Fascial Strain and Counterstrain

Introduction:

Not long after Dr. Lawrence Jones D.O. discovered neuromuscular tender points, he came to the realization that these areas of contracted, painful, tissue could be used to identify and treat all types of somatic dysfunction.

Utilizing 1970's physiology, primarily derived from an article by Irvin Korr Ph.D., Dr. Jones postulated that these neuromuscular tender points were reflexively connected to the spinal cord and were most likely related to a dysfunction in the local muscle proprioceptors.

Can Muscle Proprioceptors Really Account For Somatic Dysfunction?

The idea that muscle proprioceptors could be responsible for chronic somatic dysfunction was a revolutionary idea in that it went against the accepted joint fixation theories of the era. Korr's neuro-reflexive rationale for somatic dysfunction was definitely an advancement over the older scar tissue based theories, however, muscle spindle based rationales have some significant limitations when we consider more recent research. For example, it has been shown experimentally that:

- Other tissue receptors besides muscle spindles have been identified that could produce a similar clinical presentation. (3)
- Somatic dysfunction can be triggered from non-muscular tissues including the viscera, in which, no muscle spindles are present. (2)
- Studies show that muscle spindle firing is not necessary, nor is it usually sufficient, to produce alpha motor neuron activation. (2)
- Some muscle spindles afferents actually have an inhibitory, not excitatory effect on alpha motor neurons (4)

In addition to the above problems, researchers have found autonomic nervous system "arousal" to be a major component of chronic somatic dysfunction (2). Considering that muscle spindles have no known connection to the autonomic nervous system, it is highly doubtful that they are responsible for this phenomenon.

Type III and IV Peripheral Neurons

So what proprioceptive model could possibly account for our ability to perform strain and Counterstrain on the fascial structures of the viscera, vascular and nervous systems? The answer involves a specific type of peripheral neuron that is found almost exclusively in the body's deep fascia called type III and type IV peripheral neurons.

Type III and IV peripheral neurons have both mechanoreceptive (movement sensing) and nociceptive (pain sensing) capabilities and are found throughout the body's connective tissues including ligaments, tendons, joint capsules, the outer wall of all larger blood vessels, visceral fascia (peritoneum,) and in the neural fascia (epineurium / dura.) 5,6,7

In addition:

- Type III and IV neurons are sensitive to mechanical, chemical and thermal stimuli thus can be stimulated by trauma, surgery, inflammation or many other of the common causes of myofascial pain such as postural strain. (5,7)
- These neurons travel into the dorsal horn of the spinal cord where they can branch, traveling up to 5 segments above or below the injured segment (5). This offers a plausible explain for the tendency of somatic dysfunction to spread and refer pain into neighboring regions.
- Nociceptive neurons release inflammatory chemicals such as *substance P* when triggered, and are sensitized by the same chemicals, thus it takes a smaller stimulus to cause pain once they have been activated (6). This relates to the diminished pain thresholds observed in patients suffering from chronic somatic dysfunction.
- Not all signals from peripheral nociceptors reach consciousness (5). This accounts for the presence of "latent" tender/trigger points (areas of neuromuscular dysfunction present in our bodies to which we are unaware until palpated.)
- Of all the known tissue receptors found in muscle and connective tissue, only these type III and IV neurons are known to connect to the autonomic nervous system and have been shown experimentally to change heart rate, blood pressure and respiration (2,7.) This explains the autonomic arousal associated with chronic somatic dysfunction and the observed viscerosomatic effects of manipulation.
- These fascial neurons connect in the spinal cord to **motor neurons in the ventral horn,** and to **preganglionic neurons of the autonomic nervous system** thus can trigger **nocifensive** and **nociautonomic reflexes** (5,2). These reflexes are crucial to the understanding of the effects of fascial Counterstrain and will be discussed in the next section.

Nocifensive, Nociautonomic and other Important Reflexes

Nocifensive reflexes are defined as specific segmental and multi-segmental **contractile** responses that exist to minimize nociceptor stress (pain) in the involved tissues (2, 5). In other words, these are skeletal muscle contractions, activated through a spinal cord reflex arc, which the body utilizes to protect inflamed or damaged tissues. Realize since **all** types of fascia contain these nociceptors, this reflexive contraction can be associated with visceral, vascular, ligamentous or neural fascia!

Nociautonomic reflexes are neural connections to the autonomic nervous system that can result in autonomic responses such as vasodilation, bronchodilation, or gastrointestinal stasis (2,5). Skeletal muscle, for example, (due to the presence of beta adrenoreceptors) will "engorge" or swell under the influence of these reflexes due to the release of chemicals such as histamine and bradykinin. Again, **all** types of fascia (visceral, vascular, neural etc.) containing these Type III and IV proprioceptors can trigger this autonomic reflexive response.

The Vascular Stretch Reflex is another example of a protective reflex arc directly related to the deep fascial proprioceptors. The proximal portion of all major arteries and veins contain un-myelinated C fibers (Type III and IV nerve endings.) These nerve endings connect with the sympathetic neurons in the grey horn of the spinal cord, which in turn, connect via efferent fibers back to the smooth muscle of the vessel wall itself (8, 9). This entire pathway is a spinal reflex arc that causes a contraction of the vascular trunk when the stretch on the tunica adventitia (outer vessel wall – made of deep fascia) exceeds a potentially damaging threshold (8). In essence, this reflex arc gives our vessels the ability to avoid permanent elongation or weakening due to the effects of gravity or strain by contracting against those forces. This is a "smooth" muscle contraction or autonomic response, not a skeletal muscle response, initiated by the sensory apparatus contained in the vascular deep fascia (tunica adventitia.)

Fascial Contractility

The last piece of fascial physiology we need to discuss before putting this all together is the ability of fascia to contract. A series of fascial experiments performed in the 1990's by Yahia,

1993 and Straubesand in 1996 verified the ability of human fascia to actively contract and identified the presence of myofibroblasts or smooth muscle cells embedded within the fascia itself (10, 11). Subsequent experiments performed by Schleip in 2006 verified the presence of Type III and IV sensory endings, autonomic nerve fibers and contractile cells in fascia (12.) Schleip's studies also proved that the contractile force of fascia is of a sufficient force to influence musculoskeletal behavior through gamma motor neuron activation.

The Fascial Strain and Counterstrain Model

So why would all of the vital structures in our body (organs, vessels, nerves etc.) be covered by deep fascia containing a multitude of sensory endings? And why would this "sensory blanket" as I like to call it, connect via nociautonomic and nocifensive reflexes to the local contractile tissues in the area?

The answer I believe is quite simple, protection. The human body must have a method of sensing and rapidly responding to strain applied to our fragile internal structures. Organs, nerves, and vessel all have the ability, via the fascial reflex arcs described above, to "call in" the protective abilities of the muscle system, as needed, to avoid catastrophic damage to the organism.

This "protective" contractile response can be initiated by stimulation of the fascial proprioceptors present in any visceral, somatic, vascular or neural structure and can manifest clinically in the following manner:

- Generalized tissue allodynia and the formation of neuromuscular "tender points."
- A palpable increase in local skeletal muscle tone (due to nocifensive reflexes)
- A palpable increase in tissue tone. This can be observed in skeletal muscle, all types of fascia, or in the smooth muscle cells of contractile organs / vascular tissues.
- Localized edema due to sympathetic engorgement of skeletal muscle or interstitial edema due to dysfunction of the vascular bed, venous/ lymphatic portion.
- Tissue ischemia due to dysfunction (contraction) of the arterial side of the vascular bed
- Organ dysfunction due to altered organ blood flow and / or elevated smooth muscle tone in contractile organs (e.g. bladder, intestines, esophagus etc.)
- Loss of joint/segmental mobility due to increased skeletal or smooth muscle tone.
- Changes in joint resting position due to increased skeletal or smooth muscle tone.

- Changes in proprioception due to changes in joint resting position
- Weakness due to pain inhibition, neural compression or skeletal muscle reciprocal inhibition.
- Loss of function due to any of the above physiological effects.

Origin of the Technique

My initial observation that Strain and Counterstrain could be applied to fascial structures, occurred in the late 1990's. I had just returned from a visceral manipulation course and began to apply the visceral stretching techniques I had just learned to my chronic pain population. I was able to identify the deep visceral- fascial restrictions, however, was frustrated by the fact that the restrictions were "fighting back." In other words, I perceived active resistance to my direct manipulation efforts. Since this repeated observation could not be explained by the visceral adhesion rationale offered in the course, I decided to go in the wrong direction and attempted an indirect treatment or "Counterstrain" of the visceral restriction.

The result was an immediate relaxation of the surrounding tissue and the perception of a faint "therapeutic pulse" or vascular release in the surrounding tissue. I maintained the position for 90 seconds and after re-assessment, noted a complete correction of the visceral restriction. The patient also reported an instant improvement in pain and said to me, immediately after treatment, "I don't know what you just did, but that really helped!"

Further experimentation verified the success of indirect manipulation in the viscera. From that point on I began a laborious, multiple year process of identifying visceral tender points and the specific organ manipulation that would correct each restriction.

The Impact of Fascial Research

My excitement over successfully treating the viscera utilizing Counterstrain was equaled only by my disappointment when it came to trying to explain how this was actually possible. By that time, through deduction, I had come to the conclusion that I was impacting and manipulating visceral ligaments. This however did nothing to explain the effect of indirect techniques on these non-contractile structures, which would be necessary if I were to attempt to teach this material to anyone outside of my immediate friends and

colleagues. Nowhere in the literature could I find any references with regards to contractile fibers in fascia or ligaments.

All that changed with the First Fascia Congress which was held at Harvard Medical School in 2007. Yahia, Straubesand, and Schleip's groundbreaking studies on the contractile and sensory properties of connective tissue were presented and all my questions regarding how fascia could possibly be "Counterstrained" were answered. More importantly, I learned that the body's deep fascia is basically, histologically speaking, the same structure everywhere in the body. From there it was only a matter of time before I was able to develop a whole new theory regarding Counterstrain. The theory was simple, If Counterstrain worked in the visceral fascia and all fascia in the body is histologically similar, then vascular, neural and ligamentous tender points should also exist. Several thousand hours experimentation later and we now have what I like to call the systematic approach to neuromuscular dysfunction; or as most people call it, "Fascial Strain and Counterstrain."

Treatment

So what exactly happens during a fascial SCS treatment? Well, It's too early to be definitive, but based on my current observations, there appears to be both a neural and vascular component in fascial SCS.

Neural: When we perform a fascial SCS release, we slacken the involved fascial tissues (vessel, nerve or organ tunics) including the embedded Type III and IV neurons. This "decompression" of the free nerve endings would mechanically deactivate the local noci/mechanoreceptors, silencing the associated nociautonomic and nocifensive reflexes (2.)

Vascular: Fascial SCS will also have an immediate impact on local inflammatory metabolites like substance P. This occurs because as the involved tissues are decompressed; local venous and lymphatic vessels will open, draining inflammation from the region. This will eliminate the chemical irritation of the local type III and IV neurons which also will silence the nociautonomic and nocifensive reflexes. Realize that this can occur in muscular, vascular, neural and visceral structures since they all have their own lymphatic / venous drainage systems (e.g. vasa vasorum, vasa nevorum)

Closing Comments

The clinical impact of learning these techniques can be quite dramatic. Not only will you have treatments for all of those unknown tender points that you have encountered over the years but you will also begin to recognize entirely new *systems* of dysfunction in your patients. You will for example, learn to recognize the signs and symptoms of vascular dysfunction in your patients and be able to directly improve the function of the local vascular bed. This includes performing arterial SCS to improve blood flow in an injured area or by using Venous/Lymphatic SCS to resolve chronic edema related to regional dysfunction of the lymphatics. Other examples include learning what types of sciatica and back pain are caused by the visceral fascia and even how to directly treat the sympathetic chain in your Complex Regional Pain Syndrome patients. These new fascial techniques expand the scope of Strain and Counterstrain well beyond that of the musculoskeletal system and into the world of general wellness. These techniques actually improve the function of the nervous, visceral and vascular systems.

In closing, it is important to realize that in order to maximize the healing capacity of the human body, one must gain the necessary anatomical knowledge to identify and correct dysfunction in all of the body's systems, not just the musculoskeletal system. This treatment model is similar to that which was employed by the founder of Osteopathy, A.T. Still, in the late 1890's and supports his core osteopathic principle that unaddressed dysfunction can lead to tissue pathology and disease. In essence, this approach will allow you to identify and treat the true source of your patient's pain/disability, not just the surface presentation.

I believe we have only just begun to understand the variety of conditions that can benefit from Fascial SCS and look forward to the many discoveries that are sure to come as this technique gains mainstream acceptance and is employed by an ever expanding group of practitioners across the globe.

Sincerely,

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