Fibromyalgia and Chronic Myofascial Pain & Dysfunction: Bodyworkers

The effect of touch can be akin to magic if you are adept at the art of palpation. The more educated the fingers, and the mind behind them, the more value you will be to your clients. There is much misinformation about fibromyalgia (FM), lack of knowledge about myofascial trigger points (TrPs) and even less understanding of chronic myofascial pain and dysfunction (CMPD). This handout is a guide to help you help your clients. Clients with CMPD and FM must be treated differently than others, or harm may result. Inappropriate exercise and bodywork are very common yet avoidable perpetuating factors.

Fibromyalgia (FM) is not a "wastebasket diagnosis." It is not a catchall term for achy muscles, and it is not the same as TrPs or CMPD. We've long known that FM is not a muscle disorder. (15) Research describes the core of FM dysfunction in the central nervous system (CNS); the brain and spinal cord. (2; 4; 19 p17) FM is not progressive. (29) If symptoms worsen with time in spite of adequate treatment, there is some perpetuating factor that has not been properly addressed. **There are no such things as fibromyalgia trigger points.** The tender points of FM and the trigger points of CMPD are different, and in significant ways, although they often co-exist in the same client. [5; 11; 19 p18] FM tender points were not designed to be diagnostic, but only to screen subjects for FM clinical studies. Tender point numbers can vary from day to day and hour to hour, just like symptoms. It is the evidence of central sensitization, with allodynia and hyperalgesia, that help make an FM diagnosis. [22]

One of the most important concepts about FM and is wind-up, or temporal summation of second pain (TSSP). [20] When the CNS is bombarded with significant irritation, that is the "first pain." If the CNS is irritated again, its response can change. It begins figuratively tensing the brain's "shoulders" and clenching its "fists", quivering those little nerves, waiting for the next blow. It winds up like a tight spring. If the stimulation continues or recurs, the "second pain" level climbs higher. In FM, increasing central sensitization takes less stimulation and after-effects are greater and more prolonged. [21] Avoid further sensitizing your client by causing more pain. In FM, the brain can be so preoccupied with processing pain signals that other functions decrease. [7] With FM and CMP, there can be an inability to concentrate, think clearly, or remember things. This "fibrofog" can be aggravated by weather changes, cold, humidity, excessive physical activity, physical inactivity, hormonal fluctuations, sleeplessness, anxiety, stress, pain, depression, or mental or physical fatigue. Written instructions and other resources can be helpful.

FM can cause diffuse widespread pain, but it doesn't cause localized symptoms, including numbness and tingling. TrPs can entrap nerves, blood and lymph vessels. FM is not a diagnosis of exclusion and usually co-exists with other conditions. TrPs, arthritis, and other pain sources are pain **causes.** FM is a pain **amplifier.** Distinguishing myofascial TrPs and FM is simple when the TrPs are few and acute, but much more difficult when the TrPs have evolved into CMPD through neglect or inappropriate treatment. [19 p 39] Clients may come to you with the diagnosis of FM but may have CMPD instead, or have both. You can't diagnose, but you **CAN** write a report noting specific muscles or attachments with taut bands/contraction knots, and that according to the criteria of Travell and Simons' medical texts, your client has characteristic symptoms of myofascial TrPs in specific muscles. Once the diagnosing care provider receives your report, your client's life may improve. The primary care

provider may begin referring her/his patients to you for TrP assessment and treatment. You win. Your client wins. The primary care provider wins.

A TrP is a hyperirritable contraction knot located in a taut band. TrPs are areas in energy crisis, and **in contracture.** Contraction of muscle is driven by nerves, and contracted muscles can be strengthened. Physiologically contractured muscles are tight due to chemically induced changes, and cannot be strengthened by exercise. They need to be lengthened, and the TrPs treated. Simons' Integrated Hypothesis of Trigger Point Formation is the accepted mechanism. [16] Mechanical overload of the muscle causes massively excessive release of the neurotransmitter acetylcholine, causing excessive calcium ion release from the cellular area known as the sarcoplasmic reticulum. This chemical deluge causes formation of contraction knots, or TrPs, in taut bands. The taut band is primarily responsible for increased muscle tension and shortened rest position of the contractured muscle. Sarcomeres are the smallest functional contractile unit of skeletal muscle. Sarcomere changes due to TrP formation have been verified by muscle biopsy. [18] TrP contraction knots have been imaged at the National Institutes of Health USA, [14] and their taut bands have been imaged at the Mayo Clinic USA. [3] Over 30 chemicals released during a TrP local twitch response (LTR) have been identified. [13] There is no logical reason to doubt the reality of TrPs.

Latent TrPs don't cause spontaneous pain. They can restrict movement, cause muscle weakness, autonomic and proprioceptor-associated symptoms, and other dysfunctions. They hurt when you press them, often in the characteristic referral pattern. Active TrPs cause these symptoms plus spontaneous pain. Be gentle when palpating. Pressing TrPs hurts, can cause an LTR releasing irritant chemicals, [13] and can activate TrPs, causing pain for days or weeks. Extra muscle relaxers and pain meds may be required before and after TrP exam, and your client may need supportive therapies such as spray and stretch. If you are going to palpate an area, treat that area.

The term "myofascial pain syndrome" (MPS) is unsatisfactory for several reasons. The term MPS is also used for temporomandibular disorder and other jaw pain, leading to diagnostic and research confusion. [17] Consider research concluding a jaw splint to be an effective treatment for MPS. Does a mouth splint help your chronic pelvic or low back pain clients? Also, the characteristic dysfunction involved in the formation of TrPs involves both nerve terminal and postjunctional muscle fiber, thus TrPs are a neuromuscular **disease** rather than **a syndrome**. [16] I recommend the term CMPD rather than MPS for chronic TrPs. Dysfunctions can be as disabling as pain and are often overlooked. Chronic myofascial pain due to TrPs is not a diagnosis of exclusion and can co-exist with many chronic conditions. [2; 5] Once you become familiar with the concepts of myofascial medicine, you will find TrPs in most if not all of your chronic pain clients.

You may have been taught to strengthen a weak muscle, but TrPs have different needs. EMG studies indicate "...in muscles with active TrPs, the muscle starts out fatigued, it fatigues more rapidly, and it becomes exhausted sooner than normal muscles."[19 p22] The combination of delayed relaxation, increased fatiguability, referred inhibition and referred spasm in other muscles, means that **you cannot strengthen a muscle that has a TrP.** Muscles work in functional groups, but these groups become dysfunctional when one of more of the group is contractured due to TrPs. When one or more of the muscles in a group is contractured by TrPs to some extent, reciprocal inhibition is dysfunctional, producing weakness. Determine why the muscle is weak and the type of weakness it has, and if any muscles are inhibiting it. "Myofascial TrPs contributing to or causing the weakness may be in the same muscles and/or in functionally related muscles."[19 p114] The weakness can be due to inhibition by TrPs in other muscles. Treat the CAUSE of the weakness.

Some muscles move in several ways. They can belong to several different muscle groups. A muscle may work fine when it performs one action, but may be weak when it tries to perform another due to reciprocal inhibition. It may not contract at all, when, in a healthy state, it would function as a prime mover, or assist a prime mover. The treatment is not to try to strengthen the weak muscle, but to find the TrPs in inhibiting muscle(s) and treat them. Travell and Simons us the anterior deltoid as an example [19 pp26-27] The muscle that is inhibited during shoulder flexion may work well during shoulder abduction. In this case, the infraspinatus muscle is the inhibitor. A weak psoas may be inhibited by a weak iliacus or weak gluteals. After treating the inhibiting muscle(s), run the previously weakened muscle through at least 2 passive range of motion (ROM) exercises. This helps retrain the muscle patterns. “TrPs can influence motor function of the muscle in which they occur, and that their influence can be transmitted through the central nervous system to other muscles.” [19 p23] Some muscles become shortened and abnormally excitable; some weak and inhibited. [19 p24] Clients with TrPs may have disturbed motor functions, spasm of other muscles, weakness of involved muscle, loss of coordination and/or decreased work tolerance. [19 p21]

A muscle burdened with TrPs is already working as hard as it can. The contraction knot of a TP is associated with sections of muscle fibers that are already maximally contracted. (19 p73) The last thing needed is to try to tighten contractured muscles further. When a muscle is in sustained contracture, there is “increased metabolic demand and impaired metabolic supply.” “Circulation in a muscle fails during a sustained contraction that is more than 30% to 50% of maximum effort.”[19 p71] The sustained muscle shortening of TrPs can literally have a crushing effect on microcirculation. So what constitutes a contractured muscle? A muscle with one sarcomere that is dysfunctional may not have a palpable TrP, but it is still microscopically contractured. A muscle that has 10 or 100 dysfunctional sarcomeres may have multiple contraction knots of different sizes. Much depends on the location of sarcomeres and their relation to one another. **A MUSCLE THAT HAS A TRIGGER POINT CANNOT BE STRENGTHENED.** Trying to do so causes the TrPs to worsen. Satellite or secondary TrPs can develop, either from overuse as they are recruited to shore up weaker muscles, or from being in the referral zone of another TrP.

Patients may have clusters or chains of TrPs. [16] When any TrP is present in a client with chronic symptoms, consider the possibility of other TrPs adding to the symptom load and maintaining chronicity. Inappropriate treatment, including exercise, is a preventable TrP perpetuating factor. The harm that can result to your client cannot be overestimated. Contracture from TrPs can maintain displacement stress on joints (including vertebrae), and resulting abnormal sensory input from the joint can affect the muscles. [19 p40] Clients usually come for pain relief. They may not connect the pain with a dysfunction, such as the inability to open the mouth fully.

Although chronic pain is usually defined as beginning after 6 months from onset, a time span for CMPD is not logical. One client may have had chronic pain from other sources for years, yet only recently developed TrPs. Another may have multiple TrPs that developed singly and then became latent, only to have CMPD become evident after an activating factor such as trauma or infection. Adding to confusion is the common practice of substituting descriptions for diagnoses. Terms such as "atypical facial neuralgia," "chronic low back pain" and "tension headache" are descriptions. Some descriptions have been given “diagnostic” codes for insurance purposes, and some clinicians use them as if they were actual diagnoses. As of this writing, there are no insurance codes for TrPs or CMPD. Clients with TrPs in multiple quadrants are often given the diagnosis of FM without the understanding that the TrPs are often a **source** of symptoms, and the FM is the **amplifier**. Clients and care providers need to understand the cause of their symptoms and the basics of self-treatment.

Stage 1 CMPD consists of simple TrPs that are chronic due to lack of adequate treatment and control of perpetuating factors. If Stage 1 CMPD is inadequately addressed, it tends to develop into Stage 2 CMPD, (CMPD with one or more central sensitization conditions such as FM, migraine, and/or IBS). CNS sensitization enhances and prolongs pain experiences. Complex overlapping referral patterns may develop, with greater spillover pain. Stage 2 CMPD may develop rapidly in clients with potential perpetuating factors such as co-existing conditions, or during infections or trauma as multiple latent TrPs activate. Become familiar with non-pain symptoms associated with each TrPs. They are clues that help you find where to look for symptom-generating TrPs. Palpation of muscles and observation of movement may indicate which muscles are contractured, inhibited and/or painful. Symptoms that your client might not recognize as being related to TrPs include: cold or hot spots on the body, sweating, teary eyes, a constantly runny nose, buckling knees or ankles, balancing problems, cardiac arrhythmia, dizziness, shortness of breath, or ringing in the ear. Many non-pain symptoms are described elsewhere (23; 19; 25) Your client may come in with complaints caused by the most recently activated TrP. Don't become discouraged if, once that TrP is resolved, pain shifts to another TrP. Expect the pain to switch sides as secondary TrPs that have developed in muscles used to compensate for those weakened by primary TrPs scream for attention. Explain this possibility to your client. If there are active TrPs on one side of the body, corresponding muscles on the other side should be checked for latent TrPs. If those TrPs are present, they need treatment. [1]

There is a misconception that people with FM are pain intolerant. "Pain sensitivity" and "pain tolerance" are different. FM causes hyperalgesia, a heightened sensitivity of pain, but FM clients may have great pain tolerance. They endure more than is recognized. [10] Myofascial TrPs can cause pain as severe, or more severe, than other causes. [28] Even a few TrPs can cause "agonizing incapacitating pain." [19 p13] What of a client with hundreds of TrPs amplified by FM? One cannot treat these clients adequately without understanding both FM and CMPD. It's the combination of pain sources such as TrPs and pain amplification of FM causing the pain severity.

Clients with CMPD cannot be treated the same way you would treat someone with a few TrPs. Everything you do may amplify their discomfort/pain, and their reaction to treatment may be delayed. Encourage your clients to give you feedback as you work, to minimize pain. It's not uncommon for clients to feel nausea and/or a dramatic increase in muscle aches, especially headaches, and/or exhaustion. Warn clients to rest after bodywork. If a therapy is effective, wastes and toxins trapped in the ground substance of the myofascia are released into the bloodstream. These include triglycerides, bilirubin, uric acid and melanin, among other substances. [8] Ensure sure that your client has recovered from one session before you begin another, and remind them how beneficial it is to get those substances out of their bodies.

Even the initial assessment can cause extreme pain and effectively sideline these clients. A good medical history can often tell you where to look for TrPs. Concentrate on the 4 most life-altering symptoms first, including any that disrupt sleep, and encourage your client to talk with you throughout the treatment, and after. Clients with Stage 2 CMPD may have fibrotic muscles, or muscles swollen with interstitial infiltrates; you may be unable to palpate the TrPs, or even to feel the ropy bands. In those cases, use associated specific TrP symptoms, referral patterns, and pain at the end of range of motion. Often, it is easier to palpate ropy bands in limbs if the limb is extended three-quarters of the way, and the same is true of other extensible areas such as the jaw, and positioning the muscle can be a key factor in TrP availability. You may be able to feel the taut band by gently rubbing across the direction of the muscle fibers. [19 p21] Pressing on one TrP can cause a spasm in another muscle. [19

p24]. For example, if you press on the soleus muscle, you can cause a spasm in lumbar paraspinal on the same side. [19 pp 24-25.] The upper trapezius, masseter, posterior cervical and lumbar paraspinal muscles are often referred spasm target muscles. [19 p 26]

Clients with the combination of FM and CMPD may have metabolic problems which cause exhaustion from the slightest exertions. Their exercise programs must begin conservatively. Care must be taken to ensure that they recover between exercise sessions. At first, treatment might be limited to brief sessions, with long spaces between for recovery. Your client may be able to tolerate only a half-hour of therapy or less, once a week or less, depending on the severity of the TrPs, their number and consistency, the level of CNS sensitization, and the health of the client's detox systems. This process can't be rushed. Continuing to exercise in spite of pain can aggravate the CNS sensitization. Any new client, new therapy, or new body area must be approached carefully and cautiously. Delayed onset muscle soreness (DOMS) is especially common in these clients. [19 p25] Avoid friction massage and "strumming." Excess pain can further sensitize the already hypersensitized CNS. Although at first you will be focusing on the most life altering/disabling symptoms, including pain generation, the general guide is to first restore flexion, then side bending, rotation, and then, finally, extension.

When your client is experiencing pain while at rest for a considerable part of the time, TrPs are very active and rarely respond favorably to anything more than gentle, passive stretches and hot packs. Gentle therapies such as cranio-sacral release may be tolerated. Nerve stripping massage should be avoided on clients with CMPD and any central sensitization state, such as FM, IBS or migraine. Muscles with active scar tissue usually have TrPs. [27] Scars may significantly impair mobility and increase symptom severity. Abdominal scars can affect back mobility. These scars and the tissue around them can often be treated successfully with manual methods, but be gentle. Over-treating TrPs may result in a TrP cascade throughout one or more anatomy trains. In a client with FM and CMPD, pain spikes can result in shock symptoms. Be prepared to minimize stimuli such as dimming lights and turning off music. Monitor the client for need of emergency support. I was urged to stress this possibility by Dr. David Simons himself. It can happen.

A self-therapy program is an important part of any chronic pain program. Exercise intolerance is common in FM clients, with worsening of pain, fatigue and stiffness after a new type of exercise. [24; 9] TrPs may cause at least some of this. Exercise should be regarded as a prescription, needing the right dose, the right timing, and the right kind, and these will change as your client improves or has setbacks. I have seen too many people with Stage 2 CMPD into weight-training and work- hardening programs, resulting in total disability. A contractured muscle **cannot** relax. It's in static overload, which accelerates muscle fatigue. [19 p24] **Repetitions worsen TrPs.** In women with FM, the level of pain, rather than depression or sedentary nature, drives the level of activity and function.[26] Clients with Stage II CMPD cannot gage how much exercise will hurt them. In FM, exercise causes a reduction in temperature and cerebral blood flow. Clients may not be able to think clearly enough during exercise to set limits. Tell them to set a timer, or to exercise with a friend who can keep track of the time. Increased referred TrP pain during or after exercise is an indication that the exercise should be stopped. Remember the cognitive deficits. Have handouts for exercise limits too. Be cautious in recommending exercise, as FM clients have reduced growth hormone secretion, which plays an important role in muscle tissue repair. They may not get deep level sleep, which is when cellular healing occurs. TrPs must be resolved and the muscles returned to healthy length before muscles can be strengthened. You can't rush that process.

Teach your client how to use a tennis ball or other exercise ball, but be sure that they limit the time they use it at first. Tell them, often, that this process cannot be put on a time table. Everything depends on how the individual person reacts. Teach your client one exercise per muscle, to gently lengthen it a fraction of an inch beyond current range of motion. No bouncing or otherwise forcing of the muscle. As TrPs are inactivated, your client should begin carefully graded nonrepetitious exercises in sets, to eventually increase strength and endurance. Demonstrate each stretch, and then have the client demonstrate the stretch to be sure they understand. Have handouts for each one, as cognitive deficits can play a part in compliance. Work on the 4 most life-altering symptoms. If the TrPs are not too active, ask your client to do one "set" the first day, with a little ball work. If they are a little sore the next day, that is acceptable, but if they are in more pain, they need to adjust. They may be able to do the one set and ball work every other day. If they tolerate one set well, ask them to do two sets the next day, but separated –one in the morning and one in the evening. Again, how they do the following day will decide if they can increase stretch sets and the ball work. If your client becomes too tense, ill, cold, or has other problems, exercise may need to be adjusted. Specify relaxation and breathing between each cycle of exercise. When they are able, encourage them to find an exercise such as t'ai chi or a game, with the realization that it may need modification. Any teacher, trainer, or coach needs training in FM and CMPD. Find something that your client enjoys and build around it.

A pool with a temperature outside the 88- to 94 degree F range can cause long-range worsening of TrPs. Cramps can also result from cooler temperatures. Hotter temperatures may bring nausea and extreme fatigue as toxins and other materials from the myofascia flood the system faster than detox can occur. Hot tubs and hot baths should be limited to fifteen minutes or less at first. Some bodyworkers are trained in interior pelvic treatment. TrPs in the vaginal walls often don't have palpable contraction knots, but you can feel the taut bands, at times, in layers. CMPD clients often have short, tight pelvic floors, affecting their breathing and abdominal pressure, and worsening reflux. Be mindful not to over-treat, starting with limited therapy of 15 minutes or less, using numbing agent such as xylocaine approved for internal work. See how your client responds during the next few days.

The key to managing FM is to identity the pain sources, including TrPs, and control them as much as possible. The secret to controlling the TrPs is to identify and eliminate perpetuating factors if possible, including co-existing conditions, or if they can't be eliminated, to manage them to minimize symptoms. A perpetuating factor is anything that initiates, aggravates, or perpetuates a symptom. If TrPs recur after effective treatments, there are perpetuating factors that have not been brought under control. When TrP symptoms ease after treatment but then recur, something is perpetuating them. It is time to look for another cause, including cancer and other systemic diseases, mechanical stresses, such as body asymmetry, head-forward posture, and paradoxical breathing perpetuate TrPs. For FM, perpetuating factors include anything that irritates the CNS. New TrPs perpetuating factors are being discovered, including long torso, short stature, and short lower legs. Hand held devices are becoming more common, and so are TrPs that they generate. The head-forward position is worsened by use of many computer laptops and other devices, and "texting thumb" is a newly recognized. Ehlers Danlos Syndrome Hypermobility Type, now called HEDS or Joint Hypermobility Syndrome (JHS), is common, often missed, and greatly affects therapy. You do NOT stretch a client with HEDS.

When perpetuating factors cannot be eliminated, the TrPs cannot be eliminated. Chronic means exactly that; chronic. Clients, medical team members, and insurance companies need to understand this. That being understood, bodyworkers have the power to effect great change in these clients. For example, surgery and other expensive procedures can often be prevented if TrPs are treated and control of perpetuating factors is done promptly and thoroughly. [12] Specific TrP myotherapy training is

available through Myopain Seminars and the National Association of Myofascial Trigger Point Therapists (NAMTPT). Help with history taking, exam and treatment of clients with FM and CMPD is also available. (23) Your client education tasks will be simplified and your sessions more efficient once that book is in their hands, and in yours. Fact sheets and articles are available at www.fmcmpd.org There are in-depth treatments for individual TrPs in the definitive myofascial medical texts (19; 25), and it is appropriate to have them open for reference if needed while palpating, positioning, or otherwise treating your clients. The diagrams in those books are often misunderstood, as the "X" marks were intended by the authors as guidelines only. TrPs can occur in any part of the muscle and its attachments. TrPs can also occur in other tissues. As your client learns more self-therapy techniques—tennis ball acupressure, stretching, good body mechanics and posture—he or she will become responsible for more therapy required. As you get a reputation for superior results, you will have no shortage of clients. Healing begins when the cycle of pain/contracture is broken. Guiding FM and CMPD clients along the healing path can be an exceedingly fulfilling experience, once you know the way.

References:

1.Audette JF, Wang F, Smith H. 2004. Bilateral activation of motor unit potentials with unilateral needle stimulation of active myofascial trigger points. Am J Phys Med Rehabil. 83(5):368-374.
2.Bennett R. 2007. Myofascial pain syndromes and their evaluation. Best Pract Res Clin Rheum 21(3):427-445.
3.Chen Q, Bensamoun S, Basford JR et al. 2007. Identification and quantification of myofascial taut bands with magnetic resonance elastography. Arch Phys Med Rehabil. 88(12):1658-1661.
4.Clauw DJ, Arnold LM, McCarberg BH; FibroCollaborative. 2011. The science of fibromyalgia. Mayo Clin Proc. 86(9):907-11.
5.Gerwin, RD. 1999. Differential diagnosis of myofascial pain syndrome and fibromyalgia. J Musculoskel Pain 7(1-2):209-215.
6.Gerwin RD. 2014.Diagnosis of myofascial pain syndrome. Phys Med Rehabil Clin N Am. 25(2):341-355.
7.Glass JM, Williams DA, Fernandez-Sanchez ML et al. 2011. Executive function in chronic pain patients and healthy controls: Different cortical activation during response inhibition in fibromyalgia. J Pain. 12(12):1219-29.
8.Leahy M, Mock LE III. 1992. Myofascial release technique and mechanical compromise of peripheral nerves of the upper extremity. Chiro Sports Med 6(4):139-140.
9.Ortega E, García JJ, Bote ME et al. 2009. Exercise in fibromyalgia and related inflammatory disorders: known effects and unknown chances. Exerc Immunol Rev.15:42-65.
10.Petzke F, Harris RE, Williams DA et al. 2005. Differences in unpleasantness induced by experimental pressure pain between patients with fibromyalgia and healthy controls. Eur J Pain 9(3):325-335.
11.Pongratz DE, Spath M. 1998. [Myofascial pain syndrome: frequent occurrence and often misdiagnosed]. Fortschr Med 116(27):24-9. [German]
12.Rigberg DA, Gelabert H. 2009. The management of thoracic outlet syndrome in teenaged patients. Ann Vasc Surg. 23(3):335-340.
13.Shah JP, Phillips TM, Danoff JV et al. 2005.An in-vivo microanalytical technique for measuring the local biochemical milieu of human skeletal muscle. J Appl Physiol. 99(5):1977-1984.
14.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. Arch Phys Med Rehab. 90(11):1829-1838.
15.Simms RW. 1998. Fibromyalgia is not a muscle disorder. Am J Med Sci 315(6):346-350.
16.Simons DG. 2004. New aspects of myofascial trigger points: etiological and clinical. J Musculoskeletal Pain 12(3/4):15-21.
17.Simons DG. 1995.Myofascial pain syndrome: One term but two concepts; a new understanding. .J Musculoskeletal Pain 3(1):7 14.
18.Simons DG, Mense S.[2004] Diagnosis and therapy of myofascial trigger points. Schmertz 17(6):419-424. [German]
19.Simons DG,Travell JG, Simons LS. 1999. Travell and Simons'Myofascial Pain and Dysfunction: the Trigger Point Manual: Volume I, edition 2: The Upper Body. Baltimore: Williams and Wilkins.
20.Staud R. 2004. Predictors of clinical pain intensity in patients with fibromyalgia syndrome. Curr Rheumatol Rep. 6(4):281-286.
21.Staud R, Cannon RC, Mauderli AP et al. 2003. Temporal summation of pain from mechanical stimulation of muscle

tissue in normal controls and subjects with fibromyalgia syndrome. Pain 102(1-2):87-95.
22.Staud, R, Vierck CJ, Cannon RL et al. 2001. Abnormal sensitization and temporal summation of second pain (wind-up) in patients with fibromyalgia syndrome. Pain 91(1-2):165-75.
23.Starlanyl DJ, Sharkey J. 2013. Healing through Trigger Point Therapy: A Guide to Fibromyalgia, Myofascial Pain and Dysfunction. Chichester, England. Lotus Publishing.
24.Torgrimson-Ojerio B, Dieckmann NF, Avery S et al. 2014. Preliminary evidence of a blunted anti-inflammatory response to exhaustive exercise in fibromyalgia. J Neuroimmunol. 277(1-2):160-167.
25.Travell, JG, Simons DG. 1992. Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol.II: The Lower Body. Baltimore: Williams and Wilkins.
26.Umeda M, Corbin LW, Maluf KS. 2013. Pain mediates the association between physical activity and the impact of fibromyalgia on daily function. Clin Rheumatol. 2015 Jan;34(1):143-9.
27.Valouchova P, Lewit K. 2009. Surface electromyography of abdominal and back muscles in patients with active scars. J Bodywork Move Ther. 13(3):262-267.
28.Webber TD. 1973. Diagnosis and modification of headache and shoulder-arm-hand syndrome. J Am Osteopath Assoc 72(697-710.
29.Wolfe F, Anderson J, Harkness D et al. 1997. Health status and disease severity in fibromyalgia: results of a six-center longitudinal study. Arthritis Rheum. 40(9):1571-9.