Treating Myofascial Pain

With

Biotensegrity Informed Touch

Tutor: John Sharkey MSc

Welcome to this short course delivered to the community of Embodied Health.
I hope you enjoy the course and find the content relevant and informative.
My hope is that you will be capable of helping chronic myofascial pain patients in your communities to manage or eradicate their chronic myofascia pain issues. Critical to model I teach is change in sensations. Pain is one aspect of nociceptive modulation; however, many people suffer from changes in sensations which can be as debilitating.

Your tutor

John Sharkey is a contributing author to the bible of myofascial pain (the big red books) by Travell, Simons and Simons- "Myofascial Pain and Dysfunction-The Trigger Point Manual" 3rd Ed and two best-selling books on the topic of Fibromyalgia and Trigger Point Dry Needling. A clinical anatomist, exercise physiologist and founder of European Neuromuscular Therapy John Sharkey is a recognised authority on biotensegrity, pain science, exercise science and fascia research. John is also associate editor fascia science and clinical applications for the Journal of Bodywork and Movement Therapies (JBMT).

What is myofascial pain?

Actually, myofascial pain is known as a 'syndrome' and is characterised by local and referred pain as a consequence of the myofascia trigger point (1). Dr Janet Travell coined the term *"Myofascial Trigger Point" piggy-backing on the already established terms "trigger point"* and the separate term "myofascial" both of which were coined by Dr Arthur Steindler as early as 1940. In Europe and elsewhere painful muscles due to what people called "knots" were well recognised. What was not known at the time (and is still actively debated) is the source and pathophysiology of this pain. Many terms have been used including myogelosis, rheumatics, fibrosis and myalgia.

A myofascial trigger point, according to Myofascial Pain and Dysfunction: The Trigger Point Manual, by Travell, Simons and Simons 3rd Edition 2018, is a focus of hyperirritability located in a taut band within a muscle that, when compressed, is locally tender and, if sufficiently hypersensitive, gives rise to referred pain and tenderness. Myofascial Trigger Points (MtPs) can mimic and/or compound the symptoms of other conditions. MtPs can be associated with endometriosis, interstitial cystitis, irritable bowel syndrome, dysmenorrhea and prostatitis but also can mimic toothache, plantar pain, knee pain and others. Pain is a very personal experience. Pain is influenced by belief systems, past experiences, conceptualised era-specific norms all shaped by life events (2)

Pain can be a liar. Research has changed the way we think about pain (Moseley 2012). Pain is a child of the brain—to fully understand it we must meet the entire pain family. Peripheral tissues are close relations and could be viewed as brothers and sisters, while tissues such as muscle fibers and sarcomeres would be first cousins.
Mechanoreceptors, proprioceptors, and nociceptors might be the irritated family members, constantly taking information to and from mom and dad concerning irritating older siblings. These various members of the family can have a tendency to exaggerate or distort the truth. For example, mom and dad can overreact, underreact, or misread the situation, dishing out a response that is disproportionate and not appropriate to the reality; this is ultimately referred to as *allodynia*.

Chronic pain states are defined by significant changes in neuronal activity; such changes are profoundly influential in pain matrix mechanisms. Neuroplastic changes occur in the spinal cord, thalamic nuclei, cortex, and limbic system, and can alter pain thresholds, degree of sensitivity to pain, and the overall pain experiences of our patients (Woolf 2010).
Research by Staud (2011) describes spinal segmental sensitization (SSS) as being caused by heightened dorsal horn activity, brought about by constant bombardment of nociceptor impulses from the periphery (due to damaged or sensitized somatic or visceral tissues).

Clinical experience of thousands of practicing therapists across the globe identifies pain referral patterns that cannot be of nerve origin. Travell and Simons (1992) reported myofascial trigger points within the soleus muscle that refer deep pain to the ipsilateral sacroiliac joint. Additional myofascial trigger points in the soleus refer exceptional pain to the face and jaw. Some mechanism or mechanisms other than nerve pathways must be at play in such situations, as nerves exclusively refer pain inferiorly (the face is the exception).
Far too few therapists and medical doctors are aware of the perpetuating role of myofascial trigger points as a combining source of sensory bombardment (Shah and Gilliams 2008), with the possible result of chronic pain in various guises. Myofascial pain, according to Fogelman and Kent (2015), is an "eminently treatable condition" yet almost "universally underdiagnosed by physicians and undertreated by physical therapy modalities." Constant noxious bombardment of the dorsal horn neuron causes a release of glutamate and substance P at the segmental level. By binding to their respective receptors on post-synaptic neurons, these chemicals induce sensitization of wide dynamic range (WDR) neurons, thus further sensitizing adjacent spinal segments. The sustained release of glutamate and substance P leads to apoptosis (programmed cell death) of inhibitory neurons. This perturbation leads to a sustained sensitized state, which in turn lowers neuronal pain thresholds, activates previously inactive synapses (expansion of the receptive field of pain), and leads to allodynia and hyperalgesia (Shah and Gilliams 2008).

Central sensitization that is maintained by myofascial trigger points and other peripheral sources can be reversed over time. Myofascial trigger point dry needling has been shown to be effective in that regard and therefore a worthwhile therapeutic intervention (Srbely et al. 2010). A more recent study measuring the concentrations of a diversity of biochemicals—including β-endorphin, substance P, tumor necrosis factor-α, cyclo-oxygenase-2, hypoxia-inducible factor 1-alpha, inducible nitric oxide synthase, and vascular endothelial growth factor—found that dry needling of trigger points modulates the concentrations of these noxious chemicals in a dosage-dependent manner (Hsieh et al. 2011).

Pain

Pain can have a crepuscular aspect to it. Time can also be a healer. However, chronic myofascial pain patients know all too well the feeling of misery and despair when neither time nor medication reduces the untiring relentlessness of pain or changes in sensation.
Note: When I refer to *pain*, this is taken to mean "pain and changes in sensations."

Examples of changes in sensations

Numbness, itch, cold, heat, increased sensitivity to light, sound, other, feeling of water running on skin, difficulty breathing, headache, dystonia, limb or body-part disassociation.

Indications and Contra-indications

Myofascial trigger-point therapy is contraindicated for clients who are seeking a relaxing massage or those who cannot provide appropriate feedback . This form of treatment can result in pain experienced during treatment or can leave muscles feeling sore several hours after the treatment. It is not advised to provide treatment to patients who recently had tattoos applied to the skin or have undergone recent surgery. Also people who are medically ill, frail, or who have conditions such as oedema, cardiovascular disease, osteoporosis and those currently undergoing medical care. Arteriosclerosis- because of the risk of blood clot formation, a GP's or medical specialist's approval must be given in writing. Information regarding all medications should be provided. Myofascial trigger-point treatments may also be contraindicated for cancer patients. Malignancies- should a patient state they have a malignancy, their GP or specialist must give written permission before any treatment can be given.

Persons with severe osteoporosis or atherosclerosis, patients who have been prescribed to take blood-thinning medications, and patients who have had a hip replacement, spinal fusion, decompression, or discectomies will require clearance from their doctor. Open wounds or broken skin should be avoided. Hematomas- these should never be pressed, massaged, or stretched.

General Standards and Guidelines—Pre-treatment

- **Ensure you have your patient's signed consent before treatment informing them clearly that they may experience soreness following the treatment.**
- **Hands must be properly washed, with soap and lukewarm water, and be clean before beginning every treatment.**
- **Nails should be smooth and short.**
- **Single-use disposable paper towels are recommended.**
- **Therapists should follow national guidelines and standards provided by a competent authority within their own geographical location or follow international best practice if such standards are not locally in place.**
- **Therapists must carry out a full medical health screening in advance of any treatment to ensure the patient's suitability for treatment, and to provide a comprehensive description of the procedure, including all potential risks.**
- **Target the four most significant symptoms. Identify all of the known and suspected perpetuating factors: control the known ones and investigate the suspected ones. Include tests (such as a sleep study), exercise regimens (including correct breathing technique), and dietary changes.**
- **Therapists should also be qualified in emergency first aid (first responder), and, although not essential, I recommend courses that include defibrillation.**

General Standards and Guidelines—Post-treatment

- **Allow the adult patient appropriate time on the treatment table before they return to standing.**
- **If myofascial trigger point treatment causes the eyes to water post-treatment, advise your patient to allow appropriate time before driving.**
- **Encourage your patient to take it easy over the following day or so, and to avoid repetitious or stressful movements. The patient will require energy to accommodate and facilitate change post-treatment. This treatment can also cause some muscle soreness, similar to delayed onset muscle soreness, which can last from one to three days. Therapists should ensure that their patients are aware of this and have received written information in this regard (which they must sign)**
- **Patients should avoid very cold (ice) applications, hot baths or showers, saunas, or steam rooms for a number of days following treatment.**
- **Cool water is the post-treatment of choice.**

General Standards and Guidelines—During Treatment

- **Take appropriate steps to ensure patient comfort.**
- **To ensure the best possible treatment outcomes, it is advisable to avoid treating too many muscles in a single treatment: it is recommended to limit treatment to between three and five muscles in any one treatment. Keep in mind, however, that this could constitute many hundreds of myofascial trigger points. The patient must have the capacity to facilitate changes as a result of the treatment. Less in this case is more. Practitioners can complement the treatment with other non-invasive modalities.**
- **The hand and fingers are used to locate the myofascial trigger point and to identify key anatomical landmarks**
- **Ischemic pressure should be applied for a suitable length of time.**
- **Regular communication with the patient is advised during the treatment, ensuring feedback and information is received from the patient.**
- **Look for non-verbal signs, such as facial expression, breath holding, and clenching.**
- **For those with epilepsy more caution is required because of their low tolerance to strong sensory stimulation.**

Pathophysiology of the Myofascial Trigger Point

To date the most widely accepted theory for the MtP and myofascial pain is due to the release of substances from insults specific to the myofascia. Substances include adenosine triphosphate (ATP), bradykinin (BK), 5-hydroxytryptamin (5-HT, serotonin), prostaglandins, potassium (K+), and others which affect levels of protons (H+), from the acidic milieu, resulting in ischemia. These substances activate muscle nociceptors. They also induce the release of Calcitonin Gene-Related Peptide (CGRP) from the motor nerve terminal and from the muscle nociceptors, which in turn increases motor endplate activity.

Biotensegrity and Myofascial Pain

My work in anatomy, physiology, and touch therapy has been significantly influenced by my mentor Stephen Levin M.D., an orthopedic surgeon who coined the term *biotensegrity*. Dr. Levin was the first to promote the biotensegrity model as the new biomechanics for all biological structures. I describe biotensegrity as "anatomy for the 21st century."

Biotensegrity has emerged as the most significant development in human anatomy in recent years, with important ramifications for a wide range of medical practitioners, including surgeons, bioengineers, and human movement specialists. Bespoke Thiel soft-fix dissection techniques are providing a new vision and understanding of the continuity of the human form.
The term *tensegrity* was coined by Buckminster Fuller by combining the words "tension" and "integrity." Fuller's student Kenneth Snelson built the first floating compression structure of tensegrity in 1949, while Dr. Stephen Levin was the pioneer of "biotensegrity," which was born out of his publications on the topic in the early 1970s. As a clinical anatomist, I have investigated this model and the role of fascia in my dissections in order to better understand the mechanisms of human movement and chronic pain, while providing new anatomical knowledge and awareness, leading to less invasive surgical and non-surgical therapeutic interventions.

Borrowing from Fuller's term *tensegrity*, Levin added the prefix *bio*, which refers to all living structures. Biotensegrity is the application of Fuller's tensegrity concepts to biological structure and physiology. In the biotensegrity model the limbs are not a collection of rigid body segments: the upper and lower limbs are semi-rigid, nonlinear, viscoelastic bony segments. These segments are interconnected by nonlinear, viscoelastic connectors, including cartilage, joint capsules, and ligaments, and have an integrated nonlinear, viscoelastic active motor system—the muscles, tendons, and fascia (connective tissue).

Biotensegrity counters the notion that the skeleton provides a frame for the soft tissues to hang upon; instead, biotensegrity structures are integrated, pre-tensioned (self-tensioned), continuous myofascial networks with floating discontinuous compression struts (the skeleton) contained within them.

A column whose center of gravity is constantly changing while its base is rapidly moving horizontally would require forces too great to consider. The forces become incalculable if the column comprises several rigid bodies, hinged together by flexible, virtually frictionless, joints.

Daniele-Claude Martin, a pioneer in the world of biotensegrity and a member of the Biotensegrity Interest Group (B.I.G.), co-authored a chapter with Dr. Levin in the excellent book *Fascia: The Tensional Network of the Human Body* (Schleip et al. 2012).
The title of the chapter was "Myofascia as the tensioner in the biotensegrity model," and Levin and Martin made the following important points regarding tension:
"Central to this concept is the understanding that the fascia imparts a continuous tension to the system. Fascia displays the nonlinearity characteristic of all biologic tissues. In nonlinear tissues, the stress/strain relationship never reaches zero (a characteristic of linear materials); and there is always tension inherent in the system. It gives the 'continuous tension,' an essential component of tensegrity, that helps set the tone of the organism. There are active contractile elements in fascia (Schleip et al. 2012) and the fascial network is intimately bound to muscle (Passerieux et al. 2007). Muscle also has intrinsic 'tone' and is never completely lax, and the entire fascial network is continually tensed, by both intrinsic tension and active contractions that can be 'tuned".

This concept of tuning the fascia blends well with the fascial response to touch therapy. The change in tissue tension is obvious as the connective tissue is restored to normal parameters, creating a redistribution of tension and compression (biotensegrity).
This new model for biological structures that is based on the concept of biotensegrity identifies fascia as the major tensional member. In a tensegrity model, continuous tensile forces (from the myofascial tissues) provide an "ocean" within which the struts float (in the human body, the "struts" are loosely representing the bones, which are also continuous, as they are fascia, yet virtually separated and do not directly transmit compression forces directly to each other). The tensional members are continuous and directly distribute their tensional load to all other tensional members, as described by Fuller in 1961.

Why Consider Myofascial Trigger Point Therapy in the Treatment of Chronic Myofascial Pain?

Myofascial pain arises from muscle and its connective tissue (Shah and Heimur 2012). According to Simons, Travell, and Simons (1999), and supported by numerous researchers over the preceding years (Mense 2010), myofascial trigger points are responsible for, or play a role in, as much as 85% of musculoskeletal pain. Myofascial trigger points constitute commonly overlooked or ignored causes of common musculoskeletal pain conditions, chronic or acute. My colleague and researcher Jay Shah has demonstrated that active myofascial trigger points have a noxious biochemical milieu—including substance P, bradykinins, and other substances—which is at the root of the pain. Many drug-based therapies have in fact been demonstrated to be no better than a placebo.

A double-blind controlled dry needling study by Mayoral et al. (2013) demonstrated that the treatment of myofascial trigger points was superior to a placebo. In clinical practice, pain management or the eradication of pain is the primary focus for many patients and health care practitioners. It is worth noting that changes in sensation already mentioned but including a constant itch, numbness, tingling, burning, crawling, or feelings of water running on the skin are all components across the spectrum of pain.

These are real sensations that patients feel on an on-going daily basis—for some, twenty-four hours a day, every day. Not necessarily a pain per se, a change in sensation is rather a variation on the theme of pain. A pain experienced radiating down the anterior upper limb and terminating in the wrist and palm connotes a brachial nerve insult. When all avenues of traditional medical assessment have been exhausted without identifying any underlying pathophysiological cause, or etiology, then soft tissue myofascial trigger points must be considered. Myofascial trigger points can mimic or play significant contributing roles in migraines, cervicogenic headaches, frozen shoulder and associated pain issues, carpal or tarsal tunnel syndromes, frozen or lower back pain, sciatic pain, radiculopathies, knee and ankle pain, and a host of other conditions. Put simply, myofascial trigger points can mimic anything. For those patients who have "tried everything" with little or no therapeutic benefit, myofascial trigger point therapy is worth considering.

Contractures versus Contractions

Contractures are sustained by the chemistry at the innervation site, not by action potentials; they are to be differentiated from contractions (voluntary with action potentials) and spasms (involuntary with action potentials). The actin/myosin filaments glide into a fully shortened position (a weakened state) in the immediate area around the motor endplate (positioned at the center of the fiber). As the sarcomeres shorten, a contracture *nodule* forms—a palpable characteristic of a myofascial trigger point. The remainder of the sarcomeres on either side of this nodule within that fiber are lengthened (shape changing), thereby creating a palpable taut band—another common trigger point characteristic. Other characteristics are spot tenderness of a nodule in the taut band, and the patient's recognition of pain or sensation when pressure is applied to the tender nodule.

Additionally, there may be:

1. **Visual/tactile/autonomic evidence of local twitch response (LTR).**
2. **Pain or altered sensation in the target zone associated with that trigger point when provoked.**
3. **An EMG demonstration of spontaneous electrical activity (SEA) in the nidus (nucleus) of the trigger point.**
4. **A painful limit to range of motion and reduced range of motion.**
5. **A positive test of weakness of the muscle housing the myofascial trigger point.**
6. **Changes in cutaneous humidity (dry or moist), temperature (cool or hot), or texture (rough).**

7. **A "jump sign" or exclamation by the patient because of extreme tenderness of palpated tissues.**

Myofascial trigger points are often associated with the feeling of ropy bands beneath the palpating fingers. Locating and identifying these bands requires excellent palpation skills and knowledge of the cardinal signs. Placing the muscle in a lengthened position may exaggerate the ropy bands and should make them more noticeable to "listening" fingers.
Contraction knots can be small or large, depending on a variety of factors, such as the number of myofascial trigger points making up the contraction knots, the tissue consistencies, and the amount of fluid infiltration involved. When a muscle is burdened with multiple myofascial trigger points, there is pain when that muscle is elongated or the myofascial trigger point is compressed. Pain occurs at the end range of motion (EROM) of the muscle in question, restricting ease of movement.

The myofascial trigger point, in each muscle, causes a recognizable referral pattern. Sometimes those patterns are in the locality of the myofascial trigger points, but they may also cover several muscles. The patterns may not even include the muscle that holds the myofascial trigger points at all, as myofascial trigger points can *refer* pain and can alter sensations. Imagine having a constant itch you cannot scratch, ever.
Imagine a noise in your ear that will not go away, ever. You have to find the myofascial trigger points that cause the symptoms and treat them. Myofascial trigger points mimic everything.

Each myofascial trigger point has its own recognizable pattern—a portrait of pain or changes in sensations. Simons, Travell, and Simons (1999) highlighted the difference between what are known as *active* myofascial trigger points and *latent* myofascial trigger points. Pain and changes in sensations from active myofascial trigger points are recognized by the patient as "their pain." Latent myofascial trigger points, on the other hand, cause pain that is not necessarily recognized by the patient, however, it may be contributing to the patient's problems. Latent and active myofascial trigger points provoke motor dysfunction and impaired muscle activation patterns (Lucas et al. 2004, 2009), weakness, and muscle imbalances. It is vital to appreciate that latent myofascial trigger points can develop into active myofascial trigger points.

The pain patterns offered in this course do *not* have an "X" to "mark the spot" of the myofascial trigger point, as may be found in older texts and books. It is important for everyone to understand that myofascial trigger points can occur *anywhere* in *any* muscle fiber. This short course provides all suitably qualified therapists with safe, effective, and appropriate clinical applications as part of a multidisciplinary approach.

If a muscle is sensitive and shortened or has active myofascial trigger points within it, the patient may feel a combination of sensations and pain. On compression the patient can often feel a reproduction of "their" pain (active trigger point). This is a helpful diagnostic indicator for the practitioner when attempting to identify the true cause of the patient's symptoms.

A New Hypothesis

I wish to offer a new hypothesis while making a stark differentiation between myofascial trigger points and muscle spasm. A muscle spasm requires neural input (monosynaptic reflex arc) and ATP. Wearing my clinical anatomist's hat, I appreciate the fact that, on death, muscles take on a contracted state we call *rigor mortis* (Latin *rigor* means "stiffness," and *mortis* means "of death"). My hypothesis is that some, if not all, myofascial trigger points are a rigor contraction—an electromagnetic entity requiring no neural input and no need for ATP. This is an issue arising at the microscopic level, where the sarcomeres involved are only doing what they have evolved to be excellent at doing—contracting. To differentiate this "out of the normal" type of contraction, we refer to it as a *contracture*. The all-or-none principle would have all the sarcomeres contracting, or none contracting. In the case of a myofascial trigger point, approximately 100 sarcomeres are in a state of contracture within a cluster of myofascial trigger points. Higher up than the microscopic level, at the gross level, what is occurring would require manual therapies, such as positional release, soft tissue release, or other. Once normal electromagnetic activity has been restored, tensional and compressional forces also return to normal, and regular cellular activity can resume.

A Few Words About Dietary Influences

Adequate quantities of minerals and vitamins are essential for healthy muscles and tissues. Many patients presenting with chronic pain are found to be deficient in a number of vitamins and minerals. Vitamins B_1, B_6, and B_{12}, along with vitamin C and folic acid, are important in the war on pain; the minerals calcium, magnesium, iron, and potassium are critically important. All too often, people are confused as to why they are deficient in these important minerals and vitamins, because they will report that they eat well and have normal dietary habits. The problem may not be their diet but rather their personal health choices, such as smoking and drinking alcohol or caffeine. Smoking, for example, annihilates vitamin C, while oral contraceptives affect vitamin B_6 levels. Antacid medication can leave many individuals with the symptoms of chronic fatigue; even writing their signature becomes an effort. Patients with vitamin and/or mineral deficiencies may report feeling unusually cold, bouts of diarrhea, restless leg syndrome, headaches, disturbed sleep, and trigger point pain. Other symptoms include feeling fatigued, muscle cramping, and depression. Metabolic disorders should be ruled out, particularly thyroid problems and hypoglycemia. Referral of patients with vitamin/mineral deficiencies is recommended.

Central Sensitization and Control of Perpetuating Factors

Chronic pain syndromes display significant neuroplastic changes, altered neuron activity, and excitability and adaptations affecting pain matrix structures, specifically the spinal cord, thalamic nuclei, cortical areas, amygdala, and periaqueductal gray areas. In essence, central sensitization is characterized by an amplification of normal neurological activity (Giamberardino et al. 2011a).

Continuous bombardment of the dorsal horn by noxious afferent activity leads to a release of glutamate and substance P; this in turn leads to the activation of previously inactive synapses in the wide dynamic range (WDR), resulting in central sensitization. In normal circumstances, there is a balance between inhibitory and facilitatory neuronal activity in terms of pain management and control (Willard 2008). This results in *spinal segmental sensitization (SSS)*—a hyperactive state of the dorsal horn caused by constant noxious afferent bombardment, originating from damaged or sensitized tissues (e.g. myofascial trigger points or other soft tissue/connective tissue trauma, or visceral structures, such as a gall bladder that has become inflamed because of gallstones). A diagnosis of SSS includes observation of dermatomal allodynia, hyperalgesia, soft tissue pain/tenderness upon palpation, and myofascial trigger points (Giamberardino et al. 2011b).

Hypersensitivity initially occurs at the local segmental level. However, through the process of sensitization of adjacent spinal segments (spillover), a state of "wind-up" caused by *temporal sensory summation (TSS)*—an increased rate of nociceptive pulsing at the dorsal horn—facilitates widespread segmental sensitization, leading to body-wide peripheral pain.
TSS is caused by increased C-fiber input at the dorsal horn and can maintain a state of hyperalgesia in chronic pain patients (Staud, 2011). The stimuli that activate and sensitize the WDR neurons ascend the spinothalamic tract to reach the higher brain centers, where the thalamus and limbic systems are activated (anterior cingulate gyrus, insula, and amygdala). The limbic system is involved in modulating muscle pain, but it also modulates fear, anxiety, and distress. Therefore, increased activity in the limbic system, influencing the perpetuation of pain syndromes, can contribute to the fear or emotional stress associated with chronic pain syndromes (Niddam et al. 2007).

The rostral ventral medulla (RVM), acting as a relay point for descending activity from the periaqueductal gray (PAG), contains a number of "on" and "off" cells that can increase or decrease levels of pain. In the acute phase of injury, the "on" cells provide a protective mechanism—significant pain is evoked, thereby preventing undue movement/activity that might cause more damage. In chronic pain mechanisms, "on" cells remain active, and there appears to be an "on" cell dominance, rather than a balance of "on" and "off" cells that would maintain a balance between facilitation and inhibition (Willard 2008). Additionally, normal descending pain-inhibiting signals are disrupted, leading to a further sensitization of muscle tissue (Niddam et al. 2007).

Spinal Facilitation

Spinal facilitation is an increase in spinal cord neuron activity as a result of noxious peripheral nociceptor bombardment. In normal circumstances, this noxious stimulus is modulated by local mechanisms, or by descending pathways from the cerebral cortex and brainstem. Abnormal constant bombardment leads to cell apoptosis, wind-up, and segmental sensitization (Bishop, Beneciuk, and George 2011). As a consequence, the dorsal, ventral, and lateral horn circuits in the spinal cord may become more readily activated by lower intensity stimuli. Spinal facilitation is characterized by: 1) increased ventral horn output, which results in increased muscle tone (corresponding to the segmental level); 2) increased lateral horn output, which increases nociceptive activity (reflex mechanisms); and 3) increased dorsal horn activity, resulting in an increase in the production of neuropeptides, which can increase inflammatory activity in the affected tissues. The result is increased hyperalgesia, local tissue tenderness, and spillover, which affect adjacent spinal segments (Camanho, Imamura, and Arendt-Nielsen 2011).

Note: Too often doctors and therapists fail to consider the role of myofascial trigger points in chronic pain patients. They therefore fail to treat what may be, at least, a significant underlying perpetuating factor.

Many therapists deactivate myofascial trigger points as they find them, without giving due consideration to the mechanisms that caused them. In such cases, patients enjoy temporary relief but continue to have recurring pain issues that never fully resolve. Therapists can identify symptoms of spinal segmental sensitization and spinal facilitation by evaluating presenting symptoms of allodynia, hyperalgesia, pain pressure sensitivity, and motor and sensory responses (reflex tests, dermatome assessment, and local muscle endurance assessment). Continuous nociceptive bombardment of the spinal cord leads to increased peripheral sensitivity and a state of central sensitization (Shah and Gilliams 2008).
Active and latent myofascial trigger points are found in the tissues of both symptomatic and asymptomatic individuals. Dorsal horn neurons may manifest neuroplastic changes as a result of nociceptive bombardment if left unresolved. Cortical changes amplify the pain state, creating a pain cycle that may be difficult to break, as in the case of chronic pain conditions such as fibromyalgia, chronic fatigue syndrome, and myalgic encephalomyelitis (Camanho, Imamura, and Arendt-Nielsen 2011).

Many people do not appreciate that stress is a normal part of living. It is how our body deals with stress and how we cope and deal with our sensory impressions, and how they stack up against our internal view of our world, that results in distress or eustress. This is part of our fight or flight system, or our ability to confront, avoid, or submit. Failure to resolve a stressful situation by one of these means results in high sympathetic tone, increased cortisol production, increased resting muscle tone, and the possible formation of myofascial trigger points.

Myofascial trigger points are more likely to develop in tissue which has neurological deficits that have been caused by compression, tension, disc dysfunction, facet joint dysfunction, vascular compression, metabolic stress, biomechanical stress, postural stress, etc.

When muscles develop myofascial trigger points, they remain tight, causing local compression of vascular, neurological, and joint/biomechanical structures, thereby hampering the normal function of that tissue. All tissues distal to the nerve involved will likely be affected. Dry needling can release the muscle tension in order to resume normal function, with improved neurological conduction and vascularity. Dry needling should be supported by other appropriate soft tissue manipulation modalities and suitable physical activity.

Keys to Symptom Management

The following ten key aspects should be considered when treating myofascial trigger points:

1. **Differentiate the myofascial trigger points from pain points by using the cardinal signs, which must include palpable nodule and taught band, jump sign, twitch response, painful EROM, referred pain, and autonomic responses.**
2. **Treat the myofascial trigger points that are most superior and medial first.**
3. **The deltoid seldom develops its own active myofascial trigger points. Instead, most are "baby" or "satellite" myofascial trigger points; therefore treat associated muscles within the functional units of the deltoid first.**
4. **The upper trapezius is the "Grand Central Station" of myofascial trigger points and is a major contributor to neck, shoulder, upper back, and head pain.**
5. **Active myofascial trigger points, when irritated by a competent therapist, will result in referred pain or changes in sensation that the patient recognizes.**
6. **Latent myofascial trigger points generally result in pain or change in sensations that the patient does not recognize. These myofascial trigger points may be contributing to, but are not the true source of, a patient's problem.**
7. **Myofascial trigger points can form in any muscle fiber (Sharkey 2008) and not just in the center of a muscle, or where the "X" marks the spot (which is misleading) on so many myofascial trigger point charts. Identify and remove/change the perpetuating factor(s).**
8. **Excellent palpation skills are necessary for locating myofascial trigger points.**
9. **Upper or lower limb tension tests should be administered in order to rule out nerve insults, including compression and/or inflammation.**
10. **Any patient suffering from unresolved pain or changes in sensations should have the possibility of myofascial trigger point involvement ruled out as a primary or secondary cause or contributor.**

Initiating, Aggravating, and Perpetuating Factors

Anything that perpetuates a myofascial trigger point is called a *perpetuating factor*. What initially activates a myofascial trigger point may be different from what aggravates (worsens) or perpetuates (maintains) it, but they are all commonly called perpetuating factors. The key to controlling any symptom is the control of as many perpetuating factors as possible.
An appropriate medical history will indicate whether pain patterns are stable or evolving. Chronic myofascial pain (CMP) is not progressive. The development of satellite myofascial trigger points that worsen symptoms, and the appearance of new symptoms, are indicators that there are perpetuating factors at play. To control symptoms, first identify and control perpetuating factors. Controlling perpetuating factors is vital. Perpetuating factors include whatever impairs muscle function, such as anything that diminishes the cells' access to oxygen and nutrients, hampers the removal of cellular wastes, or adversely affects the metabolism of the neurotransmitter acetylcholine (ACh). Anything that enhances the formation of myofascial trigger points is a perpetuating factor. For instance, anything that constricts the flow of blood to the area will lessen its supply of oxygen and nutrients, adding to the energy crisis. A perpetuating factor can be anything that increases energy demand (trauma, overwork), decreases energy supply (inadequate nutrition, insulin resistance), sensitizes the CNS (pain, noise), decreases oxygen supply (congestion), enhances the release of sensitizing substances (allergies, infections), or increases endplate noise (increased ACh release, reduced acetylcholinesterase).

Perpetuating Factor Types: A Long Short War

We are fighting a war on pain. The foot soldiers of the enemy are those perpetuating factors such as mechanical stressors, including paradoxical breathing, body disproportions, myofascial or connective tissue abuse, and articular dysfunctions. Metabolic perpetuating factors include impairments to energy metabolism, and coexisting conditions, such as pain and a lack of restorative sleep. Environmental perpetuating factors include pollution, medications, trauma, and infections.
Psychological perpetuating factors are also an important area to investigate. The remedies for lifestyle perpetuating factors are often the least expensive, but may be among the most difficult to maintain. To further complicate matters, perpetuating factors often have perpetuating factors of their own. Cognitive therapy and mindfulness can be useful interventions to help us change the way we, and our patients/clients, think about and perceive pain.

Paradoxical Breathing

***Paradoxical* (or *abdominal*) *breathing* is a term used to describe an abnormal chest movement, with the patient's chest moving inward (or not moving at all) during inhalation rather than outward or forward. This means that your patient cannot take a functional breath and is most likely a shallow breather. Paradoxical breathing is a common perpetuating factor but is easy to check if a patient is presenting with this breath rhythm issue.**
To assess for correct rhythm, place one hand on your patient's abdomen. As the patient breathes in, their abdomen should swell as the abdominal cavity extends after the lungs expand. On breathing out, the patient's abdomen should come back in. When this occurs, it indicates that their respiratory muscles are healthy: the patient can move through their physiological range to accommodate the air required and expel residual air.
If the patient's chest is moving in as the breath comes in, and is moving out as the breath goes out, then this is paradoxical breathing. This inconsistent breathing includes mouth breathing, which is inefficient and shallow. Paradoxical breathing may indicate that your patient's body is not getting the oxygen it needs; it can occur temporarily during a time of congestion, such as a cold, and then may be maintained out of habit, or because myofascial trigger points have formed in the diaphragm or other respiratory muscles, thus inhibiting their function. Training and awareness of proper breathing technique are important, but they are only part of the remedial process. Is adequate air coming in through the nose, or is there congestion? If so, why? Check into the possibility of allergies, low-grade sinusitis (sometimes caused by fungal infection), or other problems. A myofascial trigger point assessment is also needed, as myofascial trigger points can cause congestion, and their presence in respiratory muscles prevents these muscles from working properly. An assessment for myofascial trigger points includes accessory respiratory muscles, such as the scalenes and the serratus muscles.

Body Disproportion Examples

A structural leg inequality of 3/16" (0.5cm) can significantly tilt the body.
What is more common, however, especially in people with myofascial trigger points, is pelvic torsion, where one thigh is drawn higher into the pelvis, thereby creating a functionally short leg. In this case, if one adds a heel lift to the shorter leg, the problem is compounded and reinforced. True leg length inequality is a clue to check the whole body for proportional shortness on that side and for compensation on the opposite side. When the horizontal core stabilizers, including the deep ligaments and tendons, are not healthy, a spiral compensating effect can occur, called *rotoscoliosis*; this is a twisting of the tissues around the spinal column and can begin anywhere, with an end result of torsion of the feet, ankles, knees, hips, and shoulders. One area rotates right and the other rotates left to compensate. The body seeks harmony and balance, but this compensatory twisting can create functional hypermobility or restriction of numerous areas.

Another consideration is a one-sided small hemipelvis. The right and left hemipelvis should match. When the hemipelvis is smaller on one side, if a patient is sitting on a flat surface, the upper curve of one hip is higher than the other. Scoliosis and pelvic rotation can develop as other muscles struggle to compensate. The quadratus lumborum muscle is greatly affected by an asymmetrical hemipelvis, with the sternocleidomastoids and scalenes struggling to adjust to the overload from tilted thoracic muscles.

Muscle Insults

Any postural habit that causes prolonged muscle fiber contraction, repetitive low-intensity overload, and muscle strain (such as high-intensity muscle contractions) can cause myofascial trigger points (Edwards 2005). Posture affects respiration and can interact with vestibular dysfunction, compounding the symptoms (Yates, Billig, and Cotter 2002). Close attention must be paid to standing, sitting, and sleeping postures. One of the most preventable types of perpetuating factor is inappropriate physical activity (exercise). It is almost impossible to strengthen muscles that have myofascial trigger points or that are inhibited without first resolving the hypertonic muscles.

Articular Dysfunction

Joint dysfunction can interact with myofascial trigger points. Any mechanical stress affecting joint position can initiate the process of osteoarthritis (AO) (Solomon, Schnitzler, and Browett 1982). Treating myofascial trigger points improves neuromuscular function and coordination, and anything that improves neuromuscular function can prevent or slow the progression of OA (Loeser and Shakoor 2003). Any arthritis treatment and prevention program need to include the treatment of coexisting myofascial trigger points (Cummings 2003).
Myofascial trigger points can cause uneven contraction of muscles. An uneven contraction can cause or contribute to temporomandibular joint dysfunction (TMJD) and may cause bone misalignment (Koolstra and van Eijden 2005). Uneven muscle contraction may also be sufficient to cause jaw articular disc erosion (Liu et al. 2000).

Vertebrae and myofascial trigger points interact. Active myofascial trigger points are associated with neck vertebral-disc lesions (Hsueh et al. 1998). As surrounding soft tissues are unevenly contractured because of myofascial trigger points, vertebrae may shift slightly out of alignment; this misalignment irritates the intervertebral discs. Intervertebral disc adjustments, and their associated ligamentous attachment compensations, cause changes in the angular motion of the body, which further stresses the inferior and superior intervertebral discs of the cervical spine (Kumaresan, Yoganandan, and Pintar 1999).
Soft tissue is often neglected. Disc deterioration may further alter motion and muscle compensation, which can contribute to additional pathologies in facet joints, muscles, and ligaments, resulting in a chronic pain state (Brisby 2006). Disc deformities or bone spurs that show up on imaging, however, may not be the cause of pain.

Surgery performed without soft tissue evaluations can result in failed back surgery (Dubousset 2003). Subsequent surgical scars, adhesions, and postsurgical tightening of soft tissue can cause added stress to adjacent vertebrae, leading to the formation of myofascial trigger points.

I do hope you enjoyed the course.

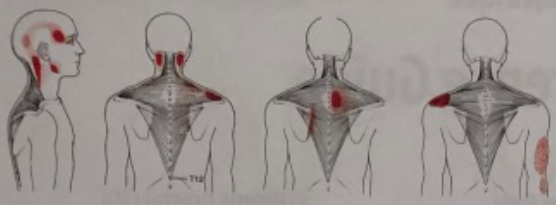
I wish you success in healing,

John Sharkey MSc
Clinical Anatomist, Exercise physiologist
Founder European Neuromuscular Therapy

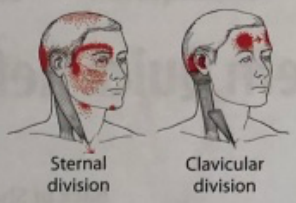
Charts from: Travel, Simons and Simons. 2018 Myofascial Pain and Dysfunction-The Trigger Point Manual 3rd ~Edition. Joseph M. Donnelly, Editor. Wolters Kluwer.

Section 2 Head and Neck Pain

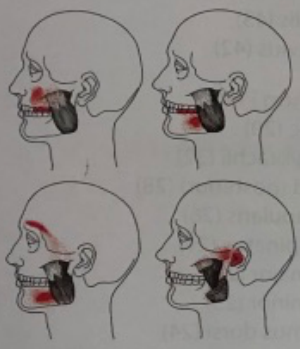
Trapezius



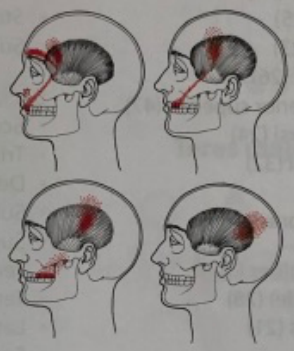
Sternocleidomastoid



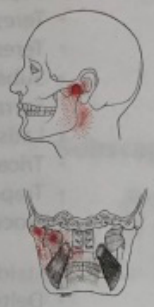
Masseter



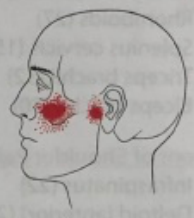
Temporalis



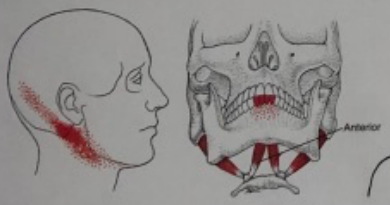
Medial pterygoid



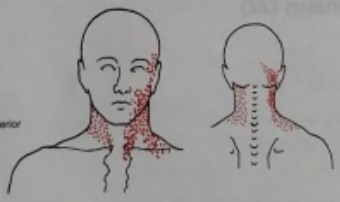
Lateral pterygoid



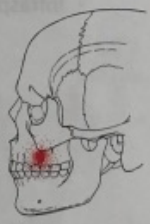
Digastric



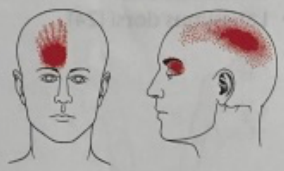
longus colli



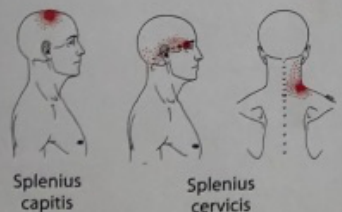
Buccinator



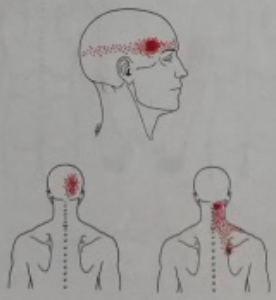
Occipitofrontalis



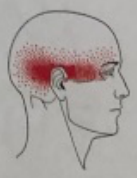
Splenius capitis and splenius cervicis



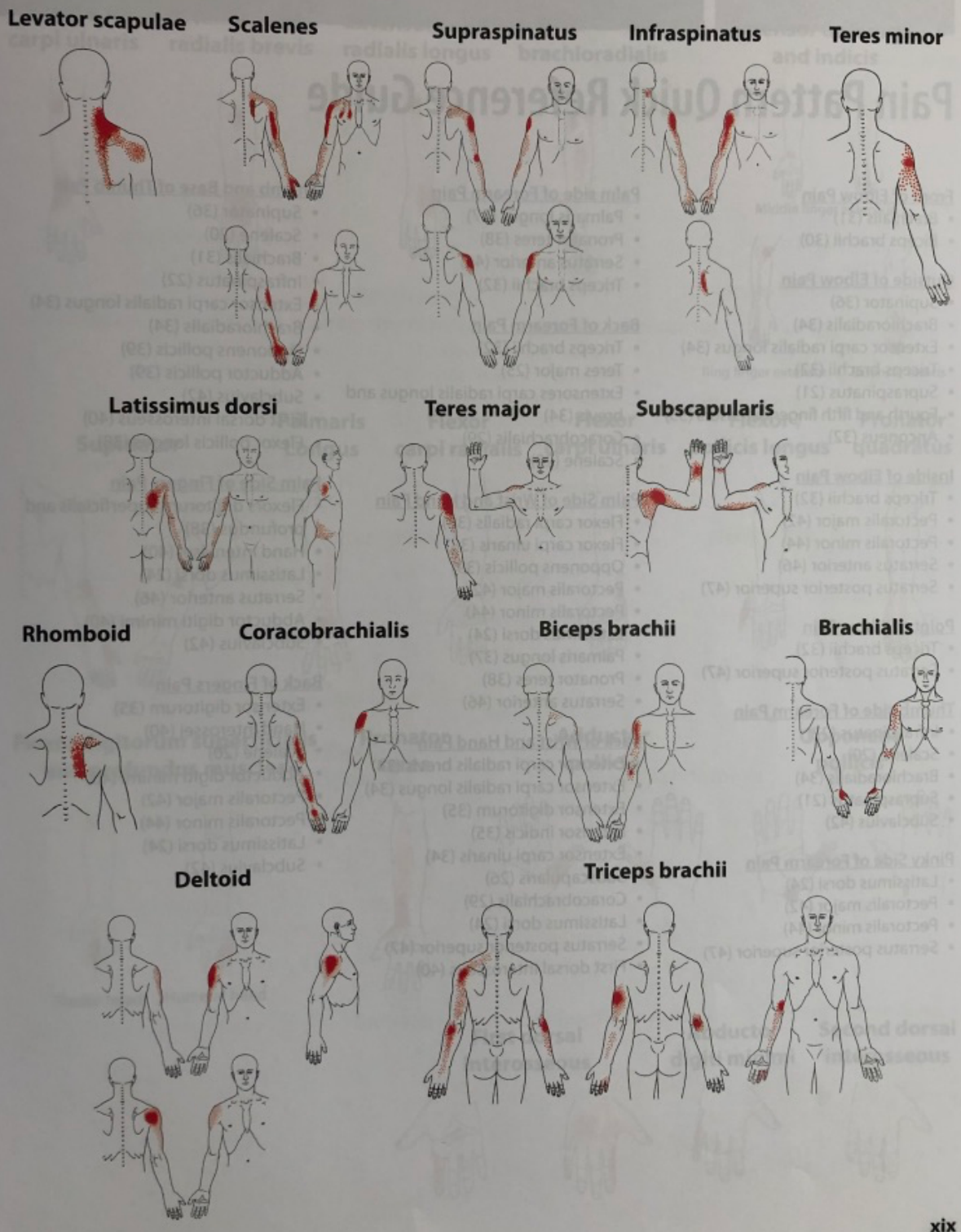
Posterior cervical muscles



Suboccipital muscles

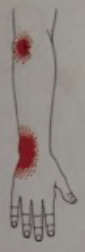


Section 3 Upper Back, Shoulder, and Arm Pain



Section 4 Forearm, Wrist, and Hand Pain

Extensor carpi ulnaris

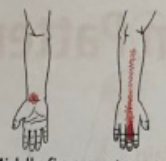


Extensor carpi radialis brevis

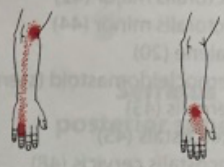
Extensor carpi radialis longus

brachioradialis

Extensor digitorum and indicis

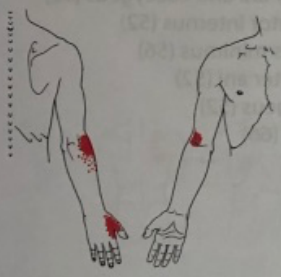


Middle finger extensor

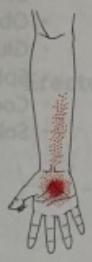


Ring finger extensor Extensor indicis

Supinator

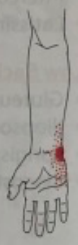


Palmaris Longus



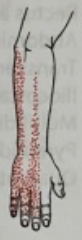
Flexor carpi radialis

Flexor carpi ulnaris

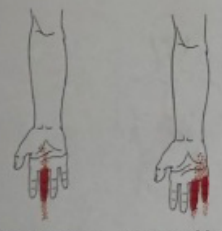


Flexor pollicis longus

Pronator quadratus



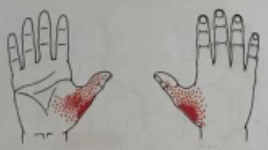
Flexor digitorum superficialis and profundus muscles



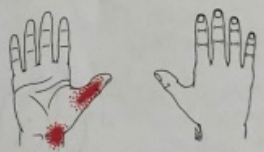
Radial head Humeral head

Pronator teres

Adductor pollicis

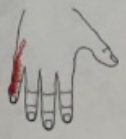


Opponens pollicis



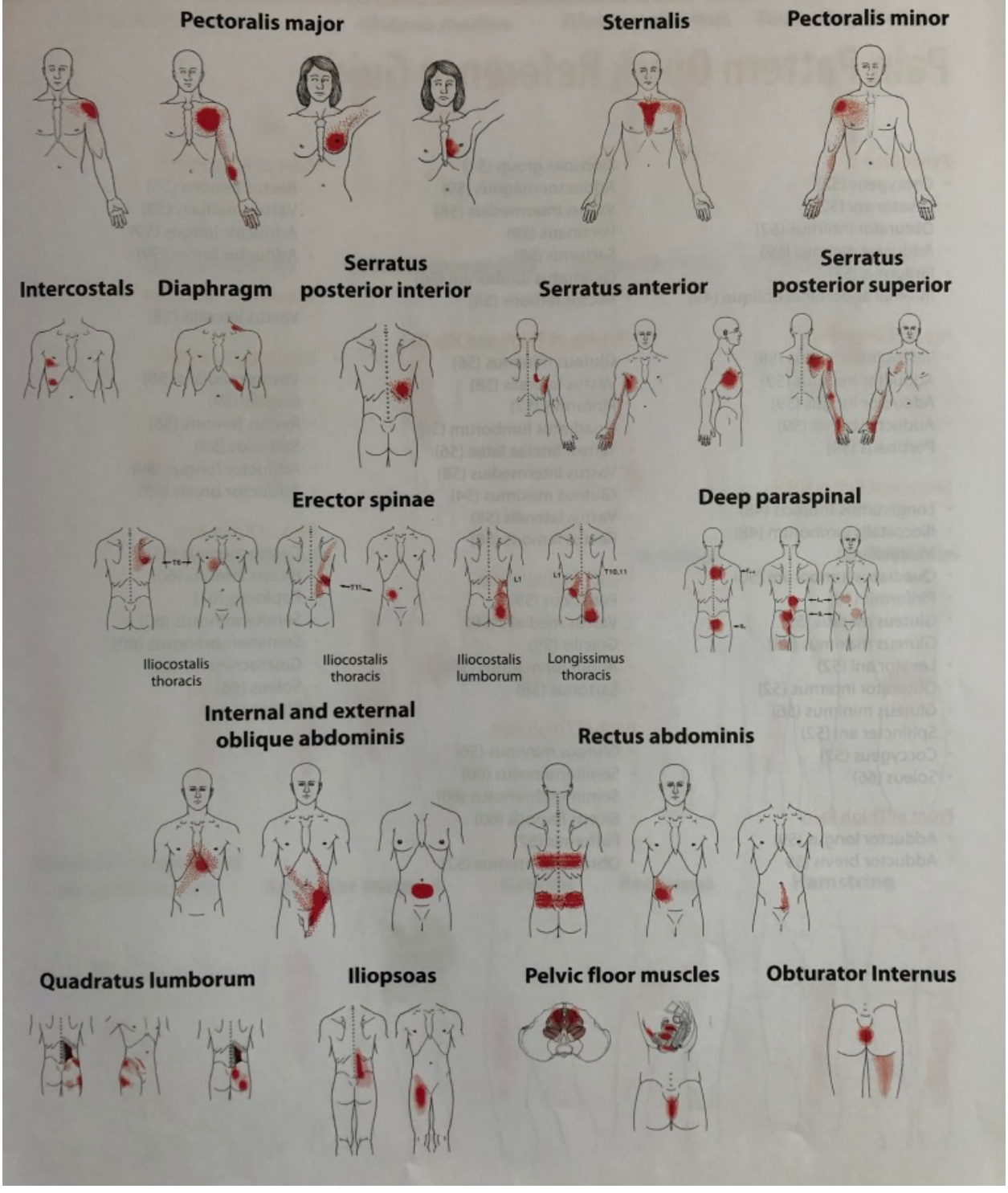
First dorsal interosseous

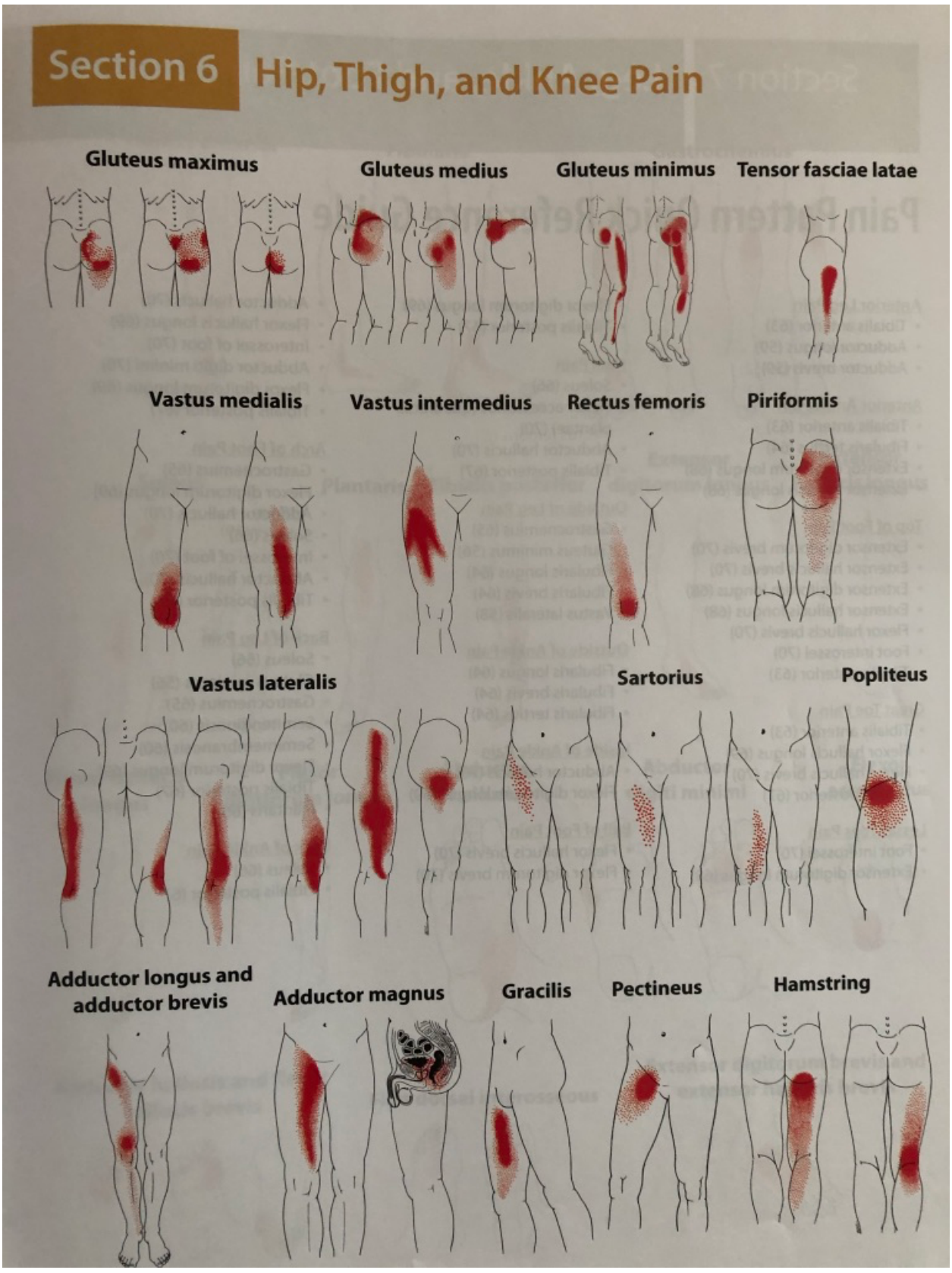
Abductor digiti minimi



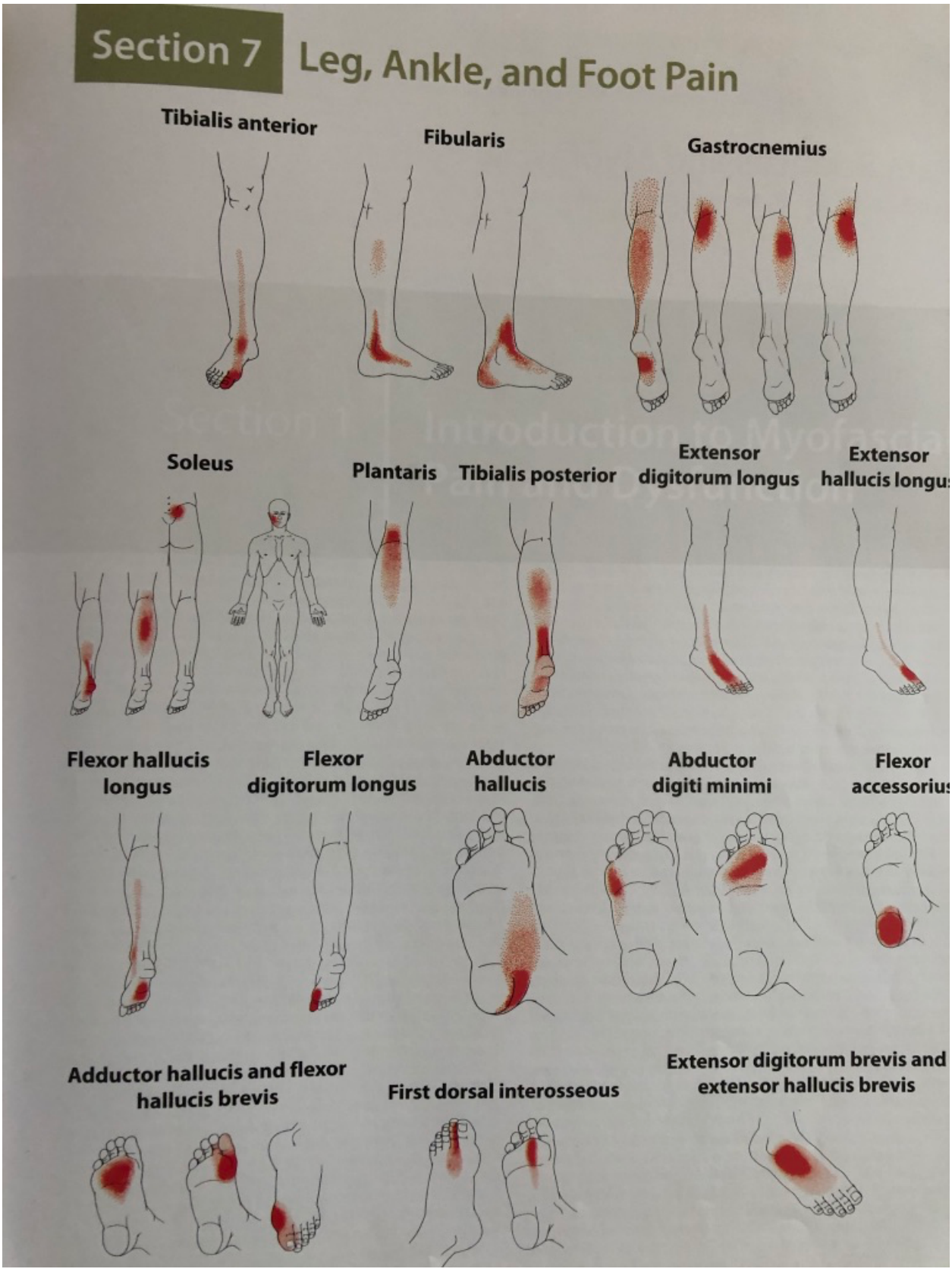
Second dorsal interosseous

Section 5 Trunk and Pelvis Pain




Gluteus maximus
Gluteus medius
Gluteus minimus
Tensor fasciae latae
Vastus medialis
Vastus intermedius
Rectus femoris
Piriformis
Vastus lateralis
Sartorius
Popliteus
Adductor longus and adductor brevis
Adductor magnus
Gracilis
Pectineus
Hamstring

Section 7 Leg, Ankle, and Foot Pain



References

1. **Sharkey, J. 2017. Concise Book of Dry Needling. A Practitioners Guide to Myofascial Trigger Point Applications. North Atlantic Books**

2. **Travel, Simons and Simons. 2018 Myofascial Pain and Dysfunction-The Trigger Point Manual 3rd ~Edition. Joseph M. Donnelly, Editor. Wolters Kluwer.**

3. **Bishop, M.D., Beneciuk, J.M., and George, S.Z. 2011. Immediate reduction in temporal sensory summation after thoracic spinal manipulation. *Spine J* 11(5), 440–446 (doi. 10.1016/j.spinee.2011.03.001).**

4. **Brisby, H. 2006. Pathology and possible mechanics of nervous system response to disc degeneration. *J Bone Joint Surg Am* 88(Suppl 2), 68–71.**

5. **Camanho, L.G., Imamura, M., and Arendt-Nielsen, L. 2011. Genesis of pain in arthrosis. *Revista Brasileira de Ortopedia* 46(1), 14–17.**

6. **Cotchett, M.P., Munteanu, S.E., and Landorf, K.B. 2014. Effectiveness of trigger point dry needling for plantar heel pain: A randomized controlled trial. *Phys Ther* 94(8), 1083–1094.**

7. **Cummings, M. 2003. Referred knee pain treated with electroacupunture to ilipsoas. *Acupunct Med* 21, 32–35.**

8. **Dommerholt, J. and Fernández-de-las-Peñas, C. 2013. *Trigger Point Dry Needling: An Evidence and Clinical-Based Approach*, London: Churchill Livingstone.**

9. **Dubousset, J. 2003. Spinal instrumentation: Source of progress but also revealing pitfalls. *Bull Acad Natl Med* 187(3), 523–533.**

10. **Edwards, J. 2005. The importance of postural habits in perpetuating myofascial trigger point pain. *Acupunct Med* 23(2), 77–82.**

11. **Fogelman, Y. and Kent, J. 2015. Efficacy of dry needling for treatment of myofascial pain syndrome. *J Back Musculoskel Rehab* 28(1), 173–179.**

12. **Fuller, R.B. 1961. Tensegrity. *Portfolio Art News Ann* 4, 112–127, 144, 148.**

13. **Gerwin, R.D., Dommerholt, J., and Shah, J. 2004. An expansion of Simons' integrated hypothesis of trigger point formation. *Curr Pain Headache Rep* 8(6), 468–475.**

14. **Gerwin, R., Shannon, S., Hong, C.Z., Hubbard, D., and Gervitz, R. 1997. Interrater reliability in myofascial trigger point examination. *Int Assoc Study Pain* 69(1–2), 65–73.**

15. **Giamberardino, M.A., Affaitati, G., Fabrizio, A., and Constantini, R. 2011a. Myofascial pain syndromes and their evaluation. *Best Pract Res Clin Rheumatol* 25(2), 185–198.**

16. **Giamberardino, M.A., Affaitati, G., Fabrizio, A., and Constantini, R. 2011b. Effects of treatment of myofascial trigger points on the pain of fibromyalgia. *Curr Pain Headache Rep* 15(5), 393–399.**

17. **Guimberteau, J.-C., Delage, J.-P., and Wong, J. 2010. The role and mechanical behavior of the connective tissue in tendon sliding. *Chirurgie de la Main* 29(3), 155–166.**

18. **Hsieh, Y.L., Chou, L.W., Joe, Y.S., and Hong, C.Z. 2011. Spinal cord mechanism involving the remote effects of dry needling on the irritability of myofascial trigger spots in rabbit skeletal muscle. *Arch Phys Med Rehab* 92, 1098–1105.**

19. **Hsueh, T.C., Yu, S., Kuan, T.S., and Hong, C.Z. 1998. Association of active myofascial trigger points and cervical disc lesions. *J Formos Med Assoc* 97(3), 174–180.**

20. **Huijing, P.A. 2009. Epimuscular myofascial force transmission: A historical review and implications for new research (International Society of Biomechanics Muybridge Award Lecture, Taipei, 2007). *J Biomech* 42(1), 9–21.**

21. **Kietrys, D.M., Palombaro, K.M., and Mannheimer, J.S. 2014. Dry needling for management of pain in the upper quarter and craniofacial region. *Curr Pain Headache Rep* 18(8), 437.**

22. **Koolstra, J.H. and van Eïjden, T.M. 2005. Combined finite-element and rigid-body analysis of human jaw joint dynamics. *J Biomech* 38(12), 2431–2439.**

23. **Kumaresan, S., Yoganandan, N., and Pintar, F.A. 1999. Finite element analysis of the cervical spine: A material property sensitivity study. *Clin biomech* (Bristol, Avon) 14(1), 41–53.**

24. Langevin, H. 2006. Connective tissue: A body-wide signaling network? *Med Hypotheses* 66(6), 1074–1077.

25. Levin, S. 1995. The importance of soft tissues for structural support of the body. *Spine: State of the Art Reviews* 9(2).

26. Lewit, K. 1979. The needle effect in the relief of myofascial pain. *Pain* 6(1), 83–90.

27. Lewit, K. and Kolar, P. 2000. Chain reactions related to the cervical spine. In: Murphy, D.R. (ed.), *Conservative Management of Cervical Spine syndromes*, New York: McGraw-Hill, 515–530.

28. Liu, Z.J., Yamagata, K., Kuroe, K., Suenaga, S., Noikura, T., and Ito, G. 2000. Morphological and positional assessment of TMJ components and lateral pterygoid muscle in relation to symptoms and occlusion of patients with temporomandibular disorders. *J Oral Rehabil* 27(10), 860–874.

29. Loeser, R.F. and Shakoor, N. 2003. Aging or osteoarthritis: Which is the problem? *Rheum Dis Clin North Am* 29(4), 653–673.

30. Lucas, K., Polus, B., and Rich, P. 2004. Latent myofascial trigger points: Their effects on muscle activation and movement efficiency. *J Bodywork Move Therap* 8, 160–168.

31. Lucas, N., Macaskill, P., Irwig, L., Moran, R., and Bogduk, N. 2009. Reliability of physical examination for diagnosis of myofascial trigger point: A systematic review of literature. *Clin J Pain* 25(1), 80–89.

32. Manheim, C.J. 1994. *The Myofascial Release Manual 2nd Edition*, Thorofare, NJ: Slack Inc., 124.

33. Martin, D.-C. and Levin, S. 2012. Myofascia as the tensioner in the biotensegrity model. In: Schleip, R., Findley, T.W., Chaitow, L. and Huijing, P.A. (2012).

34. Mayoral, O., Salvat, I., Martin, M.T., Martin, S., Santiago, J., Cotarelo, J., and Rodriguez, C. 2013. Efficacy of myofascial trigger point dry needling in the prevention of pain after total knee arthroplasty: A randomized, double-blinded, placebo-controlled trial. *Evid Based Complement Altern Med* 2013, 694941.

35. McMakin, C.R. 2011. *Frequency Specific Microcurrent in Pain*, Edinburgh: Churchill Livingstone Elsevier.

36. Mense, S. 2010. How do muscle lesions such as latent and active trigger points influence central nociceptive neurons? *J Musculoskelet Pain* 18, 348–353.

37. Modell, W., Travell, J.T., Kraus, H., et al. 1952. Relief of pain by ethyl chloride spray. *NY State J Med* 52, 1550–1558.

38. Moseley, G.L. 2012. Teaching people about pain: Why do we keep beating around the bush? *Pain Management* 2(1), 1–3.

39. Niddam, D.M., Chan, R.C., Lee, S.H., Yeh, T.C., and Hsieh, J.C. 2007. Central modulation of pain evoked from myofascial trigger points. *Clin J Pain* 23(5), 440–448.

40. Passerieux, E., Rossignol, R., Letellier, T., and Delage, J.P. 2007. Physical continuity of the perimysium from myofibers to tendons: Involvement in lateral force transmission in skeletal muscle. *J Struct Biol* 159(1), 19–28.

41. Reynolds, K.K., Ramey-Hartung, B., and Jortani, S.A. 2008. The value of CYP2D6 and OPRM1 pharmacological testing for opioid therapy. *Clin Lab Med* 28(4), 581–598.

42. Russell, I.J., Holman, A.J., Swick, T.J., et al. 2011. Sodium oxybate reduces pain, fatigue and sleep disturbance and improves functionality in fibromyalgia: Results from a 14-week, randomized, double-blind, placebo-controlled study. *Pain* 152(5), 1007–1017.

43. Samuel, A.N., Peter, A.A., and Ramanathan, K. 2007. The association of active myofascial trigger points with lumbar disc lesions. *J Musculoskel Pain* 15(2), 11–18.

44. Schleip, R. and Müller, D.G. 2013. Training principles for fascial connective tissues: Scientific foundation and suggested practical applications. *J Bodywork Move Therap* 17(1), 103–115.

45. Schleip, R., Findley, T.W., Chaitow, L. and Huijing, P. A. (eds) 2012. *Fascia: The Tensional Network of the Human Body. The Science and Clinical Applications in Manual and Movement Therapies*, Edinburgh: Churchill Livingstone.

46. Shah, J.P. and Gilliams, E.A. 2008. Uncovering the biochemical milieu of myofascial trigger points using in-vivo microdialysis: An application of muscle pain concepts to myofascial pain syndrome, *J Bodywork Move Ther* 12(4), 371–384.

47. Shah, J.P. and Heimur, J. 2012. New frontiers in the pathophysiology of myofascial pain. *The Pain Practitioner* 2012, 26–32.

48. Sharkey, J. 2008. *The Concise Book of Neuromuscular Therapy*, Chichester, UK/Berkeley, CA: Lotus Publishing/North Atlantic Books.

49. Sharkey, J. 2015. Biotensegrity-anatomy for the 21st Century. *Terra Rosa J* 16 <terrarosa.com.au>.

50. Simons, D.G. 2004. Review of enigmatic myofascial trigger points as a common cause of enigmatic musculoskeletal pain and dysfunction. *J Electromyogr Kinesiol* 14(1), 95–107.

51. Simons, D.G., Travell, J.G., and Simons, L.S. 1999. *Travell and Simons' Myofascial Pain and Dysfunction: The Myofascial Trigger Point Manual*, Vol. 1, 2nd edn, Baltimore, MD: Lippincott Williams & Wilkins.

52. Skorupska, E., Rychlik, M., Pawelec, W., Bednarek, A., and Samborski, W. 2014. Trigger point-related sympathetic nerve activity in chronic sciatic leg pain: A case study. *Acupunct Med* 32(5), 418–422.

53. Snapp, R.R., Goveia, E., Peet, L., Bouffard, N.A., Badger, G.J., and Langevin, H.M. 2013. Spatial organization of fibroblast nuclear chromocenters: Component tree analysis. *J Anat* 223(3), 255–261.

54. Soloman, L., Schnitzler, C.M., and Browett, J.P. 1982. Osteoarthritis of the hip: The patient behind the disease. *Ann Rheum Dis* 41(2), 118–125.

55. Srbely, J.Z., Dickey, J.P., Lee, D., and Lowerison, M. 2010. Dry needle stimulation of myofascial trigger points evokes segmental anti-nociceptive effects. *J Rehabil Med* 42(5), 63–68.

56. Staud, R. 2011. Peripheral pain mechanisms in chronic widespread pain. *Best Pract Res Clin Rheumatol* 25(2), 155–164.

57. Travell, J.G. and Simons, D.G. 1992. *Myofascial Pain and Dysfunction: The Myofascial Trigger Point Manual*, Vol. 2, Baltimore, MD: Lippincott Williams & Wilkins.

58. Willard, F. 2008. Basic mechanisms of pain. Future trends in CAM research in Integrative Pain Medicine: The Science and Practice of Complementary and Alternative Medicine. In: Audette, J.F. and Bailey, A. (eds), *Pain Management*, Totowa, NJ: Humana Press.

59. Woolf, C.W. 2010. Central sensitisation: Implications for the diagnosis and treatment of pain. *J Int Assoc Study Pain* 152(3), S2–S15.

60. Yates, B.J., Billig, I., and Cotter, L.A. 2002. Role of the vestibular system in regulating respiratory muscle activity during movement. *Clin Exp Pharmacol Physiol* 29(1–2), 112–117.