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Physiology, Counterstrain/FPR

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Introduction

Strain counterstrain (SCS) is a type of indirect osteopathic manipulative soft tissue technique developed by Lawrence Jones in 1955 to passively treat musculoskeletal pain, decreased range of motion, and somatic dysfunction by bringing about a change in cellular function of the tissues being treated.[1][2] Strain counterstrain uses palpation and physician feedback to manipulate the soft tissues or joints into a position of ease, away from the restrictive barrier, usually by compressing or shortening the area of dysfunction, encouraging the body to relax a tender-point.[1][3] A tender-point is a small, round tissue texture change found in muscles, tendons, ligaments, or fascial tissues that is tender or painful when palpated at a pressure that would not usually cause pain.[1][3]

Facilitated positional release (FPR), developed in 1990 by Stanley Schiowitz, is similar to strain counterstrain in that the physician places the patient in a position of ease based on palpation of the tender-point.[3][4] However, it differs in that an additional activating force is applied by the physician to compress or distract for a much shorter period than standard SCS techniques to achieve a state of palpated ease (relaxation of the tender-point).[3][4]

Issues of Concern

While many studies have shown promising results with the use of SCS and FPR for specific dysfunctions, there is limited research on the physiology. The most significant area of concern is the limited number of clinical studies to determine the physiology of SCS and FPR.[5] Much of the research done in the physiology of SCS and FPR has been done in animal models and in vitro. The physiological explanation of SCS and FPR is early-on in research and requires more in vivo evidence.[1][6]

Other studies have also questioned the ability of SCS to reduce pain and disability as well as questioned the length of relief provided from treatment.[1][7] Strain counterstrain's effectiveness as a stand-alone treatment compared to other manipulative techniques has also been criticized.[8][9]

Despite studies that question the effectiveness, use, and physiologic foundations of SCS and FPR, the number of promising studies for treating specific dysfunctions suggests a positive impact on medical practice implementation.

Cellular

Strain Counterstrain and FPR work on a cellular level to relieve pain, somatic dysfunction, and range of motion limitations by acting on muscle spindles, Golgi tendon organs, and inflammatory pathways.

Proprioceptors are end organs that sense physical changes in musculoskeletal tissues, muscle length, joint position, and tendon tension.[10] The proprioceptors are an important contribution to somatic dysfunction, limited range of motion, and tender-points. Tender-points and muscle pain appear to be created by aberrant neuromuscular activity caused by spindle fibers and nociceptor activation.[1][11] Muscle spindles, a mechanosensory receptor, inform the CNS of the contractile status of each muscle required to maintain stable posture and movements.[12][13] Each spindle contains several thin muscle fibers (intrafusal fibers), primary type 1a sensory fibers, type II sensory fibers, and fusimotor neurons made up of gamma-motor neurons and beta-motor neurons.[14] The muscle spindle is encapsulated within a connective tissue sheath and is parallel to the muscle fibers within the muscle.[10][14]

When a muscle stretches, type 1a sensory fibers and type II sensory fibers fire at increased frequencies to denote velocity and length stretched, respectively.[10][14][15] The signal transmits to the dorsal root and monosynaptically conveyed to the alpha motor neurons of the muscle fibers surrounding the muscle spindle to elicit a muscle contraction and stop the stretch, as seen in the stretch reflex.[10][14][16][17] Conversely, when a muscle contracts, the firing rate of the muscle spindle slows, and the resulting muscular contraction decreases because it decreases the number of excitatory signals elicited by the alpha motor neurons.

Intrafusal fibers, the thin muscle fibers found in the muscle spindle, can intensify the strength of the afferent signal produced if the muscle stretches and the intrafusal fibers become contracted, rather than at rest.[10] The CNS can alter the tonicity of the intrafusal fibers of the spindle and fine-tune the stretch reflex, the amount at which the muscle can and should contract at a certain length.[10][14][15] The constant changing of the intrafusal fiber length is achieved via gamma neurons and is called "automatic gain control."[10][14] Gamma neurons are efferent lower motor neurons that regulate the intrafusal fibers' contraction and are responsible for the tonicity of the intrafusal fibers alone, thus impacting the stretch reflex.[10][14]

Golgi tendon organs are proprioceptors/mechanoreceptors found in tendons and joint capsules and inform the CNS of the tension/force created by the muscle contraction, not stretch.[10][18] Fast-conducting type Ib afferent fibers innervate Golgi tendon organs and transmit directly to the dorsal horn, synapsing with inhibitory interneurons that will act on the motor neurons to inhibit the continued contraction of the muscle attached to the specific tendon.[10][18] The inhibitory signal created by the Golgi tendon organ (GTO) to decrease the excitability of efferent motor neurons and decrease contractile force is referred to as the Golgi-tendon reflex, autogenic inhibition or the inverse stretch reflex.[19]

Inflammatory pathway initiation by strain or stress to muscle tissue causes fibroblasts to release IL-1alpha, IL-1beta, IL-2, IL-3, IL-6, and IL-16.[20] These pro-inflammatory cytokines can function as damage-associated molecular patterns (DAMP's), recruit neutrophils, activate neutrophils, macrophages, and eosinophils, encouraging increased blood flow and swelling and increase in temperature.[20][21] If tissue damage or muscle injury occurs, the cells release ATP, the pH becomes lowered, and bradykinin and E2 prostaglandins endogenous neuropeptides are released, which produces an inflammatory cascade that activates nociceptors and subsequently releases neuropeptides substance P and calcitonin G related Protein (CGRP).[11]

Substance P and calcitonin G relation protein (CGRP) increase the microcirculation by dilating blood vessels and increasing permeability.[11] The activation of the nociceptors in this manner is the proposed cellular pathophysiology of tender-points treated during SCS and FPR.

Development

Derived from mesenchymal stem cells, skeletal muscle fibers and myofibers are created during primary and secondary myogenesis, which occurs during the embryonic and fetal stage, respectively.[22][23] The few muscle fibers developed during primary myogenesis serve as templates in secondary myogenesis.[22] During the fetal stage of embryogenesis, around week 11, muscle spindles begin to develop from flat mesenchymal cells surrounding small myofibrils arranged near nervous tissue fibers.[24][25]

Muscle spindles become definitive structures by week 20 and continue to lengthen and grow after birth.[24][25] After birth and into adulthood, muscle development is mostly an increase in muscle fiber size and relies on muscle satellite cells for the healing of damaged cells.[26] Golgi tendon organs develop in the late stage of fetal development with the thin collagen bundles forming myotendinous junctions at the tips of the myotubules.[27] GTOs continue to develop until a few weeks after birth when the subcapsular space divides, and the 1b fiber is myelinated.[27]

Organ Systems Involved

The musculoskeletal and nervous systems are the major organ systems treated with strain counterstain and facilitated positional release techniques. The musculoskeletal system, including their proprioceptors, and the local release of cytokines that can create nociceptive pain is responsible for the tender-points treated in SCS and FPR. Additionally, the stretch reflex and gamma-motor neurons that are part of the nervous system are integral to the muscle spindle's function.

Function

The function of strain counterstrain is to correct somatic dysfunction, pain, and tissue texture changes that have resulted in tender-points. A benefit of SCS is that it may be useful for patients that require a gentler osteopathic technique or patient’s who have not responded to other osteopathic techniques. As described above, SCS is a passive, indirect technique.

Facilitated positional release (FPR) also functions as an osteopathic treatment for somatic dysfunction, pain, and tissue texture changes (tender points) in a shorter amount of time than SCS by adding additional compression or distracting force to initiate a quicker cellular response.

Mechanism

Treatment using strain counterstrain will begin with the practitioner identifying the patient's tender point associated with dysfunction. Once identified, the tender-point will be monitored during and after SCS.[1] There is no one standard pain scale measurement used for assessment of the tender-point. However, several palpation pain scale measurements can be implemented, such as the visual analog scale, dichotomous (present or absent), "jump-sign," or sudden physical withdrawal from palpation.[1][28] Due to a lack of established reliability and diagnostic validity for these measurements, additional assessments of dysfunction are recommended to be used, such as range-of-motion, joint mobility, strength, and functional ability.[1]

After identifying the TP, the practitioner moves the patient into a position of comfort in which the TP is at least 70% less tender.[1][29] The ideal position will have a completely non-tender TP with relaxed fascia.[1][3] To produce the ideal position, the practitioner will bring the tissue associated with the TP into a shortened position by bending the joints around the TP.[1] The position of comfort is then maintained passively for 90 seconds.[3][28][3] Throughout the treatment, the practitioner will remain lightly touching the TP to assess the tenderness and fascial tightness continually.[1] Monitoring for decreasing tenderness and tissue ease via palpation throughout the treatment is used to determine if more or less than ninety seconds of palpation is necessary for treatment.[1] The practitioner will then slowly and passively return the patient to a neutral resting position and perform a reassessment of the TP.[1][3][28][3]

Facilitated positional release differs from SCS because when the patient is placed in the position of comfort, the practitioner will apply a facilitating force of compression or distraction to the tissues in that position for an amount of time that is much less than 90-seconds, such as 5 seconds.[3][4][3]

Related Testing

The tests and imaging capabilities found to be effective on Fibromyalgia patients who suffer from trigger points, small round painful tissue texture changes found in musculotendinous tissue

bands, have also been found to help image Tender-Points.[1][30][1] Ultrasound imaging techniques, specifically Sonomyography and sonomyoelastography, will show elliptical hypoechoic areas when imaging Tender Points and can be used for diagnosis and treatment efficacy.[30]

Pathophysiology

Currently, there are three proposed theories to explain the formation of tender-points and their treatment via strain counterstrain: proprioceptive theory, local inflammatory circulatory effects, and ligamento-muscular reflex.

The proprioceptive theory is the most common explanation for SCS effectiveness.[1] The theory argues that in response to the stretch reflex, the antagonist muscle spindles will activate muscle counter-contraction creating a perpetual muscle spasm resulting in neuromuscular imbalance, hypertonicity, and referred pain, termed the tender-point.[1][10] The neuromuscular imbalance caused by the proprioceptive reflex resulting in an increase in intrafusal muscle tone inside the muscle describes the "rope-like" quality of some tender points.[10] Underlying muscle imbalance/tender-Points are considered an active process/injury and can last long after strain has occurred because the muscle is actively shortening as if it were being stretched.[1][10]

The shortened muscle can also limit the joint and range of motion.[10] Counterstrain treats the somatic dysfunction by slowly shortening the muscle to reset the muscle spindle gamma-motor neuron output and decrease the intrafusal and extrafusal fiber disparity so that the muscle spindle is no longer activated and decreases its firing frequency (contraction reflex) when the muscle resets to resting length.[10]

Quantifiable studies have shown that symptomatic individuals with tender-points: (1) felt tender at lower electrical thresholds, (2) had reduced stretch reflex amplitudes when treated with SCS, and (3) experienced reduced pain and improved range of motion after SCS.[29][31] [32] However, studies that tried to test the proprioceptive theory directly have produced conflicting results.[7][31]

Local inflammatory and circulation effects appear to be another cause of the effectiveness of strain counterstrain and FPR. Repositioning an individual can increase circulation to that area, help with nutrients and waste removal, decrease swelling, and ameliorate tender-point pain caused by ischemia.[1][33] A study measuring cytokines released by fibroblasts in tissues during and after strain, stretch, and when returned to the resting position, found that after one minute of SCS fibroblast production of pro-inflammatory interleukin-6 decreased, suggesting local circulatory effects.[2][3][20][8]

The least studied explanation for SCS and facilitated positional release (FPR) effectiveness is the ligamento-muscular reflex. Ligamento-muscular reflex is defined by the contraction/relaxation of muscles in response to the ligament mechanoreceptors to prevent ligament damage.[34] SCS and FPR can correctly position joints or add light compression/detraction, respectively, to stretch the ligament or tendon to relax the muscle using the ligamento-muscular and GTO reflex.[3] The ligamento-muscular and GTO reflex inhibit muscular contraction of the corresponding muscle.[3] Although SCS's and FPR's physiological effects on the body are unclear due to conflicting research results, the current supporting literature and theoretical knowledge of their effects support the clinical use of SCS and FPR.

Clinical Significance

SCS and FPR are commonly used osteopathic techniques and are especially useful when treating individuals with chronic conditions that cause severe pain, individuals with osteoporosis, the elderly, individuals with acute strain, those who have had recent surgeries, had recent trauma, or are hospitalized or individuals that do not respond to other techniques or desire gentleness.[1] [35] Evidence shows that SCS is indicated in the treatment of the following areas:[1][28][29][33] [36][37][38][39]

- Hip tender points
- Trapezius pain
- Mechanical neck pain
- Chronic ankle instability and sprains
- Plantar fasciitis pain
- Shoulder pain
- Sacral torsion
- Lower back pain
- Cervical hysteresis
- Iliotibial band friction syndrome
- Headache
- Tendonitis
- Epicondylalgia
- Knee pain
- Rotator cuff syndrome
- Fibromyalgia
- Osteoarthritis

While there is limited experimental research done to determine the efficacy of FPR in treating pain and somatic dysfunctions, FPR's similarity to SCS would suggest it can be useful for many of the same indications. Research on the use of SCS and FPR for the treatment of piriformis syndrome, iliacus, and psoas dysfunction is also limited; however, it is part of the osteopathic physicians' curriculum and practice. However, like most osteopathic techniques the use of SCS and FPR is contraindicated in individuals whose tender-point is in an area with an inflammatory process that may be caused by problems other than somatic dysfunction (such as infection), a patient is unable to communicate for tenderness feedback, or the patient cannot handle manual therapy.[1]

Continuing Education / Review Questions

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