Tracking Down the Source of Trigger Points For Effective Pain Management

By Frank Jarrell, D.C. and Cathy Lavallee, DPT, M.Ed., LMT

Why Do We Hurt So Much?

Americans consume over 80% of the prescription opioid pain killers in the world with only 4.6% of the world's population[1] and the cost of treating chronic pain was 635 billion dollars[2] in 2010 and increasing at a rate of 9% annually.[3] Chronic pain shared the top reasons for primary care visits to doctors with musculoskeletal pain affecting 1 in 3[4] persons in 2014. Not including pain medication addiction, the cost of treating chronic pain was approximately 1 trillion dollars in 2016. In light of all this pain, we may ask ourselves: 'Is America just in the throes of an opioid epidemic or do we experience an underlying level of pain profoundly greater than any other population in the world?

The health and social implications of America's pain epidemic are immense. A rapidly increasing demand for skills that effectively manage pain and demonstrate definitive "functional objective improvement" (FOI) must be employed in today's health care market to not only provide the best care possible, but to also achieve economic success as a provider. Effective pain management, rapid treatment strategies and measurable functional objective improvement demands place a burden on virtually all professions in the American health care system. With the right tools, Therapeutic Massage is a natural for delivering what I call "immediate functional objective improvement" to millions of people in need.

To effectively manage pain and accomplish consistent functional objective improvement, neuro-musculoskeletal (NMS) professionals must move beyond the premise that local pain is the center point of the complaint. In essence, an FOI is a measurable outcome apart from the client's subjective pain feedback only. Thus, a discernable change for the better in a repeated related test, not just a change in the point of pain only, is the hallmark of improved function. Local pain in and of itself becomes increasingly limited as a reliable clinical marker as we further understand the interactive sources of cascading and chronic pain.

A classic example is the trigger point phenomena. Trigger point development is a prime example of a local reaction being driven by upstream physiological processes. Trigger points are more of a secondary reaction as opposed to being a primary problem. This implies that treating the secondary reaction, i.e. trigger point alone rarely affects the cause and will thereby marginally change the long term FOI. Therefore, reoccurrence and further degenerative changes are inevitable and the same complaint will persist over the long run and this is why trigger points typically return and/or persist in the same client. It is now possible to resolve trigger points without actually touching or treating them. However, as a health care provider, you will have to suffer the temporary pain of learning new and exciting ideas that will expand your horizons. We can also manage this type of pain with science and good logic.

More than Just Joints

The human body contains more than 200 joints or areas where 2 or more bones come together. Joints provide skeletal movement and stability. Of the 3 different types of joints (fibrous, cartilaginous and synovial), synovial joints allow the greatest degree of movement while both maintaining dynamic stability throughout their range of motion and limit excessive motion that can compromise the joints integrity.

Not all joints simply manage motion. The following example illustrates the intrinsic difference between spinal and non-spinal joints. In roughly the same length as the lower extremity having 5 joints; the spine is comprised of 75 joints that protect and channel 99% of the body's nerves that control information and bodily functions.

Eighty-five percent of these nerve pathways carry afferent (sensory) information to the brain for feedback while only 15% carry efferent motor, viscera and glandular information to the body for action. Your brain on average needs a 5:1 feedback ratio to make decisions and direct function. Of particular interest within this system are the spinal facet joints. They are synovial plane joints comprised of bone, cartilage and a capsular ligament that forms a cavity containing

synovial fluid. There are 28 pairs of spinal facets for a total of 56 left and right facet joints. They are technically called zygapophyseal articulations. Each one packs a pretty powerful reflex that is at the center of our trigger point mystery, so hang in there as we dig deeper and deeper into the science.

Every Mystery Has a Suspect

Like a good mystery, we are unsure of whom, or in this case, which one is the guilty party in a group of 56 suspects from a gang of spinal facet joints. To find the culprit, we must first view the segments of the spine as a highly organized kinetic chain that functions as a flexible floating lever from which all limbs and appendages can move freely from. This system is dynamically stable if it has sufficient muscle mass to control it and if it is free of damage to any given link in the chain. Understanding those two facts will allow you to move from the myopic view of the spine as a purely anatomical array of bones and nerves and instead, view it as a functional conduit for the nervous system and all movement that ultimately is dependent entirely upon our favorite organ to accomplish its objectives: MUSCLES.

Traditionally, neuromusculoskeletal pain and dysfunction is presumed to originate from stress or trauma to local tissues, i.e. if it hurts where we strained muscle, sprained the tendons, tore the ligaments or our poor postural, ergonomics, lifestyle or activities ended in tissue failure, it must also be the central cause of pain. This is certainly true of acute trauma, but rarely true for the pain of recovery, non-traumatic pain and referred pain.

With the exception of acutely traumatized tissue, most physical pain in the NMS field is a product of some form of weakness or instability in the kinetic chain that is creating neurological reactions, soft tissue strain and joint tracking error. For example, if the knee is weak, overstretched or torn; would it be stable throughout its range of motion? No. If it is unstable, is it subject to injury, failure, pain or degenerative changes over time? Yes. Does the knee have nerves that run through it and control other areas of your body? No. If the spine is weak, overstretched or torn; would it be unstable and affect the nerves entering and exiting it at different levels? Answer: Absolutely yes. So much so that one unstable facet joint can activate spinal reflexes that will over-facilitate (contract) 50-125 muscles until they are metabolically fatigued, weak upon testing and grossly disruptive of hundreds of physiological processes.

In this model the million dollar questions are: Which facet joint is suspect and proves to be guilty of creating the cascade of pain and dysfunction in this client? Do you know how to find it and what will you do about it if you do find it? If you stop the chaos, will you see an objective functional improvement in the client's status and how will you measure it?

Under normal defense neurology, a facet driven reflex swiftly moves the body out of harm's way and then returns it to a safe and non-defensive state. We call this a "spondylogenic reflex" (spondylo = vertebrae and genic = origin)(SR). The origin of this type of reflex is in the spine, not the extremities. We need these reflexes to protect us, however when a SR remains stuck on due to overstretch, tears or damage; it now becomes a "spondylogenic reflex syndrome" (SRS).

The objective as a trained Massage Therapists is to identify the suspect in less than 30 seconds, therapeutically arrest it and then prove to the client that it is guilty of hijacking their nervous system involuntarily, firing multiple muscles to the point of fatigue, disrupting joint tracking efficiency and producing trigger points and an array of pain throughout their body.

A Clue

Yes, the SRS will generate myofascial trigger points. Why? Facet joints are rooted in the embryological origin of the boney spine and its' respective cartilage and ligaments[5] and understanding dysfunction in these structures is a critical factor in understanding trigger point development and the role they both play in the amount of NMS pain a client will experience over a given lifetime.

Essentially, trigger points are an adaptive response at the end of a cascade of physiological events and the primary objective is to globally resolve multiple trigger points by treating the SRS, rather than treating one trigger point at a time. This approach results in much less physical work, a substantially faster response and will lead to a long term FOI in most of your clients.

A Modern Problem

Instability in the facet joint begins with traumatic and non-traumatic facet capsular ligament overstretch due to injury, lack of paraspinal muscle development, poor posture, detrimental ergonomics and lifestyle choices. Underlying all of this; poor back strength exacerbates a complex, modern evolutionary crisis in which paraspinal muscle mass is too low to support the increasing length of the modern spine.

As our spine increases in length with each new generation and our supporting muscle mass decreases with poor lifestyle choices from childhood on, we are now the weakest generation in the history of humanity. Given the current research and modern lifestyle trends, each successive generation will become notably weaker than the one we are in right now.

Moving from Analogies to More Facts

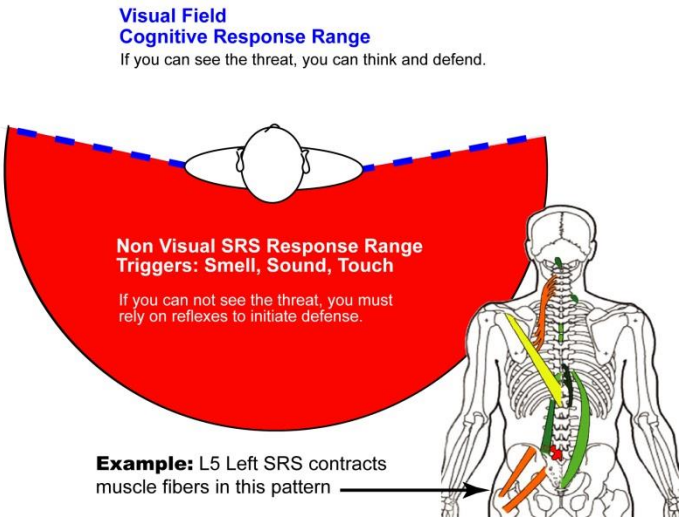
The facet capsular ligament is in fact the smoking gun that fires the muscles that drive the reactions that create trigger points and an array other forms of pain. This gives the Massage Therapist a much larger playing field over and above conventional techniques focusing only on where it hurts. Knowing that you can treat any one of a set of specific muscles on a given SRS pathway and grossly affect a client's pain, spasms and range of motion in seconds is thrilling. How does this work?

The facet capsular ligament is embedded with "slow stretch mechanoreceptors" (SSM) that send increasingly stronger signals to the spinal cord for an indefinite period of time. When they are over-stretched or torn through trauma, weak muscle support, poor posture or low blood sugar levels the facet joint becomes unstable and sets a predefined neurological cascade of reactions into motion.

If you treat the muscles specific to the SRS, you shut down all reciprocally facilitated muscles and activate reciprocally inhibited muscles through the nerves of the spine. We call this homeostasis. The client calls it pain free, lighter, increased range of motion, stronger, more vitality and a positive emotional affect.

Deeper into the Mystery

Now that we know our suspects and we have a clue or two as to how the facet joint pulled it off, let's dig through the challenging stuff and discover the trigger point's true relationship in all of this.



Shrinking the SRS definition further, it is a normal defense withdrawal reflex that originates from the facet joints of the spine in response to an unseen or unheard contact stimulus or sound occurring outside of your visual field (behind you). It will activate and return to normal on its own if the threat subsides without injury to the spine or back of the body. An example is to imagine having ice-cold water thrown on your back. You did not smell, hear or see it coming as the skin on your back felt the sudden cold.

One or more spondylogenic reflexes will fire neuromuscular pathways that activate and contract a large number of muscles throughout your body in a specific pattern that causes you to jump up and twist to push the irritant (threat) away as you rotate your eyes and head in the same direction to identify who threw the ice cold water at you.

Reflexes are hardwired in all of us and if I have 700 or 7 billion people experiencing the same stimulus, they will all contract the exact same reflexive muscle pattern without variation. From this neurological fact, I can now rely on the SRS to produce limited patterns of muscle contractions that are predictable, dependable and reproducible - every time.

Now, imagine what will happen if the SRS and its reactive cascade is "stuck-on". It gets real messy, real fast. You only need to learn how to find them and how to treat them to stop the process.

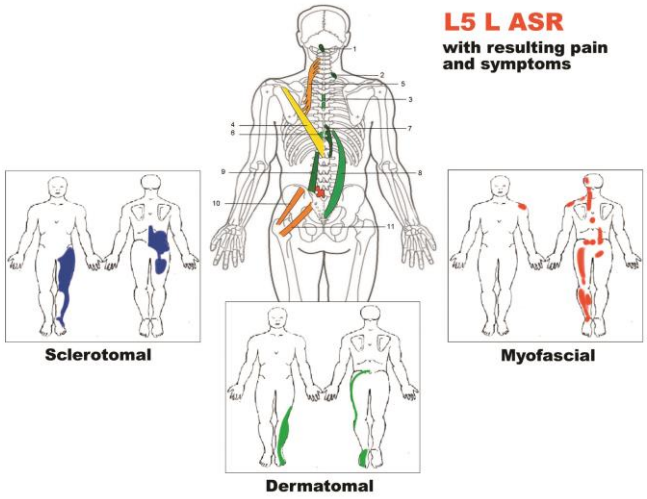
Deeper into the Technical

Hang in there! Overstretch or trauma to the facet capsular ligament's slow stretch mechanoreceptors result in ligament laxity[6], sclerotome pain[6-8] and spondylogenic reflex syndromes[9] (SRS). Innervated through a single spinal nerve or its branches[5], the SRS was discovered by Kelligren[6] and researched by numerous authors over the past 75 years.[11-13, 15-17]

The term "spondylogenic reflex syndrome (SRS)" was originally coined by Sutter[9] and ongoing research into the underlying pathophysiology of facet joint muscle activation was published as recently as 2014.[8,14]

Acute or chronic facet ligament laxity[6], mechanoreceptor signaling, reflexive effector target tissue facilitation[11-13] and progressive pain signaling via neuronal excitability, glutamate signaling[8], nerve growth factor and peptidergic joint afferents[14] form a complicated relationship between function, dysfunction and pain.

As a comprehensive pathological entity, we can further define SRS activation as a causative factor in trigger point development. With the exception of one addition critical aggravating factor; our traditional understanding of trigger point physiology remains unchanged. Everything you know about trigger points are true, however it is just part of a larger story.



Trigger Points are Reactions

The hallmark of an active SRS is persistent multi-muscle "over facilitation or hypertonicity". Over-facilitation eventually leads to muscle metabolic fatigue and the migration of accumulated metabolic waste to the muscle/fascia interface due to insufficient clearing. Clearing rates are controlled by numerous complicating factors including, but not limited to low core temperature, nutritional deficiencies, prolonged muscle loading, traumatic muscle overload, joint tracking error, aberrant biomechanics, physical deconditioning, poor lymphatics and poor posture.

Nociceptor fibers in the muscle/fascia interface complete the trigger point profile by responding chemically to the noxious waist buildup and in turn refer pain to other regions of the body. The least realized "aggravating factor" controlling the number, severity and frequency of trigger point activity is the role of "lower than normal core body temperature" on venous and lymphatic drainage of muscle metabolic waist.

Low Core Temperature

A client's core temperature reflects the internal metabolic rate of the body (CT°) and is the result of an average set of metabolic processes. For humans, average CT° is 98.6 F° orally and 99.4 F° auricular.
CT° allows for efficient circulation, appropriate enzyme activity, protein synthesis, nerve and muscle fibril activity, cellular detoxification and lymphatic drainage. All are critical factors in muscle power, endurance and recovery.

If the CT° approaches 104 F°, muscle metabolism will cease functioning and lose its contractile ability. If the core temperature is too low (>.8 F° below normal), muscle will proportionally fail to clear metabolic waste and will lose its ability to relax or lengthen efficiently. Similarly, we are well aware that muscle will contract or spasm in response to cold air (chill reflex) and will struggle to return to its resting state after contracting. A good example of this involves muscle shortening and spasms when subjected to prolonged periods of cold air, i.e. as in the case of a vehicle air conditioner blowing on your neck. A clinically relevant note: If your client exhibits a lower than normal CT°, they will develop delayed onset muscle soreness (DOMS) 12-24 hours after a deep tissue massage. Solution: Lighten the pressure on your low CT° clients.

Core Temperature and Trigger Point Pain Simplified

Trigger points are products of prolonged muscle facilitation or muscle overload that generates metabolic waste, hypoxia and restricted nutrient supply faster than lymphatics and venous circulation can clear. Low CT° further compounds this process by impairing muscle relaxation phase and prolonging muscle contractions through increased tissue acidosis.

When the muscle cannot completely relax or recover between contractions, the belly of the muscle again cannot eliminate metabolic byproducts through normal clearing channels. Muscle metabolic waste will then migrate and concentrate at the muscle/fascia interface. The fascia, not the muscle belly contains nociceptors (pain receptors) and accumulated waste will generate myofascial trigger point pain referral. The waste site becomes the palpable location of the trigger point and the source of myofascial pain and dysfunction.

SRS Drives Trigger Point – The Technical

Facet capsular ligament overstretch will persistently fire slow stretch mechanoreceptors and in turn activate the SRS. The SRS will always activate or facilitate predefined effector target tissues (muscle, viscera and gland) to include partial or whole muscles in the head, neck, torso, pelvis and all extremities.[12-14]

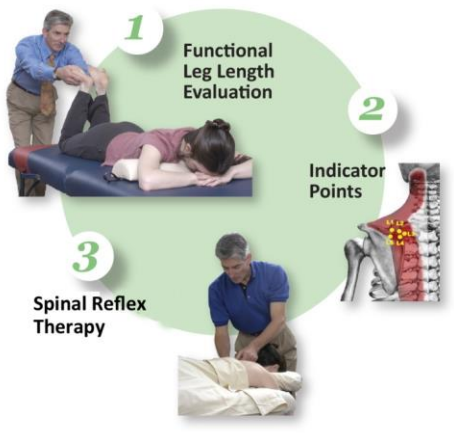
Once activated, the SRS becomes a cascade of reflexive facilitation of extraneous muscles that create a specific, yet broad subset of dysfunction as described above. As defined in the following flow chart, the potential for pain can occur at any or all steps of the cascade and selective intervention can assist in partial or whole pain management. Systematic spondylogenic reflex syndrome management through available techniques such as Spinal Reflex Therapy will comprehensively target all of the following reactions listed below, whereas many professions focus on a limited number of reactions only.

Follow the SRS Cascade

FACET CAPSULAR LIGAMENT OVERSTRETCH ►SRS activation ►Reflexive muscle shortening along the spine and ►Multi-level hydrostatic nerve root compression ►Myotome muscle over-facilitation ►Attachment tendonitis in over facilitated muscles ►Reciprocal muscle inhibition and weakness ►Metabolic fatigue, toxicity and weakness in over-facilitated muscles ►Myofascial trigger point activation in over-facilitated muscles ►Muscle compartment tone and strength imbalances ►Joint tracking error ►Joint strain, increased risk of injury and degenerative changes ►Local pain from the histological stress associated with any number of the above stated reactions ►Increased whole body inflammation and fatigue ►Increasing myofascial trigger point complications in the below normal CT° client

Much like tendonitis, myofascial trigger points require a reason for prolonged states of muscle over facilitation to activate. Whereas tendonitis is predominantly a process of prolonged muscle shortening coupled with repetitive overload leading to tendon inflammation, trigger points involve muscle shortening with lower than average core temperature with or without repetitive overload. When treating either condition, the joint-nerve-muscle sequencing of events caused by the SRS quickly illustrates how a simple, yet little known reflex originating from the facet joint capsular ligaments can become a powerful source of both tendonitis and trigger point pain and dysfunction.

Caption: SRT is a 3 step process



Deriving an SRS Assessment

There is much to learn about this topic and a first article can only be an introduction. Read and explore more information on this topic to better understand what to do to treat spondylogenic reflex syndromes.

Deriving an SRS assessment involves a quick and effective small set of tools necessary for identification combined with a requisite knowledge on how to effectively treat this problem through technique development and clinical practice.

I have already found that learning to identify and treat this weak link in the pain management arena can drive the massage profession into a major gatekeeper role for the majority of NMS problems. As a time

tested and evidenced based strategy, this novel approach to identifying and treating those muscles involved in the management of facet dysfunction and secondary trigger point activation is relatively easy to learn and practice and is extremely rewarding due to its “immediate functional objective improvement” capabilities.

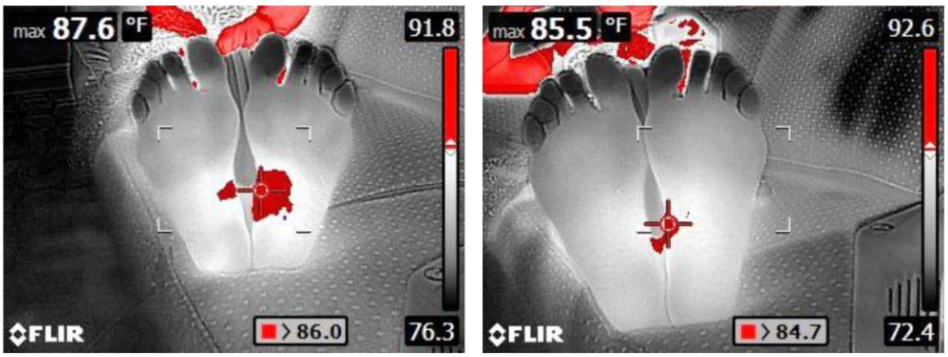
The following case studies illustrate functional objective outcomes from an SRS management perspective and how science can take Massage into a new era of predictable, dependable, reproducible and immediate objective improvements.

Confirming SRS Activity through Infrared Imaging

Infrared imaging can be used to visually qualify and quantify pre and post treatment assessment and FOI. For each case, note the area of complaint and the SRS facet level being treated. The impact of facet instability on remote and seemingly unrelated areas of pain and dysfunction is notable. If the client has a normal CT°, risk of trigger point development is nominal.

Note the visual scale on the right of each image: White illustrates the highest temperature areas within the image and blue illustrates the coolest temperatures. It is normal for torso skin temperature to proportionally display 91-95 F° in a room with ambient temperatures between 73 – 93 F°. Infrared temperatures above 96 F° are indicative of inflammation.

Case Study – Plantar Fasiitis



C5/C6 SRS and Plantar Fasciitis

Case Study: 67 year old female patient with plantar fasciitis for 9 months. SRT therapy applied to C5R SRS (muscles governing the facet articulation) only resulted in a 2.1 F° drop in target muscle temperature within 10 minutes post treatment. The patient stated a subjective reduction in discomfort of 70%. Frank Jarrell, D.C. 2016

Case Study - Facet Inflammation from Back Muscle Weakness

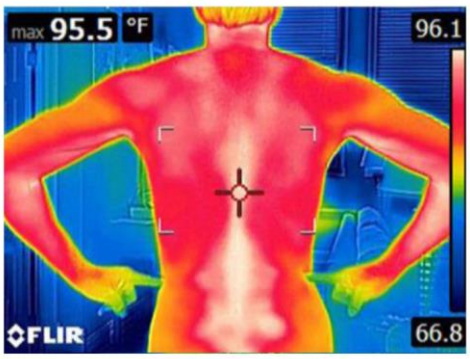


Fig. 9d IR Imaging before 5Minute Back Dynamic Spinal Stabilization session. Note: Elevated midline thoracic and lumbosacral inflammation (white) with a peak temperature of 95.5 F° at the T8-9 level.

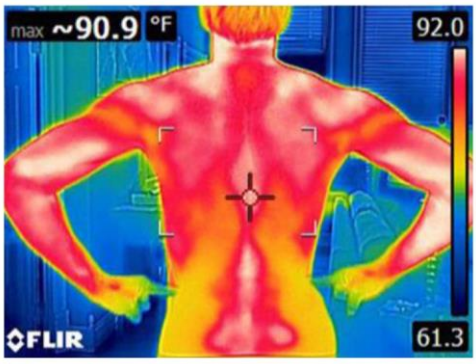
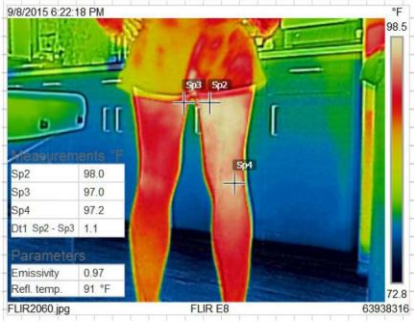


Fig. 9e 58 min. after 5MB. Note: Continued reduction in inflammation and continued elevated skeletal muscle resting metabolism. Peak is 90.9 F° w/reduction of 4.6 F° at T8-9.

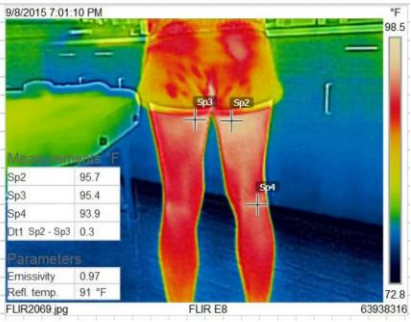
58 year old male with generalized thoracic, lumbar and sacral inflammation, spinal stenosis, intermittent right foot numbness and a history of L4R micro discectomy. Note bilateral S1 SRS impact on midline spinal pre and post treatment inflammation.

Case Study – Runner with C4 Right SRS Over-Facilitating Tight Hamstring

4 - Sp1 references a focal infrared inflammatory profile at the L4 level with a max temperature of 99.2 F°, well above the average 86-93 F° resting temperature of paraspinal muscle.



5 - Sp2 for the right thigh is 98.0 F° compared to left Sp3 at 97.0 F°. Right hamstring metabolism is operating at 1.0 'F higher than the left. Sp4 illustrates popliteus facilitation at 97.2 F°.



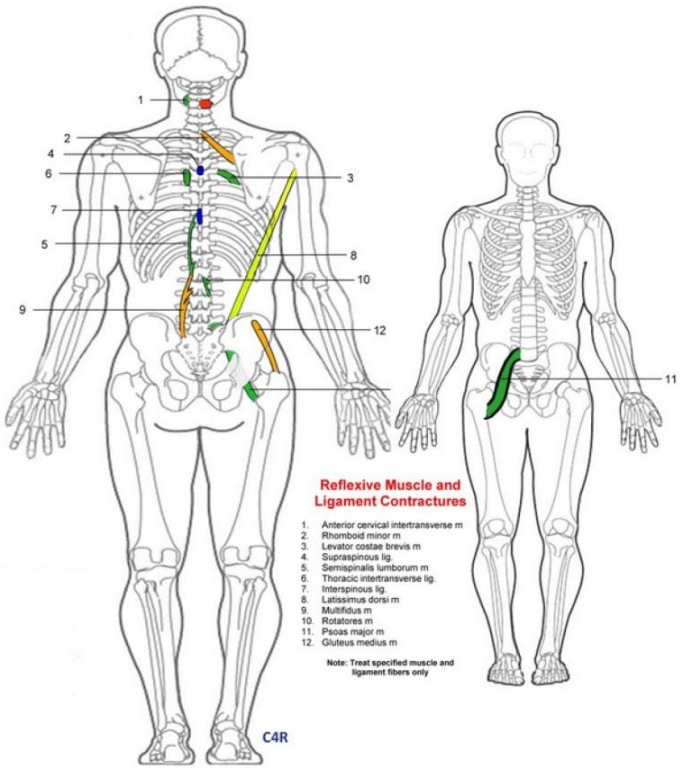
6 - SP2 reduction of 1.3 F° (95.7 F°), SP3 reduction of 1.6 F° (95.4 F°) and Sp4 reduction of 3.3 F° (93.9 F°) reflect a reduction in inflammation associated treating C4 SRS via a decrease in psoas and paraspinal muscle over facilitation at 40 minutes post treatment.

A seasoned 36-year-old female marathon and trail runner presents two weeks prior to the Imogene Pass Run unable to train. She developed progressive and debilitating right hamstring pain, spasms and trigger points over the previous year while training for this and other running events. Her complaint progressed into unrelenting right buttock pain, burning in the right posterior thigh and right hamstring muscle spasms. Her MRI/Medical diagnoses included tight hamstring muscle, moderate DJD and L4/5 Grade II spondylogenic spondylolisthesis (SS).

She was prescribed 3 months of physical therapy. The Initial steroid injection provided moderate relief while continued physical therapy spinal manipulation aggravated her condition. A second injection proved ineffective. Her initial evaluation revealed an SRS or primary unstable facet joint at the C4 spinal level on the right. Specific soft tissue therapy to paraspinal muscles only resulted in a significant reduction in reflexive psoas muscle contractures and associated trigger points aggravating her low back condition and lower extremity.

Summary

In essence, treating the SRS resulted in a decrease in lumbar facet periarticular nerve compression at the L4/L5 innervation of the biceps femoris, gracilis and popliteus muscles. It further reduced metabolic fatigue, spasms, pain and causalgia in the target tissue and increased the patient's muscle load capacity, endurance and power. This study illustrates how the SRS soft tissue facilitation cascade limits a client's ability to perform and how mitigation can return function and performance. After 1.5 weeks of treatment, the client not only completed the 17.1 mile run over the 13,114 ft. mountain pass with marginal discomfort; she ranked 1st in her age group and achieved her best time to date.



Treating the SRS (C4R) and reflexively facilitated core muscles using reflex maps. Note the C4R SRS (red), its psoas muscle facilitation relationship (muscle #11) and its potential for multiple levels of facet neurocompression.

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Biographies

Dr. Jarrell designs treatment techniques, educates on the science, interpretation and application of Spinal Reflex Therapy (SRT) and is Director of the Spinal Reflex Institute, Intl. educational portal and Spinal Reflex Research and Development nonprofit foundation.

Cathy Lavallee, PT, DPT, M.Ed., LMT has used Spinal Reflex Therapy in her Georgia practice for 13 years, is a Certified SRT Instructor, and is a coauthor on SRT Soft Tissue articles.

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