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Strain and Counterstrain for Structural Integrators

Caryn (Davidson) Pierce



Caryn Pierce, PT, JSCC, BCSI, MTC, graduated from Andrews University with a BS in anatomy and physiology in 1994 and an MS in physical therapy in 1995. After 17 years of clinical practice and three professional certifications—strain and counterstrain (www.jiscs.com), structural integration (www.anatomytrains.com), and manual therapy-joint manipulation (www.usa.edu)—she is just now starting her own cash-based mobile practice out of her home in North Bend, WA. She hopes the increased flexibility will not only help her meet clients' needs but also allow her to pursue PhD and teaching goals, so the awareness of these fascial therapies will be available to entry-level healthcare practitioners in an academic setting. Caryn can be reached at bodymechanic@hotmail.com.

Strain and counterstrain (SCS) is an indirect approach to manipulating fascia. In addition to its usefulness as an integration tool, knowledge of the reflexive mechanisms involved in SCS deepens our understanding of how fascia functions to protect vital structures from injury and how unbalanced posture and movement patterns develop after trauma.

History and Description

The phenomenon originally known as *positional release* or *spontaneous release by positioning* was discovered by Lawrence Jones, DO, in 1955 while he was trying to help a patient with persistent low back pain and a stooped posture find a comfortable position in which to sleep. The patient was so comfortable propped in an extremely flexed and twisted posture that Dr. Jones left him there while he went to treat another patient. When Dr. Jones returned to help the patient up off the treatment table, the patient was able to stand fully erect without pain for the first time in several months. This led to nearly a decade of experimentation and practice before Dr. Jones first published his findings in *The DO* (1964). He continued to practice, publish, and teach until his death in 1996, by which time he had founded the Jones Institute of Strain and Counterstrain with Randall Kusunose, PT, as its director.

The technique is incredibly simple. A counterstrainer holds the body in a finely tuned three-dimensional position of ease away from muscle guarding or movement restriction and waits for the body to reflexively “let go” of the restriction. Once the body lets go, normal motion in the opposite (previously restricted or guarded) direction is restored (Jones, 1964). This takes about 90 seconds

unless facilitation techniques are used concurrently to speed up the release (Kusunose, n.d.b). The results can be immediate and dramatic even if the original strain occurred years prior to treatment.

Newer fascial strain and counterstrain techniques developed by Brian Tuckey, PT, one of the original Jones Institute instructors, call for manually gliding the restricted fascia in a direction of ease rather than positioning the whole body around it. With these fascial gliding techniques, the release is often more widespread and takes less time. Tuckey was initially inspired to counterstrain this way in 1992 while studying direct visceral manipulation techniques. He felt active, involuntary resistance from the stretched fascia and tried performing the techniques backward (Tuckey, n.d.c.).

The advantage of SCS over some other indirect approaches is the fact that its developers have mapped out specific tender points—autonomic projections of pain—that consistently correlate with specific structures and specific positions or directions of ease and bind. These tender points are palpable as localized areas of hypertonicity and edema in addition to the involuntary *jump sign* they elicit from the client. While the treatment is not directed at the tender point, the effectiveness of the release position or direction can be monitored by palpating the tender point, which melts away and begins pulsing when you've got it right and stays away after slowly returning the body to a neutral position. The existence of these consistent tender points makes evaluation and treatment more objective and easier to study and teach (Jones, 1964).

When performed correctly, SCS treatment is comfortable and painless for even the most fragile

client. I would even go so far as to describe the experience of receiving the treatment as affirming because counterstrain gives additional support to involuntary reflexes set off by an initial strain and thus reinforces what the body was already trying to do to protect itself. At first, the treatment positions may seem counter-intuitive, as they exaggerate asymmetrical and unbalanced postures, but the slack provided by each position or glide takes the stretch off dysfunctional proprioceptors that are hypersensitive to stretch and thus allows the body to stop trying to prevent overstretching (Jones, Kusunose, & Goering, 1995). Notice also that counterstrain can be performed with the client fully clothed. Even many of the pelvic floor techniques can be performed externally through loose-fitting clothing.

Example #1

Tender points on the lateral aspect of the L5 spinous process and the superior medial aspect of the PSIS both correspond with the same dysfunction—limited flexion at L5-S1, or an L5 that is stuck in an extended position on the same side as the tender point, appearing possibly as exaggerated lordosis that won't reverse, or a contralateral rotation with forward bending. The classic Jones positional release (Figure 1) uses the leg as a lever to exaggerate the extension deformity, usually in a prone position (Kusunose, n.d.a). Tuckey's fascial glide (Figure 2) sinks manually through the gluteal musculature to the depth of a corresponding branch of the superior gluteal artery and pushes it in a direction of ease back toward the heart, or more specifically where it

branches off from the internal iliac artery (Tuckey, n.d.a). Either technique works because it slackens the fascia around the superior gluteal artery and thus turns off hypersensitive stretch reflexes that were restricting movement and projecting the tender point.

Example #2

A tender point on the anterior aspect of the transverse process of C6 corresponds to limited extension at C6-7, or a C6 that is stuck in flexion on the same side as the tender point and contributes to a forward head posture. For the classic Jones positional release performed with the client supine (Kusunose, n.d.a), the head and neck are lifted into a significant amount of forward bending with rotation and side bending away from the tender point, bringing the corresponding transverse process forward and down toward the chest (Figure 3). Tuckey's fascial glide (Tuckey, n.d.c) sinks through the rib cage to depth of the lung and pushes it up toward the neck, which can be positioned on a pillow in neutral or slight forward bending with rotation and side bending away (Figure 4). These techniques are both effective because they slacken Sibson's fascia at the apex of the lung where it blends with the occasionally present scalenus minimus muscle and attaches to transverse processes of C7, turning off hypersensitive stretch reflexes there.

One of the advantages to the fascial glides is the specificity of contact on the structure in which the hypersensitive stretch reflex originates. Another advantage is that most of the fascial glides can be performed in neutral postures for clients who have



Figure 1. The classic Jones positional release for limited flexion at L5-S1



Figure 2. Tuckey's fascial glide for limited flexion at L5-S1



Figure 3. The classis Jones positional release for limited flexion at C6-7

poor tolerance for movement. Positional releases can be good for clients who don't tolerate manual contact or pressure on the fascia, or if the practitioner is uncertain what structure contains the hypersensitive stretch sensors.

Reflexive Mechanisms in Somatic Dysfunction

Though effective for years, counterstrainers have not always known what they were treating. An early hypothesis based on an article by Irvin M. Korr (1975) implicated a hypersensitive stretch reflex mediated by muscle spindles. Korr described how the central nervous system could effectively turn up the volume in order to hear from these momentarily silent stretch sensors in a muscle on the hyper-shortened side of a strained joint, such as the medial side of an ankle inversion sprain. By increasing excitatory outflow from the sensorimotor cortex to gamma motor neurons that signal the contraction of intrafusal muscle fibers, the muscle spindle's sensitivity to stretch is increased. This amplification or *gain* enables the body to sense changes in muscle length while the muscle is on slack. However, it can also result in a faulty report of overstretching or potential strain if the muscle returns too quickly back to its normal length.

This would explain why the body holds a sprained ankle in an inverted rather than neutral position even though it increases stress to the lateral side of the ankle where tissue damage is most likely to have occurred. While there may be pain and inflammation on the lateral side of the ankle, the exquisite tenderness to palpation and resistance to stretch associated with somatic dysfunction will



Figure 4. Tuckey's fascial glide for limited flexion at C6-7

often be found on the medial side, subsequently limiting eversion range of motion and providing faulty proprioceptive input on which the body bases further attempts at motor planning. This is the strain. Counterstrain resets the inappropriate gain by shortening the affected muscle even further in order to remove all stretch from the spindles, zeroing their output. (Notice that this would simulate the original strain position.) Then, by slowly lengthening it back toward a neutral position rather than jerking it back quickly, the hypersensitive stretch reflex is not reactivated. Free of somatic dysfunction, the ankle will rest in a neutral position once again, allow both inversion and eversion range of motion, and provide correct proprioceptive input for motor planning (Jones, et al., 1995). Oh, and the pain goes away!

You can picture a more complex version of this for a spine that experiences whiplash and ends up with exaggerated primary and secondary curves and perhaps a twist in the direction it was flung around the shoulder strap of the seatbelt.

Interestingly, according to Brian Tuckey, PT and SCS instructor for the Jones Institute, Dr. Jones was not fully satisfied with this explanation. He did not think counterstrainers were treating muscles because the release positions did not correspond directly to muscle origins and insertions, neither did they correspond to joint mechanics, and they had autonomic effects.

After Tuckey began experimenting with performing direct visceral release techniques backwards, he discovered a paper by Richard L. Van Buskirk (1990) that describes complex *nocifensive* and *nociautonomic* reflexes that recruit skeletal muscle

and autonomic processes to avoid stimulating the pain-sensitive free nerve endings in fascia while at the same time lowering the threshold of stimulation through central sensitization in the spinal cord as well as through the excretion of inflammatory cytokines in a positive feedback loop. Van Buskirk hypothesized that, by taking all the tension out of the fascia, counterstrainers facilitate the free flow of fluid to wash out noxious chemicals from around the nociceptors, and thus turn off these persistent reflexes. This model is definitely an important piece of what happens during SCS, but it was developed before research had led to the discovery of the stretch reflex (Yahia, Pigeon, & DesRosiers, 1993), myofibroblasts and smooth muscle (Staubesand, & Yi, 1996), and proprioceptors in fascia (Schliep, 2003). Robert Schliep's neurobiological explanation for fascial plasticity (2003), especially in response to the direct myofascial manipulation of Rolfing®, also illuminates an explanation for the body's reflexive response to indirect manipulation in counterstrain. He describes free nerve endings that are not just pain sensitive but serve as slow- and quick-adapting type III and IV mechanoreceptors that exist everywhere in the body. He also describes myofibroblasts in fascia that contract a latticework of smooth muscle cells (Schliep, 2006) in response to either mechanical or chemical stimulation, thus transmitting forces more efficiently to mechanoreceptors (Schliep et al., 2006). In combination, these provide proprioceptive information similar to that received from primary and secondary nerve endings in the muscle spindles modulated by contraction of intrafusal muscle fibers. The combined result is a fascial stretch reflex that behaves very much like the one we see from muscle spindles and was described by Korr as the basis of somatic dysfunction, but is applicable globally and has autonomic connections in the spinal cord that interact as described by Van Buskirk.

Fascia as a Proprioceptive Blanket

At this point counterstrainers see fascia functioning as a proprioceptive blanket that surrounds and protects vital structures by providing sensory input for complex reflexes that not only control tension in the fascia but recruit skeletal muscle to prevent overstretching or strain to the fascia around visceral organs, arteries, veins and lymphatic vessels, nerves, and even bones and joints (Tuckey, n.d.c.). These reflexes are similar to posture and righting reactions that keep our eyes facing the front, level with the horizon, and keep vestibules upright perpendicular

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to gravity. During normal function they help maintain spatial relationships among the structures as we move, and prevent excessive elongation of connective tissue in response to the pull of gravity, no matter what position we are in. However, when this stretch-sensitive fascia experiences forces outside the range of motion, speed, or duration the body can successfully manage, somatic dysfunction develops. Even without mechanical disruption to the tissue, an area of fascia that has been tugged on too hard, too fast, too many times, or for too long will become hypersensitive to stretch, and the reflexes will persistently disallow motion through that part of the fascia when planning movements or maintaining a posture. (This is the same as in the previous example of an ankle sprain, except now we understand that the somatic dysfunction is maintained by a protective reflex around the vital blood vessels and nerves, not just hyper-sensitized muscle spindles.)

Unfortunately, fascia can remain painfully hypersensitive to stretch long after its protective function was required during a straining event, but counterstrain corrects this by a number of different mechanisms.

System Phenomenon

Through his study and practice of SCS, Tuckey was increasingly able to identify and treat somatic dysfunction around specific structures, turning off tender points as he went. In doing so, he encountered constellations of tender points that lit up together when related structures came under strain and turned off or dimmed together when counterstrain was applied. He also noticed that as he turned off tender points related to dysfunction in one part of a constellation—putting slack into that system, it was immediately taken up by the next strongest area of dysfunction within the same system. Tender points in that area also had to be treated in order to turn off the entire constellation. He called this the *system phenomenon* and thus identified five separate fascial tensegrity systems organized around

the vital structures: visceral, lymphatic and venous, arterial, neural, and periosteal which includes the ligaments of the skeletal system. Though dysfunction in more than one system does co-exist, for the most part it only jumps around within the same system during treatment.

Another interesting manifestation of the system phenomenon is that when somatic dysfunction develops with its associated tender points and movement restrictions in one part of a system, it actually increases tension evenly throughout the whole system according to the principles of tensegrity. A client's subjective reports of pain and tightness are not always located in the tender and restricted area but in the area where greatest excursion of that system occurs during movement. For example, dysfunction in the lymphatic and venous fascia could be maintained in the left shoulder and right foot, though the client reports pain and tightness in the low back as the taut vessels are stretched with forward bending. Palpating and treating tender points located in the restricted areas around the shoulder and foot release the tension throughout the entire lymphatic and venous system and relieve the back pain with forward bending.

Cranial Representation and Scan

Tuckey's most exciting discovery related to the system phenomenon is that somatic dysfunction in each of these five systems is consistently represented as rigidity in the deep fascia of a corresponding region of the cranium, which likely has its roots within embryologic development. He discovered this while trying to determine which structures and systems Dr. Jones' original cranial tender points and treatment positions were associated with. Scanning the cranium for rigidity leads to efficient sequencing of treatment by directing the counterstrainer to the system and quadrant of the body where the strongest dysfunction is held. In the same way that local somatic dysfunction resolves immediately with counterstrain, so does the corresponding cranial rigidity. The cranial scan can be used repeatedly throughout a treatment session to assess effectiveness of treatment in clearing an entire system of dysfunction and to identify the next most significant area of dysfunction. Though the cranial scan is accessible to beginners, more familiar methods of assessment such as body reading, motion testing, and palpating for characteristic tender points are also informative.

If . . . somatic dysfunction is present in one of your clients, . . . it will be difficult or impossible to sink into the layer of restriction and push it in the way you want it to go.

Simplicity and Power

Despite its complex mechanisms and far-reaching effects, the application of SCS techniques remains simple: palpate and monitor the tender point, position the corresponding body part or glide the fascia in the direction of ease, wait a few seconds for the body to let go of it, and then slowly return it to a neutral position. A recent study of cervical hysteresis published in the *Journal of Bodywork and Movement Therapies* (Barnes et al., 2012), compared five different osteopathic manipulation techniques (OMTs) to sham intervention. While all five OMTs did better than the sham, SCS did better than the other OMTs.

Clinically observable effects of SCS include decreased pain, decreased tenderness, decreased swelling, increased circulation, normalization of aberrant muscle tone, increased muscle flexibility, increased joint range of motion, improved proprioception, improved posture, improved efficiency of muscle recruitment patterns, and overall improved functional performance. There are also physiologic effects specific to the vital structures involved and a general shift in the autonomic nervous system from sympathetic to parasympathetic tuning (Jones, et al., 1995; Tuckey, n.d.b). You may recognize many of these parameters as characteristics of structural integration and, much like structural bodywork, a collection of SCS techniques can be applied effectively to specific areas of the body to solve specific problems, but the full benefit of the treatment approach is not appreciated unless it is applied systematically throughout the entire structure.

So, the question arises: How does this relate to the practice of structural integration as we have learned it in our various schools, systematically applying direct manipulation with movement to release restricted myofascial structures?

Somatic Dysfunction and Structural Integration

Somatic dysfunction is likely a *first cause* underlying some of the habitual movement patterns that develop after trauma and lead to adaptive changes in the myofascia. If the somatic dysfunction were cleared using SCS immediately after the traumatic event, it would not become part of the history that makes up the cumulative pattern, and structural bodywork would not be necessary for integration. However, if somatic dysfunction persists, adaptive changes will occur over time according to the principles Tom Myers eloquently describes in the first chapter of *Anatomy Trains* (2009). In that case, if only the somatic dysfunction is addressed with SCS, the adaptive changes will remain. It is possible they will dissipate over time the way they accumulated in the first place, now that the underlying hypersensitive stretch reflexes have been turned off. However, this process of integration can be accelerated and more thoroughly accomplished with structural bodywork. Conversely, if only structural bodywork is applied in the presence of somatic dysfunction, the work will be difficult and painful and the habitual pattern will probably return—maybe even immediately after the mechanical restriction is released—because the hypersensitive stretch reflex remains untreated.

Sometimes the somatic dysfunction from recent trauma is superimposed on an underlying adaptive pattern that predisposes the body to strain. In that case, it is important to address the old pattern with structural bodywork in order to prevent new somatic dysfunction from becoming persistent due to recurring strain. Occasionally I have worked with very stiff, crooked clients who did not manifest the tenderness and hypertonicity associated with somatic dysfunction until after receiving structural bodywork, evidence that the adaptive changes were effectively preventing them from stretching the hypersensitive mechanoreceptors. Once they were able to move more freely, these clients began to experience pain with what was perceived as “overstretching.” Counterstrain relieved it. It is also possible to develop adaptive changes for other reasons besides somatic dysfunction: habitual body postures related to emotional states, repeated movements below the threshold of strain, or immobilization due to splinting or paralysis, etc. All of these still need to be integrated with structural bodywork.

If you are wondering whether or not somatic dysfunction is present in one of your clients, see if the quality of the fascia is exquisitely tender and defensive to palpation. It can be difficult or impossible to “sink in to the layer of restriction and push it in the direction you want it to go” as called for by direct myofascial release techniques. Instead of lengthening and letting go, the body fights back, reflexively tightening against the applied force. Cuing active movement does help some, but when it doesn't, structural body work can be very painful for your client and frustrating for both of you. Even if you are both able to gut it out and achieve some lengthening of the fascia in this manner, you may be surprised and disappointed to see that, upon returning to standing upright in gravity or performing functional movements, your client's body still prefers to hold it in a shortened position or move around it instead of through it. By clearing this somatic dysfunction using indirect techniques before attempting direct release techniques, both you and your client will enjoy moving more freely toward the goal of integration.

Some somatic dysfunction is undoubtedly cleared on a regular basis without specific application of SCS techniques when we rest in comfortable positions or free dance. Stretching may also inadvertently clear somatic dysfunction in maximally shortened structures opposite those being stretched. You may even happen upon an indirect release if you intuitively follow a layer of fascia into a direction of ease and hold it there during bodywork. I would even go so far as to say that if you perform your direct myofascial manipulation strokes slowly enough, you may be turning off hypersensitive stretch reflexes in the slackened fascia ahead of your contact. Other systems of indirect visceral mobilization or myofascial unwinding touch on a similar phenomenon. However, none of them will be as thorough or efficient as intentionally performing properly sequenced indirect releases to specific structures while monitoring the associated tender points systematically in SCS.

SCS and SI

As a structural integrator, I incorporate SCS into my practice in a number of ways. With clients where somatic dysfunction is evident during their initial visit and widespread throughout the body, I will do six to eight 45-minute sessions of counterstrain as preparatory work prior to beginning the integration

series. This allows me to work with people who would not otherwise tolerate structural integration—those with acute conditions such as whiplash or systemic conditions such as fibromyalgia. With other clients where the somatic dysfunction is localized to a specific area, I may counterstrain a few relevant tender points during a specific session of the series to improve tolerance and effectiveness. With still other clients, somatic dysfunction becomes apparent only after they are able to lengthen up out of their previously compressed posture near the end of the series, and I will address it with counterstrain during the integration sessions.

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Resources

Strain and Counterstrain is easy to learn. The Jones Institute posts course schedules, and downloadable research articles, case studies, and other published papers on its website: www.jiscs.com.

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