Tracking Down the Source of Trigger Points For Pain Management

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Why Do We Hurt So Much?

Not including the expense of treating pain medication addiction; the cost of treating chronic pain in the U.S. was approximately 1 trillion dollars in 2016. Cost aside, the health and social implications of America's pain epidemic are immense. With the following information and the right tools, therapeutic massage is a natural option for effectively managing most acute and chronic pain while simultaneously achieving a cost effective position in America's pain management model.

More Than Just Joints

With over 200 joints, the human skeleton provides stability for movement and structural support. It also provides a flexible, floating lever from which all limbs and appendages move freely. It is tough and efficient when strong, and an incredible source of chaos and pain when weak. This floating lever is also a highly organized kinetic chain with 27 bones and 75 joints that essentially functions as the crossroads for 95% of all nerve activity to and from the body. Buried deep within the kinetic chain are 56 facet joints that conceal a surprisingly complex secret to unlocking the relationship between joints, nerves, muscles, trigger points and our pain epidemic.

To get to the root of these discoveries, we must first accept that most pain originates from remote tissues. Trigger point referred pain is a prime example. Then, we must ask: Is it the local tissue that is causing the trigger point and its referred pain to develop or is it just another layer in a fascinating chain of events?

A Hidden Mechanism

When any one of the facet joints becomes unstable, a little known capsular ligament overstretch reflex is activated that systematically facilitates muscle fibers throughout the body to shorten to the point of metabolic fatigue. Muscle over-facilitation and subsequent metabolic fatigue is the primary mechanism behind muscle weakness, ache, pain, hypertonicity and spasms. This is also the cause of trigger points.

Traditionally, neuromusculoskeletal (NMS) pain and dysfunction is presumed to originate from stress or trauma to local tissues. We presume that if it hurts where we strained muscle, sprained tendons, tore ligaments or just ache; then these tissues must be the source of "the pain". This is generally true in acute injuries, but is rarely true in chronic pain presentations. As an acute injury repairs, sharp pain turns to a dull, chronic ache that may or may not appear related to the original area of injury.

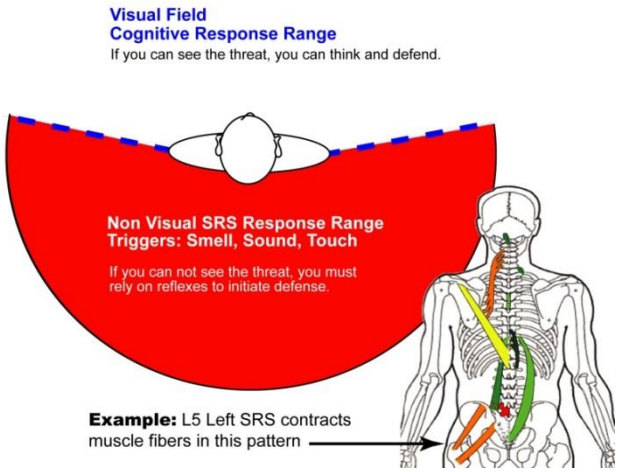
Leading up to the acute injury, it is also inherently difficult to injure a part of the body without destabilizing the facet joint. After all, motion revolves around the spine. Even your eyes moving seemingly isolated from the rest of the body will fire deep rotatories and intertransversalis muscles connecting on facet to another and moving your big toe will change tone in the masseter muscle. When a trauma damages a limb or neck or back, etc. , it also initiates sensory stimulation or capsular ligament overstretch of which in turn we call a "spondylogenic reflex" (SR) (spondylo = vertebrae and genic = origin).

As with most reflexes, an SR swiftly moves the body out of harm's way so that it may return to a safe and non-defensive state and heal. We need these reflexes to protect us; however when an SR remains stuck on due to prolonged damage and/or overstretch of the capsular ligament, it now becomes a "spondylogenic reflex syndrome" (SRS). Once activated, the SRS turns on a massive cascade of tissue reactions that hijack nerves, muscles and joints throughout the body for an indefinite period of time.

The SRS Begins

An SRS is rooted in the embryological origin of the boney spine and its' respective cartilage and ligaments[5]. Understanding its dysfunctional state is a critical factor in understanding trigger point development and its role as another source of chronic pain over a given lifetime.

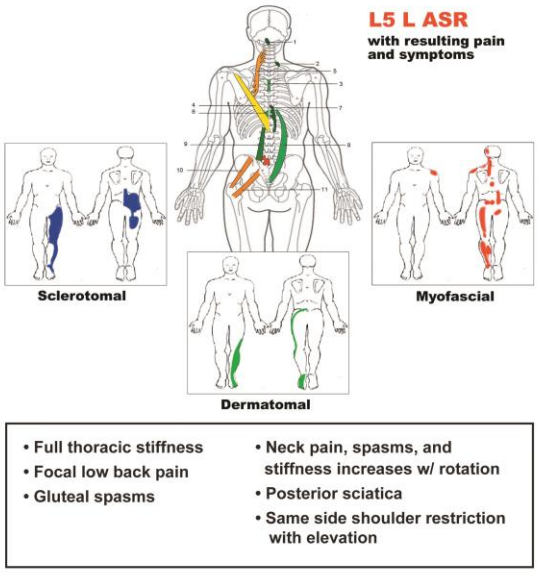
Overstretch or trauma to facet capsular ligament's slow stretch mechanoreceptors result in ligament laxity[6], sclerotome pain[6-8] and spondylogenic reflex syndromes[9]. Innervated through a single spinal nerve or its branches[5], the SRS was discovered by Kelligren[6] and researched by numerous authors over the past 75 years[11-13, 15-17]. The term (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. Spondylogenic reflexes are hardwired into all of us and if we have 7 or 7 billion clients experiencing the same SRS, they will all involuntarily contract the exact same muscle fibers without variation. From this neurological fact, we can now therapeutically rely on the SRS to produce specific patterns of muscle contractions that are predictable, dependable and reproducible - every time. And we can then find them swiftly, treat them systematically and globally reduce or eliminate most trigger point pain and dysfunction without treating the trigger point itself.

The SRS Initiates Trigger Point Activity

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 extremities[12-14]. Once activated, the SRS becomes a cascade of reflexive muscle facilitation and a specific, yet broad subset of dysfunction as described above.



Trigger points develop within the muscles activited by the SRS due to accumulated metabolic waist driven by the unrelenting over-facilitation. The longer the SRS is "stuck-on", the greater the incidence and severity of the trigger points. The hallmark of an active SRS is persistent multi-muscle "over facilitation and hypertonicity". Persistent over-facilitation restricts arterial blood flow and venous-lymphatic drainage. This in turn, leads to muscle hypoxia, nutritional deprivation, metabolic toxicity and tissue acidosis. Overall muscle fatigue and the migration of accumulated metabolic waste to the muscle-fascia interface (trigger point development) are essentially due to insufficient clearing.

The fascia, not the muscle belly contains nociceptors (pain receptors) subject to the accumulated waste under the fascia. Noxious stimulation generates pain referral to other regions of the body. The metabolic waste site also becomes the palpable location of the trigger point (myofascial dysfunction) and the source of pain (myofascial in origin).

Clearing rates are further complicated by factors to include, but not limited to nutritional deficiencies, prolonged muscle loading, traumatic muscle overload, joint tracking error, aberrant biomechanics, physical deconditioning, poor lymphatics, poor cardiopulmonary function, poor posture and low core temperature*.

The SRS can last indefinitely and further drive an expanding cascade of reactions to include secondary reciprocally facilitate muscles which will in turn reciprocally inhibited opposing muscles throughout the body. We feel those reflexively taught and slack fibers throughout our massage session. The client feels the tenderness and pain consistent with their complaint(s). They also feel the relief from pain when you we identify and shut down those very reactions.

Globally resolving multiple trigger points rather than treating one at a time is one of many therapeutic benefits of treating the spondylogenic reflex syndrome. This approach results in much less physical work, is substantially faster and leads to long term functional objective improvement (outcome) in most of our clients.

*A Complicating Factor

When an SRS drives muscle over-facilitation at rates faster than lymphatics and venous drainage can clear, muscle cannot completely relax or recover between contractions. When the client has a *low average core temperature* (CT°), further impairment of the muscle relaxation phase occurs along with additional reductions in lymphatic and venous drainage. The net effect: Trigger point activity increases when CT° decreases. A drop of approximately 1° F. in CT° will increase trigger point activity approximately 20% and a 2° F. drop below normal will increase activity 60% or more.

In Summary

Joint-nerve-muscle-trigger point sequencing of events caused by the SRS quickly illustrate how a simple, yet little known reflex originating from the facet joint capsular ligaments can become a powerful source of both chronic and trigger point pain and dysfunction.

The science and strategies involved in managing spondylogenic reflex syndromes can take massage into a new era of predictable, dependable, reproducible and immediate functional improvements in client care.

It also becomes a win for all when managing the SRS by grossly reducing trigger point pain and dysfunction in the client and notably lessening the physical wear and tear on the therapist.

Caption: SRT is a 3 step process

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