

Nociceptive considerations in treating with counterstrain

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The proprioceptive mechanistic model of somatic dysfunction proposed by Korr is accepted as the neurophysiologic basis of counterstrain by the developer of that manipulative technique. We suggest that the physician should also take into account the physical damage, if any, that the original trauma produced. We propose that with tissue injury, nociceptive reflexes could produce patterns of motion restriction opposite that predicted by a solely proprioceptive model. A nociceptive component is suggested as an explanation for the origin and maintenance of somatic dysfunction and its response to the counterstrain technique. In actuality, both proprioceptive and nociceptive responses may occur in dysfunctional states. Other physiologic responses also may be involved. These views are consistent with clinical experience.

(Key words: Counterstrain, osteopathic manipulative treatment, nociceptive reflexes, somatic dysfunction.)

Counterstrain is a passive, positional-release manipulative technique, developed by Lawrence Jones.^{1,2} Counterstrain techniques place somatic systemic elements into the position of greatest perceived ease, or comfort, by passively approximating the attached surfaces of those elements. Osteopathic physicians have demonstrated time and again that counterstrain is a clinically valid and useful entity. However, the theoretical physiologic mecha-

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nism by which counterstrain techniques ameliorate somatic dysfunction has not been explained conclusively. (Somatic dysfunction is defined³ as "Impaired or altered function of related components of the somatic [body framework] system: skeletal, arthrodiagonal, and myofascial structures, and related vascular, lymphatic, and neural elements." Korr^{4,5} has described a proprioceptive mechanism of somatic dysfunction that Jones² accepts as underlying counterstrain. This explanation is compatible with personal clinical impressions of changes observed during counterstrain treatment.

However, it seems unlikely that proprioceptive reflexes represent the sole element involved in how counterstrain works, a position with which Korr agrees (conversation, May 1989). Advances in physiologic knowledge of the proprioceptive functions have not changed Korr's concepts. These advances, in fact, have clarified the theoretical basis for somatic dysfunction and explanations for response to manipulative treatments. In this paper, we (1) suggest an updated theoretical basis for somatic dysfunction involving nociceptive stimuli, and (2) examine the responses to counterstrain treatment. Our proposed theoretical role of nociceptive mechanisms in initiating or maintaining somatic dysfunction (or both) and proposed basis for effects of counterstrain treatment extend the concepts related to proprioceptive functions.

Basic reflexes

First, let us apply Korr's proprioceptive model to a simple hinge-type joint, the elbow. To facilitate the discussion, let us concern ourselves with only two antagonistic muscles that act across the elbow joint: the lateral head of the triceps brachii muscle, an extensor of the forearm, and the brachialis muscle, a forearm flexor (*Figure 1*). Now let us assume that while the elbow is semiflexed, a weight is placed in

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the hand. To lift the load, the brachialis muscle shortens (contracts), and the triceps muscle is concurrently stretched. Both brachialis and triceps muscles contain specialized encapsulated receptors called muscle spindles⁶ that are responsive to stretch. When the muscle is stretched, the spindles are activated. The innervated muscle is reflexively induced to contract, and the contraction is accompanied by the reciprocal inhibition of its functional antagonists.⁷ This simple reflex arc is the familiar myotactic or stretch reflex. When the muscle shortens, the spindles are "unloaded" and, as a result, their responses are quieted or even silenced.⁸ Thus, in our example, the spindles in the triceps muscle increase their response rates as the muscle is stretched. Similarly, the spindles in the brachialis muscle decrease their rate of firing momentarily as the muscle shortens.

In simple terms, we have now defined the primary muscle-spindle afferent response. Muscle spindles, however, are complex receptors, and the primary afferent response is only one constituent of an entire informational spectrum. In addition to primary spindle information, there is another component provided by their gamma-efferents.⁹ The functioning of the gamma-efferent system and its interactions with the spindle primaries is both subtle and complex. For the purpose of this discussion, it is sufficient to state that the gamma-efferents augment and fine-tune the activity of the primary afferent system.¹⁰ In our example, the primary afferent spindle response of the brachialis muscle is quieted, so that the central nervous system is provided with little information regarding the degree of brachialis stretch.

The gamma efferent system subserving the brachialis muscle would "turn up" the sensitivity of its primary afferents and restore some level of spindle activity and information inflow to the central nervous system. The primary spindle afferent stretch reflex functions largely at a segmental level.¹¹ This means that the specific segment of the spinal cord responsible for the innervation of the muscle is also responsible for its myotactic reflexes. In contrast to the primary afferents, the gamma system is controlled from suprasegmental levels

of the central nervous system, that is, from the brain rather than the spinal cord.¹² Collectively, the muscle spindles provide proprioceptive information, and the reflexes they mediate are called *proprioceptive reflexes*.

Proprioceptive reflexes and somatic dysfunction

What does this elementary neurophysiology have to do with somatic dysfunction and, more specifically, its treatment by counterstrain? To answer this question, let us return to our previous example in which a weight was placed on the semiflexed upper extremity (*Figure 2A*). Furthermore, assume that the weight is applied suddenly. The upper extremity is abruptly "loaded," and the forearm is forced immediately toward extension. The brachialis muscle is suddenly stretched (the *strain* part of Jones' original strain/counterstrain), and the triceps muscle is shortened (*Figure 2B*).

Subsequent reflexive reactions establish and maintain the somatic dysfunction. Proprioceptive reflexes come into play (*Figure 2C*), and the brachialis muscle is contracted, such contraction applying physiologic "brakes" to the runaway movement of the forearm. According to Korr's^{4,5} explanation, this sudden shortening and concurrent silencing of the spindles in the triceps muscle (*Figure 2E*) causes both the gamma "gain" in the triceps muscle to be reflexively turned up, and the muscle itself to be reflexively contracted (the *counterstrain* of strain/counterstrain) (*Figure 2F*). The central nervous system regains its all-important spindle information but at the expense of a shortened triceps muscle. This muscle now reports to the central nervous system that it is being stretched, even before it attains its neutral length. On recovery from the sudden extension movement, flexion is resisted by a triceps muscle that is now tonically shortened by its inappropriately high gamma gain.

Clinical findings associated with this example include the forearm's moving easily into extension (the direction of ease), but resistance to movement into flexion (the direction of bind) and pain. Thus, normal symmetry of motion about the elbow joint is disrupted. There are tender points, or painful foci, located on the

posterior aspect of the upper arm/forearm, with associated changes in tissue texture. Jones² pointed out that the pathologic alteration in this type of somatic dysfunction involves tissues with an essentially atraumatic history; for example, the only external events to which the triceps muscle (the counterstrained element) was exposed was that it was suddenly allowed to shorten. Only the brachialis muscle (the strained element) was suddenly stretched.

The proprioceptive model of somatic dysfunction as applied to our example may be summarized as follows: The forearm is suddenly moved from the resting position (*Figure 2A*) into extension; the brachialis muscle is stretched (loaded) and its spindles increase their firing rate (*Figure 2B* and *2E*), that is, the brachialis muscle is *strained*. The triceps muscle is shortened (unloaded) and its spindles decrease their firing rate (*Figure 2B* and *2E*).

Bereft of triceps spindle information, the central nervous system turns up the gain of the triceps gamma system; the triceps muscle, reflexively contracts (*counterstrain*), and increases its rate of spindle firing (*Figure 2C* and *2F*). The triceps muscle now reports a "neutral" position even though the forearm is positioned toward extension.

The brachialis muscle responds to being stretched by reflexively contracting; because the triceps muscle is now shortened; flexion is limited and the somatic dysfunction is established. Such dysfunction is maintained by the continued increased gamma gain in the triceps muscle so that the forearm's neutral position is reset toward extension; normal symmetry of forearm motion is compromised.

Counterstrain treatment

Counterstrain treatment of the just described dysfunction would be accomplished by passively placing the forearm into a position of extension, thereby approximating the ends of the tonically contracted triceps muscle. This procedure recreates the direction of the original injury. With the tension removed from the counterstrained muscle (triceps), its spindles are unloaded and slow their firing. Released

from the influence of the primary afferent spindle stimuli, the inappropriately exaggerated gamma gain can be reset by the central nervous system. The forearm would be held by the physician in this position for a short time (typically, about 1 minute). Accompanying the nervous system-mediated release are palpable tissue texture changes and a resolution of the tender points. The upper extremity is then *slowly* returned to a more neutral semiflexed position. All movements of the forearm must be accomplished by the physician without active assistance from the patient.¹³ On reexamination, a pain-free, symmetric range of forearm motion is expected.

Painful comparisons

Now that we have reviewed the mechanisms that are proposed by Korr^{4,5} to underlie somatic dysfunction and discussed their application to counterstrain, let us add another dimension to our previous example. Assume now that the weight is applied to the forearm with sufficient intensity and abruptness to injure the brachialis muscle and to cause pain. Except for this addition, all other conditions are identical: same weight, same forearm. Will the predicted results, such as ease of motion and localization of pain, be the same? To fully answer these questions, we must first consider the outcomes of activating pain-mediated (nociceptive) reflexes.

Nociceptive reflexes are powerful and capable of overriding voluntary behavior.¹⁴ Even by conscious design, it is nearly impossible to abrogate nociceptive reflexes. When we step on a sharp object, it is exceedingly difficult to avoid withdrawal of the foot and possibly stumbling, even if circumstances, such as carrying a dozen eggs at the time, forbid.

Flexor withdrawal is a multisegmental, nociceptive reflex that moves the affected body region away from a noxious stimulus. In our example, the noxious stimulus is insult to the brachialis muscle and its associated tissues, resulting in either frank tears or less severe stress, both of which result in the initiation of nociceptive responses. The nociceptive reflex then takes the form of contraction of the brachialis muscle, drawing the forearm into

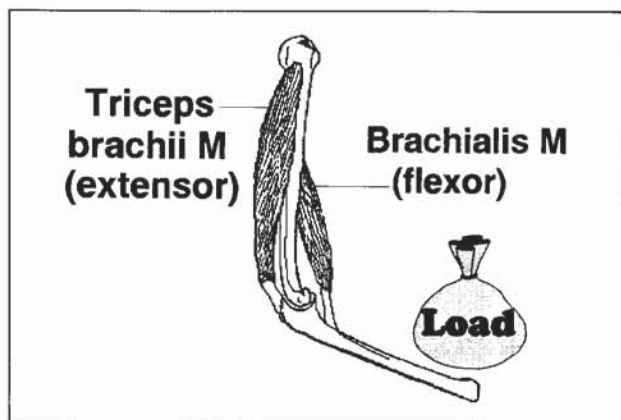


Figure 1. Lateral head of triceps brachii muscle and brachialis muscle with bony attachments on upper extremity.

flexion,¹⁵ guarding and protecting the injured extremity.

Note that application of the same weight applied across the same joint produces physical findings opposite to those described in the proprioceptive model. The damaged brachialis muscle is held in a state of contraction. Ease of motion is now toward *flexion*, not extension as before. The anterior aspect of the extremity is tender and painful. What happened to the proprioceptive reflexes that acted in our first example? We propose that the proprioceptive reflexes are still present and probably functional, but are being masked by the more potent nociceptive reflex. It is possible that the nociceptive reflex represents a predominately acute phase of the injury, whereas the proprioceptive mechanism functions in more chronic aspects. This could certainly be the case if the damage to the brachialis muscle were minimal and the noxious stimuli were resolved quickly. In that circumstance, we expect the proprioceptive reflex to be unmasked on resolution of the noxious input. However, given the more potent nature of the nociceptive reflex, if the damage to the brachialis muscle were severe, we expect tonic brachialis contraction to be both the acute and the chronic effect, restricting the range of motion about the elbow joint for a potentially extended period. If the somatic dysfunction is not resolved, there is evidence that the connective tissues in the upper extremity will reorganize. Fibrocytes will undergo proliferation, but with their

fibers oriented in random directions, so that normal joint motion is even more restricted.¹⁶

The following summarizes the nociceptive component of somatic dysfunction as applied to our example: The forearm is moved into extension with sufficient force to cause some degree of trauma to the brachialis muscle or its surrounding tissues (or both). Pain receptors are activated both directly by the tissue damage and by subsequently released tissue factors. The brachialis muscle is reflexively induced to contract, so that the forearm is pulled into flexion. As long as the noxious stimulus is present, the brachialis muscle will continue to contract. Symmetry of motion is disrupted.

It is possible that both proprioceptive and nociceptive reflexes function in synergistic fashion. Recall that in the original example of proprioceptive reflex functioning, the direction of restricted motion was toward flexion, because of a tonically contracted triceps muscle. Also recall that there were painful tender points associated with the posterior aspect of the arm/forearm. Given this scenario, it is consistent with a nociceptive model that the triceps muscle, which is anatomically associated with the area of painful stimulation, tends to contract.¹⁷ Local tissue conditions such as ischemia, circulatory stasis, and edema associated with the somatic dysfunction¹⁸ maintain nociceptive

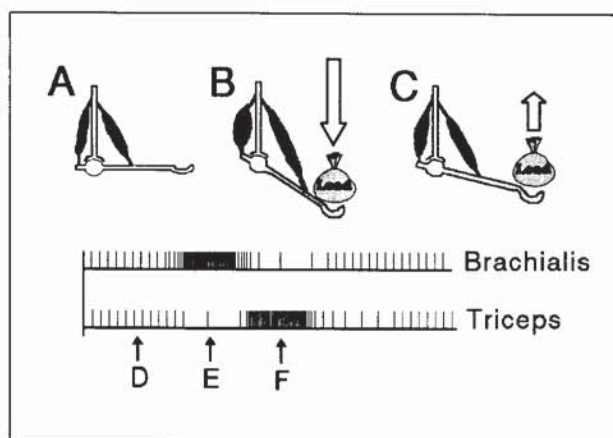


Figure 2. Position of upper extremity (A,B,C) and the primary muscle spindle activities (D,E,F) of brachialis and triceps muscles during a sudden extension of forearm. A and D, resting conditions. B and E, immediate effects of sudden extension movement. C and F, proprioceptive reflexive response.

stimulation, and further aggravate the dysfunctional state. Similarly, in the second example in which the injured brachialis muscle is the source of the noxious stimulus, the painful muscle contracts, so that the forearm tends to be drawn into flexion.

The previous example of the injured brachialis muscle provides a simple example of proprioceptive or nociceptive mechanisms (or both) creating and maintaining a pathologic functional state. Let us now examine a more complex physical insult. Whiplash is a relatively common injury characterized by sudden hyperextension of the cervical spine. It is clear that the consequences of such an injury would be profound and widespread. However, for the purposes of our discussion let us focus our attention on two functional groups that act as agonist/antagonist pairs in the cervical spine. The anterior group (flexors) includes the scalene, suprahyoid, infrahyoid, and prevertebral muscles. The posterior group (extensors) consists predominantly of the erector muscles of the spine, but includes the transversospinal, suboccipital, splenius, and trapezius muscles.

On physical examination of the whiplash patient, clinical findings may include edema, tenderness, and increased muscle tension in the anterior tissues. The posterior tissues typically have multiple cervical tender points, increased muscle tension, and ease of motion toward extension. In the initial impact, the patient's cervical spine is thrown violently into sudden hyperextension. The tissues of the cervical *flexor* region are rapidly stretched, while those of the cervical *extensor* region are suddenly shortened. Proprioceptive mechanisms are activated in the extensor muscles so as to result in their reflexive contraction. The findings in the anterior cervical region, which indicate tissue damage, are more easily attributed to nociceptive mechanisms. The direct traumatization of these tissues during the initial impact initiates local tissue inflammatory responses (accompanied by initiation of nociceptive stimuli) and sets the stage for muscular guarding. Clinically, the result is a patient with a stiff, painful neck.

This example exposes the difficulties of imposing simplistic explanations for the complex

phenomenon of human injury. Both proprioceptive and nociceptive mechanisms find expression in our whiplash example. Local tissue injury can play a role in both the genesis and the maintenance of somatic manifestations.¹⁹ The intimate association of the cervical portions of the sympathetic chains and ganglia with the affected anterior tissues should be considered. Similarly, the affected posterior tissues are associated with the suboccipital and occipital nerves and the vertebral artery. It seems likely that these important structures will bring their own influences into the clinical presentation. Which effects predominate is largely dictated by the individual circumstances of the injury and the individual patient.

Further complicating the whiplash example is the fact that whiplash is usually not solely a hyperextension injury. More commonly, the cervical spine is thrown violently first into hyperextension and then, with almost equal force, into extreme flexion. In effect, each forward/backward movement of the head creates its own "layer" of somatic dysfunction. The question of which tissues were "strained" and which were "counterstrained" becomes moot, as most of the tissues in the cervical region receive varying degrees of damage from such an event.

To treat whiplash with counterstrain technique, the physician must decide which component is treated first. This decision is usually made by determining the direction of greatest ease of passive cervical motion, as well as assessing the quantity and quality of tender points in the associated tissues. The head would be positioned in such a way as to allow those most painful regions to relax, effectively treating at least the most acute and noxious "layer" of the whiplash injury. Subsequent treatments would be necessary to address the other "layers."

If we assume that at least some of the pathologic alteration has a nociceptive component, how would we expect counterstrain treatment to help? Empirically, we find that somatic dysfunction resulting from painful tissue damage responds quite well to counterstrain treatment. Possibly counterstrain treat-

ment resolves underlying proprioceptive elements of the somatic dysfunction and allows more effective healing of the tissue damage component. Counterstrain techniques appear to restore physiologic motion by affecting the origins of somatic dysfunction, allowing pathologically perpetuated reflexes to normalize and the symptom complex to resolve. Clinically, counterstrain treatment is appropriate for both the simple and the complex patient presentation, irrespective of the physiologic model being applied.

Comment

Probably very few dysfunctional states result from either a purely proprioceptive, or nociceptive response. Both are likely to occur simultaneously. Additional factors such as autonomic responses, other reflexive activities, joint receptor responses, or emotional states must also be accounted for. What predominates is largely a matter of degree, determined by conditions both external and intrinsic to the injured tissues.

The symptomatic patient represents the accumulated total of an intricate variety of physiologic responses. Our understanding of such complexity begins at the level of the simplistic neurophysiologic analysis proposed here. It must progress with the incorporation of such concepts into the awareness that our patients are more than a complex collection of reflexes. Human beings represent a gestalt, that is, more than the sum of the parts, and as such cannot be fully comprehended by reductionistic analysis. Realizing this, we are returned to a primary osteopathic tenet that rational therapy is based on an understanding of body unity, self-regulatory mechanisms, and the interrelatedness of structure and function.

Conclusions

A nociceptive component—stimuli, reflexes, reactions, and responses—is proposed as a theoretical explanation for the initiation and maintenance of somatic dysfunction and its response to counterstrain manipulative treatment. This component and others that may be present have characteristics similar to those of the proprioceptive component. The ideas, ex-

pressed in simplistic form, are consistent with clinical experience.

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