Counterstrain Tender Points as Indicators of Sustained Abnormal Metabolism:

Advancing the Counterstrain Mechanism of Action Theory

Paul R. Rennie

Proposed Theory

Previous explanations for the mechanism of action in counterstrain theory have centered prominently on the role of the muscle spindle apparatus triggered from a sudden "panic lengthening" of the muscle fibers during injury.[8,25] This theory asserts that the muscle spindle maintains the abnormal tone.

This article will review the various effects on muscle metabolism that result from injury and the resultant forces placed on all structures associated with the muscle. Key to this process is the critical balance of oxygen delivery, blood flow, sympathetic tone, and intramuscular pressure on metabolic recovery after muscle effort.[5,10] The resultant alteration in muscle effort may exert a traction/compression effect on the nerve fibers, blood vessels, and lymphatic channels as they course through the myofascial tissues.[38,41,43,46]

Anatomical consistency of many of the tender point and motor point locations throughout the body will also be explored. Viewed in relation to the metabolic alterations found within injured muscles, the following discussion will provide additional insight into the tremendous overlap in physiological and anatomical processes leading toward a possible explanation for the shared phenomenon of tenderness and treatment approaches.

Therefore, sustained altered metabolism is at the center of the establishment of tender point manifestations. Proper positioning of the tissues during counterstrain treatment reduces the tender point manifestation while enhancing circulatory movement and, therefore, normalization within these tissues.

Postural Integrity

Postural integrity is a vital function of the musculoskeletal system. Ideally, the body is not overly challenged, and the system is kept at equilibrium. However, when tissues become injured or deconditioned, an adaptation must take place in order to attempt to maintain postural orientation. Alteration in muscle coordination with resultant reorchestration of the muscle efforts becomes evident, and these findings offer a roadmap to the rehabilitation needs of the patient.[4,36,40,46]

An exploration of what underlies postural integrity requires an understanding of how the systems of the body functionally relate. The osteopathic philosophy stresses the concept of unity of the living organism's structure (anatomy) and function (physiology).[72] Osteopathic principles are founded on the principles that the human body is a dynamic unit of function, possesses self-regulatory mechanisms that are self-healing in nature, that structure and function are interrelated at all levels, and that rational treatment is based on these principles.

Somatic dysfunction is defined as the impaired or altered function of related components of the somatic (body framework) system: skeletal, arthrodial, and myofascial structures, and related vascular, lymphatic, and neural elements.[72] Therefore, the diagnosis of somatic dysfunction confirms that osteopathic neuromusculoskeletal treatment is indicated and appropriate as part of the treatment plan. A better understanding of the anatomical and physiological matrix of the body reveals how these systems can be influenced in a manner that will contribute to the postural integrity of the body.

Effect of Reduced Blood Flow and Increased Nociceptive Activity

Proper balance in blood flow is required for normal metabolic activity for all tissues, including the musculoskeletal system. Reduced blood flow leads to reduced oxygen and metabolic support, along with reduced waste by-product removal and reduced overall force generation from the muscles.

Capillaries course between the muscle fibers through spaces so small that the red blood cell discs must travel in a horizontal orientation to traverse these channels.[13] Any further compression or reduction in muscle pumping effort reduces the movement of these cells. Ischemia is a local anemia due to mechanical obstruction (mainly arterial narrowing) of the blood supply.[14] The ischemia that results from this reduction in blood flow can stimulate the nociceptive receptors and trigger a pain response, especially with the release of acidic hydrogen ions and bradykinin.[29,44] Altered blood flow therefore triggers a neural response signaling that a problem exists within the tissues and that there is a state of low-energy formation in the muscles.

Blood flow is one necessary component for muscle contraction. The other is a coordinated and effective neural stimulus. Therefore, muscle contraction requires both a chemical and an electrical reaction. The neural (electrical) process involves sensory and motor activity conducted between the central nervous system and the periphery. Blood flow is mediated through the arterial, capillary, and venous conduits connected with various organ systems, particularly the heart, lungs, gastrointestinal, and genitourinary systems.

Sufficient blood flow must be maintained in order to regenerate the ATP necessary to break the actin-myosin cross-links so that muscle fiber movement may be continued.[2,71] Therefore, even without considering the electrical effects, reduced blood flow will alter the metabolic support necessary for normal muscle movement. This process demonstrates the observation of Andrew Taylor Still, MD, DO that "the rule of the artery is supreme". Without appropriate blood flow, suboptimal functional activity remains and likely maintains the somatic dysfunction.

The neural tissues also depend on proper balance within the circulatory system. Nociceptors are found in close proximity to the arterioles and provide for early warning of dysfunction within the tissues. Once activated, the nociceptive input triggers an axon reflex that activates adaptive mechanisms in order to protect the body from further injury and to generate the reparative response needed in tissues that become damaged. This response can augment the vascular response and requires adequate blood flow. Nociceptors are active in releasing the chemical environment by which tissue edema is generated from increased tissue permiability and vasodilation. If maintained, this leads to congestion and reduced blood flow.[12]

Nociceptive input is also transmitted to the spinal cord and brainstem. Normally, nociceptive threshold response, and therefore neural signal propagation, should be able to discriminate an event that is tissue threatening and therefore, not activate in the presence of weak local applied pressures, normal physiologic contractions, and normal joint ranges of motion. However, some input through the spinal cord and brainstem may induce long-term changes in synaptic processes in dorsal horn neurons.

This afferent input is maintained in the central nervous system and remains despite apparent resolution at the original tissue injury site.[8] Experiments have shown that a morphological change occurs in the CNS that produces a fixed functional change.[48] It is thought that this process may be the mechanism behind hyperalgesia (increased sensitivity to nociceptive stimuli). Therefore, central sensitization increases the tenderness of the peripheral structures due to spinal rewiring with non-nociceptive inputs stimulating nociceptive pathways.[12] This central fixation may be one reason why it takes more time for pain to diminish in chronic pain patients.

Intramuscular Pressure

Muscle tone is the degree of muscle tautness at rest. It is measured by the degree of stiffness or resistance to passive movement.[9] This tone is established by the viscoelastic properties within the muscle fibers and fascia and by the degree of activation of the contractile elements.[17]

Intramuscular pressure (IMP) is that pressure contained within the muscle. IMP becomes elevated if increased external or internal compression is applied to the muscle tissues. Causes of this include internal tissue damage (compartment syndrome as an extreme example), sustained muscle contraction and overuse syndromes, and pressure placed on the muscle tissues via taut fascial compartments and boney elements that surround the muscle tissues.[39]

Each muscle, due to its morphologic arrangement, may attain a different maximal intramuscular pressure during contraction effort as compared to other muscles in the body. As an example, maximal intramuscular pressures measured in one study of shoulder abduction revealed the trapezius to average 86 mmHg, deltoid 146 mmHg, infraspinatus 439mmHg, and the supraspinatus 524 mmHg.[6] Additionally, when the supraspinatus muscle was measured during shoulder flexion at 30 degrees, the IMP average was 58 mmHg. Therefore, morphologic and positional factors are involved in the IMP generated.

Sustained muscle contractions maintain a higher IMP. If the muscle contracts at 30% or more of its maximal contraction force (MCF), it will compress its own blood vessels.[12] If sustained, such as with overuse syndromes, repetitive strain injury, and chronic compartment syndrome, this will reduce nutrient and oxygen delivery necessary for the mitochondria to regenerate ATP via oxydative phosphorylation for the uncoupling of actin-myosin cross- bridges.[49,50] This sets-up a vicious cycle of venous and lymphatic congestion, ischemia, further release of vasoactive and nociceptive sensitizing chemical edema and thus, a perpetuation of the dysfunction. And so the process begun as a metabolic abnormality results in a cycle in which the nociceptive afferent system elicits alterations in the motor response to either increase or decrease a particular muscle effort secondary to actual or perceived tissue damage, and the nociceptive response further alters metabolism.

Increased and sustained sympathetic tone with increased exercise or labor activities will also affect blood flow. Intracellular ph is reduced with resultant reduction in blood flow and mitochondrial respiration.[51] Despite the compensatory metabolic vasodilatory effect, the sympathetic vasoconstrictive effect on the blood vessels is not overcome. Therefore, oxygen support does not adjust for the metabolic needs in the muscle tissues. Phosphocreatinine and oxydative ATP recovery is dependent primarily on the oxydative capacity in the muscle tissues. Higher levels of exercise can result in a worsening of the imbalance in oxygen delivery[64] and blood flow.[55] This, coupled with a limitation of oxygen at the onset of exercise, leads to a greater reliance on anaerobic ATP turnover.[49]

Increased IMP has been found to be associated with an increased fluid content after repeated maximal isokinetic contractions. IMP is affected by the fascial compliance and fluid content in the muscle compartment.[53] Increased IMP may affect blood flow particularly in the low-pressure venous system, thereby reducing waste product removal from the tissues. On the other hand, increased IMP during repetitive contractions does assist in venous flow return to the heart.[54] The emphasis here is with a proper balance of muscle contractions to augment low-pressure fluid flow. Static work and inactivity aggravate the effect of sustained elevated IMP. However, with the development of fatigue, a drop in IMP toward mean arterial pressure (MAP) may allow for maintenance of muscle perfusion and oxygen delivery. Again, the metabolic environment is paramount and normally dictates a response in blood flow that signals to the individual to adjust their activity level to support recovery to the metabolic environment.

Metabolic Recovery

Metabolic recovery within the muscle requires that IMP return to proper resting levels. These levels vary within different muscles and have not been fully researched for each muscle.

Prolonged elevated IMP and static muscle positioning lead to impairment of intramuscular blood flow to the muscle and tendons. EMG studies on the biceps muscle have found that IMP had to return to below 20 mmHg before metabolic recovery was possible.[10] It has also been found that IMP as low as 15 mmHg can decrease microcirculation to the margins of an injured and edematous site and to more fragile sites such as the tendons.[5] Removal of interstitial fluid with ultrafiltration has been shown to reduce the risk of developing acute compartment syndrome in patients prone to this condition.[56] The ideal is to offer a conservative means to maintain proper resting IMP levels in all people before further metabolic compromise or ongoing degeneration becomes established.

Other influences also play a role on blood flow to the tissues. In one study involving pain to the trapezius muscle, IMP measurements indicated the muscle had no significant blood flow impairment. However, laser doppler flowmetry (LDF) revealed a lowered local blood flow due to impaired regulation of the microcirculation.[57] This effect was thought to be created through a defect in the release of vasodilatory substances that are excreted axonally.

Therefore, muscle and its blood vessels possess the capacity to "squeeze-out" their blood supply, the muscle's metabolic support. The resultant loss of mechanical effort from these fibers must be taken-up by other healthy muscle fibers.[44] This increases the workload on these fibers, possibly increasing the IMP in these muscle groups. This process has the potential of spreading to yet other regions of the body in order to adapt to the mechanical needs of the body.[39] If sustained, more muscle fibers will suffer metabolic exhaustion due to sustained elevated IMP and loss of metabolic support.

Skeletal muscle fibers can be categorized into slow and fast twitch types. These types distinguish metabolic and functional differences between the muscle fibers. Slow twitch (tonic) muscle fibers are smaller than fast twitch fibers but have more mitochondria and blood capillaries than do fast twitch fibers. The sarcoplasm also has a high content of myoglobin that carries additional oxygen for use by the mitochondria. These features account for the red coloration of these fibers as opposed to the pale-colored fast twitch muscle fibers.

Fast twitch (phasic) muscle fibers are metabolically and functionally designed for more ballistic activities requiring power movement performed over a short duration of time. In order to accomplish this, the fast twitch fibers utilize the glycolytic pathway with a more extensive sarcoplasmic reticulum to allow faster movement of calcium ion transport. However, these fibers are more easily subject to fatigue than the slow twitch type. Yet, if there is a disturbance in the blood flow characteristics to these slow twitch fibers, there may be the increased use of the more fatiguing fast twitch fibers leading to potential early fatigue. Also, since the slow twitch fibers tend to have higher proprioceptive input, this may have an influence on the balance efforts from the muscles resulting in more uncoordinated movement. This leads to the condition of muscle imbalance, to be discussed as follows.

Altered Joint Function

Joint inflammation or increased joint fluid pressure will stimulate joint afferent neurons in the same way that injured muscle fibers and elevated intramuscular pressure stimulates intramuscular nociceptive afferent receptors. The common end result is inhibition of muscle and joint movement to the injured sites, with protective spasm from other healthy, non-injured muscle groups.

The differing metabolic profiles of specific muscle and the affect they have on joint arrangements are associated with a pattern of recognizable muscle inhibition and joint restriction. For instance, the gluteus maximus muscle appears to become inhibited with ipsilateral sacroiliac restriction, the gluteus medius with acetabular restriction, the multifidi with zygapophysial restriction, and the rectus femoris with knee joint restriction.[1,3,11,15] Knowing these associations allows for focused treatment approaches.

Correlation between Motor Points and Tender Points

Counterstrain tender points are hyperalgesic areas found at consistent anatomical locations throughout the body.[7,8,18,19,20,21,23,45,47] On further review of many of the known and reliable tender point sites, a clear correlation between these sites and motor point sites may be found. The majority of tender point sites appear to be consistent with neural tissue locations whether it be motor points[46,61,62,63,64,65,66,67,68] or more deeply invested neural fibers into the ligamentous structures such as the collateral ligaments at the knee.[70]

This provocative association suggests that the accessible neural components found at these regions reveal the facilitated status of the connected structures. These sites do not, in their entirety, indicate that the problem is exclusive to this site but may be part of a chain attached to deeper and more elaborate structural dysfunction. Maintained muscle tightness, elevated IMP, ischemia, and sustained nociceptive activation, either through an activated axonal reflex and/or sustained neuroplastic response may trigger the necessary environment to create the manifestations encountered on palpating counterstrain tender points.

Many of the neural and circulatory conduits follow similar courses through the body. The "rule of the artery" also applies to the neural system. It is no coincidence that these two vital conduits commonly wind together through various connective tissue elements to reach the tissues they serve.[42] Functional integrity requires this intimate connection in order to provide for organized movement and responsiveness of the body systems. This view differs from the "boney" model wherein we may view that a vertebral misalignment may be the sole cause of our somatic dysfunction. The view should rather be that of a contiguous mechanism that requires all elements of the somatic framework and visceral system to maintain homeostasis.

Various methodologies also view the presence of tender areas on the body that are associated with somatic and visceral dysfunction. In addition to the current discussion regarding counterstrain tender points, there has been much debate about the nature and qualities of trigger points, fibromyalgia tender points, and acupuncture points. However, there has been less

debate over motor point sites, and this may be due to anatomical consistency and diagnostic methods largely correlated to EMG studies. Dr. Angus Cathie stated in 1960 that "many so called 'trigger points' correspond to the points where nerves pierce fascial investments".[58] Others have sited the connection between neural positions in relation to the muscles and fascia and a possible role in pain and functional alterations.

In "Muscles, Testing and Function," Henry O. Kendall suggests that nerves could be irritated from the muscles being drawn taut and firm, thus exerting a compressive or friction force on these nerves.[46] Muscles that are pierced by a peripheral nerve may become symptomatic if the muscle develops adaptive shortening moving through a shorter range of motion and becoming tight before reaching its full length. Examples include:

- Radial nerve with the supinator and lateral head of the triceps
- Median nerve with the pronator teres
- Ulnar nerve with the flexor carpi ulnaris
- Greater occipital nerve with the trapezius and semispinalis capitis
- C5 & C6 root of the plexus and the long thoracic nerve with the scalenus medius
- Musculocutaneous nerve with the coracobrachialis
- Lumbar plexus nerves with the psoas
- Iliohypogastric nerve with the transversus abdominis
- Obturator nerve with the external oblique
- Fibular nerve vith the biceps femoris and gastrocnemius

In the *JAOA* article, "Nerve compression syndromes as models for research on osteopathic manipulative treatment" (Luchenbill-Edds, Bechill), a question presented by Dr. Irvin Korr asked "How many compression, angulation, or other deformations of nerves and nerve roots by surrounding structures influence neural chemistry and metabolism and the synthesis and axonal transport of macromolecules and subcellular structures"?[59] Further review indicated that the effect of compression on the nerves can produce ischemia at pressures of 30 mmHg, affecting the vessels of the subperineural region and leading to decreased venular outflow. Additionally, the effect of ischemia increases the permeability of the endothelial linings of the capillaries, which increases edema. Compression also blocks anterograde and retrograde axonal transport necessary for nutrient support. It was also suggested that neurapraxia (axonal conduction and transport compromise but no axon degeneration resulting from chronic or acute nerve compression) may be relieved with OMT with counterstrain listed as one of the possible treatment methods.

Chronic myofascial tenderness has not been found to be associated directly with ongoing inflammation.[2] Local tenderness is commonly found over nerve trunks at sites of entrapment or metabolic insult. This has been attributed to the sensitization of free nerve endings within neural connective tissues, the nervi nervorum.[62] Additionally, it appears that unmyelinated sensory fibers are the afferent limb of trigger points.[60] Trigger points can be reduced by lidocaine infiltration or by transection of the motor nerve innervating the trigger point.[60] However, transection of the spinal cord above the level to the innervation site to the muscle has been shown to fail to abolish the twitch response of the trigger point. This interesting study therefore demonstrated that the local twitch response is a spinal reflex and not mediated in the cortex.

Acupuncture points have not only been associated with trigger points but also with motor points.[60,61,63,64,65,66,67,68] There has also been the suggestion that acupuncture loci be categorized into types that involve motor points, superficial nerves, and nerve plexi.[63] Clearly, in common is the presence of tenderness at these sites. Tenderness at motor points located in the myotomes has been correlated to segmental spinal injury.[67] Further, the degree of tenderness has been found to correlate with the severity of symptoms with greater involvement to both the anterior and posterior primary rami. Muscle tenderness is found to be maximal at the motor point location (neurovascular hilus). This tenderness has also been associated with positive EMG changes that may or may not be present in the mildest form of tenderness but become more clearly significant with greater neuropathic findings.[68] Therefore, early neuropathic changes that may not be detected by EMG could be best elicited by palpating for tender motor points.

Concept of Neurocirculatory Integration (Fascial Release)

It is evident that the aforementioned physiologic changes play a role in the associated manifestations of the counterstrain tender point. Sustained alteration within the muscle fibers that have become inhibited and taut can be expected to demonstrate poor metabolic activity and sustained nociceptive input.[22,30,33] Tender points found long after the injury occurred demonstrates a memory effect locally within the tissues and through the central nervous system.

It is interesting to note the correlation of counterstrain treatment positions with the position of the patient's body at the time of injury.[8] How is it that osteopathic physicians remedy the somatic dysfunction by returning the body to the position of injury? How is it that treatment decreases the sensitivity and improves the quality of function of these tissues?

Particularly interesting is the additional manifestation of a palpable pulsation response felt at the tender point site as treatment is delivered.[23,45,47] It is commonly the case that when the patient reports the most significant reduction in tenderness, thus when the nociceptive input is terminated, the pulsation amplitude is found to be at its greatest intensity.

Because neural and circulatory conduits tend to follow together, and tender points and motor points are often found in close proximity, the anatomical explanation why osteopathic physicians are able to perceive this pulsation phenomenon becomes more evident. This represents an objective manifestation of improved metabolic recovery within the muscle tissues. This phenomenon suggests improved intramuscular perfusion necessary for the muscle tissues to recover metabolically, thus reversing the effects from an injury process.

Muscle imbalance is defined as the existence of inequality in the strength of opposing muscle groups wherein one muscle group is weak and its opposing group is strong (tight). This imbalance leads to inefficient and potentially injurious movements, particularly to the joints.[4,46] Both weak and tight muscles reflect

→

abnormal metabolic activity. Counterstrain treatment requires a reduction in the tension (and, secondarily, a shortening) of a particular muscle or group of muscles along with the associated myofascial structures and joints in order to reduce the nociceptive afferent stimulus found at the tender point site.[7,8,23,45,47]

Muscles that are tight (with limited range of motion) and do not possess tender points typically require therapeutic lengthening, which can be performed with various manipulative techniques such as with muscle energy technique. Muscles that contain tender points may not be as accommodating to aggressive lengthening without added discomfort. Thus, counterstrain methodology has provided a more "indirect" means of reducing discomfort and assisting in the proper lengthening of the affected myofascial tissues.

Typically, injured muscle tissues are protected from further movement through a spastic neural response generated from muscle tissues, the muscle spindles that are capable of providing this adaptation.[12] Counterstrain treatment can be applied concurrent with the application of post-isometric relaxation to the antagonist muscles to allow further unloading of tension to the myofascial group containing the tender point. This process transforms "classical" counterstrain approach that requires the patient to be totally passive to a more integrated process that incorporates group Ia inhibitory interneurons that not only function locally on the antagonist muscles but also at higher centers of control opposing muscles at the joint in reciprocal fashion.[9]

Together with the reestablishment of improved circulatory flow and reduced nociceptive input, this neurocirculatory integration fascial release approach incorporates an effective and efficient means of addressing the integrated neurocirculatory needs of the musculoskeletal system. This integration of methods provides for the correction of the structural and postural responses the body has manifested with the original injury. This allows for improved muscle balance through the action on the agonists, antagonists, and synergists.

Integrative Thinking – The Challenge

We are therefore at the point long hoped for in the osteopathic profession where the manifestations we have attempted to describe receive support from dynamic technologies that bring to life the anatomical and physiological manifestations of somatic dysfunction.

We have established the central role that altered metabolic processes contribute in the initiation and maintenance of tender points, a manifestation of the presence of somatic dysfunction. The coupled role of the neural and circulatory systems now have to be viewed as a unit in order to more completely understand the pathophysiology of somatic dysfunction and the methodologies required to treat these dysfunctions. To neglect the importance of either system leads to a suboptimal understanding of the underlying physiology.

The primary goal in the provision of medical services should be to offer, through better understanding of the body's response to injury, a conservative means to restore and maintain proper resting IMP levels, circulatory flow, and reduce nociceptive stimulus before further metabolic compromise or ongoing degeneration and muscle imbalance becomes established. This conservative emphasis should be encouraged throughout the health care system.

Neurocirculatory integration fascial release that utilizes counterstrain along with other osteopathic manipulative principles enhances neural and circulatory normalization within the tissues, providing a conservative approach that is both diagnostic and therapeutic. The concepts explored here offer a window into a better understanding of the complexity and yet the opportunity to evolve with a greater appreciation for what we can do to address the needs our patients. This understanding can lead to improved diagnostic assessments and treatment outcomes that impact the healthcare system and, most importantly, the treatment of our patients.

Special thanks to Claudio Carvalho, DO, MS. Additional thanks to the Fellowship Committee of the American Academy of Osteopathy and to Dennis J. Dowling, DO, FAAO, Richard L. Van Buskirk, DO, PhD, FAAO, Robert Kessler, DO, and to Gabriele Rennie for their editorial assistance.

References

1. Dorman TA, Brierly 5, Fray J, Pappani K. Muscles and pelvic clutch: Hip abductor inhibition in anterior rotation of the ilium. Journal of Manual and Manipulative Therapy. 1995;3:85-90.
2. Ashina M, Stallknecht B, Bendtsen L, Pedersen JF, Schifter 5, Galbo H, and Olesen J. Tender points are not sites of ongoing inflammation-in vivo evidence in patients with chronic tension-type headache. Cephalgia. 2003; 23:109-116.
3. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine. 1996;21:2763-2764.
4. Janda V. Muscles and back pain, assessment and treatment, physical medicine research foundation. CME Seminar Workshop Series. 1994.
5. Jarvholm U, Palmerud G, Karlsson D, Herberts P, Kadefors R. Intramuscular pressure and electromyography in four shoulder muscles. Journal of Orthopaedic Research. Jul 1991;9(4):609-619.
6. Jensen BR, Jorgensen K, Huijing PA, Sjogaard G. Soft tissue architecture and intramuscular pressure in the shoulder region. European Journal of Morphology. 1995;33(3):205-220.
7. Jones LH, Kusunose RS, Goering EK. Jones Strain -Counterstrain. Boise, Id: Jones Strain-Counterstrain mc; 1995:24.
8. Jones LH: Strain and Counterstrain. Colorado Springs, Co: American Academy of Osteopathy; 1981:16.
9. Kandel ER, Schwartz JH, Jessell TM. Principles of Neural Science. 3rd ed. Norwalk, Ct: Appleton and Lange; 1991:387,577,578,585.
10. Korner L, Parker P, Almstrom C, Herberts P, Kadefors R. The Relation between spectral changes of the myoelectric signal and the intramuscular pressure of human skeletal muscle. European Journal of Applied Physiology. 1984;52:202-206.
11. Lee D. Instability of the sacroiliac joint and the consequences for gaits. In: Vleeming A, Mooney V, Dorman T, Snijders C, Stoedkart R (eds). Movement, Stability, & Low Back Pain. Edinburgh, UK: Churchill Livingstone; 1997:233.
12. Mense S. Pathophysiologic basis of muscle pain syndromes: an update. In: Physical Medicine and Rehabilitation Clinics of North America. Philadelphia, Pa: WB Saunders Company. Feb 1997;8(1):23-53.

13. Hammersen F. Histology. 2nd ed. Baltimore-Munich: Urban & Schwarzenberg; 1980:69.
14. Stedman TL. Stedman's Medical Dictionary. 26th ed. Baltimore, MD, Williams and Wilkins; 1995:894.
15. Stokes M, Young A. Investigations of quadriceps inhibition; implications for clinical practice. Physiotherapy. 1984;70:425-428.
16. Vecchiet L, Giamberardino MA. Referred pain, clinical significance, pathophysiology, and treatment. Physical Medicine and Rehabilitation Clinics of North America. Philadelphia, Pa: WB Saunders Company. Feb 1997. 8(l):119-136.
17. Ward R. Foundations for Osteopathic Medicine. Baltimore, Md: Williams and Wilkins; 1997.
18. Jones LH. Spontaneous Release by Positioning. The DO. 1964;4:109-116.
19. Jones LH. Missed Anterior Spinal Lesions: A Preliminary Report. The DO. Mar. 1966;6:75-9.
20. Jones LH. Foot Treatment Without Hand Trauma. Journal of the American Osteopathic Association. Jan 1973;72:481-489.
21. Schwartz HR. The Use of Counterstrain in an Acutely Ill In-Hospital Population. Journal of the American Osteopathic Association. Jul 1986;7:433442.
22. Travell JG, Simons DG. Myofascial Pain and Dysfunction, The Trigger Point Manual. Baltimore, Md: Williams and Wilkins; 1983.
23. Yates HA, Glover JC. Counterstrain Handbook of Osteopathic Technique. Tulsa, OK. Y Knot Publishers; 1995.
24. Hoppenfeld S. Physical Examination of the Spine and Extremities. Norwalk, Ct: Appleton and Lange; 1976.
25. Korr IM. Proprioceptors and Somatic Dysfunction. Journal of the American Osteopathic Association. Mar 1975;74(7).
26. Willard FH. Autonomic Nervous System. In: Foundations for Osteopathic Medicine. Baltimore, Md: Williams and Wilkins; 1997.
27. Vleeming A, Mooney V, Snijders CJ, Dorman TA, Stoeckart R. Movement, Stability, and Low Back Pain: The Essential Role of the Pelvis. Edinburgh, UK: Churchill Livingstone; 1997.
28. Kadi F, Waling K, Ahlgren C, Sundelin G, Holmner S, Butler-Browne G, Thornell L. Pathological Mechanisms Implicated in Localized Female Trapezius Myalgia; Pain. Dec 78;3:191-196.
29. Determe D, Rongieres M, Kany J, Glasson IM, Bellumore Y, Mansat M, Becue J. Anatomic Study of the Tendinous Rotator Cuff of the Shoulder. Surgical and Radiologic Anatomy. 1996; 18(3):199.
30. Fischer AA. Documentation of Myofascial Trigger Points. Archives of Physical Medicine and Rehabilitation. Apr 1988;69(4): 290.
31. Fischer AA. Pressure Algometry Over Normal Muscles, Standard Values, Validity and Reproducibility of Pressure Threshold. Pain. Jul 1987;30(1):115126.
32. Fischer AA. Tissue Compliance Meter for Objective, Quantitative Documentation of Soft Tissue Consistency and Pathology. Archives of Physical Medicine and Rehabilitation. Feb 1987;68(2):122-125.
33. Fischer AA. Pressure Threshold Meter: Its Use For Quantification of Tender Spots. Archives of Physical Medicine and Rehabilitation. Nov 1986;67(11):836-838.
34. Fischer AA Pressure Tolerance Over Muscles and Bones in Normal Subjects. Archives of Physical Medicine and Rehabilitation. Jun 1986;67(6):406-409.
35. Van Buskirk RL. Nociceptive Reflexes and the Somatic Dysfunction: A Model. Journal of the American Osteopathic Association. Sep 1990;90(9).
36. Solomonow M, Guanche C, Wink C, Knatt T, Baratta RV, Lu Y. Mechariorecеptors and Reflex Arc in the Feline Shoulder. Journal of Shoulder And Elbow Surgery. Mar-Apr 1996; 5(2 Pt 1):139-146.
37. Johansen RL, Callis M, Potts 3, Shall LM. A Modified Internal Rotation Stretching Technique for Overhand and Throwing Athletes. Journal of Orthopaedic and Sports Physical Therapy. Apr 1995; 21(4): 216-219.
38. Berry H, Kong K, Hudson AR, Moulton RJ. Isolated Suprascapular Nerve Palsy: A Review of Nine Cases. Canadian Journal of Neurological Sciences. Nov 1995;22(4):301-304.
39. Palmerud G, Kadefors R, Sporrong H, Jarvholm U, Herberts P, Hogfors C, Peterson B. Voluntary Redistribution of Muscle Activity in Human Shoulder Muscles. Ergonomics. Apr 1995;38(4):806-815.
40. Otis JC, Jiang CC, Wickiewicz TL, Peterson MG, Warren RF, Santner Ti. Changes in the Moment Arms of the Rotator Cuff and Deltoid Muscles with Abduction and Rotation. Journal of Bone and Joint Surgery, American Volume. May 1994;76(5):667-676.
41. Kukowski B. Suprascupular Nerve Lesion as an Occupational Neuropathy in a Semiprofessional Dancer. Archives of Physical Medicine and Rehabilitation. Jul 1993;74(7):768-769.
42. Ringel SP, Treihaft M, Carry M, Fisher R, Jacobs P. Suprascapular Neuropathy in Pitchers. American Journal of Sports Medicine. Jan-Feb 1990;18(1):80-86..43
43. Ferretti A, Cerullo G, Russo G. Suprascapular Neuropathy in Volleyball Players. Journal of Bone and Joint Surgery, American Volume. Feb 1987;69(2):260-263.
44. Herberts P, Kadefors R, Hogfors C, Sigholm G. Shoulder Pain and Heavy Manual Labor. Clinical Orthopaedics and Related Research. Dec 1984;(191):166-174.
45. Rennie PR, Glover J, Carvalho C, Key LS. Counterstrain and Exercise: An Integrated Approach. 2d ed. Williamston, Mi: RennieMatrix; 2004.
46. Kendall FP, McCreary EK, Provance PG: Muscles, Testing, and Function. ed. Philadelphia, Pa: Lippincott, Williams and Wilkins; 1993.
47. Glover J. Rennie PR. Strain and Counterstrain Techniques, In: Foundations for Osteopathic Medicine, Baltimore, Md: Lippincott, Williams and Wilkins; 2002.
48. Sperry MA, Goshgarian HG. Ultrastructural changes in the rat phrenic nucleus developing within 2 h after cervical spinal cord hemisection. Experimental Neurology.-1993; 120:233-244.
49. Hughson RL, Shoemaker 3K, Tschakovsky ME, & Kowalchuk 3M. Dependence of muscle Vo2 (oxygen uptake) on blood flow dynamics at onset of forearm exercise. Journal of Applied Physiology, 1996;81(4):1619-1626.
50. Jansson E, Dudley GA, Norman B, Tesch PA. Relationship of recovery from intensive exercise to the oxidative potential of skeletal muscle. Acta Physiologica Scandinavica. May 1990;139(1):147-152.
51. Shoemaker 3K, Pandey P, Herr MD, Silber DH, Yang QX, Smith MB, Gray K, Sinoway LI. Augmented sympathetic tone alters muscle metabolism with exercise: lack of evidence for functional sympatholysis. Journal of Applied Physiology, 1997;82(6):1932-1938.
52. Kagaya A, Homma S. Brachial arterial blood flow during static handgrip exercise of short duration at varying intensities studied by a Doppler ultrasound method. Acta Physiologica Scandinavica. Jul 1997;160(3):257-265.

→

53. G Sjogaard, BR Jensen, AR Hargens, K Sogaard. Intramuscular pressure and EMG relate during static contractions but dissociate with movement and fatigue. Journal of Applied Physiology. 2004;96:1522-1529.
54. Ameredes BT, Provenzano MA. Regional intramuscular pressure development and fatigue in the canine gastrocnemius muscle in situ. Journal of Applied Physiology. Dec 1997;83(6):1867-1876.
55. Saltin B, Radegran G, Koskolou MD, Roach RC. Skeletal muscle blood flow in humans and its regulation during exercise. Acta Physiologica Scandinavica. Mar 1998;162(3):421-436.
56. Odland R, Schmidt AH, Hunter B, Kidder L, Bechtold JE, Linzie BM, Pedowitz RA, Hargens AR. Use of tissue ultrafiltration for treatment of compartment syndrome: a pilot study using porcine hindlimbs. Journal of Orthopaedic Trauma. Apr 2005;19(4):267-275.
57. Larsson R, Oberg PA, Larsson SE. Changes of trapezius muscle blood flow and electromyography in chronic neck pain due to trapezius myalgia. Pain. Jan 1999;79(l):45-50.
58. Cathie AG. The fascia of the body in relation to function and manipulative therapy. In: 1974 Yearbook of Papers Selected from the Writings and Lectures of Angus G. Cathie, D. 0., M.Sc. (Anatomy), F.A.A.O. Colorado Springs, Co: American Academy of Osteopathy; 1974:81.
59. Luckenbill-Edds L, Bechill GB. Nerve compression syndromes as models for research on osteopathic manipulative treatment. Journal of the American Osteopathic Association. 1995;95(5)319-326.
60. Rivner MH. The neurophysiology of myofascial pain syndrome. Current Pain and Headache Reports. Oct 2001;5(5):432-440.
61. Melzack R. Myofascial trigger points: relation to acupuncture and mechanisms of pain. Archives of Physical Medicine and Rehabilitation. 1981;62:114-117.
62. Quintner JL, Cohen ML. Referred pain of peripheral nerve origin:an alternative to the "myofascial pain" construct. Clinical Journal of Pain. Sep 1994; 10 (3):243-51.
63 Gunn CC, Ditchburn FG, King MH, Renwick GJ. Acupuncture loci: a proposal for their classification according to their relationship to known neural structures. American Journal of Chinese Medicine. 1976;4:183-195.
64. Gunn CC, Milbrandt MD, Little AS, Mason KE. Dry needling of muscle motor points for chronic low-back pain. Spine. May 1980;5(6)279-291.
65. Liao SJ. Acupuncture points: Coincidence with motor points of skeletal muscles. Archives of Physical Medicine and Rehabilitation. 1975;56:550.
66. Liu YK, Varela M, Oswald R. The correspondence between some motor points and acupunctue loci. American Journal of Chinese Medicine. 1975;3:347-358.
67. Gunn CC, Milbrandt WE. Tenderness at motor points. A diagnostic and prognostic aid for low-back injury. Journal of Bone and Joint Surgery (Am]. Sep 1976;58(6):815-825.
68. Gunn CC, Milbrandt WE. Tenderness at motor points: An aid in the diagnosis of pain in the shoulder referred from the cervical spine. Journal of the American Osteopathic Association. Nov 1977;77:196-212.
69. Walko EJ, Janouschek C. Effects of osteopathic manipulative treatment in patients with cervicothoracic pain: pilot study using thermography. Journal of the American Osteopathic Association. Feb 1994;94(2):135-141.
70. McDougall JJ, Bray RC, Sharkey KA. Morphological and immunohistochemical examination of nerves in normal and injured collateral ligaments of rat, rabbit, and human knee joints. The Anatomical Record. May 1997;248(1):29-39.
71. Boron WF, Boulpaep EL. Medical Physiology, Philadelphia, Pa: Saunders; 2003:239,1220,1245.
72. The Glossary Review Committee of the Educational Council on Osteopathic Principles. Glossary of osteopathic terminology. In: Ward R. Foundations for Osteopathic Medicine. Baltimore, Md: Lippincott, Williams and Wilkins; 2002:1242,1249.
71. Boron WF, Boulpaep EL. Medical Physiology, Philadelphia, Pa: Saunders; 2003:239,1220,1245.
72. The Glossary Review Committee of the Educational Council on Osteopathic Principles. Glossary of osteopathic terminology. In: Ward R. Foundations for Osteopathic Medicine. Baltimore, MD: Lippincott, Williams and Wilkins; 2002:1242,1249.

Accepted for publication: March 2006

Address correspondence to:
Paul R. Rennie, DO, FAAO
CAOBNMM, CAOBFP, DAAPM
Associate Professor
TUCOM/NV
874 American Pacific Drive
Henderson, NV 89014
E-mail: prennie@touro.edu ❒

CME QUIZ

The purpose of the quiz found on the next page is to provide a convenient means of self-assessment for your reading of the scientific content in the *"Counterstrain Tender Points as Indicators of Sustained Abnormal Metabolism – Advancing the Counterstrain Mechanism of Action Theory"* by Paul R. Rennie, DO, FAAO. For each of the questions, place a check mark in the space provided next to your answer so that you can easily verify your answers against the correct answers that will be published in the June 2007 issue of the *AAOJ*.

To apply for Category 2-B CME credit, transfer your answers to the AAOJ CME Quiz Application Form answer sheet on the next page. The AAO will record the fact that you submitted the form for Category 2-B CME credit and will forward your test results to the AOA Division of CME for documentation.