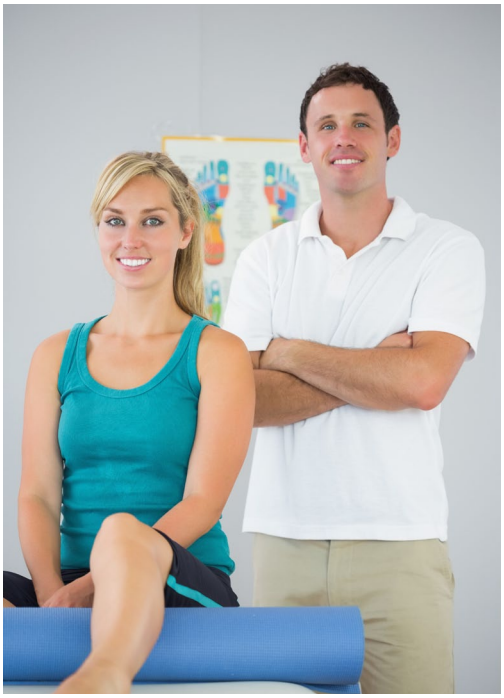
The Science Behind Spinal Reflex Therapy

An Unwavering Event

The Spondylogenic Reflex Syndrome (SRS) is an unwavering neurolophysiological event across all populations. It is also the primary missing factor in the assessment and treatment of most NMS pain and dysfunction, and it severely limits therapeutic outcomes across all professions.

The prevalence of spondylogenic reflex syndrome in the global population is rising exponentially due to rapidly decreasing paraspinal muscle strength, adverse lifestyles and dietary habits and ever increasing environmental stress.

When properly utilized in the identification and treatment of the ensuing cascade of reflex pathophysiology, SRT Assessment and Treatment protocols set a higher standard for predictable, dependable and reproducible outcomes.



Capsular Ligament Overstretch

Spondylogenic reflexes (SR) are normal, neurologically hard wired defensive responses activated via threshold dependent noxious stimulation of posterior pelvic, torso and/or cervical dermatome(s), and/or overstretch and tears to facet capsular ligaments.

Dysfunction within the facet capsular ligament occurs with soft tissue trauma, paraspinal muscle weakness, chronic overstretch, poor posture, adverse ergonomics and in certain conditions involving chronic muscle fatigue and/or metabolic disruption such as hypothyroidism, glucose metabolism dysregulation, etc.

Facet capsular ligament overstretch, or tears temporarily or permanently destabilize one or more functional levels of the spine and induce definitive and progressive sensory/motor facilitation. Spondylogenic reflexes are polysynaptic (multiple nerve pathways) and directly affect multiple neuroeffector junctions throughout the body. When chronically activated due to facet instability, they form a cascade of pathophysiology, dysfunction and pain.

"Predictable, dependable and reproducible outcomes are critical for an efficient and successful patient and provider experience."

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Capsular Ligament Neurophysiology

On a neurophysiology level, capsular ligament overstretch, or tearing initially fires intrinsic capsular nociceptor fibers followed by progressive nociceptor inhibition (seconds to hours later) via capsular ligament stretch mechanoreceptors. Specifically, the propensity for capsular stretch mechanoreceptors to fire at a lower voltage will override nociceptor activation. Facet capsular mechanoreceptor over-stimulation at a given vertebral motor unit may produce afferent sclerotomal pain sensation and will facilitate efferent (effector) junctions of various muscles, glands and viscera within the body. The net effect is an initial, transient, marginal to severe pain sensation (fast pain pathway) localized to the overstretched or torn facet tissue. This is immediately followed or subsides into a progressive cascade of chronic cord mediated effector facilitation with remote pain and tissue dysfunction.

In a normal state, spondylogenic reflexes will activate when triggered and normalize once the stimulus is removed. If a facet articulation is functionally compromised due to a net reduction in overall capsular ligamentous tensile strength due to tears, overstretch, or deficiency in appropriate paraspinal muscle support (regardless of the cause), the resulting spondylogenic reflex syndrome will drive a definitive hard wired reflexive polysynaptic effector tissue (muscle, etc.) facilitation pattern into a chronic state of metabolic fatigue and/or a chronically stressed and compromised physiological state.

The Clinical Side Of Trigger Points

Trigger points, along with their respective myofascial pain and dysfunction profiles are a common topic of soft tissue therapy with most protocols resulting in mixed outcomes and high rates of recurrence. Fundamentally, chronically activated (shortened) muscle tissue will result in metabolic waist migration to the sub fascial regions of the muscle belly due to chronic restriction in venous and lymphatic drainage, arterial hypoxia and restricted nutritional supply; all factors necessary for the recovery phase of muscle contraction.

The resulting metabolic waist accumulation results in what is commonly referred to as myofascial trigger points. Trigger points are frequently complicated further by a subpar core (metabolic) temperature found in approximately one-third of the patient population, and in those with chronic disease conditions. Low core metabolism contributes to reduced lymphatic drainage with increased muscle cytotoxicity, further complicated by a net impaired muscle tissue relaxation/recovery phase and these patients will have multiple recurrent or chronic trigger points. Patients with good cardiopulmonary function, muscle conditioning and normal core temperature can sustain an acute muscle overload event, develop localized trigger points and recover with minimal intervention or chronicity.

Spondylogenic Reflex Syndromes

Spondylogenic reflex muscle facilitation is the primary cause of trigger point development followed by low core temperature, and is a determinant in long term trigger point management. If the SRS is not identified and arrested, trigger point activity will recur and persist due to the inherent tonal increase in all reflexive muscle fibers on the spondylogenic reflex neurological pathway. Further, reflexive muscle activation will

propagate reciprocal facilitation and inhibition in related muscles: another neurological overlay. Chronically activated “spondylogenic reflex syndromes” will continue indefinitely until identified and functionally stabilized through effective patient treatment to include passive procedures and therapies coupled with concise patient dependent spinal stabilization exercises.

The SRS has been evident in research since 1938 and is responsible for spine related sclerotomal pain, most myofascial trigger point activation, non-radicular pain syndromes, facet syndrome (facet joint loading with foraminal hydrostatic neuro-compression) muscle weakness, stiffening and fatigue, attachment tendonitis, joint stiffness and joint tracking error.

Applying systematic SRT procedures and tools in the assessment process allows for rapid identification and localization of the SRS. Given that all reflexes are neurologically hard-wired, that there are a limited set of zygapophyseal capsular ligament reflexes in the spine, that there are a limited number of sensory/motor facilitation pathways in this system, and that their motor profiles are consistent across all populations; strategies in identifying and treating the SRS can and have been standardized. Once identified, a clinically functional profile or thumb print of the SRS can be formulated, an average symptom profile can be constructed and effective treatment strategies can be implemented. These are some of the hallmarks of Spinal Reflex Therapy.

Spinal Reflex Therapy

Mitigating the SRS through Spinal Reflex Therapy involves the rapid identification of an activated SRS through its fingerprint or reactive profile and applying SRT procedures to Ruffini, slow pain nociceptor fibers and Golgi tendon organelles that are subjected to local tendon traction edema triggered by the SRS. SRT procedures are uniquely formulated to activate periaqueductal grey (PAG) matter and adjacent pain analgesic systems located in the thalamus of the brain as a means to reflex inhibition. Specificity and technical accuracy in the application of the technique are essential and assures the release of the endogenous neuropharmacology necessary to reduce slow pain nerve transmission and inhibit cord mediated reflexes.

The net therapeutic effect is the simultaneous reduction in reflex driven effector (motor) metabolic fatigue and weakness, joint compression, joint tracking error, sensory disturbance and pain in multiple regions of the body. Reducing these physiological impediments greatly enhances all subsequent therapeutic procedures and results in a net gain in physiological performance.

In summary, SRT procedures are uniquely formulated to reduce pain and dysfunction, safely reduce the risk of pain medication, improve function and performance and grossly improve the quality of human life.

Research and Supportive Literature