Jones Institute Europe

Formation Lawrence H. Jones

Chapter 13.

Strain and Counterstrain

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Strain and Counterstrain

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Observing a skilled strain and counterstrain practitioner you are immediately impressed with how gentle and nontraumatic this technique is for the patient and the operator. How quickly they are able to assess the musculoskeletal system for the areas of dysfunction and the involvement of the patient in assisting to guide the operator to the final treatment position.

This innovative system for the treatment of somatic dysfunction was developed by Lawrence Jones, DO, FAAO. He defines strain and counterstrain as a "passive positional procedure that places the body in a position of greatest comfort, thereby relieving pain by reduction and arrest of inappropriate proprioceptor activity that maintains somatic dysfunction."

From the definition it is clear that the strain and counterstrain concept is not directed toward tissue injury or tissue damage but aberrant neuromuscular reflexes within that tissue. Specifically, the primary proprioceptive nerve endings are singled out as reporting false information to the central nervous system and maintaining somatic dysfunction.[1] The operator will affect this system by passively positioning the patient's dysfunctional segment toward comfort or ease and away from pain, bind, and restricted barriers. The position results in maximal shortening of the involved muscle and its proprioceptors and eventual reduction of neuromuscular firing to tonic levels. Strain and counterstrain is an indirect technique because its action is away from the restricted barrier.

ORIGIN

Jones was motivated to experiment with the concept of positional release in part from his frustration with the rationale of his time for the osteopathic lesion (which has since changed names to somatic dysfunction). He was schooled to believe that somehow joints became locked or subluxed and the only way to treat them was to burst them loose via high velocity thrust techniques. His results were generally good, but occasionally a case would enter his office that resisted all of his manipulative skills, until Jones states, "only stubbornness kept me from admitting I was stumped." He recounts that he was treating just such a case when he discovered positional release.[2,3]

A young man with psoasitis (stooped posture, unable to come completely erect with severe pain across the low lumbar area) had been treated by Jones using high velocity techniques for 6 weeks with no relief of symptoms. He had been treated previously by two chiropractors for 2-½ months with similar results. He complained of pain in bed and an inability to find a comfortable position that he could stay in for any longer than 15 minutes. So, Jones devoted one treatment session to finding a reasonably comfortable position for the patient to sleep in. After 20 minutes

of experimentation, a position of amazing comfort was found. Jones relates that: "He was nearly rolled into a ball with the pelvis rotated about 45 degrees and laterally flexed about 30 degrees." This was the first positive response the patient had had after 4 months of treatment, so Jones propped him in the position and went off to treat another patient. When he returned, he helped the patient upright and was astonished to find he could stand completely erect in total comfort. Examination revealed full and near pain-free range of motion. All Jones had done was put the patient in a position of comfort and the results were dramatic after his best efforts had repeatedly failed.

This was the inspiration that started Jones' experimenting with positional release and applying it to all somatic dysfunctions. During this developmental period he observed that the return to neutral done very slowly was important to the outcome of the positional release. If the patient was moved too quickly, especially in the first 15 degrees of motion, the benefit from the positioning was lost. Also, after initially supporting the patient in the position of release for 20 minutes, he was systematically able to reduce the period to 90 seconds. Anything less than 90 seconds and his results were inconsistent; but more than 90 seconds did not appear to increase the benefit to the patient.

The second feature to strain and counterstrain was the discovery of palpable myofascial tender points and their correlation to specific somatic dysfunction. Jones describes tender points as "small zones of tense, tender, edematous muscle and fascial tissue about a centimeter in diameter." These points, found by moderate palpatory pressure, are directly related to somatic dysfunction and with such consistency that they became his diagnostic tool. Tender points are four times more tender than normal tissue. Palpation with less than sufficient pressure to cause pain in normal tissue will elicit a sharp local pain characteristic of a strain and counterstrain tender point. Most of the tender points are found overlying the muscle involved in the dysfunc-

tion. Tender points found in the paravertebral musculature or over spinous processes are especially valuable for diagnosing segmental dysfunction in the vertebral column.

RESEARCH DATA

Prior studies have shown the efficacy of palpation on pressure-sensitive points in accurately diagnosing spinal dysfunction.[4–6] Quantitative studies done by Denslow and associates[7] showed how spinal dysfunction could be objectively confirmed with gauged pressure on the spinous processes and measurement of the motor reflex threshold.

Denslow[7] observed that when he pushed on a dysfunctional vertebral segment on either side of the spinous process or in the paravertebral area he would elicit local pain and a muscular contraction in the erector spinae group. From this clinical observation he designed a study to measure the amount of pressure it would take to elicit an initial muscle response from these points, which was called motor reflex threshold. Pressure was applied to the spinous processes by a self-designed pressure meter, and electromyographic electrodes were placed in the paravertebral musculature. The exact amount of pressure necessary to elicit a muscle response was measured. What he found was that at levels where he had made a palpatory diagnosis of vertebral dysfunction he was consistently able to correlate a lower motor reflex threshold. It took less pressure to elicit pain and a corresponding muscle contraction. Nondysfunctional segments responded with little or no pain and muscle contractions at the highest pressure settings.

Denslow correlated a second characteristic of joint dysfunction with a lower motor reflex threshold, this being differences in tissue texture. At sites of low threshold he describes palpable changes in tissue texture as "doughy and boggy." He used these terms to describe the tense, edematous feel of the tissue. Tissue texture changes and reflex muscle contraction with palpatory pressure were so consistent

that he was able to predict low motor threshold levels with 95% accuracy.

Another observation of Denslow's, using electromyograms, was that muscle completely at rest was characterized by an absence of action potentials. At low threshold segments, despite the apparent relaxation of the subject, he found "rest activity," action potentials from the paravertebral musculature even at rest. He states, "It was often necessary to position and reposition the shoulder girdle, upper extremity, head and at times the lower extremities in order to eliminate rest activity."

Denslow concluded that "low threshold segments are apparently hyper-excitable not only to pressure stimuli applied to the corresponding spinous process but also to impulses from proprioceptors associated with positioning."

The phenomenon of the elimination of EMG "rest activity" may be associated with strain and counterstrain. Denslow's study tested the thoracic levels 4, 6, 8, and 10. The strain and counterstrain treatments for posterior thoracic dysfunction at those segments would include passive positioning of the shoulder girdle, upper extremity, head, and lower extremities to find the position of release.
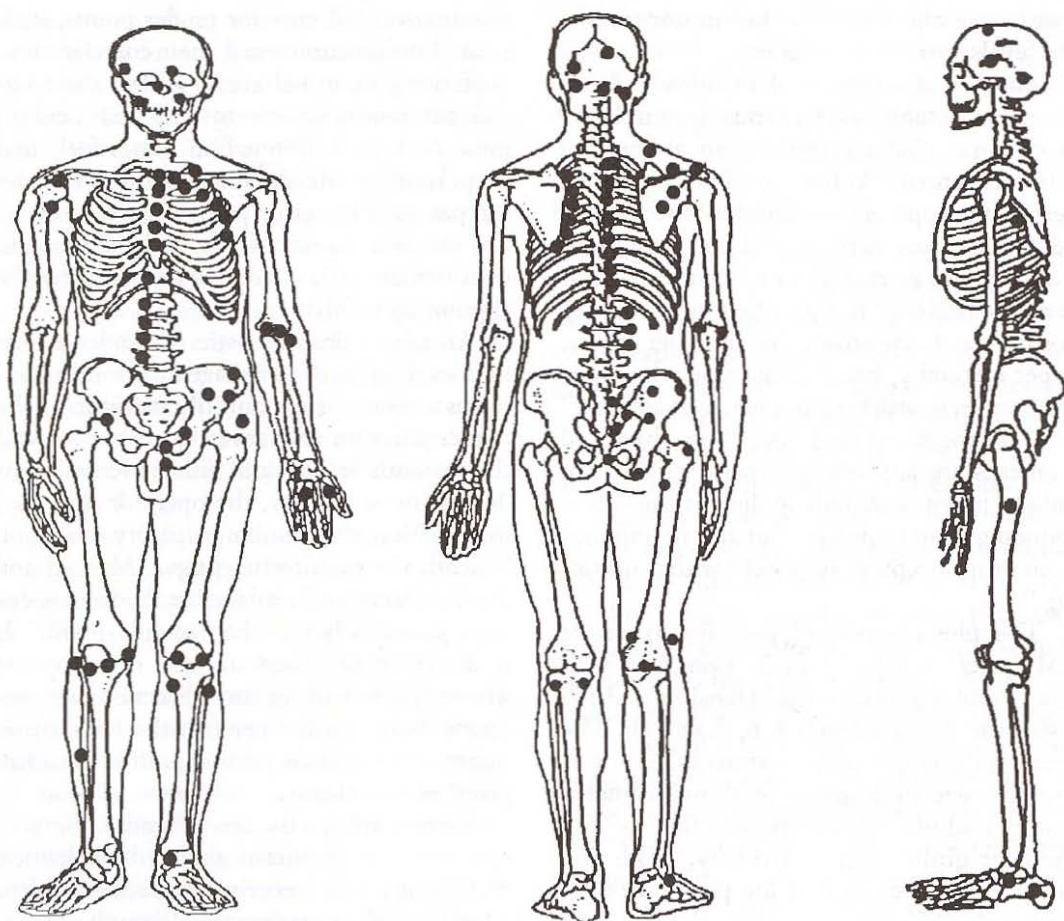
TENDER POINTS

Tender points are not only found over spinous processes or paravertebral musculature. Figure 13.1 shows the magnitude of the number of diagnostic tender points that Jones has mapped out over the entire body. This illustration represents just a small portion of the close to 200 tender points that Jones has correlated with specific dysfunction. Tender points in the posterior torso over spinous processes or paravertebral musculature are closely associated with the area and level of posterior pain complaint. Tender points in the anterior torso and pelvis are also closely associated with an area and level of posterior pain. Patients usually have no awareness of these anterior tender points until they are probed. Many osteopathic clinicians believe the discovery of anterior tender points, their related dysfunctions, and their correlation to posterior pain to be one of Jones's most significant contributions to the treatment of musculoskeletal dysfunction. Jones feels that 50 percent of the dysfunction that produces the patient's posterior pain is represented on the anterior aspect of the body. Failure to consider these dysfunctions may lead to disappointing results.

An added characteristic of tender points besides their value as a diagnostic tool is their use as a monitoring point. By monitoring the tender point for changes in tissue tension and the patient's feedback of either increasing or decreasing sensitivity, the operator is guided to a position of maximum palpatory relaxation beneath the monitoring finger. Marked and prompt decrease in subjective tenderness ensues. Jones calls this the "mobile point." It is the point of maximum ease or relaxation where movement in any direction will increase tissue tension beneath the monitoring finger. The mobile point signifies the ideal position for release.

Jones[2] explains the use of tender points in this way. "A physician skilled in palpation techniques will perceive tenseness and/or edema as well as tenderness, although the tenderness (often a few times greater than that of normal tissue) is for the beginner the most valuable diagnostic sign. He maintains his palpating finger over the tender point to monitor expected changes in tenderness. With the other hand he positions the patient into a posture of comfort and relaxation. He may proceed successfully just by questioning the patient as he probes intermittently while moving toward the position. If he is correct, the patient can report diminishing tenderness in the tender area. By intermittent deep palpation he monitors the tender point, seeking the ideal position at which there is at least two thirds reduction in tenderness." Finding the position of release in this way, holding this position for 90 seconds and returning to neutral very slowly are the major components of a strain and counterstrain technique.

A common question is the relationship of

Figure 13.1. Location of tender points. With permission, Jones LH. Strain and counterstrain. Newark, OH: American Academy of Osteopathy, 1981.

strain and counterstrain tender points to Travell's trigger points, Acupuncture points, Chapman reflex points, Shiatsu points, etc. There is, of course, a great deal of overlap in point locations and the palpatory feel of the tissue. However, there are two major differences. First, strain/counterstrain tender points tend to be more segmental in origin. Points along the vertebral column designate segmental dysfunction at the corresponding vertebral level. The other philosophies[8] identify points as related to full body systems and are more holistic in nature. Second, Jones feels strain and counterstrain tender points are a sensory manifestation of a neuromuscular or musculoskeletal dysfunction. The points are used to

make the diagnosis and to monitor the effectiveness of the treatment technique. Treatment is not directed at the tender point but at the dysfunction that produces the tender point. If the treatment is effective the tender point diminishes in tenderness, tissue tension, and edema. In the other philosophies the treatment is directed toward the painful point, by injection, needling, deep pressure, electrical stimulation, and vapocoolants.

RATIONALE

The rationale for strain and counterstrain is based on a neurologic model first proposed by Dr. Irvin Korr in 1975.[6,9] His hypothesis

incriminated the muscle spindle or primary proprioceptive nerve endings as the basis for joint dysfunction. His concept is derived from: a) the consensus on the importance of decreased joint mobility or decreased joint range of motion for determining somatic dysfunction, and b) on the muscle's function as a "brake" to retard or resist joint motion. Korr explains, "While usually thinking of muscles as the motors of the body, producing motion by their contraction, it is important to remember that the same contractile forces are also utilized to oppose motion. By the application of controlled counteracting forces, contracting muscle absorbs momentum (for example, of a swinging limb) and regulates, resists, retards and arrests motion." He expanded on his observation of "ease" and "bind," the behavior of a dysfunctional joint to move freely and painlessly in certain planes of motion and the painful resistance to motion in the opposite direction. Korr reasoned that impairment of joint motion in distinct planes was produced by a unilateral active contraction of muscles pulling the joint in a certain direction. Contraction of these muscles around the joint would resist (bind) motion in directions that would tend to lengthen or stretch the muscles and surrender (ease) to motion in directions that would shorten or approximate the muscles.

Korr's premise is that high gamma discharge exaggerates afferent firing from the muscle spindle producing a reflex muscle spasm which fixates the joint in a certain direction and resists any attempts to return to neutral. At this point a review of the structure and function of the muscle spindle[11,12] is in order to establish a common understanding (Fig. 13.2).

MUSCLE SPINDLE

Muscle spindles are highly specialized sensory receptors which are scattered throughout the extrafusal fibers of muscle. Muscle spindle density will vary with function. Phasic muscles have more muscle spindles than postural muscles due to the precision of control required. Each spindle is fluid-filled and contained in a connective tissue sheath about 3–5 mm long, enclosing 5–12 thin specialized muscle fibers known as intrafusal fibers. They lie in parallel to the extrafusal fibers and are attached to them at each end. There are two types of intrafusal fibers: larger fibers with centrally located nuclei aggregated into a bag-like pouch called a nuclear bag fiber, and smaller fibers containing only a single row of nuclei in their central portion called a nuclear chain fiber. These fibers can be thought of as having three regions, a central or equatorial portion where the nuclei are concentrated, and the
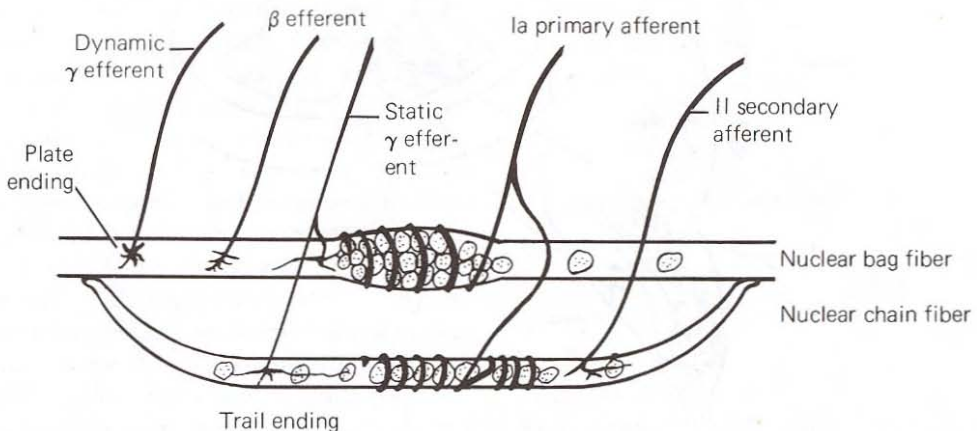
Figure 13.2. Muscle spindle. With permission, Ganong WF. Review of medical physiology. 9th ed. Los Altos, CA: Lange Medical Publications, 1979.

two polar ends which contain the contractile material. In the equatorial portion lie the primary afferent nerve endings also called the annulospiral endings which coil around the nuclear regions. Secondary or flower spray afferent nerve endings terminate on either side of the primary endings closer to the contractile polar ends.

Innervating the intrafusal fibers are gamma motor neurons whose cells originate in the ventral horn, pass through the ventral root, and terminate on the contractile polar ends. In contrast to the alpha motor neurons innervating the extrafusal fibers, these neurons are small and their axons thin.

The muscle spindle is sensitive to length changes. When the extrafusal fibers are stretched the muscle spindle is stretched, causing the annulospiral and flower spray nerve endings to fire. These fibers end monosynaptically directly at the motor neurons of the muscle containing the excited spindles. The excitatory effect produces a reflex contraction of the extrafusal muscle fibers, resisting the stretch. This is the familiar stretch reflex.

The frequency of firing of the annulospiral

and flower spray nerve endings is in direct proportion to the change in length. The annulospiral nerve ending has the additional characteristic that its frequency of firing is in proportion to changes in the rate of stretching. Therefore, the annulospiral nerve ending measures length plus velocity of the stretch and the flower spray nerve ending measures only length. Though the effect of these nerve endings is excitatory on the motor neuron of the involved agonist muscle, accessory impulses are transmitted to adjacent interneurons which form an inhibitory pathway to the motor neurons of the antagonist muscle. This is called reciprocal inhibition (see Fig. 13.3).

Gamma efferent stimulation of the intrafusal fibers will also stimulate afferent spindle firing. Impulses transmitted through the gamma efferent neurons will evoke contraction of the polar ends of the intrafusal fibers. Contraction of the polar ends stretches the nuclear portion stimulating the annulospiral and flower spray nerve endings to fire. The response is equal to that produced by stretching the extrafusal fibers. By controlling the contraction of the intrafusal fibers through gamma stimulation the central nervous system
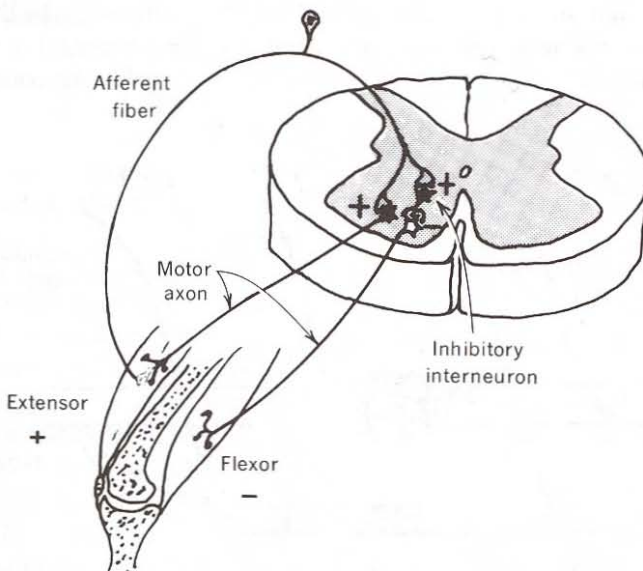
Figure 13.3. Stretch reflex and reciprocal inhibition. Astrand P-O, Rodahl K. Textbook of work physiology. New York: McGraw-Hill, 1970.

is able to set and reset muscle length, muscle tone, and muscle spindles' sensitivity to stretch. This mechanism provides for a state of preparedness for the muscle to respond to slight changes in length. Thus the higher the gamma stimulation the greater the spindle sensitivity to stretch. Stretch of the muscle with high gamma stimulation produces a more intense spindle discharge and therefore a stronger reflex muscle contraction (see Fig. 13.4).

Approximation of the muscle by active contraction or passive shortening will decrease spindle discharge proportionately, and with maximal shortening may even silence it. With high gamma stimulation the muscle must shorten even more to approach the same proportionate reduction in spindle discharge.

MUSCLE SPINDLE AND SOMATIC DYSFUNCTION

Discussion of the role of the muscle spindle in somatic dysfunction would be best initiated with a definition of somatic dysfunction. The current, accepted definition is: impaired or altered function of the related components of the somatic (body framework) system, skeletal, arthroidial, and myofascial structures; and related vascular, lymphatic and neural elements. It is widely accepted that somatic dysfunction involves alterations in systems other than the musculoskeletal. Sympathic involvement in somatic dysfunction is well documented but poorly understood and not within the scope of relevance for this topic. The somatic dysfunctions considered here are primarily produced during mechanical trauma.

The components of somatic dysfunction important to a strain and counterstrain diagnosis would be: first, tissue texture changes described as tense, ropey, and boggy. This is most commonly represented as muscle hypertonicity and tissue edema involving a muscle or muscles investing a particular joint; second, specific tender points which, when palpated, elicit exquisite local pain. Each point indicates a specific somatic dysfunction; and third, impairment in the amplitude and quality of joint
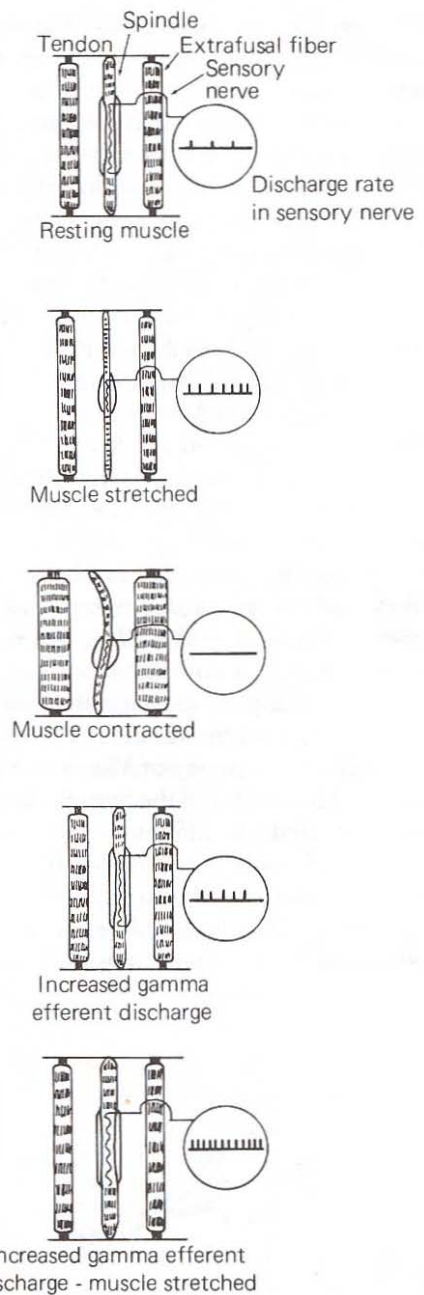
Figure 13.4. Effect of various conditions on muscle spindle discharge. Adapted from Ganong WF. Review of medical physiology. 9th ed. Los Altos, CA: Lange Medical Publications, 1979.

range of motion. Jones[2] states, "It is well known that for each painful joint there is a specific direction of position that greatly aggravates the pain and stiffness. Movement of

the joint in this direction results in immediate reflex and voluntary muscle resistance, to the point of rigidity. The converse is also true; for each painful joint there is a specific direction of position that greatly relieves pain and muscle tension. Movement of the joint in this direction results in immediate and progressive reflex and voluntary muscle relaxation, to the point of complete relaxation and comfort."

Muscle spindle involvement in somatic dysfunction will be described using an illustration (see Fig. 13.5). This is a sequence of a generic joint. It has a Muscle A and a Muscle B. Below is represented the firing frequency of the annulospiral nerve endings. Plate 1 depicts a joint in neutral. Muscles A and B are in balance and the annulospiral firing frequencies are equal, indicating a tonic rest condition of the muscles. Plate 2 depicts a joint in "strain." Muscle A is severely overstretched and Muscle B is maximally shortened. The annulospiral firing frequency is increased because of the stretch on Muscle A and its spindle. The firing frequency of Muscle B is practically nil. Shortening of the muscle slacks the spindle and reduces afferent firing and the stretch on Muscle A reciprocally inhibits Muscle B. Now, if the body reacts to this strain position in a slow and deliberate manner to return to the neutral position then the

stretched Muscle A is eased back to resting length with no pain and afferent firing returns to tonic levels. What occurred was an overstretching and nothing more. But if the body reacts to this strain position with a quick, sudden, or forceful movement, a panic reaction, to restore the joint to neutral then Muscle B and its spindle are quickly stretched.

Now, since the responsibility of the spindle in Muscle B is to detect the rapid rate of change of the extrafusal fiber lengths and the frequency of firing of the annulospiral nerve ending is in direct proportion to that rate of change, the spindle in Muscle B begins to report a stretch to the CNS even before the muscle reaches its normal resting length. This results in a sharp reflex muscle spasm, not in Muscle A, the overstretched muscle, but in Muscle B, the hypershortened muscle. Plate 3 depicts a joint in dysfunction. Muscle B in spasm fixates the joint in a certain direction and resists any attempts to lengthen and return the joint to neutral. The annulospiral firing frequency of Muscle B is immensely increased, reporting to the CNS a continuing message of strain which maintains the muscle in spasm. Korr[6] explains, "Under the influence of gravitational forces, antagonists and postural reflexes, which would be tending to stretch the muscle back toward resting length,
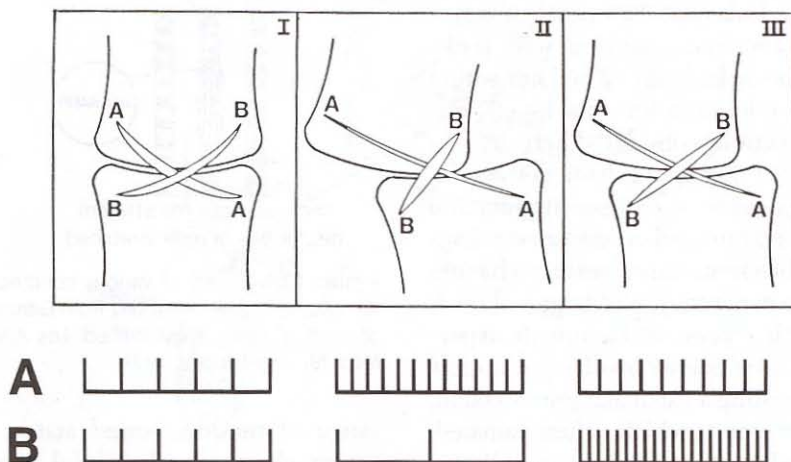

Figure 13.5. Somatic dysfunction at a joint.

the spindle would be continually discharging and through the CNS ordering the muscle to resist. The more the stretch, the much more the resistance."

It comes to mind that if Muscle B is spasmed and its attachments approximated this would shorten and slack the spindle, reduce the afferent discharge to the CNS, and relieve the spasm. Korr postulates that in the position of strain (with Muscle B maximally shortened and its afferent firing practically nil) the CNS, receiving no information from Muscle B, would greatly increase gamma neuron discharge to the intrafusal fibers until the spindle resumes reporting. This is what osteopaths refer to as "high gamma gain." With "high gamma gain" you increase spindle sensitivity to stretch. Now, with a panic reaction stretch to the hypershortened Muscle B the resultant spasm is of such an intensity that the body is unable to reduce it on its own. Korr[6] states, "The higher the gamma activity because of its influence on the excitatory spindle discharge, the more forceful the muscle contraction and the greater its resistance to being lengthened. During high gamma activity the spindle may, in effect, be calling for a contraction when the muscle is already shorter than its resting length."

Therefore, somatic dysfunction occurs not because of strain, but because of the body's reaction to strain. If the reaction is slow and deliberate somatic dysfunction is avoided. If the reaction is panic-like, the velocity of movement sets off the reflex muscle spasm producing dysfunction.

Patient accounts of their mechanism of injury bear this out. The person who is bending forward or squatting and experiences an excessive strain forward will react with a strong backward movement toward neutral. The patient will describe pain not in the strain position but with the return to neutral. The person involved in a minor motor vehicle accident is rear-ended at a speed which would not appear to cause tissue injury, but the person's cervical spine in maximal flexion underwent a quick extension. The complaint of

posterior cervical pain is aggravated with extension movement, and muscle guarding would be noted. Flexion movement is pain-free and relaxing. Examination reveals numerous anterior cervical tender points and related anterior cervical joint dysfunction.

PURPOSE OF STRAIN AND COUNTERSTRAIN

What strain and counterstrain attempts to accomplish with its position of comfort is to relax the muscle spasm by reducing aberrant afferent flow from the muscle spindle. This is accomplished by mimicking the original strain position or applying a "counterstrain." By passively mimicking the original strain position the operator moves the joint in a direction of ease and maximally shortens the involved muscle. Holding for 90 seconds allows the spindle to slow down its afferent firing frequency. Returning to neutral in a slow and deliberate manner avoids reexciting the previously spasmed muscle. Korr[6] explains, "The shortened spindle nevertheless continues to fire, despite the slackening of the main muscle, and the CNS is gradually enabled to turn down the gamma discharge, and, in turn, enables the muscle to return to 'easy neutral' at its resting length. In effect, the physician has led the patient through a repetition of the lesioning process with, however, two essential differences: first, it is done in slow motion with gentle muscular forces, and second, there has been no 'surprise' for the CNS; the spindle has continued to report throughout."

CASE STUDY

Most of the knowledge about the nature of somatic dysfunction and what strain and counterstrain accomplishes with its position of comfort is based on patient accounts of their mechanisms of injury, their response to positional treatment, and the observations of skilled practitioners. Neurophysiologic studies in this area are woefully limited. Therefore, the presentation of a case study seems

an appropriate way to lend support and give the reader insight into the rationale.

This case, Jones's favorite, involves a middle-aged man who had a habit of falling asleep supine on the sofa. While asleep his right arm would occasionally fall off the edge and hang in marked extension at the elbow. For years, his wife, noticing her husband napping in this position, would slowly and gently replace the arm across his chest without awakening him (a slow return to neutral). And for years the man would awaken from his nap with no complaint of discomfort. One day, while his wife was out, he was awakened abruptly by the ring of a telephone near his head, while his elbow was in marked extension. He was so startled that he violently jerked his right elbow into flexion. He immediately began to feel pain in the right bicep especially with movement into flexion. A diagnosis of bicep strain was made for his condition on the basis of painful elbow flexion, even though palpation revealed no clinical evidence of strained or injured tissue. By the time he saw Jones he had been disabled for 2 years with pain and progressive weakness of the biceps. Examination of the biceps failed to reveal any information as to the nature of the problem. However, palpation of the distal triceps uncovered exquisitely sharp tender points (evidence of triceps dysfunction). The triceps had been maximally shortened then suddenly lengthened with a panic response.

Treatment consisted of positioning the elbow in hyperextension so that the triceps was maximally shortened (mimicking the original strain position), holding the position for 90 seconds while monitoring the tender points, and slowly returning to the neutral position. After three treatments full and pain-free function was restored.

This case demonstrates: first, how important the slow return from a strain position is in avoiding joint dysfunction; second, that the palpable evidence of dysfunction is frequently found on the opposite side of pain in the antagonist of the overstretched muscle; and third, how treatment techniques mimic the original strain position. The apparent weakness in the biceps was attributed to disuse and reciprocal inhibition due to the continuous contraction of the triceps.

STRAIN AND COUNTERSTRAIN IN THE MANUAL MEDICINE ARMAMENTARIUM

Strain and counterstrain can be used as a sole treatment modality or as an adjunct to other manual medicine techniques. Its therapeutic uses range from the very acute to the chronic patient.

Its value with the acute patient is unmatched because it is so gentle and nontraumatic. The operator is guided by what feels good to the patient, and often dramatic changes are made in subjective pain, muscle guarding, and edema.

The gentleness of strain and counterstrain makes it safe and effective for treating somatic dysfunction on fragile patients (i.e., elderly, osteoporotic, fractures, pregnancy) and infants.

Strain and counterstrain is valuable with chronic patients for two reasons, first, a scan for tender points provides a quick assessment of the problem areas of the body and allows the operator to delineate the areas of dysfunction contributing to the pain complaint, and second, the treatment will reduce the aberrant flow of afferent impulse in the involved muscles which have maintained the joint in chronic dysfunction.

The approach with strain and counterstrain is to passively put a slight strain into a dysfunctional joint. Patients with severely limited range of motion (adhesive capsulitis, cervical spondylosis) find strain and counterstrain helpful to reduce secondary muscle guarding. Positions of comfort are easily found, but within the available range which will be in lesser degrees of motion than patients with full range of motion. Measurable gains in range and quality of motion can be made.

Pain associated with hypermobility can also be treated. The approach is still to put a strain upon the joint; therefore hypermobile

patients are usually treated in greater degrees of motion than patients with normal range.

Strain and counterstrain can make a significant contribution when integrated with other manual medicine techniques. Used in conjunction with articular techniques (i.e., joint mobilization, high velocity manipulation) which restore position and motion, it will normalize the imbalance of muscle tension affecting the joint so that recurrence of dysfunction is decreased.

Strain and counterstrain and muscle energy can be combined with effective results.[9,10] Isometric muscle energy's inhibitory effect on contracted muscle by increasing Golgi tendon organ discharge or through reciprocal inhibition can enhance strain and counterstrain's inhibitory effect on the same muscle. Muscle energy can also be valuable to strengthen the antagonist muscle (weakened by reciprocal inhibition) to bring the joint back to postural balance.

Strain and counterstrain can be used before myofascial release techniques. By clearing corresponding tender points, counterstrain can assist in reducing neurophysiologic barriers, allowing myofascial release to break down biomechanical barriers with greater ease.

CONCLUSION

Strain and counterstrain is an indirect manipulative technique of extreme gentleness for the treatment of somatic dysfunctions. It is based on a neurologic model that proposes, for some, a new concept for the production of somatic dysfunction. The hypothesis is aberrant afferent flow from the muscle spindle produces a reflex muscle spasm that fixates a joint in a certain direction and resists any attempts to return the joint to neutral. Diagnosis is made by the presence of a specific tender point that overlies the muscle. Using the tender point as a monitor the operator is guided into a position of comfort that reduces aberrant afferent flow and returns the muscle to "easy neutral." Holding the position of comfort for 90 seconds and returning to neutral slowly following the positional release

are two very important aspects of this procedure.

Though research data are limited in this area to support the model, the observations of practitioners, recounting the immediate changes in palpable pain, tissue tension, and ease of movement following positional release, point to a neural basis.

Recognition must be given to Lawrence Jones for decades of arduous experimentation on patients and his own body to develop strain and counterstrain. His book, *Strain and Counterstrain*,[2] has mapped out hundreds of the most common tender points and positions for treatment. To the beginner, the treatments appear straightforward and easily mastered, but development of the palpatory skills required to find the optimal position of release takes practice and perseverance. A complete study of the book and the teachings of Lawrence Jones are highly recommended.

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