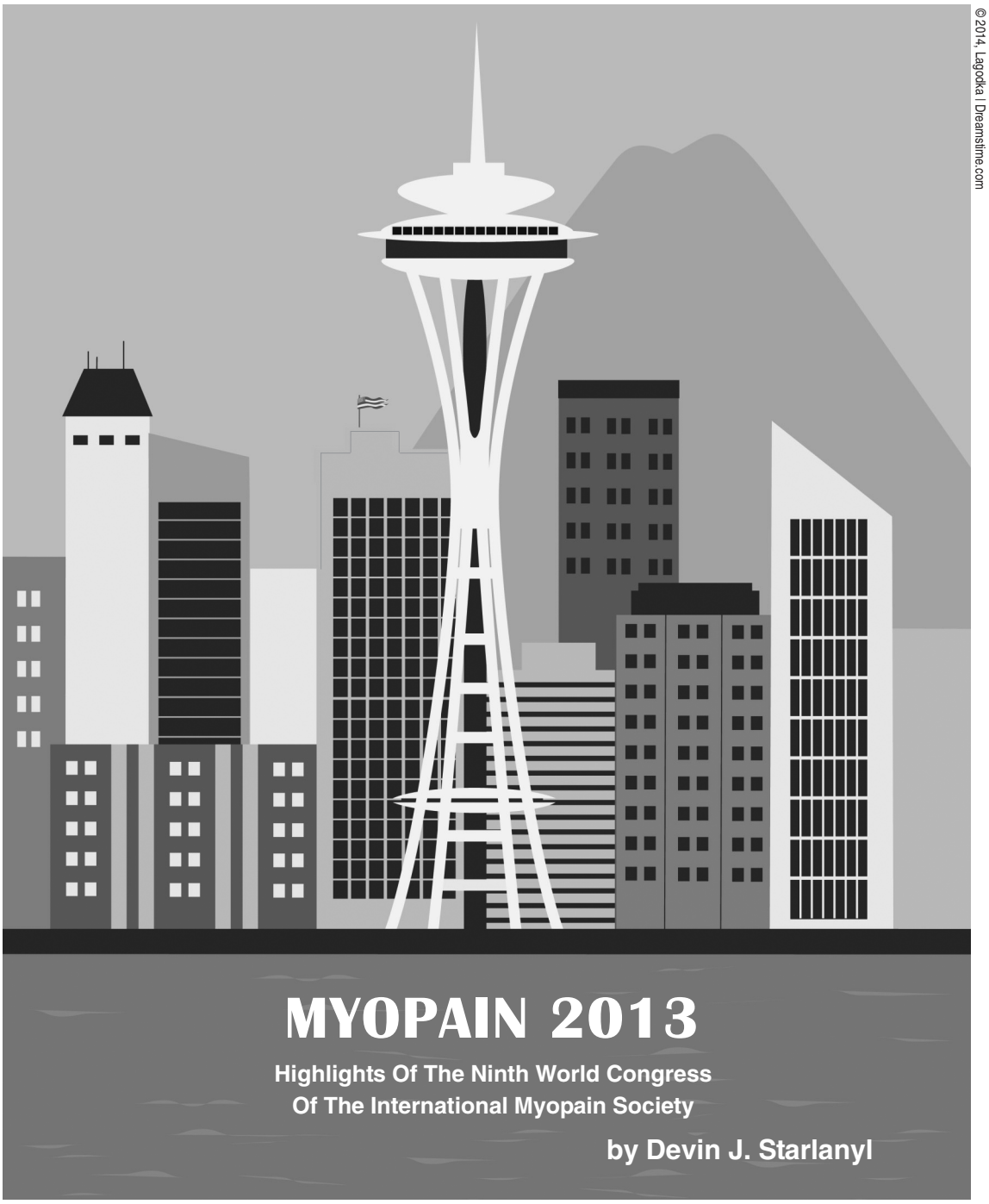


MYOPAIN 2013

Highlights Of The Ninth World Congress Of The International Myopain Society

by Devin J. Starlanyl

The International Myopain Society (IMS) is a nonprofit, international, interdisciplinary group of health care providers, researchers, educators, and others who work to improve care and transform delivery of treatment for soft tissue pain. They are "…dedicated to information exchange relating to recognition, neurobiology, and management of soft tissue disorders".. such as chronic myofascial pain and fibromyalgia.

The IMS holds a World Congress every two years in a variety of member cities, worldwide. The Ninth World Congress was held August 15-18, 2013, in Seattle, Washington, at the Grand Hyatt Hotel. Four days of presentations, papers, question and answer segments, research posters, and workshops offered a wealth of information. The article which follows presents highlights of the proceedings and offers a glossary of technical terms (see page 10).

Key Messages and Direction

by Phillip Mease, M.D., Convention Chair
Department of Medicine, Division of Rheumatology
University of Washington School of Medicine
Seattle WA USA

Fibromyalgia is defined as non-inflammatory, chronic widespread pain (CWP). Non-inflammatory CWP is an important contributor to all rheumatic diseases, therefore it follows that FM is an important part of all rheumatic diseases. Every patient with rheumatic disease should be evaluated for CWP, whatever the primary diagnosis. Every patient with CWP requires evaluation of sleep (poor sleep and depression are independently associated with pain). Remember that there are more treatment avenues than medications alone. Consider all options. For example, cognitive behavioral therapy (CBT) and exercise can help mood, stress, function, and pain levels. By controlling peripheral pain sources, such as arthritis or myofascial trigger points (MTrPs), one can decrease central pain.

Patient education is an important part of treatment and should include the understanding of the shared neurobiology of pain and depression. Patients and prescribers need to know that antidepressants also have an analgesic effect. Because some of the initial research on central mechanisms and pharmaceutical treatment parallels that for some types of mental illness, some of the medications used for depression and similar conditions are also used for central pain, but in different amounts and for other effects.

If centrally-acting agents improve pain and function, care providers must investigate to determine whether the central pain is coming from the primary illness, such as arthritis, MTrPs, cancer, etc.; the primary illness plus FM; or the primary illness plus FM with the addition of mood disorder. All co-existing conditions must be identified and treated.

Patient-related barriers to pain management include:

- the erroneous belief that the level of pain indicates the progress of a rheumatic disease, and that relief of pain will allow the disease to spread without the patient knowing.
- poor adherence to treatment recommendations
- fear and distrust of medications because of side effects, concerns about loss of efficacy of pain treatments, and risk of addiction
- cost of medications

Health care provider-related barriers to pain management include:

- lack of basic and clinical training in pain
- considering pain management of secondary importance to disease management
- additional time investment needed to treat pain
- the belief/disbelief that patients always report pain honestly (is the pain worse, less, or different, and how is it impacting ability to function?)
- concerns about addiction and side effects of medications, especially opioids

Rheumatology professionals need to:

- recognize that chronic pain is a disorder in its own right
- think outside the box of usual rheumatic disorders for treatment options
- manage peripheral pain generators that are not part of the underlying rheumatic disorder
- understand the basics of pain pathophysiology
- learn how to diagnose and treat chronic pain in terms of the underlying pathophysiology

Myofascial Pain: Systemic Review of Treatments

by IMS President James Fricton, D.D.S.
Senior Researcher, HealthPartners Institution for Education and Research
University of Minnesota Head and Neck Pain Clinic
Minneapolis, MN USA

One of the points emphasized in this talk was that the quality of the studies we have available on myofascial pain syndrome (MPS) is poor, often with conflicting results. Dr. Fricton and others are working to standardize criteria for use of terms such as MPS. Patient education is very important, as is a personalized approach and focus on self-care.

In his related poster presentation, Dr. Fricton noted that "The Institute of Medicine has made musculoskeletal pain one of the highest priority areas of research because of its high prevalence and significant functional limitations and disability, missed work, and high health care costs. The most common cause of musculoskeletal pain is myofascial pain."

[*Note:* David G. Simons, M.D., wrote an article on one basic cause of this seeming confusion. (Simons DG.

1995. "Myofascial Pain Syndrome: One Term but Two Concepts; A New Understanding." *J Musculoskeletal Pain* 3(1):7-14.) Some researchers, primarily in dental and mental health, use MPS as synonymous with temporomandibular joint dysfunction (TMJD) or masticatory pain, with ill-defined criteria. Medical researchers generally use MPS to mean myofascial pain caused by trigger points, using criteria of Travell and Simons. Reviews and meta-analyses frequently mix papers using both definitions, leading to mass confusion. For example, one research article may conclude that oral splints are a *major treatment* of MPS, leaving medical readers to wonder how a mouth splint will help their MPS patients with chronic pelvic pain and/or plantar fasciitis caused by MTrPs. DJS]

Overlapping Features and Mechanisms of Myofascial Pain and Fibromyalgia

by Hong-You Ge, M.D., Ph.D.
Associate Professor, Center for Sensory-Motor Interaction
Aalborg University, Denmark

Dr. Ge reported that he and his colleagues became aware that all of their fibromyalgia patients had co-existing myofascial trigger points (MTrPs), although not all patients with MTrPs had FM. They wanted to find out if there were a relationship between the two conditions, and, if so, what it might be. They deduced that there were three kinds of possible relationships (see figure 1) between MTrps and FM:

1. Myofascial trigger points lead to general fibromyalgia.
2. Fibromyalgia may induce trigger points.
3. MTrPs and FM are parallel and co-existing, but not interactive.

Multiple studies were conducted, with the following conclusions:

Study 1: MTrPs contributed significantly to neck and shoulder pain in FM patients.

Study 2: All defined FM tender point sites are MTrPs, either active or latent, and the number of active MTrPs at the 18 tender point sites correlates with spontaneous pain intensity.

Study 3: The FM patients' pain can be reproduced by stimulating active MTrPs.

Study 4: Inactivating MTrPs significantly reduces pain and dysfunction in FM patients.

Study 5: Irritating latent MTrPs can cause the pain and generalized hyperalgesia (amplified pain) that is associated with FM.

Study 6: Irritating a latent MTrP (with glutamate) increased the area of hyperalgesia.

Study 7: Irritating a latent MTrP caused increased sensitivity of another latent TrP, indicating one way satellite MTrPs can form, and how pain and dysfunction can spread to more areas.

Study 8: There is reduced efficiency of reciprocal inhibition due to MTrPs, with increased antagonist muscle activity (contraction of agonist muscle with relaxed antagonist muscle).

Study 9: There is increased synergistic muscle activity due to MTrPs.

Study 10: MTrPs cause muscles to become fatigued more easily, overloading normal muscle fibers in the proximity of MTrPs.

Study 11: MTrPs cause sympathetic hyperactivity generally associated with FM.

Ge et al. also discovered that the MTrPs caused microcirculation problems. They found decreased blood flow around even the latent MTrPs.

The myofascial trigger points also contributed significantly to mental and physical dysfunction in FM patients. The usual MTrP treatment of twice-a-week dry needling proved to be too irritating for the sensitized fibromyalgia patients. Once a week needling was tolerated much better. Symptoms improved with each treatment, except in study controls who received sham needling treatments. After the weekly needling on the most severe MTrPs, the FM patients slept better.

The improvement remained as long as factors contributing to MTrP perpetuation were brought under control. These studies showed that "TrPs play a significant role in the development of FM."

Conclusion: (see figure 2)

1. MTrPs constitute an important and major peripheral pain and other symptoms generator in FM.
2. MTrPs contribute significantly to physical and mental function in FM.
3. Treatment of myofascial trigger points is beneficial to fibromyalgia patients.

FIGURE 1

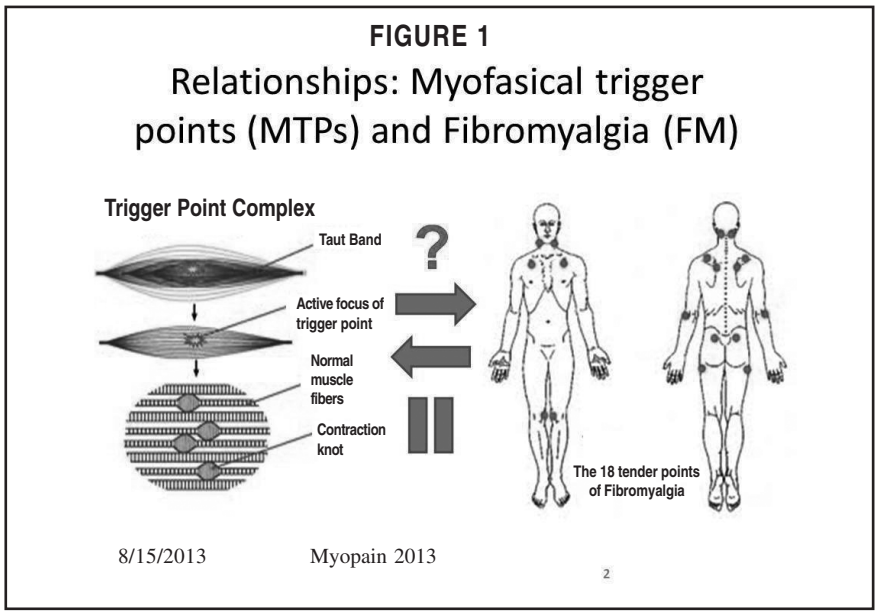
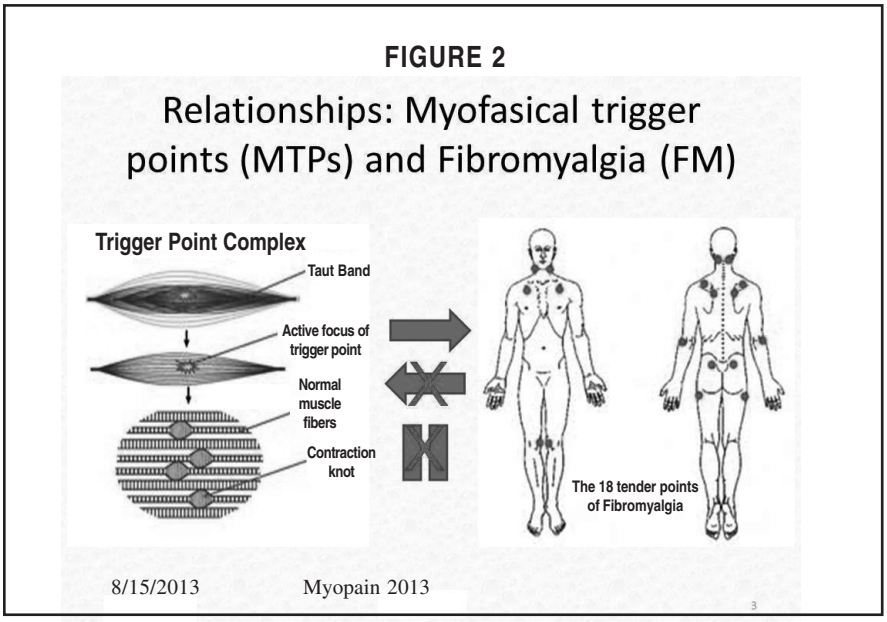


FIGURE 2



These studies indicate that treatment of myofascial trigger points, part of the co-existing condition of chronic myofascial pain, holds the key to successful improvement of many symptoms causing misery to FM patients. This does not indicate that dry needling in any painful location will treat FM. The treatment must be directed to the source of the pain and dysfunction; the myofascial trigger points. They are significant irritants that help maintain altered central nervous system responses to irritating stimuli in fibromyalgia.

In a related poster by Brady, McEvoy, and Dommerholt et al. "Adverse events following trigger point dry needling: a prospective survey of chartered physiotherapists," 39 physiotherapists and 7,629 treatments were included. While mild adverse effects, such as bruising and/or pain during treatment were common, no significant adverse effects occurred. Dry needling is not only effective, it is safe.

Translation Studies and Human Models of Musculoskeletal Hyperalgesia Relevant for Myofascial Trigger Points

by Thomas Graven-Nielsen, Ph.D.
Center for Sensory-Motor Interaction
Aalborg University, Denmark

The mechanisms of pain, referred pain, and the transition from acute to chronic pain are complex. The more we learn about pain, the more we realize that it isn't as simple as we once thought. Boundaries between central sensitization and peripheral sensitization are no longer as clear.

[*Note:* For example, we now know that a process occurs as the central nervous system (CNS) becomes sensitized (central sensitization). This process is called windup, or temporal summation of second pain (TSSP). With prolonged or repeated pain or other irritating stimuli, the brain and spinal cord change. This ability to change is called neuroplasticity. The first time the CNS encounters a nasty stimulus, that is "first pain".

Like a surprised puppy, your CNS metaphorically cringes and tries to recover. If the disagreeable stimulus continues or repeats, or if it is replaced by another irritant, that is "second pain." The CNS puppy, metaphorically again, tenses its little shoulders, quivering and waiting for the next blow to fall. Then it becomes irritable. With each blow, the central nervous system response gets stronger, and the hypersensitivity takes longer to come down, until one day it doesn't come down all the way. Eventually, the CNS can react to irritating stimuli like a junkyard dog on meth. In FM patients, central sensitization takes increasingly less stimulation to occur, and after- effects are greater and more prolonged. DJS]

We are learning more about the processes of:

- how acute pain states transform into chronic pain
- how referred pain may develop
- the mechanisms of how pain messages get to the brain, how the brain filters (or loses the ability to filter) those pain messages, and how it responds to the peripheral tissues
- how delayed onset muscle soreness (DOMS) develops, and
- some of the reasons for deep tissue hyperalgesia; hyperalgesia and referred pain are typical manifestations of MTrPs.

It is the fascia, rather than the muscle itself, that is crucial in DOMS-associated sensitization.[1] A subject feels more pain when there is less time between each sensitizing stimulation. Changes in the way the brain sends messages to the body in response to pain affect increasing central sensitization and TSSP facilitation. So, in cases of irritable bowel syndrome (IBS), FM, vulvodynia, neuropathy, osteoarthritis (OA), tension headache, etc., we don't always know how much of the pain and sensitization is due to dysfunction in the way the brain filters pain messages and responds, and how much is due to central sensitization. We are learning more about what drives the mechanisms of descending pain control (DNIC).

[*Note:* When multiple irritating messages from different areas of the body, often spatially distant, are transmitted to the brain, some of the pain is inhibited or filtered out. The DNIC has a greater effect on second pain than on first pain, so dysfunction in the DNIC system is stronger in patients with central sensitization. This pain modulating effect of the DNIC does not work well in healthy women or in FM patients of both sexes. DJS] [2]

Single myofascial trigger points begin as peripheral pain, outside the central nervous system. They cause peripheral sensitization, and, if allowed to persist, can result in eventual central nervous system (CNS) sensitization. Universal forms of diagnosis of MTrPs are needed. Right now, diagnosis requires palpation, which is difficult to standardize and dependent on variables such as training, experience, and ability. Using an algometer (a pressure tool used to measure tissue resistance) to locate trigger points is not the equivalent of palpation. For example, pressure algometry will miss latent TrPs, which are important causes of dysfunction.

In their studies, Dr. Graven-Neilson and his colleagues used a variety of sensitizing agents, including saline, glutamate, and bradykinin to study referred pain in healthy subjects. Not only muscle, but also visceral organs, joint-related structures, and areas such as the fat-pad of knee, can be involved in referred pain. Sacroiliac ligaments can also cause impressive referral pain patterns. Time (prolonged pain) and intensity of pain are large factors in sensitization and referred pain. About 80% of healthy subjects develop referred pain. We don't yet know why 20% do not.

If we can remove the sources of peripheral pain generation, such as myofascial trigger points, we can normalize central nervous system sensitization.

Referred pain is facilitated in patients with OA, chronic low back pain, fibromyalgia, and whiplash. Chronic myofascial pain may be a frequently coexisting condition in chronic pain conditions. For example, patients with OA have many more TrPs than controls. Dr. Ge has treated several children for TrPs at the ages of five or six.

During the question and answer session, Dr. Robert Gerwin mentioned that spinal cord L5 pain can occur outside typical known referral patterns, and may sensitize the entire spinal cord.

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2. Staud R, Robinson ME, Vierck CJ JR, et al. 2003. "Diffuse Noxious Inhibitory Controls Attenuate Temporal Summation of Second Pain in Normal Males But Not in Normal Females or Fibromyalgia Patients." *Pain* 101(1-2):167-174.

Contribution of Peripheral Factors to Fibromyalgia Pain

Roland Staud, M.D.
Professor of Medicine, Division of Rheumatology and Clinical Immunology, Department of Medicine in the College of Medicine
University of Florida at Gainesville, USA

Studies about fibromyalgia patients have reported vascular abnormalities (Olsen and Park, 1998), impaired capillary microcirculation (Lindman, 1995), and reduced capillary blood flow and oxygenation (Grassi, 1994). When muscle tissues become hypoxic (low in oxygen), unmyelinated pain receptors become activated (Mense and Stahnkke, 1983), muscle pain increases during activity and blood flow decreases (Elvin, 2006), and there is delayed blood flow after activity (Quiao, 1991). Temporal summation of second pain, also called wind-up, increases in FM patients after exercise (Vierk, 2002).

Fibromyalgia pain and hyperalgesia affect peripheral tissues that are not overtly injured but are showing changes. The signature characteristic of FM is **central** sensitization; hyperalgesia (amplified pain) and allodynia (pain from normally non-painful sensations). There are both peripheral and central pathways for pain (see figure 3). Central hyperactivity requires a driving force that causes and/or maintains sensitivity and pain. What is happening outside the CNS in the peripheral areas? What causes and/or maintains fibromyalgia?

Dr. Staud and his colleagues found that the peripheral tissue, not including skin, caused C-Fiber abnormalities in chronic pain. Peripheral tissue irritation, including pain, caused the hyperalgesic priming leading to temporal summation of second pain (TSSP), also known as wind-up, which leads to the central nervous system sensitization seen in fibromyalgia and other central sensitization states. Abnormal activity of peripheral and central pain pathways is fundamental to the development and maintenance of chronic pain. Once central sensitization is established, it requires minimal stimuli to sustain (see figure 4).

Their work indicates that peripheral stimuli, such as those caused by myofascial trigger points, initiates and maintains central sensitization. Reduction of peripheral pain

FIGURE 3

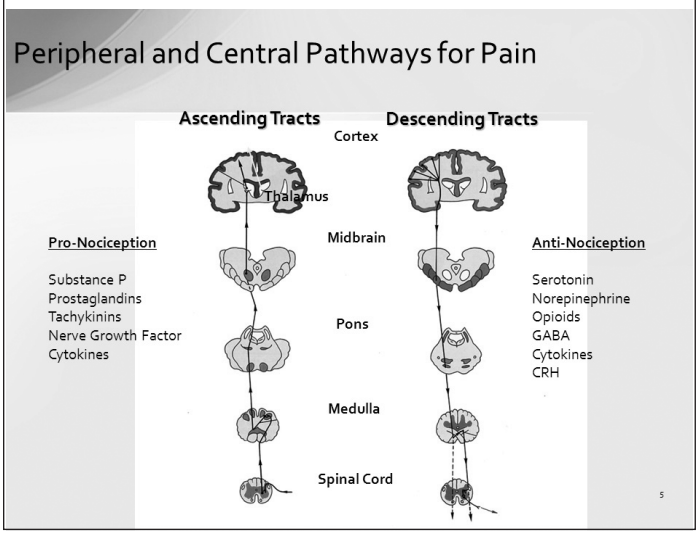
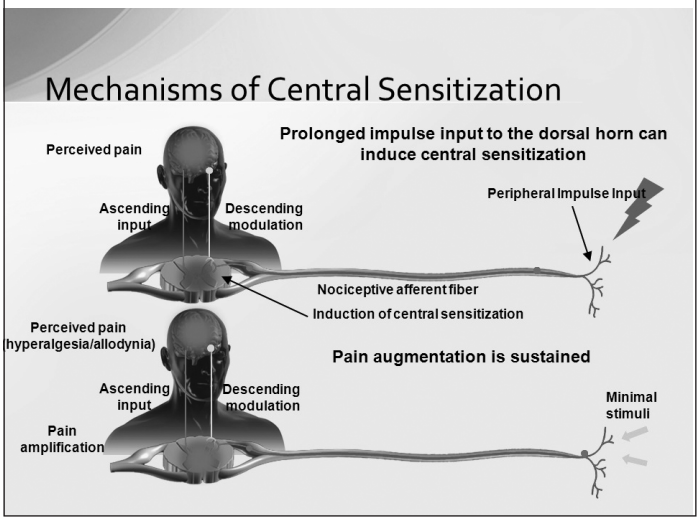


FIGURE 4



may improve FM hyperalgesia and pain. Grey matter in the brain changes after the cessation of pain. This can mean that the neuroplasticity that allows central sensitization is reversible. When the cause of pain is removed, brain atrophic changes recover, even after a year and longer, although there are also genetic contributions to chronic pain.

Myofascial trigger points of the neck and shoulder are important pain-initiating factors, especially tonically (tight muscle) maintained pain, in FM. Injection of MTrPs using nonsteroidal, lidocaine electrophoresis resulted in clear and significant reductions in pain, even distant pain, and tender point score in FM patients, even when only injecting the four worst areas. There was no systemic effect from the lidocaine, just local effects on the MTrPs, so the reduction of hyperalgesia was not due to systemic lidocaine. The pain reduction was maintained for at least seven days.

There is a lot of overlap in chronic pain types, but the studies of Staud et al. show that one can reduce central sensitization by reducing or abolishing peripheral noxious input. Myofascial trigger points can significantly impact chronic pain. Peripheral sensitization is critical in the development of FM.

The effectiveness of NSAIDS is not conclusive in FM. There are groups of individuals with FM who do respond to opioids, so we must identify those and use opioids responsibly. In the question and answer session, Dr. Staud answered: "You can treat patients peripherally, and over time the central pain will resolve." This offers great hope for FM patients.

Improving Access to Evidence-Based Non-Pharmaceutical Management of Fibromyalgia and Chronic Pain

David A. Williams, Ph.D.
Professor of Anesthesiology, Medicine (Rheumatology), Psychiatry, and Psychology
University of Michigan at Ann Arbor, USA

This presentation emphasized the importance of patient self-management, empowerment, the use of non-pharmaceutical intervention (such as exercise and cognitive behavior therapy), and providing educational resources for chronic pain patients. Cognitive behavioral therapy (CBT) had its origins in talk therapy to change temperament and behaviors. Chronic pain patients can learn to cope better, advocate for themselves, and manage their own treatment. Patient empowerment is not the same as hand-holding. Guides, tools, and teachers are needed. Many skills can be learned to improve function and diminish pain, including learning to find joy and have fun in life in spite of chronic pain conditions. Tools include relaxation response, sleep hygiene, exercise tailored to the patient's changing abilities, and learning to schedule pleasant activity. This often requires lifestyle changes that involve removal of barriers to needed change.

FIGURE 5

Pain Medicine Versus Pain Management: Ethical Dilemmas Created By Contemporary Medicine and Business

John D. Loeser, MD* and Alex Cahana, MD, PhD*

"Despite the talk about evidence-based medicine (EBM), the primary driving force behind changes in health care has become economics."

*Loeser J, Cahana A. (2013) *Clinical Journal of Pain* 29(4):311-316.

Courtesy of David A. Williams, M.D.

Interventions can include stress management, goal setting, structural problem solving, reframing, and communication skills. This requires changes on the part of the patient, the care providers, and the system. Currently, interventional pain medicine has an established business model, but the system of interdisciplinary pain management does not. We are still figuring out how to develop a business model around interdisciplinary pain management and patient self-management. "Despite the talk about evidence-based medicine (EBM), the primary driving force behind changes in health care has become economics."

* * * * *

There were question and answer remarks relevant to the topics so far reported. **Robert Gerwin, M.D.,** stressed that both fibromyalgia and chronic myofascial pain are interrelated, and it is important for care providers of both conditions to understand that the two often co-exist not only with each other but also with other soft tissue conditions. They need to know both. We cannot exclude myofascial trigger points or any peripheral pain sources during FM management, and FM central sensitization must be taken into consideration during MTrP treatment. The combination of MTrP pain and dysfunction generation and FM amplification can result in considerable pain, which must be adequately managed.

Daniel Clauw, M.D., mentioned that MTrPs are part of a continuum to FM. The system must be changed so that primary care providers can be reimbursed for the time it takes to listen to chronic pain patients and find out what is needed. For example, Medicare and other insurance often will reimburse for cognitive therapy but not for treatment of myofascial trigger points. Policy makers need to get the message.

James Fricton, D.D.S., spoke of how very little preventive work is being done on chronic pain. How do we engage primary care docs about predisposing factors of chronicity? All of the resources are going to the chronic pain front lines, putting out fires. There isn't enough attention on adequate treatment of acute pain to prevent the conflagration of chronic pain. This must change.

Genome-Wide Peripheral Blood Gene Expression Profiling in Fibromyalgia

by Sunil Kurian, Ph.D.
Staff Scientist, Department of Molecular and Experimental Medicine
The Scripps Research Institute
La Jolla, CA USA

Fibromyalgia causes persistent widespread pain, fatigue, stiffness, disrupted sleep, and cognitive difficulties. It is frequently accompanied by anxiety and/or depression, further causing functional impairment of daily activities. Recent clinical and research findings classify FM as a central neurobiological sensitivity syndrome. There can be comorbidity with illnesses characterized by systemic inflammation, such as chronic hepatitis C infection, autoimmune diseases such as RA and SLE.

[In these cases, the other condition may be the primary condition, may co-exist with myofascial trigger points, and may be an interactive and perpetuating factor of both FM and MTrPs. DJS]

All current diagnoses of FM rely on the ACR research criteria for FM, which includes soft tissue pain of greater than > 3 months duration and pain at least at 11 of 18 paired designated tender points. The 2010 preliminary criteria remove some subjectivity and incorporate some non-pain symptoms such as fatigue, cognitive impairment, and sleep disturbances. Other pain-related diagnoses were excluded to establish these diagnoses, making it difficult to use these criteria on patients with multiple conditions. So we have two major sets of criteria that have no reliable objective lab test to make the diagnosis, but researchers are looking for FM biomarkers.

Functional neural imaging is helping, reflecting how the brain processes the sensory experience of pain. Included are fMRI, positron emission tomography (PET), and single-photon emission computed tomography (SPECT). The fMRI images have shown the most promise, revealing that clinical pain intensity corresponds with an increase in cerebral blood flow, which has shown to respond favorably to drug treatment. Right now, studies suffer from small sample sizes, prohibitive costs, and problems standardizing the techniques. No high sensitivity and specificity as yet has been found. Researchers are also looking at gene profiling and molecular diagnostics. The differential expression profiles of the individual cell subsets show little in common, suggesting that each subset has a unique set of genes. There is just so much we don't know. FM is not homogeneous. There may be many genetic subsets in FM, and there may be a combination of genes causing the tendency to develop FM, or even several combinations of genes.

Deconstructing the Fibromyalgia Phenotype

by Robert Bennett M.D., FRCP, FACP
Professor of Medicine and Nursing
Oregon Health and Science University
Portland OR, USA

Stedman's Medical Dictionary defines a phenotype as the manifestation of the genetic constitution of an individual. Researchers have been on a hunt for "the" gene responsible for FM. Dr. Bennett is a doctor who understands both FM and chronic myofascial pain due to trigger points. He asked us to consider some important questions. Do people understand fibromyalgia? In the light of our evolving understanding, is the concept of FM dead? What will we think of FM 10 years from now? He reminded us that definitions must be based on a careful analysis of up-to-date evidence.

Then he took us through the history of FM. This included reminding us that the American College of Rheumatology (ACR) definition of 1990 was intended for research use only, and that the 2010 criteria for FM is preliminary. Is the concept of fibromyalgia dead? Not at all. There is simply a need to redefine our understanding of the basic traits of FM, so that it concurs with the all of the new information we have at hand.

Definition of Terms

agonist muscle: a prime mover muscle that is in a state of contraction to perform a specific task

antagonist muscle: a muscle relaxing to allow its agonist to contract to perform a specified task

allodynia: pain sensation from normally non-painful stimuli such as touch, smell, noise or lights

central nervous system (CNS): The brain and spinal cord. The remainder of the nervous system is called the peripheral nervous system.

central sensitization: sensitization of the central nervous system

cognitive behavioral therapy (CBT): a psychotherapy approach that uses behavior modification to change dysfunctional patterns of thoughts and feelings. This is based on talk therapy.

cytokines: These are informational biochemical proteins produced by many types of cells. They include lymphokinins, interleukins, and chemokines. They work to regulate the immune modulation of the cells through their cytokine network. Some are anti-inflammatory and some are pro-inflammatory, and none are specific to fibromyalgia.

delayed onset muscle soreness (DOMS): muscle soreness and/or pain that begins hours or even days after the provoking activity.

descending noxious inhibitory control (DNIC): the DNIC prevents the brain from being overloaded by multiple pain stimuli. Thus using one noxious stimulus, such as ice, to block pain stimuli.

dysfunction: abnormal, impaired, or incomplete function. Trigger points can cause dysfunction such as blurred vision, reduced lung tidal volume, dizziness, or muscle weakness, in addition to, or instead of, pain.

fibromyalgia: chronic widespread pain condition associated with hyperalgesia, allodynia, unrestorative sleep, cognitive deficits, and other global symptoms.

peripheral pain generators: sources of pain which are located outside of the central nervous system

reciprocal inhibition: a neuromuscular reflex that allows the antagonist muscle to relax so that its counterpart on the opposite side of the joint, the agonist muscle, can contract safely. The contracture of the trigger-pointed muscle can weaken the other muscles using this mechanism, causing dysfunction.

sympathetic hyperactivity: when the sympathetic nervous system, the part of the autonomic nervous system which prepares the body for stress or emergency, is unusually sensitive or overly responsive

visceral organs: body organs (heart, liver, intestines) located in the large cavity of the trunk

Terms for Myofascial Pain

myofascial trigger points: a hyper-irritable nodule found in a taut band in the myofascia. The nodule is painful on compression, and can cause characteristic referral patterns of pain and associated dysfunction. MTrPs have been imaged at the Mayo Clinic and the National Institutes of Health, and their patterns documented by many research articles and in medical texts.

active trigger points: TrPs that are always: tender, prevent full muscle lengthening, cause muscle weakness, and produce a spontaneous clinical pain complaint. They may be accompanied by associated autonomic and proprioceptive phenomena.

attachment trigger points: These trigger points are found in the area of the tendon/muscle attachment, in the tendon muscle, or both, or in the muscle near a bony attachment. Attachment trigger points are more likely to become calcified, and are often more difficult to treat manually, than are trigger points in the belly of the muscle.

dry needling: the use of mechanical stimulation by acupuncture needles on trigger points without injection of local anesthetic

latent trigger points: TrPs that do not cause spontaneous pain, but have the other characteristics of an active trigger point.

palpation: touching the body with the hands and fingers with the intent to acquire information on the health of the tissues, including changes in texture and the presence of taut bands, barriers, and trigger points.

referral pattern: a recognizable configuration of pain or altered sensation and/or dysfunction that is associated with one or more myofascial trigger point(s). The location of referral patterns for given trigger points are, in general, anatomically similar in the body from patient to patient.

satellite trigger points: TrPs that form in response to the activity of a primary TrP. These TrPs can occur when a muscle is being recruited to perform a task normally done by another muscle weakened by TrPs, in addition to its normal task, or they can occur in a referral pattern.

Overview of Fibromyalgia

I. Jon Russell, M.D., Ph.D.
Editor, *Journal of Musculoskeletal Pain* and retired Professor of Medicine/Rheumatology, University of Texas Health Science Center, San Antonio, Texas USA

Fibromyalgia (FM) is a medical condition of central nervous system dysfunction associated with chronic widespread pain, lowered pain threshold (allodynia, hyperalgesia), and typical co-existing conditions. It is common in the general population. Some people are genetically predisposed to it, and it is biochemically mediated with inflammatory features such as increased cytokines. Any psychiatric symptoms are unrelated to somatic symptoms, and management is no longer difficult.

People who have had widespread discomfort for at least three months and experience pain from four kg pressure on at least 11 out of 18 anatomically-paired, tender points can be considered as having FM, even if another disorder is known to be present. If no treatment is received, the natural course of FM over time will maintain about the same central nervous system (CNS) sensitivity.

[*Note:* If there are co-existing conditions, including MTrPs, these can become more severe and give the impression of progressive disease. An increase in peripheral pain generators, including trigger points, arthritis, infection, or trauma, can worsen central sensitization and pain level. If co-existing TrPs increase in number and/or severity, patients will not be able to exercise normally without aggravating their condition because muscles with TrPs are in energy crisis even at rest. Patients and their care providers must be aware of both FM and myofascial trigger points, as well as their interactions with other co-existing conditions. DJS]

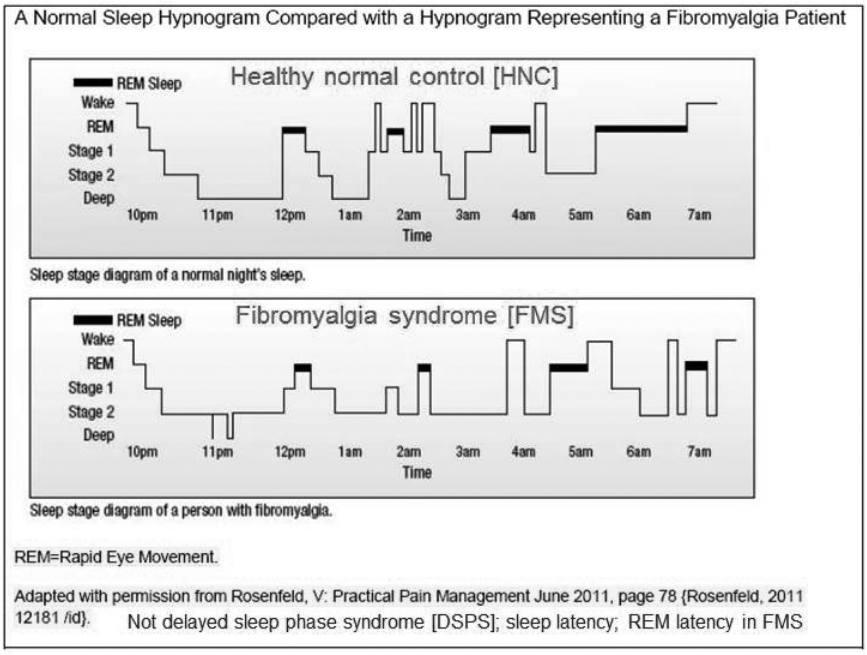
Dr. Russell remarked that the American College of Rheumatology's 1990 Diagnostic Criteria for Fibromyalgia were compiled for the purpose of research classification. The criteria included widespread pain of three months duration and 11 or more of 18 designated tender points, but they addressed only the pain/tenderness domain and were never validated for community clinical care.

Both pain pathways in FM (transmission and modulation), are dysfunctional. FM is heterogeneous and has many subsets of patients, symptom profiles, and triggers. It is not one uniform illness. In some ways, the FM brain is similar to a brain in which age-related changes have been accelerated. The regions showing these changes may be linked to affective symptoms disturbances and chronic widespread pain.

FM is more than one chronic pain syndrome; there are often co-morbidities. Controlling FM depends on controlling the co-morbidities. One example is sleep dysfunction. Sleep problems are often mistakenly considered to be a normal part of aging. More than 50% of older people have insomnia, and it is often under-treated. The pattern of sleep, called a hypnogram, is different in FM patients than in healthy controls (see figure 6), and polysomnography (in-lab sleep study) is recommended for them, even in the elderly. Non-pharmacological

FIGURE 6

Sleep - Polysomnography



Graphic courtesy of I. Jon Russell, M.D., Ph.D. Adapted with permission from Rosenfeld, V: *Practical Pain Management*, June 2011, page 78. Note lack of deep sleep cycles in FM patient.

interventions for insomnia are under-used by health care practitioners, and more training is needed.

*See Kuchinad A, Schweinhardt P, Seminowicz DA et al. 2007. Accelerated brain gray matter loss in fibromyalgia patients: premature aging of the brain? *J Neurosci.* 27(15):4004-4007. The authors state "…fibromyalgia patients had significantly less total gray matter volume and showed a 3.3 times greater age-associated decrease in gray matter than healthy controls." The longer the individuals had FM, the greater the gray matter loss, with each year of FM equivalent to 9.5 times the loss in normal aging. In addition, FM patients had significantly less gray matter density than healthy controls in several brain regions.

Legal Issues with Chronic Pain and Fibromyalgia Cases

Steven P. Krafchick, M.P.H., J.D.
Krafchick Law Firm
Seattle, Washington USA

Public and medical perception of the reality of fibromyalgia has been helped by drug commercials. The challenge is proving that FM patients cannot work in their own or any other occupation. There is usually some triggering factor that sets off fibromyalgia, although there is a genetic tendency to develop FM. It may be spinal problems, arthritis, injury, or other types of physical stress. Emotional stress also may trigger this illness. The result is a change in the way the body "talks" with the spinal cord and brain. Levels of brain chemicals and proteins may change. For the person with FM, it is as though the volume control is turned up too high in the brain's processing centers.

Social Security Disability Insurance (SSDI): Is there any objective evidence to help prove disability? Social Security Disability 2012 regulations consider FM a medically determined impairment, with sufficient objective evidence. SSR 12-2P defines FM as a common, complex medical condition characterized primarily by widespread pain in the joints, muscles, tendons, or nearby soft tissues that has persisted for at least three months. They also recognize post-exertional longitudinal fatigue.

To prove disability, the opinions of treating physicians, if they are M.D.s or osteopaths (D.O.), are given more importance than that of other care providers. You must have a M.D. or D.O. to certify that you have disability. The Social Security Administration can consider evidence from other health care providers and will give them some weight. Cardio-pulmonary exercise testing, neuropsychological testing and vocational evaluation help. Fibromyalgia is generally accepted in the scientific community, but the insurance industry wants to consider it junk science. You may need to go back in medical records a long time to piece together information, because it can take time until symptoms appear and longer to diagnose. It is important to document co-existing conditions as well.

Social Security Disability can offer the same benefits under mental and physical health, unlike the Employee Retirement Income Security Act (ERISA). SSDI coverage begins as of the date of disability, not the date of acceptance. Union members may be able to get help from their union getting SSDI. The Social Security Association's definition of disability is that you are unable to do any full time job in the national economy.

It is important to file as soon as possible for Social Security Disability, but this can be difficult due to the nature of FM. Patients may have been working, but not working reliably, because they have good days and bad days. Everyone requires money to live. You need good documentation and good history notes. Do symptoms impair productivity? What is your reliability for showing up for work eight hours a day, five days a week? Insurance companies can hire surveillance. Get a good form for FM vocational evaluations, provide medical literature for support, and document cognitive and somatic symptoms. The amount an attorney can charge for Social Security cases is limited to $5000.

ERISA cases are different than Social Security. There is no jury trial. There are no depositions and no expert testimony. Some policies limit disability due to FM to 24 months. You need to prove physical symptoms sufficient to disable, as there are mental health limitations for coverage.

A neuropsychiatric evaluation is helpful to document cognitive problems, but the evaluator needs to be familiar with FM and executive function and word-finding difficulties. The best neuropsychiatric evaluation for fibromyalgia patients seeking ERISA is a two-day test, for which most patients must pay out-of-pocket. One day, a FM patient may be able to do a task, but can s/he repeat it the next day? Can fibromyalgia patients deal with sustained activity – a 40-hour week? Can the patient work week after week and be productive? The physical capacity evaluator needs to know FM to ensure a comprehensive and fair evaluation.

Imaging of Muscle

by Siddhartha Sikdar, Ph.D.
Assistant Professor, Department of Electrical and Computer Engineering, George Mason University
Fairfax, Virginia USA

"A systematic musculoskeletal evaluation of people with myofascial pain syndrome reliably distinguishes them from subjects with no pain. The two groups are significantly different in their physical findings and self-reports of pain, sleep disturbance, disability, health status, and mood. These findings support the view that a "local" pain syndrome has significant associations with mood, health-related quality of life, and function."[1] TrPs don't usually exist as single lesions. Often there are multiple, closely related, ropy, lumpy nodules with a large and extended region of muscle that is affected.

About 95% of people with chronic pain disorders have chronic myofascial pain (CMP) due to TrPs, but few physicians receive training in the clinical diagnosis of MTrPs. There is a need for an objective, reliable and repeatable test to identify MTrPs and evaluate treatments. Diagnostic ultrasound has been used to evaluate other soft tissue conditions. It's low-risk, and could be used to characterize texture and elastic properties of myofascia, quantify the myofascial tissue entrapment of blood vessels, provide dynamic measures of tissue performance such as muscle contraction, and demonstrate the relationship between tissue structure and dysfunctions.

Vibration sonoelastography (VSE) imagery also has potential. Ultrasound VSE uses an external vibration source in conjunction with Doppler techniques to identify localized regions of increased tissue stiffness and has been used to investigate circulation and vasculature in myofascial trigger point sites and surrounding tissues. When external vibration is applied, stiffer tissue vibrates differently than less stiff tissue.[2]

A different technique, magnetic resonance elastography, has also been used for investigating the taut bands of myofascial trigger points. Exclusionary criteria for this study included fibromyalgia, a history of trigger point injection in the upper trapezius, and many local area conditions. On two-dimensional grayscale imaging, myofascial trigger points in the upper trapezius appeared as focal darker areas that are heterogeneous but differ from the surrounding space in the way that they reflect the imaging waves. A single two-dimensional image did not fully capture the 3D area of the myofascial trigger point and surrounding soft tissue, so three-dimensional imaging was also performed by manually sweeping the transducer over the upper trapezius in a specific manner.

Preliminary findings indicate that these imaging techniques can be used to distinguish myofascial tissue containing myofascial trigger points from normal myofascia. On 3D imaging, the discrete MTrP was clearly visible in the longitudinal, transverse, and coronal images. The image structure in the nodule was heterogeneous and markedly different from a control point in the trapezius. On color variance imaging, myofascial trigger points appeared as focal areas of reduced vibration amplitude. Color Doppler and duplex Doppler examination of the upper trapezius in the subject sample showed differences between latent and active MTrPs.

Trigger points are stiffer and more viscous than normal tissue and are more organized and contractured. A contractured muscle is not the same as a contracted muscle or a muscle in spasm. Contracture is not orchestrated by nerve impulses. When related to TrPs, a contracture is defined as a prolonged and intrinsic activation of the contractile elements of the muscle fibers without direction from motor units. The contracture, according to Simons' Integrated Hypothesis, is caused by the release of excess calcium and irritating substances at the motor end plates, rather than by nerve stimulation. Contracture of a few sarcomeres may not be noticeable by the owner of said sarcomeres, nor evident to a manual body worker such as a physical therapist, but it is still occurring, and can worsen with repetitive exercises.

In VSE imagery, TrPs have been revealed as part of a regional alteration of tissue and not as isolated lesions; the vicinity of the trigger point often may be modified. Blood vessels very close to a trigger point often show evidence of vascular reorganization near the TrP. There is a significant difference between active and latent TrPs in regards to blood flow congestion. There is evidence of compression and consistency within the vascular bed, with retrograde flow. There is extremely heavy vascular resistance with localized hypoxia (oxygen deficiency). Using this imagery, *tender* points of fibromyalgia do not have the same appearance as the *trigger* points. Attachment TrPs show an increase in vascular resistance which is consistent with blood vessel compression or entrapment caused by sustained contracture at a TrP, or constriction caused by oxidative stress or hypoxia. Blood vessel compression may be sufficient to cause diminished local microcirculation.

All findings support Simons' "Integrated Hypothesis of TrP Formation" that proposes an

energy crisis that perpetuates an initial sarcomere contraction. The sarcomere is the basic contractile unit of the muscle cell. The sarcomere contraction leads to increased local metabolic demands in the presence of compromised capillary circulation. This, in turn, leads to local energy crisis with lack of oxygen, tissue damage, or both. Results of 2D US imaging confirm that significant tissue abnormalities and morphological changes are associated with MTrPs. The darker and stiffer nodules may be indicative of contraction knots resulting from increased muscle fiber contraction and recruitment, local injury, and/or localized regions of diminished blood flow.

These techniques might eventually lead to a low-cost, in-office, objective, non-invasive way to identify myofascial trigger points, but right now that objective is far away. Further studies are needed to confirm these findings and their clinical usefulness. Presently, we cannot tell the composition of the tissue by vibration only; we need biopsies. Thus far, all studies have been done using the same operator. This technique is very expensive and research-operative-dependent, and not anywhere near clinical availability.

1. Gerber LH, Sikdar S, Armstrong K et al. 2013. A systematic comparison between subjects with no pain and pain associated with active myofascial trigger points. *PMR*. 5(11):931-938.

2. Sikdar S, Shah JP, Gebreab T et al. 2009. Novel applications of ultrasound technology to visualize and characterize myofascial trigger points and surrounding soft tissue. *Presentation to Arch Phys Med Rehabil* 90(11):1829-1838.

Ehlers-Danlos Syndrome

by Clair Francomano, M.D.
Director of Adult Genetics, Harvey Institute of Human Genetics, Greater Baltimore Medical Center
Baltimore Maryland USA

Up to 45% of all patients referred to general rheumatology clinics satisfy the criteria for Ehlers-Danlos Syndrome Hypermobility Type (EDS-HT), also called Type III, indicating that it's common but often undiagnosed.[1] Most of the pain EDS-HT patients feel is from the musculoskeletal, GI, or nervous systems. EDS predisposes to certain kinds of pain, but EDS does not cause pain in and of itself. There may be recurring joint dislocations, chronic joint/limb pain, and/or a positive family history. There is a general unawareness by practitioners of the multifaceted manifestations of this variation of Ehlers Danlos Syndrome.[2]

Although Classic EDS includes skin hyperextensibility, widened hypersensitive scars, joint hypermotility, and a number of other minor criteria, EDS-HT is characterized by hyperextensible and/or smooth, velvety skin and a generalized hypermobility that may go unnoticed. Skin manifestations in EDS-HT are much less obvious than in other forms.

There are many associated conditions, including carpal tunnel syndrome (CTS), FM, CFIDS, degenerative disc disease (DDD), CMP, gastroesophageal reflux (GERD), interstitial cystitis (IC), dysmenorrhea, back pain, impaired proprioception, dysautonomia, minor wound healing deficits, fragile capillaries, leaky (or permeable) gut, very low levels of Vitamin D, multiple allergies, syncope, insomnia, numbness, non-epileptic seizures, tremors, tinnitus, mood swings, anxiety, autoimmune overload, and weight gain. There can be tethered cord myelopathy, meaning that tissues attach to the spinal cord within the spinal column, restricting core movement. There is an increased risk of osteopena and osteoporosis, even at young ages. Almost all patients are female.

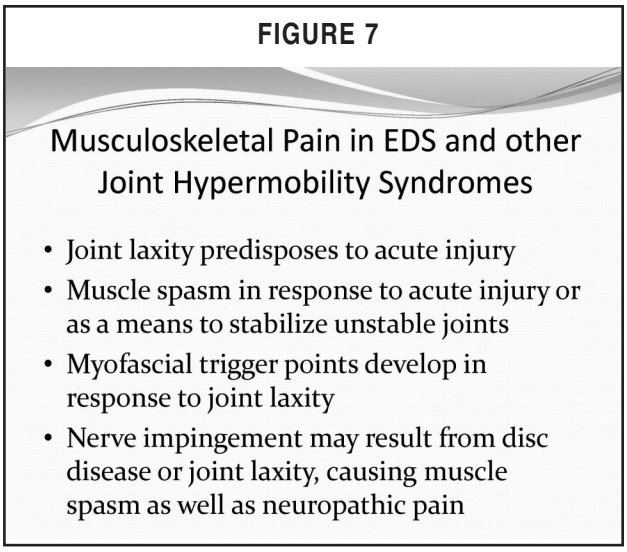
Degenerative disc disease and CMP are major factors in EDS mobility, pain, and dysfunction issues. There may be a problem draining blood from the head, along with autonomic dysfunction. As with other chronic pain conditions, sleep quality assessment is very important. When there is brain fog, fatigue, and lack of restorative sleep, it is critical to identify the initiating factor.

Ehlers-Danlos Syndrome Hypermobility Type affects many systems: Some possible manifestations include:

Musculoskeletal: Joint hypermobility, joint subluxations and dislocations, tendon and ligament rupture, stress fractures, degenerative disc disease, meniscus tears, chronic myofascial pain, headaches, sinus pain, TMJD, dental pain. Joint laxity predisposes to acute injury. Muscle spasm can occur in response to acute injury or as a means to stabilize unstable joints. MTrPs develop in response to joint laxity. Nerve impingement may develop from disc disease or joint laxity, causing muscle spasm as well as neuropathic pain, potentiating TrPs.

Neurological: Craniocervical instability, cervical instability, Chiari I malformation, low-lying cerebellar tonsils, increased intracranial pressure, and cerebrospinal fluid leaks.

FIGURE 7



Autonomic: Postural orthostatic tachycardia syndrome, neurally mediated hypotension, difficulty regulating temperature, and other signs of parasympathetic/sympathetic imbalance (see figure 8).

Sleep: The healthy progression of sleep levels may be impaired, causing irritability, cognitive impairment, memory lapses or loss, impaired judgment, severe yawning, decreased temperature, hallucinations, ADHD-like symptoms, impaired immune system, risk of type 2 diabetes, fatigue, decreased reaction time and accuracy, tremors, growth suppression, risk of obesity.

Cardiovascular: Mitral valve prolapse, aortic root dilation, aneurysms, venous insufficiency, increased heart rate variability, and risk of heart disease.

Gastrointestinal: Hiatal hernia, GERD, slow gastric motility, chronic nausea, vomiting, constipation, IBS, malabsorption, dysmotility, esophageal spasm, constipation, sphincter of odi dysfunction, abdominal wall hernias.

Immunologic: Common variable immunodeficiency, multiple allergies, mast cell activation disorder.

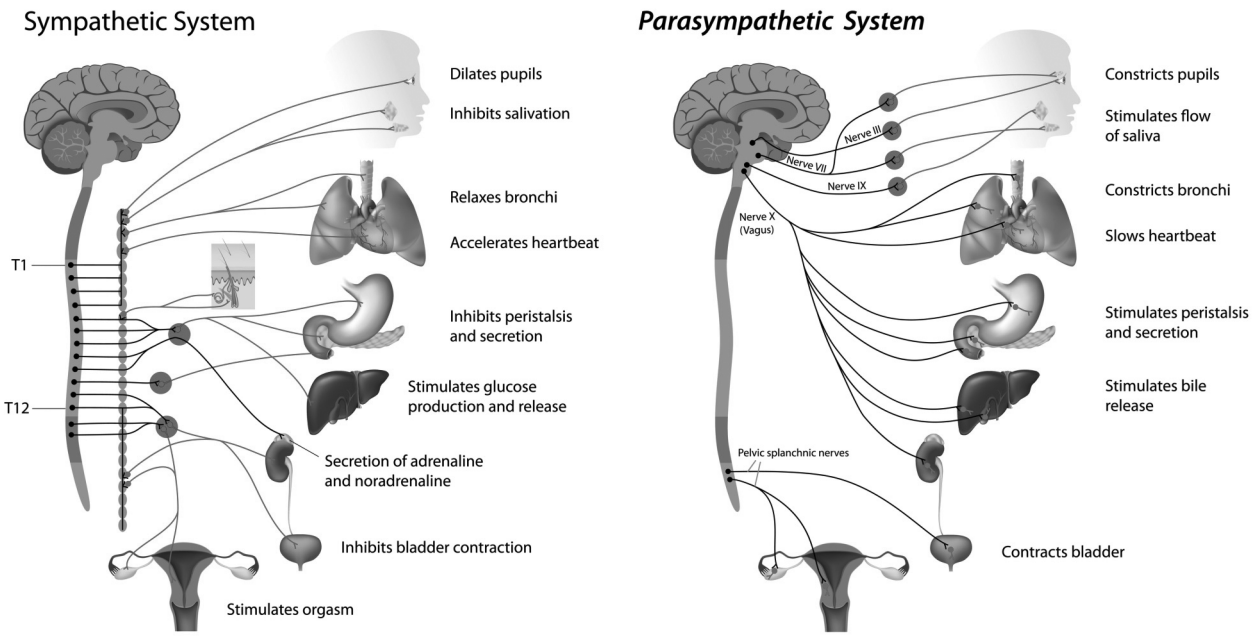
1.Hakim AJ, Grahame R. 2003. A simple questionnaire to detect hypermobility: an adjunct to the assessment of patients with diffuse musculoskeletal pain. *Int J Clin Pract* 57(3):163-166.

2. Castori M. 2012. Ehlers-Danlos Syndrome, Hypermobility Type: An under-diagnosed hereditary connective tissue disorder with mucocutaneous, articular, and systemic manifestations. *ISRN Dermatol* 2012 (Epub 2012 ahead of print) Nov 22.

A related poster, "Chronic Pain and Hypermobility," by TT Vanem, NG Juel, HL Soberg,et al. of Oslo, Norway, reported that the 240 patients they studied who considered themselves hypermobile had chronic pain. It can be difficult to identify inherited connective tissue disorders among patients with hypermobility.

FIGURE 8

The Autonomic Nervous System



Integrative Care for Chronic Muscle Disorders

Al Clavel M.D.
Neurologist, University of Minnesota
Fairview Pain Management Center
Minneapolis, Minnesota USA

We need to broaden our understanding of headache mechanisms and coordinate a variety of management techniques for chronic headaches. We need to use all the tools in our medical tool box, including headache prevention medications, relaxation training, thermal biofeedback, trigger point therapies, cognitive behavioral therapies, and electromyographic biofeedback.

Headaches can be either primary: migraine, tension-type headache (TTH), cluster, other (benign cough headaches, etc.); or secondary to another condition such as infection, hemorrhage, increased intracranial pressure or brain tumor. Risk factors for the transformation from episodic to chronic migraine include those that we cannot control, such as female gender, head trauma, genetic/epigenetic factors; and those that are controllable, such as medication overuse, caffeine overuse (more than 100mg /day), reaction to stressful life events, snoring and sleep apnea/insomnia, depression/anxiety, allodynia, obesity, or muscle dysfunction.

Medications generally work faster for headaches than behavioral treatments, but the latter tend to last longer. Combining medication with behavioral treatments is the best way to have a greater than 50% improvement. Some headaches thought to be primary could actually be secondary to myofascial trigger points or other undiagnosed co-existing conditions.

Adaptive states include Fight/Flight/Freeze reactions to a real or perceived threat and involve multiple biochemicals such as neurotransmitters, hormones, and peptides, working in different parts of the brain. They can cause arousal or dissociative states to help the body/mind cope with the threat. Dissociative states may vary widely in type and intensity, and can include changes in sense of self, identity, environment, perception, consciousness and memory. We can do this purposefully to some extent, when we get lost in a book or movie, but in a clinical dissociative state, simplistically stated, one loses the sense of what is real and what is not.

When the stressor is repetitive or sustained, the brain and its responses change. Prolonged "states" become "traits", leading to autonomic dysregulation. There are differences in adaptive responses to acute trauma and stressors, depending on age. Infants tend to dissociate, adults tend to go into fight or flight mode, and older children are somewhere in the middle. With multiple trauma events or prolonged stressors, people can move from one adaptive mode to another. These states are associated with the activity of different neurotransmitters. FM is like having the autonomic nervous system intensity switch stuck on high. Patients with the autonomic system volume stuck on low are usually diagnosed with CFS. If CFS patients are placed into relaxation therapy, it can make them worse.

If muscles are tied into old patterns of dysfunctional recruitment, they need to be retrained, and other perpetuating factors must be identified and brought under control. It takes time to retrain the brain and body. Much of the brain is designed to learn by experience.

Most patients at Dr. Clavel's clinic have more than one type of headache, and overuse of medications for acute headache often leads to more frequent headaches. They find that the most useful tools are those belonging to the realm of mind/body medicine. The evolving science of psychoneuro-immunonology (PNI) is often defined as the study of bi-directional interactions between the mind/brain, nervous system, immune system and endocrine system, but that is actually shorthand for a much more extensive process in which the limbic-hypothalmic axis plays a central role. To adequately treat chronic pain; we must treat the patient, not just medicate symptoms.

Mind/body approaches have proven to be particularly effective when there is a dysregulation of the autonomic nervous system (ANS), optimizing balance between sympathetic and parasympathetic branches of the ANS. Many of these patients have been defensive about considering psychological/psychophysiological components of their syndromes, because that issue been treated as "physical versus psychosomatic." That view is dysfunctional, and there is no justification for it. Everything is mind/body.

Dr. Muhammad Yunus theorizes that many chronic conditions (see figure 9) are manifestations of Central Sensitization Syndromes. These conditions may have an imbalance in the areas of physiological sensitivity (reactive or shut down to stress), emotional sensitivity (reactive or stoic), interpersonal sensitivity (reactive or withdrawn), and somatic or visceral sensitivity (hypersensitivity or poor awareness). As stimuli become prolonged, altered psychophysiological states become psychophysiological traits, and the patient becomes stuck.

The clinician must understand trauma physiology to be able to successfully treat chronic pain as a multi-

system issue. S/he must start by believing what the patient says, rather than causing the patient yet another traumatic experience. That sets a stage wherein the patient's brain can relearn. The brain and body learn together. The patient learns to become mindful of his or her actions and how they affect state of mind and the body. This creates a higher level of awareness in which the patient and care providers together can work to help heal the chronic pain state. The state of psychophysiological awareness is a part of what Dr. Clavel calls Yin-Yang Training.

Most of the United States is Yang-oriented. Life requires a balance of yin and yang. Patients may need to move back and forth between these modes with a variety of therapies that are tailored to their individual changing health needs. Healing of chronic pain conditions is circular in nature. Practicing the art of medicine means clinicians educate and provide whatever tools the patient needs; not just what they are good at and feel comfortable with. Chronic pain conditions require the skills of integrated medicine.

Many chronic pain patients lack self-esteem. That must be restored before the patient can handle the task at hand. Engage patients to be part of the process. Less negative feelings are not the same as positive feelings. If you feed the positive, the negative ones will diminish. The first task is for the physician to realize that the patient can be taught to self-manage.

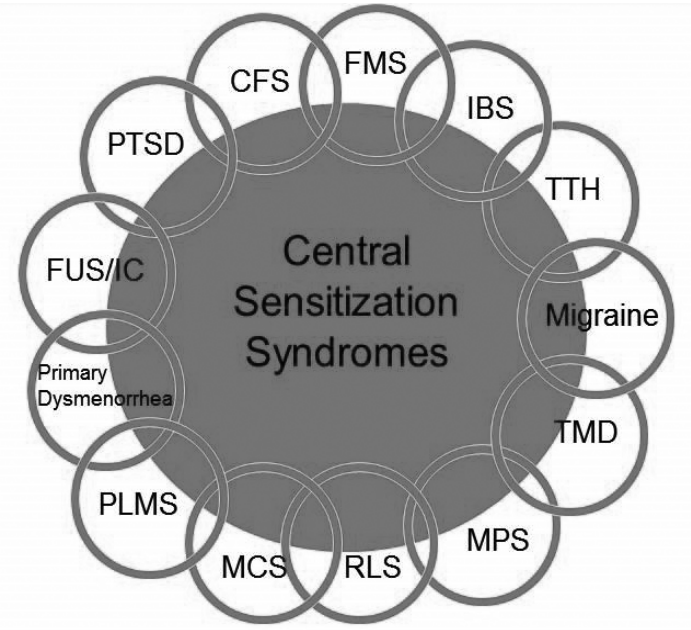
The second task is for the patient to learn this as well. Discover what state the patient is in and how he or she travels between states. This travel needs to be stabilized and the patient taught the basics. Use all available tools in the medical tool chest, and teach the patient what he or she needs to effectively manage his or her conditions. Chronic pain care must be patient-managed and self-directed. Respect the ability of the central nervous system to change, and see that the patient understands and respects it, too.

The care provider must give the patient direction, time, and space needed to affect that change. Change from the doctor saying from on high, "I want you to learn how" to a partnership with the patient saying "I want to learn how." The patient owns the healing process. Healing is a process of self-directed neuroplasticity.

FIGURE 9

Central Sensitization Syndromes

Muhammad Yunus @ U of Illinois



Graphic by Dr. Muhammad Yunus provided courtesy of Dr. Clavel

During the question and answer session, a comment was made that there is still a lot of stigma attached to FM. Some pain clinics will not allow patients to make an appointment if they say they have FM. All chronic pain patients may have trigger points, and that component can be treated. Many pain clinics fail to recognize this.

Botulinum Toxin for Myofascial Trigger Points

by Robert Gerwin, M.D.
Medical Director and President of Pain and Rehabilitation Medicine, Bethesda, Maryland USA. Also, Department of Neurology, Johns Hopkins University School of Medicine, Baltimore, Maryland USA

The discussion focused on the use of botulinum toxin for transformed migraine, those migraines that have become chronic. Most of those attending who used botulinum toxin found it helpful for transformed migraine. Some used it only after they proved that trigger point injection with local anesthetic provided good but short-lasting relief. Others started with botulinum toxin in severe cases, especially for patients on heavy drugs with severe pain.

Active trigger points were involved with migraines, and identifying and treating those TrPs was a critical part of treatment. This required palpation and careful patient positioning. Migraine patients often required MTrP assessment and treatment from the top of the head down to and including the trapezius. The headache can often be reproduced if you grasp the sternocleidomastoid (SCM) muscle in the neck, initiating the TrP referral pattern and signaling where at least some of the pain is generated. The SCM muscle is often a major factor in migraines.

Other symptoms provide clues where botulinum toxin can be used as well. For example, if there is a notable lack of ability to open the jaw, the anterior digastric trigger points may respond well to BT. One might start patients with a frozen shoulder with subscapular myofascial trigger point injections.

In a related poster, "Efficacy of Botulinum Toxin in Transformed Migraine," M.T. Jacob and B. Jacob reported finding multiple head and neck myofascial trigger points in patients with transformed migraine. They checked for inactivation with 0.5% lidocaine, repeating every 15 days for four times, observing response. If they achieved 70% pain relief or greater, they switched to botulinum toxin (BT). Of 25 patients, 21 achieved a good response with lidocaine, and were treated with BT. After one month, the patients had a relief of about 80% that remained on average of four and a half months. The BT treatment was repeated about three times until there were no more symptoms. The researchers concluded that inactivation of active trigger points was effective for transformed migraine symptoms.

The Relationship Between Obstructive Sleep Apnea and Sleep Deprivation on Muscle Pain Disorder

By Antonio Romero-Garcia, D.D.S., Ph.D.
Cranioclinic Valencia and Dental Sleep Solutions, S.L.
Valencia, Spain

Sleep disorders are common, especially among chronic pain patients, yet most patients remain undiagnosed due to lack of health care provider training. During sleep, the nervous system recalibrates, synapses consolidate, biochemicals restore balance, and the brain cleanses itself of the detritus of the day, yet we don't understand why we need to sleep, except that sleep is controlled by the brain, for the brain. Imaging studies show that areas of the brain that are deactivated by sleep can be reactivated by pain. Pain can be caused by disrupted sleep, and disrupted sleep can be caused by pain.

Chronic pain can negatively affect both the quantity and quality of sleep, and most patients with chronic pain have sleep problems. The most common include insomnia, non-refreshing sleep, daytime sleepiness, and fatigue. Chronic pain can cause sleep disturbance contributing to lack of energy, mood disorders, and lowered activity level. Loss of even four hours of sleep and Rapid Eye Movement (REM) deprivation can cause muscle aches and fatigue and affect mood even in healthy people. To manage chronic pain, refreshing sleep must be restored and maintained. Poor sleep is often associated with dysfunction in the cardiovascular, immune, and endocrine systems; affective disorders; learning and memory problems; and lack of resilience to stress. Pain decreases slow wave (restorative) sleep, increases sleep stage shifts, causes alpha intrusion into deep sleep, increases arousals (and thus chance of bruxism), and affects quantity of sleep. These may return to normal when pain is properly managed.

Sleep bruxism itself might be a compensatory mechanism to prevent the collapse of the airway. Patients who clench or grind their teeth during the night may experience sleep-interrupting pain, and sleep arousals themselves may cause bruxism. TMJD pain may be related to sleep fragmentation that negatively impacts the pain modulating system, and yet dental appliances for bruxism can themselves cause TMJD. Bruxism that occurs during awake states is caused by a different mechanism than bruxism that happens during sleep. So far, there is no direct relationship shown between the presence of bruxism and presence of pain. Medications that decrease sleep, especially REM sleep, may increase pain.

Upper Airway Respiratory Syndrome (UARS) does not cause oxygen desaturation or apnea, but it does cause daytime sleepiness and must be treated. UARS patients have a greater tendency than obstructive sleep apnea (OSA) patients to report muscle aches. A significant amount of chronic pain patients have primary sleep disorders other than insomnia, including OSA, restless leg syndrome (RLS), or periodic leg movements (PLM). Any coexisting interactive condition will complicate pain management. Medications and therapies that can positively affect the pain and the comorbid condition are most beneficial.

Headaches may occur as a consequence of specific sleep disorders, such as bruxism, OSA, and insomnia. Many headaches resolve when OSA is treated successfully. OSA-related migraines were often associated with REM and other delta sleep disruptions. About 9% of all men and 4% of all women have some form of OSA. Less than 10% of OSA cases have been diagnosed. Of those diagnosed, less than 25% have been successfully treated. Yet, it is a serious condition. OSA can result in increased work-related accidents, increased motor vehicle accidents, lower threshold for depression, family discord, and a decreased quality of life. OSA causes reductions in oxygen content and sympathetic tone during an apneic episode, and it can worsen any cardiovascular disorder.

[Note: anything that decreases the oxygen supply also increases the chance of myofascial trigger point formation. DJS]

Patients with chronic pain and any signs of OSA or fatigue require further evaluation. In-lab polysomnography (sleep study) is required as the basis for diagnosis of sleep disorders including bruxism, and mandatory for any patient with chronic pain and fatigue or other signs of non-restorative sleep. It is critical to identify primary sleep disorders such as: insomnia, sleep disordered breathing, periodic limb movement, bruxism, restless leg syndrome, and circadian phase shifts. In addition to a sleep study, assess: sleep hygiene, sleep environment, the wake/sleep cycle, lifestyle habits including diet, and excessive duration of naps (no more than 30 minutes) or evening nap habit.

Treatment includes addressing whatever is brought to light in the sleep study, co-existing conditions that may be perpetuating sleep disruption including pain, and assessment by behavioral/cognitive measures and physical management. Establish regular routines for evening relaxation, and avoid "bringing work home" or intense evening discussions. Body work including moist heat or ice massage, physical manipulation, myofascial release therapy and other manual strategies may be useful to relax muscles. Pharmacological measures include pain medications, muscle relaxants, antihistamine, antidepressants (pramipexole if PLM is present), cannabinoids, melatonin, and chamomile.

Summary: There is a close link between sleep and pain. Doctors should evaluate all patients for the presence of all sleep-related conditions. Addressing both pain and sleep problems can optimize treatment. Ignoring the presence of the sleep disturbances can limit success and jeopardize patients' health.

These are but a sample of the presentations offered at MYOPAIN 2013. There were also hands-on workshops on fibromyalgia and on dry needling. The Myopain Congress is a constant learning experience. Was your care provider there?

For information on the International Myopain Society, visit www.myopain.org

The IMS also has a link for those who wish to donate to FM & trigger point research: http://myopain.org/default.asp?page=donations/

For information on fibromyalgia and chronic myofascial pain visit Devin's website at: www.sover.net/~devstar

Join Devin's Facebook support group at: Fibromyalgia, Myofascial Pain and Dysfunction.