spindles. This would allow for sympathetic control of muscle tension independent from the standard alpha and gamma motor neuron pathways.

Although the TrP nodule feels like a single focus of contracted muscle tissue, recent studies have led to a new hypothesis (29, 30). The trigger "point" may actually be a trigger "area," composed of multiple sensitive loci within the muscle tissue. When a needle injection is performed in the region of a TrP, several small areas of needle-point-sized sensitive loci can be detected so that each elicits a separate and distinct LTR. This new research is exciting, for it again raises the possibility that the TrP phenomenon may be related to changes in muscle spindle activity and the central nervous system.

The most important distinction between TePs and TrPs is the issue of cause and effect. It seems that a TeP is an effect or symptom(s) of a systemic dysfunction of unknown etiology. In and of itself, the TeP does not cause the syndrome of FM; rather, the syndrome of FM is characterized by a certain number of TePs.

A TrP, however, is the causal agent in MPS. Although it is still unclear as to the precise mechanism underlying the TrP phenomenon, it is known that the TrP itself is a noxious focus on muscle tissue that is the generating source of the referred pain and autonomic phenomena seen in MPS. Treatment applied locally to the TrP will eliminate the pain and motor dysfunction symptoms found in this syndrome.

It is common for those inexperienced with myofascial trigger point therapy to mistake TePs for TrPs and vice versa. A confounding factor relates to the phenomenon of *referred tenderness*, in which the soft tissues within a TrP referred pain zone seem to be sensitized. For example, a TrP in the quadratus lumborum muscle may refer pain and tenderness into the soft tissues of the buttock. Palpation of the buttock muscles may detect what seem to be TePs; in reality, these "tender points" are merely the symptom of *referred tenderness* secondary to TrPs in the quadratus lumborum.

Most of the anatomical locations for TePs are found in areas that overlap the referred pain zones and locations of some of the most common TrPs in MPS. Without this knowledge, the unwary examiner may easily mistake areas of myofascial referred tenderness and/or TrPs for TePs. For this reason, it is imperative that the clinician who practices soft tissue therapy be well-versed in the diagnosis and treatment of both FM and MPS and understand clearly the differences between TePs and TrPs.

A Common Denominator Between TrPs and TePs?

At the present time, it seems that TrPs and TePs have more differences than similarities. However, many studies have attempted to detect some underlying disturbance in muscle metabolism or central nervous system control of the motor system that is a common denominator in both FM and MPS.

Needle biopsy studies have recently been performed on FM patients in which samples of muscle tissue surrounding a TeP were examined by light and electron microscopy (31, 32). Structural changes such as "moth-eaten fibers," mitochondrial changes and Type II atrophy were found in these biopsy studies, indicating the possibility of some disturbance of muscle microcirculation as the cause. The electron microscopy study also showed some alteration in muscle capillary endothelium, similar to changes seen in ischemic conditions. However, these nonspecific muscle changes still do not explain how or why the microcirculation is impaired in FM patients. A more crucial problem is that these studies did not assure that the biopsy sites were definitely TePs, not TrPs mistakenly diagnosed as TePs.

Biopsy studies of muscle tissue taken from "fibrositic nodules" (TrPs?) also reveal interesting structural changes. Awad examined the biochemical exudates found in the extracellular fluids around TrPs withdrawn by needle biopsy (33). He found serotonin, bradykinins and hyaluronic acid, which indicated damage to the sarcolemmal membrane of muscle cells. Fassbender also performed electron microscopy studies on TrP biopsy samples taken from patients in different stages of chronicity and pain intensity and found distinct structural changes that correlated with chronicity (34). The acute cases showed some myofilament destruction and mitochondrial swelling, and the most chronic cases showed complete destruction of sarcomeres and fibers, necrosis and fibrosis in the muscle cells and collagen and scar accumulation. These studies show that there are definite degenerative changes that occur in muscle cells within the TrP and that they seem to be related to ischemia and metabolic impairment.

If there is a common denominator between TePs and TrPs, it has not yet surfaced in the literature. It is becoming increasingly clear that the TeP concept is difficult to substantiate via a microcirculation disturbance model. It is quite likely that TePs will be shown to be peripheral, hyperalgesic zones caused by altered modulation of pain perception in the brain. In short, FM patients may have a lowered threshold to painful stimuli

throughout their soft tissues and the TeP locations may just be convenient sample sites for measuring this hyperalgesic state.

On the hand, TrPs show definite signs of a local disturbance in microcirculation, because systemic disturbances such as anemia and hypothyroidism will aggravate and further sensitize TrP activity. Because red blood cells carry oxygen to muscle cells, anemias will worsen the local ischemic condition in TrPs, especially during any increased activity of the muscle.

Thyroid hormones T3 and T4 both affect energy production and consumption in all cells throughout the human body. T3 has an especially strong effect on muscle metabolism and significantly increases the production of mitochondrial ATP. Because TrPs are thought to be perpetuated by impaired ATP production, it is clear how low thyroid output can directly affect muscle metabolism locally within the region of the TrP.

Nutritional factors may also play a role in the pathogenesis and treatment of MPS. Travell and Simons devote nearly an entire chapter of their text to a discussion of nutritional imbalances that adversely affect muscle metabolism (6). It is well known, for example, that patients on diuretic medications that flush out minerals such as calcium, magnesium and potassium are very susceptible to muscle cramps. These patients respond very well to oral mineral supplements.

A recent development that does lend some credence to the microcirculation model of FM is a study in which FM patients seem to respond well to oral supplements of malic acid and magnesium. The authors of this study propose that FM symptoms may be attributable to a deficiency of certain nutritional substrates necessary for ATP synthesis and a breakdown of muscle proteins by the body to enhance gluconeogenesis (35). Both magnesium and malic acid play extremely important roles in glycolysis and the Kreb's cycle; therefore, it is plausible that deficiencies could impair muscle cellular function and lead to impaired ATP synthesis. Another study showed decreased magnesium levels in the red blood cells of patients with symptoms very similar to those of FM patients (36).

Although these studies show some promise, they must be considered preliminary in nature, because it is still unknown if these metabolic changes are the *cause* of FM or merely an associated clinical sign. Regarding MPS, it is known that metabolic and endocrine imbalances may perpetuate or worsen TrP activity, but are not the ultimate cause of TrP formation. Obviously, the chiropractic model of treating "the whole person" becomes very pertinent to any discussion of treatment options for both FM and MPS.

It should now be apparent that there are major differences in the clinical presentations of FM and MPS. Both conditions are common and likely to be found daily in chiropractic practice. FM is now considered by rheumatologists to be the most common cause of chronic, widespread musculoskeletal pain. FM patients represent only about 6% of the patients found in a primary health care facility but may make up to 20% of the patient population in a rheumatology clinic (37). It is reasonable to predict that the patient population of a chiropractic clinic is very similar to a rheumatology clinic.

MPS is even more common than FM and is probably found in the vast majority of chiropractic patients. Sola examined the shoulder muscles of a group of 200 military recruits and found TrPs in 54% of the females and 45% of the males (38). Skootsky examined a series of 172 patients who entered a primary care clinic with the symptom of "pain." He found that MPS was the most common diagnosis (30%) and represented the single most common reason for these patients (with unknown pain) to visit their primary care physician (39). Fricton examined almost 300 patients with chronic head and neck pain who presented to a dental clinic and found that the vast majority (55%) had MPS as their primary diagnosis (40).

It can not be emphasized strongly enough how prevalent both FM and MPS are likely to be in chiropractic practice. The private practice setting of most chiropractors is likely to be full of patients with these two conditions, which presents an enormous opportunity for research into the effectiveness of conservative chiropractic treatment. Both conditions fall within the scope of chiropractic practice; however, FM is more likely to require a multidisciplinary approach due to its systemic nature.

The present standard of care and management of FM patients is a multidisciplinary approach that relies on three points. The first is psychotherapy and patient education. Because FM is strongly associated with depression and anxiety, psychotherapy or cognitive restructuring can help these patients to cope better with their stress and normalize their sleeping patterns. Also, merely educating FM patients about the fact that the condition can be treated and is not life threatening can reduce patient anxiety about FM itself.

The second approach is to normalize the sleeping disorder through the use of extremely low dosages of antidepressants, usually amitriptyline or cyclobenzaprine. Both of these medications have shown a statistically significant effect in reducing the number of TePs, with patients reporting noticeably less muscle pain and stiffness in the mornings. Nonsteroidal anti-inflammatory drugs and corticosteroids have also been studied but are absolutely ineffective in the treatment of TePs and FM and should not be used.

The third approach is exercise and manual therapies, including chiropractic. Several studies have shown that moderate aerobic exercise can have a beneficial effect on FM patients, possibly because of endorphin release and better sleeping habits associated with exercise. Aerobic exercise is also thought to increase oxygenation and circulation to muscle tissue. Manual therapies such as myofascial release techniques, massage, ischemic compression and stretching may help FM patients by increasing overall muscle tone and flexibility.

Dr. Frederick Wolfe, a pioneer in FM research, surveyed his FM patients and found that rest and relaxation were the most effective "treatments," with 65.7% and 46.8% of patients reporting pain relief, respectively (41). However, Wolfe then reports *chiropractic treatment* as the next most effective therapy, with 45.9% of patients reporting significant relief. Chiropractic treatment enjoys a relatively high patient satisfaction rate and it is quite possible that patients "feel better" both emotionally and physically after a chiropractic visit, which somehow reduces the central nervous system dysfunction associated with FM. Clearly, much more research is necessary

before any actual physiological mechanisms can be understood in this regard.

Chiropractic management of FM patients has not yet been researched to any great extent and represents a open field of study. A fundamental axiom of chiropractic practice has been "structure affects function," that manipulation of musculoskeletal structures can cause normalization of nervous system function. Because FM is presumed to be related to a dysfunction of the central nervous system, chiropractic methods that restore neural homeostasis should show great promise. Clearly, much more research will be needed before chiropractors can make claims of clinical effectiveness with this condition.

It is very important to recognize the importance of the *psychological component* and the *sleep disorder* found with FM patients. The average chiropractor is not skilled in psychological counseling and may inadvertently fail to recognize the subtle signs of anxiety and/or clinical depression that are associated with FM patients. Chiropractors who choose to treat FM should network with a psychotherapist and a medical doctor who can play valuable roles in the multidisciplinary management of these patients by providing psychotherapy and medication when necessary.

MPS patients present a different challenge in the chiropractor. Chiropractors who use only osseous manipulative techniques will have great difficulty when attempting to treat patients with MPS, for the TrPs found in this condition require specific treatment, applied directly to muscle tissue. There are many methods that chiropractors can use to treat TrPs, including ischemic compression (Nimmo), various muscle stretching techniques, ultrasound and electrical currents.

Two studies have substantiated the clinical effectiveness of ischemic compression in treating myofascial TrPs. Garvey studied the effectiveness of TrP injection with lidocaine and/or prednisone, acupuncture (dry needling) and vapocoolant spray with acupressure (ischemic compression) (42). Of the patients who received acupressure/vapocoolant, 67% responded favorably, compared with 61% who received acupuncture and 45% who received needle injection of lidocaine/steroid. Hong compared the effectiveness of four TrP modalities: spray & stretch, moist heat packs, ultrasound and ischemic compression (43). All four modalities were found to decrease the pain of TrPs immediately after treatment; however, ischemic compression was more effective than any other modality.

The common denominator among all TrP therapy modalities is that, in some way, they all release the contracture of taut bands within the skeletal muscle. Techniques such as spray and stretch and postisometric relaxation probably release the taut band by pure mechanical stretching. The stretch will literally pull apart the actin/myosin heads that are held in contracture. Simons believes that ischemic compression may act as a form of local intense stretch on the TrP nidus and thereby mechanically disrupt the contracted actin/myosin heads and release the TrP. If the TrP nodule is infiltrated precisely by a needle, as in dry needling or deep acupuncture, the needle may mechanically disrupt the actin/myosin heads as well.

Most chiropractors find that ischemic compression and specific stretching techniques are easily learned, natural adjuncts to chiropractic practice because they are manual skills that require no specialized equipment. The standard in TrP diagnosis is manual palpation for taut bands and TrP nodules, which are quickly located by chiropractors, who excel at the art of palpation. Many clinicians are already locating TrPs without labeling them as such, instead marking them as "taut and tender muscle fibers" or "muscle hardening/spasm."

CONCLUSION

FM and MPS are very common conditions and require differential diagnosis. FM is a syndrome that presents with a history of widespread pain and a feeling of morning fatigue and stiffness. The TePs found in FM do not exhibit any palpable texture (unless they are really TrPs), only cause local pain and are hyperalgesic zones of nonspecific soft tissues. FM is a disorder of the central nervous system that responds to a systemic, multidisciplinary approach. Treatment that consists only of direct therapy aimed at the individual TePs will not be effective.

MPS is a regionalized pain syndrome in which the patient has discrete TrP nodules that are found in association with taut, "ropy" bands of skeletal muscle. When TrPs are firmly palpated, they will often cause referred pain and autonomic phenomena in a pattern that is characteristic for each skeletal muscle. Snapping palpation of the TrP will elicit a local twitch response in the fibers of the taut band. Specific treatment applied directly over the TrPs will give rapid and often immediate relief of pain.

Chiropractic management of both conditions is based upon a comprehensive understanding of the criteria for diagnosis and treatment. It is important to differentiate between these two conditions and a plea is made for accuracy in use of the terms FM, MPS, TeP and TrP. The terms TrP and TeP are not synonymous, nor are the conditions FM and MPS. These terms should not be used interchangeably.

Traditionally, the chiropractic profession has primarily focused on the joint dysfunction and osseous manipulation. However, this model is evolving into a newer paradigm of practice, in which the importance of muscle dysfunction is becoming recognized and soft tissue techniques are taking a respectable place in chiropractic offices. For those practitioners who intend to manage patients with soft tissue pain, it is essential they understand the distinctions between TePs and TrPs and FM and MPS, which represent by far the two most common conditions seen in private practice. Management of these conditions will also require that the clinician be well-versed in soft tissue manipulative techniques.

The development of a chronic pain syndrome in a patient who has acute TrPs and is mismanaged because of ignorance or neglect is most unfortunate. MPS only becomes chronic when the patient is not properly treated while in the acute stage. This is analogous to the patient who develops degenerative joint disease after being mistreated during the joint dysfunction stage. Once the muscle tissue becomes infiltrated with scar tissue or undergoes necrosis/fibrosis, it becomes much more difficult or impossible to restore normal muscle function.

If we intend to become involved with the conservative care of FM and MPS, we must begin to research the clinical effectiveness of various chiropractic treatment procedures on these conditions. It is hoped that this article will stimulate some debate and discussion toward that end.

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REFERENCES

1. Boline PD, Anderson AV, Berk R. The various components and treatment of fibrositis: a review. Chiro Technique 1989; 1:133–9.
2. Taylor MR. Fibromyalgia syndrome; literature review. AJCM 1990; 3:118–26.
3. Duarte M. Primary fibromyalgia syndrome and myofascial pain syndrome: clinical features and criteria for diagnosis. Chiro Technique 1989; 1:97–100.
4. Simons DG. Muscular pain syndromes. In: Fricton JR, Awad E, eds. Advances in pain research and therapy. Vol. 17. New York: Raven Press, 1990:1–41.
5. Wolfe F, Smythe HA, Yunus MB, et al. The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. Arthritis Rheum 1990; 33:160–72.
6. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual. Vol. 1. Baltimore: Williams & Wilkins, 1983.
7. Travell JG, Simons DG. Myofascial pain and dysfunction: the trigger point manual. Vol. 2. Baltimore: Williams & Wilkins, 1992.
8. Fischer A. Pressure algometry over normal muscles: standard values, validity, and reproducibility of pressure threshold. Pain 1987; 30:115–26.
9. Wolfe F, Hawley DJ, Cathey MA, Caro X. Fibrositis: symptom frequency and criteria for diagnosis. J Rheumatol 1985; 122: 1159–63.
10. Bennett RM, ed. The fibrositis/fibromyalgia syndrome: current issues and perspectives. Am J Med Suppl 1986; 81(3A):1–115.
11. Fricton JR, Awad E, eds. Advances in pain research and therapy. New York: Raven Press, 1990.
12. Moldofsky H. A chronobiologic theory of fibromyalgia. J Musculoskel Pain 1993; 1:49–59.
13. Carette S, McCain GA, Bell DA, Fam AG. Evaluation of amitriptyline in primary fibrositis. Arthritis Rheum 1986; 29:655–9.
14. Bennett RM, Gatter RA, Campbell SM. A comparison of cyclobenzaprine and placebo in the management of fibrositis. Arthritis Rheum 1988; 31:1535–42.
15. Quimby LG, Gratwick GM, Whitney CD, Block SR. A randomized trial of cyclobenzaprine for the treatment of fibromyalgia. J Rheumatol 1989; 19:140–3.
16. Yunus MB, Masi AT, Aldag JC. Short term effects of ibuprofen in primary fibromyalgia syndrome. J Rheumatol 1989; 16:527–32.
17. Clark S, Tindall E, Bennett RM. A double blind crossover trial of prednisone versus placebo in the treatment of fibrositis. J Rheumatol 1985; 12:980–3.
18. Hawley DJ, Wolfe F. Depression in fibromyalgia: a comparative study of 1522 rheumatic disease patients. [Abstract] Scand J Rheumatol Suppl 1992;94 (S88):14.
19. Jacobsen S, Danneskiold-Samsoe B, Lund B, eds. Musculoskeletal pain, myofascial pain syndrome, and fibromyalgia syndrome. Proceedings of the 2nd World Congress on Myofascial Pain and Fibromyalgia. J Musculoskel Pain 1993; 1(3/4):1–324.
20. Travell JG. Pain mechanisms in connective tissues. In: Ragan C, ed. Connective tissues: transactions of the second conference, 1951. New York: Josiah Macy, Jr. Foundation. 1952:86–124.
21. Nimmo RL. Receptors, effectors, and tonus: a new approach. Journal of National Chiropractic Association 1957; Nov.
22. Lewit K. Manipulative therapy in rehabilitation of the locomotor system. 2nd ed. Oxford: Butterworth-Heinemann Ltd., 1991.
23. Bogduk N, Simons DG. Neck pain: joint pain or trigger points? In: Voeroy, Merskey, eds. Progress in fibromyalgia and myofascial pain. New York: Elsevier Science Publishers, 1993:
24. Vernon HT, Aker P, Burns S, Viljakaanen S, Short L. Pressure pain threshold evaluation of the effect of spinal manipulation in the treatment of chronic neck pain: a pilot study. J Manipulative Physiol Ther 1990; 13:13–6.
25. Russell IJ, Baeroy H, Javors M, Nyberg F. Cerebrospinal fluid biogenic amine metabolites in fibromyalgia/fibrositis syndrome and rheumatoid arthritis. Arthritis Rheum 1992; 35:550–6.
26. Bennett RM, Clark SR, Campbell SM, Burckhardt CS. Low levels of somatomedin-C in patients with the fibromyalgia syndrome: a possible link between sleep and muscle pain. Arthritis Rheum 1992; 35:1113–6.
27. Simons DG. Myofascial pain syndrome due to trigger points. In: Goodgold J, ed. Rehabilitation medicine. St. Louis: C.V. Mosby Co., 1988:686–723.
28. Hubbard DR, Berkoff GM. Myofascial trigger points show spontaneous needle EMG activity. Spine 1993; 18:1803–7.
29. Hong C-Z, Torigoe Y. Electrophysiological characteristics of localized twitch responses in responsive band of rabbit skeletal muscle fibers. J Musculoskel Pain 1994; 2:17–43.
30. Hong C-Z. Considerations and recommendations regarding myofascial trigger point injection. J Musculoskel Pain 1994; 2:29–59.
31. Schroder HD, Drewes AM, Andreasen A. Muscle biopsy in fibromyalgia. J Musculoskel Pain 1993; 1:165–9.
32. Lindman R, Hagberg M, Bengtsson A, Henriksson KG, Thornell LE. Changes in trapezius muscle structure in fibromyalgia and chronic trapezius myalgia. J Musculoskel Pain 1993; 1:171–6.
33. Awad EA. Interstitial myofibrositis: hypothesis of the mechanism. Arch Phys Med 1973; 54:449–53.
34. Fassbender HG. Non-articular rheumatism. In: Pathology of Rheumatic Diseases. Stuttgart: Springer Verlag, 1975:303–14.
35. Abraham GE, Flechas JD. Management of fibromyalgia: rationale for the use of magnesium and malic acid. J Nutr Med 1992; 3:49–59.
36. Cox IM, Campbell MJ, Dowson D. Red blood cell magnesium and chronic fatigue syndrome. Lancet 1991; 337:747–60.
37. Masi AT. Epidemiology and natural history: myofascial pain and fibromyalgia. J Musculoskel Pain 1993; 1:113–36.
38. Sola AE, Rodenberger ML, Gettys BB. Incidence of hypersensitive areas in posterior shoulder muscles. Am J Phys Med 1955; 34:585–90.
39. Skootsky SA, Jaeger B, Oye RK. Prevalence of myofascial pain in general internal medicine practice. West J Med 1989; 151 (Aug):157–60.
40. Fricton JR, Kroening R, Haley D, Siegert R. Myofascial pain syndrome of the head and neck: a review of clinical characteristics of 164 patients. Oral Surg 1985; 60:615–23.
41. Wolfe F. The clinical syndrome of fibrositis. Am J Med 1986; 81(Suppl 3A):7–14.
42. Garvy TA, Marks MR, Wiesel SW. A prospective, randomized, double-blind evaluation of trigger point injection for low back pain. Spine 1989; 14:962–4.
43. Hong CZ, Chen YC, Pon CH, Yu J. Immediate effects of various physical medicine modalities on pain threshold of an active myofascial trigger point. J Musculoskel Pain 1993; 1:37–53.