VIEW FROM THE MOUNTAIN TOP

MYOPAIN '07: The International MYOPAIN Society's 7th International Scientific and Clinical Meeting

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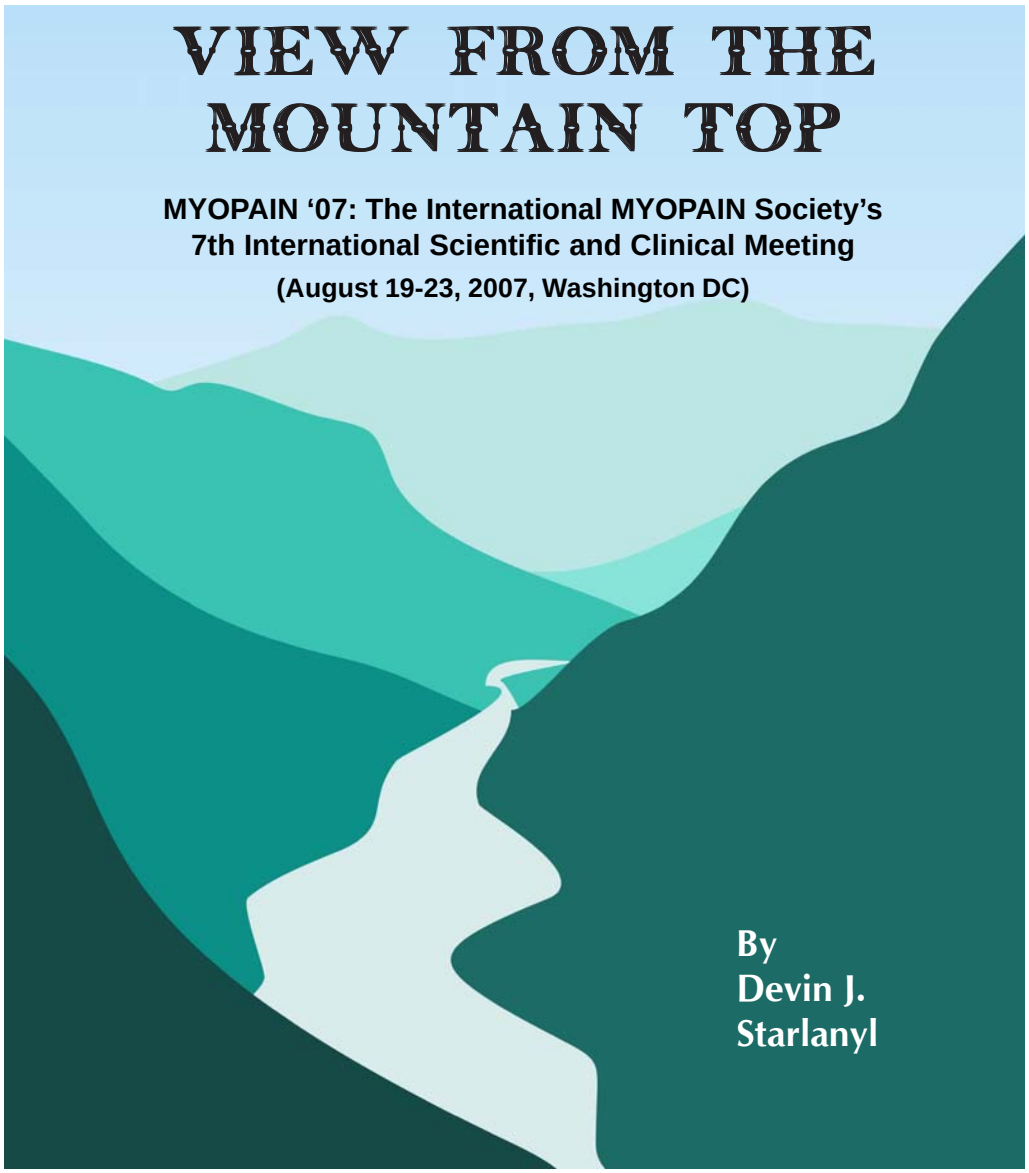

By
Devin J.
Starlanyl

Like fine wine and most patients with fibromyalgia (FM) and chronic myofascial pain (CMP), I do not travel well. I had never been able to attend an International Myopain Congress before. This one was being held relatively close to where I live. I wanted to speak with some of the people who have dedicated their lives to trying to help us, and it was a chance to see old friends and meet new ones. I planned on three and a half days of sensory overload of the most intense but pleasant kind. Myopain is more than a gathering of the top musculoskeletal researchers and clinicians. It is an experience unlike any other. In the pages that follow, I will try to give you a taste of what it was like to be there and give you highlights of some of the presentations, explain what they mean and why they are so important to us. I beg forgiveness to the many researchers I have not covered here due to lack of space.

The opening reception on Sunday began the learning experience. There was no formal presentation at that time—just good food and good company. At registration, I was given a bag which included a special edition of the *Journal of Musculoskeletal Pain* (*JMP*), the official journal of the International Myopain Society (IMS), containing abstracts of posters and a few presentations. A "poster," in the world of medical seminars, is a very large billboard filled with diagrams and data concerning research and other material from one or more investigators. A select few of these posters are chosen for oral presentation. There was a chance to sit and talk with some of the presenters over lovely food, and the first opportunity to ask and answer questions. It was a relaxing time before an avalanche of research started the next day. I get a warm and wonderful feeling remembering conversations, catching up on new projects, sharing

quick exchanges of ideas and theories, and the quiet chat my husband Rick and I shared with sleep specialist Dr. Harvey Moldofsky.

When we got back to our room, I glanced at the *JMP* issue and the 117 abstracts it contained, and I told Rick he probably wouldn't be seeing me for the next three days and that I needed to look over the mountain of information in the journal that evening to figure out which researchers I needed to track down, because they couldn't always be found near their posters.

As it turned out, at the conference there was a double ballroom-sized area arrayed with row after row of nearly wall-to-wall posters, each on a frame like an office cubicle without partitions. The research contained in each poster would be "defended" and explained by the researchers themselves, or by their representatives. I don't remember how many lines of poster walls there were, but the amount was both daunting and exciting. Around the edges of this huge space were exhibitors offering research literature and samples of new health care products. So, not only was there almost nonstop information in the main lecture room, there were the posters elsewhere, and posters would be changed daily. Meanwhile, presentations would continue through lunch, and there would be time allowed to ask questions of the speakers by microphone after each presentation.

On Monday, introductions and a sense of continuity were supplied by **Dr. I. Jon Russell** *(San Antonio, USA)*, Editor of the *JMP*. Although you may be familiar with his research, you may not know that he has one of the kindest and gentlest of voices and is a most talented artist. He and his wife, Dr. Barbara Runnels, MEd, were responsible for much of the smooth flow of the conference.

MYOFASCIAL PAIN

[Chairmen: Jo-Tung Chen (Taiwan), Bryan O'Neill, (USA)]

Monday focused primarily on myofascial pain. The first speaker was outgoing Myopain President **Dr. Dieter Pongratz** *(Munich, Germany)*. He summed up the accomplishments which had occurred in the fields of fibromyalgia, myofascial pain, and related conditions during his tenure, including the use of functional Magnetic Resonance Imaging (fMRI) to gain a better understanding of the central nature of FM and the FDA-approval of one medication (Lyrica) for FM and another (Cymbalta) in the approval process. He warned of the dangers of lumping all pain under the FM heading, and he pointed out that there are many possible causes for neuromuscular pain, many of which are being mistaken for FM. He also noted that we now have electromyographic differentiation of endplate noise for myofascial trigger points (MTPs). This means that the nerve at the area where MTPs form produces a measurable electrical signature. He reminded the audience that pain from MTPs, although the most common cause of muscle pain, is still often misdiagnosed.

Dr. Jay Shah *(Bethesda, MD, USA)* then gave a presentation—a cinematic masterpiece, entertaining and informative—that any doctor who still doesn't "believe in" myofascial trigger points should see. Dr. Shah took us to the world of latent and active MTPs, and told us that when you move a muscle, it shouldn't hurt. We could see how pain happens in response to MTP action, and why it is important to examine for and treat latent MTPs as well as active MTPs. We saw slides of the mechanism he has developed that enables him to sample substances that are released when a myofascial trigger point twitches. He has identified over 30 such substances, many of which are neurotoxic. [Note: Provoking a myofascial trigger point by needling or other treatment often produces an involuntary local *twitch* response (LTR)]. He has also discovered that the area around myofascial trigger points also becomes more acidic and that other nearby muscles without MTPs can also be affected by these biochemicals. This means that, biochemically, MTPs directly and adversely change more than just their own area. They can affect the biochemical balance of the surrounding muscles. This research was done on patients with *acute* MTPs. Those of us with chronic MTPs need to understand that this research is uncovering measurable reasons for our symptoms. Dr. Shah's research is revealing new targets for analgesic drug development and offers hope for all of us with MTPs.

Dr. John Jarrell *(Calgary, Canada)* explained how myofascial trigger points can cause specific gynecological pain, including general pelvic pain and vulvodynia. MTPs are especially common in endometriosis. Many doctors are missing the boat and the cause of much of the pain by focusing on organs rather than on the soft tissue. Most pelvic examiners try to feel an abnormal mass and don't know how to palpate for MTPs. Examiners must

learn how to search for MTPs and treat them. They can be taught to *gently* find taut bands that feel like violin strings, as well as contraction knots that may, to the finger tips, resemble fatty tumors under the skin. These often refer pain to the chest and back as well as the abdomen and pelvic areas. Many cases of vulvodynia have contributing MTPs in the perineum, the area in between the anus and the vagina. [I strongly suggest that when examining for MTPs and taut bands during pelvic exam, topical anesthetics are used, especially if the patient also has central sensitization, the name that describes a super-sensitive and dysfunctional central nervous system (CNS).]

Following the panel on myofascial pain, we were given a quick break to grab lunch and return with it to our tables in the lecture room. While we ate, **Dr. Harvey Moldofsky** *(Toronto, Canada)*, a leader in the field of sleep dysfunction research, fed our minds at a Haworth Press-sponsored lunch symposium. He reminded us that any kind of sleep disruption occurring during rapid eye movement (REM) sleep can decrease the pain threshold. Pain itself can disrupt sleep, and anything that disturbs sleep, including environmental stimuli, can produce non-restorative sleep, further increasing muscle pain and fatigue. Both FM and chronic fatigue are associated with fragmented sleep, sleep apneas, periodic limb movement, and other conditions that can cause frequent sleep arousals. Dr. Moldofsky's information indicates that many sleep dysfunctions are interactive with FM and other disorders.

During the afternoon, the difficulty of treating trigger points occurring in non-myofascial tissue (i.e., trigger points just beneath the skin or near the bone) was a topic discussed by several presenters and also included on several of the posters at the conference. As more health care providers learn to recognize MTPs and develop an understanding of the mechanisms behind MTPs, more are finding trigger points in other tissues. Non-myofascial trigger points are largely uncharted waters that require more research.

As I made my way through the gallery of posters, I saw a delightful deluge of research from **Professor Fernandez De Las Penas** *(Madrid, Spain)* and his fellow researchers covering the following topics:

- Myofascial trigger points in eye, neck and shoulder muscles as a primary cause of tension-type headaches;
- The association of at least one MTP with muscle atrophy (wasting) [Note: As MTPs can cause nerve entrapment and blood vessel entrapment, this would be logical but hasn't been documented before.];
- How some chronic low back pain patients with MTPs in the multifidi muscles (i.e., small muscles along the spine) should be treated before any stability exercise program begins because these MTPs can contribute to muscle instability; and
- How pain from eye muscle MTPs contribute to (CNS) sensitization.

Other posters of special interest included the following:

- How phantom limb pain occurs in patterns similar to myofascial trigger points' referral pain patterns, indicating that identification and treatment of the MTPs might help;
- How using environmentally-friendly vapocoolant for spray-and-stretch significantly increases both passive and active hip range-of-motion stretch over the use of stretch alone;
- How acute radicular pain can be caused by activated MTPs and how treatment of MTPs could even help chronic radicular pain. The patient must be treated, rather than the radiography; and
- How soft tissue must be assessed, rather than the current focus on the skeletal system and discs alone.
- I also learned that **Dr. H.Y. Ge** *(Aalborg, Denmark)* and his team have discovered that the local twitch response from MTPs can cause muscle cramps. They are currently working on connections between the local twitch response and spinal reflex. Their work has provided more documentation on MTPs and increased motor endplate sensitivity. [The motor endplate describes the area where the terminal branch of a motor axon contacts a striated muscle cell through an electrical junction called a synapse. Most central MTPs are found in the motor endplate area.]

- **Dr. Dimitrios Kostopoulos** *(New York, USA)* documented how treating myofascial trigger points with manual therapy in conjunction with passive stretch gives a superior result compared to either done independently.

- **Karen Lucas** *(Melbourne, Australia)* and her team offered an impressive amount of documentation showing that latent MTPs, those MTPs that are present but not causing pain, can affect the whole body as they shift muscle motions into dysfunctional patterns. Latent and active MTPs cause muscle dysfunctions and restriction of range of motion. Care must be taken not to equate MTPs only with pain. The dysfunctions are often missed.

Posters documented MTP palpation skills among trained and experienced care providers as well as new practitioners. They demonstrated that laser therapy and spray-and-stretch techniques were equally effective in reducing the soreness that sometimes occurs after dry-needling a MTP. Dry needling is the mechanical stimulation of a MTP using a needle without the use of an anesthetic.

There was also validation of surface electromyography (sEMG) for the documentation of myofascial trigger points, helping to pinpoint which muscle or muscles are being used to compensate for other muscles weakened by MTPs. I found this study personally interesting, as the MTPs were initiated due to the use of an endotracheal tube during surgery, and the referral pain pattern occurred during swallowing. Having experienced MTP cascades from endotracheal intubation myself, I know how difficult this can be and how unaware most anesthesiologists and other surgical and post-surgical team members are about this preventable possibility.

Several researchers documented the common presence of MTPs in trauma and torture survivors. I will never forget **Dr. Bente Danneskiold-Samsoe** *(Copenhagen, Denmark)*, an international expert on the subject of torture. Her soft voice carried compassion for those afflicted by unnecessary pain in a world already holding far too much suffering. We shared a moment of discussion and empathy in the noisy poster room before I moved on.

By now, you may be wondering how I managed to see the posters, speak with the researchers, and visit all the exhibits when there were presentations going on in the lecture hall, even through lunch. Even break time was brief. I usually just grabbed some bottled water and charged through the poster area like someone who had won a grocery store sweepstakes. The charge was often unceremoniously interrupted, as I took time for precious moments with my mentor Dr. David Simons, visited with his wife Dr. Carol McMakin, and occasionally called out to other friends or was called by them. The time I spent with them was far too short, but well used.

The day ended too soon for me to assimilate half of what I had taken in. I regretfully realized that I couldn't attend the Muscle Pain Symposium starting at 7:30 pm if I wanted to be functional at 6 am the next morning to start the next round.

FIBROMYALGIA

[Chairmen: Orlando Mayoral del Moral (Spain), Russell Rothenberg, (USA)]

Fibromyalgia was the focus on Tuesday**.** As I review my scribbled notes, it's apparent my own MTPs affected my ability to record them. The day started briskly, with **Dr. Laurence Bradley** *(Birmingham, USA)* summing up the research on the genetic tendency to develop FM, at least in some people. He mentioned some of the complexities of this research and how difficult it can be to separate genetic influences from environmental ones. Much of this research requires families with several siblings, some with and some without FM or other complicating conditions. Work has been focused on a few of the most likely genes, but research has discovered new associations that may lead to other connections. This research will take time and persistence due to the number of variables.

Dr. Daniel Clauw *(Ann Arbor, USA)* gave an overview of the different types of ongoing FM research. He suggested that a significant advance is the acceptance that fibromyalgia is primarily a neural disease and that central nervous system factors are the major role players. [In my opinion, this is something that many physicians in practice still don't understand.] He felt that probably the most consistent finding in FM research is the so-called "left-shift" in sensitivity of the FM patient, which can include amplification of many stimulus responses, including those to heat, noise, electricity and light, as well as pressure. [Patients with FM and other conditions and symptoms (especially

MTPs) and their care providers must understand this, as some treatment modalities for other conditions include electrical stimulation, heat, etc.] Also important to remember is that many illnesses other than FM are associated with this central sensitization. These conditions include irritable bowel syndrome (IBS), interstitial cystitis (IC), and low back pain. FM seems to be a combination of high level pain facilitation and low level pain inhibition. Some areas of the CNS pain pathway seem to be functioning normally in FM patients, but the descending pain-relieving pathway that uses serotonin and norepinephrine doesn't work well. Many new medications that act on both of these neurotransmitters, including Effexor (venlafaxine) and Cymbalta (duloxetine), may be able to help turn down the volume on the FM amplifier. If FM patients can regain restorative sleep and exercise tolerance, this may help as well.

New uses of imaging are helping researchers find areas of the central nervous system, especially in the brain, that are malfunctioning in fibromyalgia. There was recognition that the causes of central sensitization such as peripheral pain must be brought under control. FM is a high-maintenance condition, and just like other chronic illnesses, patients may require effective help without which they may have no time or energy for fun (among other things), and experience much lower quality of life.

We need to stop having to apologize about fibromyalgia, because we know enough about FM's mechanisms to know better [and our care providers should know this as well.] In addition, FM is not a homogenous medical condition, and we need to stop talking about it that way and start discussing it, researching it, and treating it in terms of subsets.

A questioner from the audience asked about using systemic neurotransmitter modification and expressed concern about flooding the whole body with non-specific medications. [The same neurotransmitter can have a variety of functions in different tissues.] The answer was to identify FM subgroups and focus treatment on the subgroups. Dr. Clauw described FM as a "hypertension of the pain processing system." During break I took my hyped-up (but well managed) pain processing system and my myofascial trigger points, grabbed a bottle of water and was off for another dash through the poster room.

Among the interesting posters I saw were the following:

- The use of the NMDA receptor antagonist Memantine, presently used for Alzheimer's, to possibly help memory deficits in FM patients;
- Some FM patients do not have depression and have healthy coping skills;
- FM patients may do well with a physician who takes FM seriously, but a physician who doesn't can have a significant negative impact on their health;
- Skin rolling technique is useless as a diagnostic test for FM; and
- The complex but important matrix of potential biochemical interactions produced by **Dr. Jean Eisinger** *(Toulon, France)* and his team demonstrated how insulin and its metabolism can have profound affects in FM.

Other studies showed:

- Tizanidine (Zanaflex)—a Substance P inhibitor which helps relax muscles and decrease muscle spasms and cramping— to be effective FM therapy for some patients;
- The SSRI drug Escitalopram (Lexapro) can help both FM and multiple chemical sensitivity (alone or together);
- Multi-drug therapy that includes gamma hydroxybuterate can be helpful for FM patients with anxiety, depression, and/or post traumatic stress disorder;
- A clear relationship between a traumatic brain injury and the development of FM;
- How to identify patients at high risk of developing FM and perhaps prevent or moderate its occurrence; and
- How to manage FM once it occurs.

I winced a little at this last one. It was a good poster by **Dr. Anne Marit Mengshoel** *(Oslo, Norway)* about the necessity for getting a diagnosis for validation and focusing treatment options, learning healthy coping strategies, adapting to the limits set by FM, and getting a support system in place. I winced because I was certainly exceeding my limits, but what I was learning was superb and so much fun!

Back in the lecture room, **Dr. Roland Staud** *(Gainesville, FL, USA)* spoke on the effects that peripheral pain stimuli have on chronic pain conditions such as FM. He mentioned temporal summation and other topics that have been covered in previous issues of *Fibromyalgia Frontiers*, and he touched upon the new recognition of the importance of dysfunctional ion channels [I'll explain those later] and of both dopamine and cannabinoid receptors, and that they would all become more important in controlling chronic pain in the near future.

There are both immediate and delayed CNS pain mechanisms involved with FM and multiple mechanisms involved in turning acute pain into a chronic state. The FM patient is different in measurable ways from the skin inward. There is an increase in histamine, mast cells, cytokines and NMDA 2D receptors in the skin. [Tests for these are not easily available nor practical for documentation in a doctor's office, but FM is demonstrably not "all in your mind."] The FM patient usually feels deep tissue pain and muscle pain but usually doesn't complain of joint or skin pain. If pain generators such as osteoarthritis (OA), MTPs, and irritable bowel syndrome (IBS), which cause a barrage of pain stimuli, can be brought under control, fibromyalgia pain can be reduced and sometimes even completely resolved. However, the pain must be effectively managed for as long as it exists.

There may be many different peripheral contributors to central pain in one patient. Although peripheral stimulation is necessary for FM pain, it may not be sufficient to explain all of the pain, as the process of pain facilitation and abnormal pain processing can be due to dysfunctional CNS areas. Most FM patients have focal tissue abnormalities such as myofascial trigger points or osteoarthritis. Treating FM central pain includes identification and control of all peripheral pain generators.

Dr. Philip Mease *(Seattle, USA)* gave us a better understanding of the process of designing FM assessment for both clinical care and for research studies. Deciding what exactly constitutes key FM symptoms is no easy task, as each patient is different. Key symptoms such as pain, cognitive problems, stiffness, sleep disturbance, fatigue, and other common symptoms have been chosen. Once there is an acceptable standard assessment, each treatment, medication, etc., can be assessed using these key symptoms. During the question and answer segment, Dr. Mease mentioned that most patients with FM have at least one MTP. It was urged that any FM assessment *must* include that co-existing MTPs be identified, as they may influence research conclusions or patient management. For example, if the patient has dizziness, is it caused by MTPs in the sternocleidomastoid, cognitive dysfunction from the FM, or a side effect of the medication or therapy? This is also true of gastrointestinal symptoms and other non-pain symptoms. Too often researchers and clinicians are unaware that MTPs can cause many symptoms other than, or as well as, pain.

During lunch, Dr. Russell suggested how one might design a medication therapy program for fibromyalgia using strategic polypharmacy based on symptom management. Patients with FM may need several medications to control a wide variety of symptoms. Medications that can reduce multiple symptoms are a bonus. Some pain medications, for example, also provide an improvement in sleep quality, which brings relief from fatigue, better brain function, and a further lowering of pain. Sleep quality is often significantly low in FM and must be improved, and so it may be necessary to search for any cause of secondary sleep disturbances.

New medications like pregabalin can have dramatic effects for some patients, but FM patients may be sensitive to medications, so it is necessary to begin slowly, working closely with the patient and increasing the dosage to find the maximum benefit with the minimum medication. In the case of gabapentin, however, it is important to realize that at a certain point if you increase the dosage, the bioavailablity–the amount of drug available for use in the system–actually goes down. On the other hand, pregabalin may affect co-existing conditions. For example, bipolar patients may become quite manic, and there may be loss of blood sugar control. Yet pain itself can cause a rise in blood pressure. Duloxetine can also help some FM patients. If duloxetine causes nausea, it is often necessary to decrease the dosage, let the patient adjust, and then try an increase again.

There is research in press that indicates FM is not just about pain. It's associated with premature aging, enhanced pain reception, biogenic amine deficiency, and changed social status. There are gender differences, too. Serotonin levels in FM patients are higher in males. In addition, the tendency of FM to run in families could be due to a biogenic amine deficiency that may be associated with prions, particles smaller than a virus that can affect the CNS. It is also

vital for the patient and the physician to understand that there is no magic wand for FM. It takes a combination of patient education, lifestyle adjustments (including attitude, dietary habits, and exercise), medication, therapy, and adequate follow-up.

Dr. Yangming Xiao *(San Antonio, USA)* presented research that indicated a specific protein defect involved in cell regulation that his team had found in FM patients. This is early but possibly significant research that it is hoped will prompt some investigation of new types of medications, as well as genetic studies. Other research showed that cannabinoids look very promising for FM pain reduction, as well as for positive effects in mood and sleep architecture. Combinations of different cannabinoids worked well for different FM types. [This agrees with the large file folder of research I have accumulated concerning chronic pain and symptom relief from cannabinoids.] There was also research supporting the conclusion that the majority of FM patients have no psychological illness, and most of us are coping pretty well, thank you very much, in spite of chronic pain, questioning the validity of FM psychological profiling.

By this time, my hands and arms were complaining every time I grasped a pen, but the Continuing Medical Education (CME) representative had been coming in periodically warning us not to tape any presentations, nor to take any photos of the posters. Before I knew it, though, they were taking down the posters, and it was time for refueling with food and t'ai chi and getting ready for the next day. I was able to have a quick word with Dr. Robert Gerwin before I left. During the reception which had taken place on the previous Sunday evening, I had asked him what he thought about the idea that myofascial trigger points might be due to channellopathy. (I promise to explain that.) He replied with a twinkle in his eye that I should be sure to come to his presentation, as if I would miss it. At some time during the next two days, he invited everyone, and added this incentive: "There will be chocolate."

GENERAL MUSCLE PAIN

[Chairmen: Joseph Donnelly (USA), Michael Spaeth (Germany)]

Wednesday's center of interest was general muscle pain, although this, of course, included FM and MTPs. **Dr. Brian Cairns** *(Vancouver, Canada)* started the day with a presentation on glutamate and glutamate receptors. Glutamate is one of the major excitatory amino acids, and its receptor is the structural protein molecule that binds it. The amount of the biochemical glutamate found in interstitial areas can change musculoskeletal pain sensitivity. Researchers are looking more carefully at interstitial space. This area is sometimes called "Third Space" because it is neither intracellular (within the cell), nor extracellular (right outside the cell). Most interstitial fluid is trapped in the gelatin-like ground substance, part of the myofascia which supports cells and fibers and through which nutrients and waste material are transported. Its importance in body function and the transfer of biochemicals from one area to another is becoming increasingly obvious. Its composition can vary tremendously from person to person and from site to site, and it plays a significant role in health. Glutamate response varies considerably between males and females, which may be one reason women feel more pain in some conditions.

Dr. Lars Arendt-Nielson *(Aalborg, Denmark)* explained some difficulties encountered in measuring muscle pain, including how skin pain differs from that of deep tissue, and the importance of understanding referred pain. It can be difficult to diagnose pain occurring in one area but generated in another. Those of us with chronic musculoskeletal pain have "...significantly larger referred pain areas..." in response to the same stimuli as healthy subjects. There are general features that may help to predict who might develop central sensitization, including a lower pain threshold enhanced after trauma, and family members with a genetic tendency to chronic pain. Prompt short-term action when a painful stimuli occurs may prevent long-term CNS sensitization.

Dr. Thomas Graven-Nielsen *(Aalborg, Denmark)* taught how muscle pain can alter a relaxed muscle, causing many changes including static muscle contraction, decreased endurance time, and prolonged fatigue. If the patient continues to work the muscle in spite of pain, that will aggravate the fatigue. In addition, pain in one muscle can spark increased muscle activity in another. If the pain persists, more perpetuating factors develop. For example, the muscle will begin bracing for the perceived threat of more pain, and other muscles will change to compensate for the affected muscle, overloading those muscle groups and contributing to the spread of pain.

Dr. Kathleen Sluka *(Iowa City, USA)* has been studying how localized pain spreads to become chronic widespread pain. If an animal is injected with an irritating substance, there is acute pain. A second injection five days later can result in significant changes, including hyperalgesia of the whole injected paw, the muscle, and reactions in the organs, similar to what occurs in humans developing widespread pain. Animals treated with lidocaine before the second injection did not develop widespread symptoms. If the lidocaine was injected *immediately after* the 2nd injection, the developing changes completely normalized. If the lidocaine injection was delayed too long after the 2nd injection, widespread symptoms developed anyway, and remained. Changes included neurotransmitter release, an increase in excitatory amino acids, and changes in parts of the brain. This pain responded to opioids intrathecally (injected directly into the spinal cord), and also to NMDA inhibitors and gabapentin. There are specific acid-sensing ion channels in pain receptors that innervate muscles, and the response to acidity can be significant and plays a role in the development of hyperalgesia. Mice lacking a specific type of acid-sensing ion channel don't develop mechanical-induced hyperalgesia under the same circumstance. Multiple pain stimuli applied to one paw can cause hyperalgesia in another paw as well. Her work with mice suggests that there is an increase of pain sensitivity in the CNS after fatigue occurs in response to muscle insult.

Needle electrical intramuscular stimulation can provide an immediate pain relief for myofascial trigger points, but the mechanism for this does not seem to be increased microcirculation. **Dr. Carol McMakin** *(Vancouver, USA)* demonstrated her frequency specific microcurrent (FSM) and presented research showing that FSM can significantly reduce facet pain (specific areas of the vertebrae). Since different frequencies of FSM have already been shown to help FM and MTPs, this may be a sign of more research to come.

Other topics which were explored in posters included the following:

- Estrogen may be involved in pain modulation. Male rats, and female rats with their ovaries removed, have a significantly higher baseline pain threshold than female rats with ovarian hormones.

- There is a significant problem separating chronic pain patients because most have multiple diagnoses, and researchers may have to specify patients as "predominantly FM," or "predominantly neuropathic," etc.

- Lightning-like electrifying pain attacks occur in over 60% of FM patients [although some of this may be due to unrecognized co-existing MTP nerve entrapment.]

- FM patients who receive inadequate care can be an enormous economic drain. Too often a pain specialist isn't consulted in a timely manner, and we need primary care physicians to send chronic pain patients for assessment and stabilization sooner. Some psychiatrists don't understand the physiological aspects of FM, and patients can be negatively impacted by what the doctors are saying to them and can become depressed after seeing these doctors.

- **Orlando Mayoral del Moral, PT** *(Toledo, Spain)*, offered a poster showing that breast cancer patients have a high incidence of myofascial trigger point development, no matter if they are treated surgically, with radiation, with chemotherapy, or with hormones. Treating co-existing MTPs may relieve a substantial amount of post-treatment breast cancer pain.

- **Dr. H. Mueller-Ehrenberg** *(Munster, Germany)* and his co-author have found myofascial trigger points to be common in sports-related injuries. They have been able to improve pain caused by these injuries by disrupting the MTPs using piezo-electric-generated focused extracorporeal shock wave therapy, decreasing the pain and measurably improving the performance and muscle function of the athletes. Central sensitization is often unacknowledged and untreated in osteoarthritis, as are co-existing MTPs, and the pain may have a spinal segmental component that may include sclerodermal hyperalgesia of the supraspinous ligaments. Peripheral pain can cause and maintain central sensitization, but the hypersensitive central nervous system must be treated to enable the peripheral areas to better respond to their treatment.

- Another poster showed that the use of prolonged vitamin E treatment in CFS patients helped their muscle fatigue and hyperalgesia.

At last, it was time for **Dr. Robert Gerwin** *(Bethesda, MD, USA)*, and yes, there was luscious fruit and some very fine chocolate to drizzle over it. Even better, Dr. Gerwin presented a hypothesis that may supply the missing puzzle pieces in the formation of MTPs. Until now, we have not had an explanation for the excess release of acetylcholine, the excess release of calcium, and the excessive motor endplate noise that occurs during this process, nor did we understand why the taut band forms. According to Dr. Gerwin, these phenomena could be explained by a dysfunctional ryanodine receptor calcium channel. This dysfunctional ion channel could promote the excessive calcium release from the sarcoplasmic reticulum, resulting in persistent muscle fiber contraction. Gates in the cell wall, like tiny air-locks in a space station, allow charged particles, called ions, to allow calcium, potassium and other minerals to flow along pathways in and out of the cell membrane. Once inside, these ions can affect the interior metabolism of the cell. The pathways are called ion channels. An illness caused by dysfunction of the gate mechanism is called a channellopathy. So MTPs could be caused by a channellopathy. This offers a whole new way of looking at myofascial pain, and perhaps a whole new way of treating it. I hope researchers will take note and mobilize forces to investigate this.

This overview is but a brief distillation of over 40 pages of handwritten notes, some handouts, discussions with presenters and poster defenders, and annotated highlights from a few abstracts. If you are interested in finding out more about the posters, I have included references and some brief annotation on my website at: www.sover.net/~devstar. (Enter the keyword "Myopain" on your computer's "Find" function.) The poster abstracts are available in a special edition of *The Journal of Musculoskeletal Pain*, the official journal of the International Myopain Society. Please urge your doctors to join this organization through the IMS' website, located at: www.myopain.org A subscription to the *JMP* is included with membership.

Last but not least, while visiting with Richard Finn, Director of the Pittsburgh School of Pain Management, at the Myopain conference, I learned that the National Association of Myofascial Trigger Point Therapists has a new resource for patients on their website at www.myofascialtherapy.org/symptomcheck showing many common MTPs and their referral patterns.

Many other important events happened "offstage," including the development of friendships that I hope will last a lifetime. It's always a bittersweet thing to look around a conference room and realize that here, at least, there is a place where I don't have to explain FM and MTPs. Now I had to go back to a place where that education is lacking.

I want patients to know about these researchers and clinicians. Whenever you feel discouraged, remember them. You aren't alone. People are working to make a difference, they are achieving a great deal, and they are turning on the light at the end of the tunnel.

About the Author: Devin Starlanyl specializes in education in the fields of fibromyalgia and chronic myofascial pain. She is co-author of the popular book, *Fibromyalgia & Chronic Myofascial Pain: A Survival Manual*, and author of *The Fibromyalgia Advocate*. In addition, she has written numerous medical journal articles and created an extensive informational website for patients, supporters and medical professionals at www.sover.net/~devstar. She is the past director of the Fibromyalgia and Chronic Myofascial Pain Institute and also serves as a consultant and clinical researcher. As someone who has both FM and CMS, she has a keen understanding of both conditions from the inside out.