

Experimental Resolution of the Myofascial Pain Enigma [Comment on Gunn Research Ideas Article]

David G. Simons

The juxtaposition of the two *Research Ideas* that were submitted by Simons (1) and by Gunn (2) provides considerable food for thought. Simons explicitly presented a research protocol to bring closure on the experimental evidence that the essential cause of a myofascial trigger point [TrP] is a population of microscopic contraction knots which are produced at individual dysfunctional endplates. The population of contraction knots produces a nodule at the TrP and the palpable band associated with the nodule. Together they cause increased muscle tension and muscle shortening. Apparently, Gunn proposes that all myofascial pain, including that of TrPs, originates as a radiculopathic neuropathy.

The Gunn article raises two serious questions. First, what is the relation between myofascial pain caused by neuropathy as he describes it and myofascial pain caused by TrPs as described by other authors: Travell and Simons (3), Simons (4), Gerwin (5), Chu (6), Rachlin (7), and Fischer (8)? Second, what research questions or ideas did Gunn have in mind? Several were implicit but I found none that was explicit.

SPECIFIC ISSUES

Neuropathic Pain

Although not all physicians are familiar with peripheral neuropathy, this condition is now thoroughly familiar to neurologists and physiatrists (9,10),

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since it has been a major topic of study and teaching by them for at least 30 years.

Disuse supersensitivity was presented as an important mechanism. However, the key clinical feature described by Gunn in his section entitled *Release of Muscle Shortening Is Key to Treatment* was muscle shortening that was illustrated in his Figure 1 and was ascribed to palpable bands in the muscle. The rationale by which disuse sensitivity and peripheral neuropathy can cause a shortened muscle syndrome was unexplained. These contracted muscles show no resting electromyographic activity. In addition, neuropraxia and denervation of muscle is associated with weakness, paralysis, and atrophy but not increased tension. The research idea proposed by Simons (1) shows why muscle shortening caused by palpable bands is a cardinal feature of TrPs.

Myofascial Pain

The statement by Gunn that "persistent nociception is not a common cause of chronic pain" flies in the face of the last five years of pain neurophysiological research. One of the big breakthroughs in pain management is the realization that even relatively brief nociceptive input can cause plastic changes in the central nervous system that potentiate pain. Increasing attention is being paid to the importance of avoiding unnecessary intensity and prolongation of acute pain because of its tendency to develop into chronic pain. Fischer (11) emphasized this with regard to the treatment of myofascial TrPs.

Apparently, Gunn is proposing to add a third definition of myofascial pain, "neuropathic pain that presents predominantly in the musculoskeletal system" in addition to the two definitions that are already established (12): general—any regional musculoskeletal pain syndrome, and specific—myofascial TrPs. Gunn's new definition adds an unnecessary third dimension of semantic confusion.

To those acquainted with the TrP literature, the subject of Gunn's article almost entirely concerns myofascial TrPs. His article adds further the confusion by introducing new terminology for established concepts. It would be helpful to readers if Dr. Gunn followed the lead of Jennifer Chu (6) who recognized that Gunn's writings and concepts relate closely to myofascial TrPs. His Table 1 could just as well have been titled "Clinical Syndromes Caused by Myofascial Trigger Points" and essentially has been published as such (3).

Gunn's article proposed peripheral neuropathy as the explanation for all musculoskeletal pain not explained by injury or inflammation. It seems much more likely that neuropathy is a common source, but not the only source, of TrPs. Clearly muscle overload plays an important role.

The seven observations identified by Gunn as leading to a radiculopathy model are equally true of TrPs and many of the observations do not depend on radiculopathy for their explanation. The proximity of TrPs to motor points [endplate zones] occurs because TrPs are characterized by dysfunctional endplates. Gunn's tender points at musculotendinous junctions are often due to enthesopathy secondary to the TrP-generated taut bands as illustrated in Gunn's Figure 2 and by Fischer (11, Figure 1). Wu et al. (13) showed that TrPs can be closely associated with the segmental distribution of lumbar radiculopathy in back and extremity muscles. Chu (6) showed a high correlation between the presence of TrPs and minimal neuropathy in paraspinal muscles. This association between TrPs and nerve root compromise had been noted previously (3). Two critical research questions are, "How commonly do TrPs develop without any evidence of neuropathy?" and "Are neuropathic changes characteristic of nerve fibers that have the endplate dysfunction of TrPs?" The endplate dysfunction may disturb the normal function of that neurone.

Palpable muscle bands are a primary characteristic of TrPs (1) and a band is palpable because of contracture of the sarcomeres in muscle fibers that have a contraction knot in the TrP.

Gunn's description of intramuscular stimulation sounds almost indistinguishable from Hong's description of the dry needling of TrPs using acupuncture needles (14). The main therapeutic difference between dry needling TrPs and injecting an anesthetic is that the anesthetic relieves much of the postinjection soreness which often appears in the next few days. The same clinical symptoms and signs typically disappear when the patient is treated by dry needling of TrPs or by intramuscular stimulation [regardless of what you call it]. Treatments other than dry needling are also effective for TrPs (3).

RESEARCH CONSIDERATIONS

With regard to neuropathic pain as described by Gunn, how much of the pain is of primary neuropathic origin due to sensitization of sensory nerves, and how much of the patient's pain is due to active TrPs? One approach would be to compare separately the degree of reduction in neuropathic and TrP findings following inactivation of the TrPs [by dry needling or any other effective method]. Measures of TrP activity and degree of neuropathic findings could be recorded before treatment, immediately afterward, and several weeks later, noting recurrence of the TrPs in the presence of persistent neuropathic findings, or the lasting resolution of both sets of findings. When all symptoms are reported to be relieved by intramuscular stimulation, one likely possibility is that the relief was due to inactivation of myofascial TrPs which were causing the symptoms. It is unclear how dry needling relieves radiculopathy.

Gunn's discussion section included several implied research considerations. He proposed that insertion potentials represent an outburst of injury potentials which relax shortened muscles instantaneously or within minutes. No rationale is given as to how action potentials initiated by the needle insertion [the positive sharp waves that he referred to as injury potentials] are able to cause muscle relaxation. Electrodiagnostic experience would indicate that these routine insertion potentials are irrelevant to release of muscle contracture. He also proposed that needling produces a sympatholytic effect which releases vasoconstriction and helps to account for the efficacious results obtained. Since the ischemia in the vicinity of active loci is the result of mechanical compression of circulation due to the muscle fiber contracture, it is hard to see how local vasodilation could be very helpful. Similarly, the stimulation of collagen formation would seem to be equally irrelevant to the release of muscle contracture.

Gunn's article raises a number of questions that deserve competent research investigation.

1. How does a mild peripheral neuropathy produced by only a modestly severe radiculopathy cause the development of TrPs? One possible mechanism would be that compromised axoplasmic flow causes the nerve terminal to be more susceptible to mechanical trauma and thereby the onset of excessive acetylcholine activation of the postjunctional membrane, which is what is responsible for the TrP-induced contracture.
2. To resolve whether the electrodiagnostic evidence of neuropathy is the result of endplate dysfunction, or if the endplate dysfunction can occur in the absence of any neuropathic process, it should be helpful to examine the propagated muscle action potentials and nerve conduction of individual nerve fibers that have dysfunctional endplates in TrPs as compared to nerve fibers that have normally functioning endplates.
3. Several other authors have also attributed the grating sensation associated with needle penetration of a TrP to fibrosis. A dense concentration of contraction knots might produce the same sensation, or fibrosis may develop in areas that suffered a concentration of contraction knots for a prolonged period that produced tissue-destructive hypoxia. Studies are needed that identify under what conditions fibrosis may occur as the result of TrPs. Production in experimental animals of contraction knots that are typical of TrPs may be possible by injecting an acetylcholinesterase inhibitor. The resultant prolonged excessive acetylcholine activity has been demonstrated to produce lesions that fit the description of contraction knots (15). Experiments could be designed to test under what conditions this type of lesion developed fibrosis.

REFERENCES

1. Simons DG: Myofascial trigger points: The critical experiment. *J Musculoske Pain* 5(4):113-118, 1997.
2. Gunn CC: Radiculopathic Pain: Diagnosis and treatment of segmental irritation or sensitization. *J Musculoske Pain* 5(4):119-134, 1997.
3. Travell JG, Simons DG: Myofascial Pain and Dysfunction: The Trigger Point Manual, Vols. 1 and 2. Williams & Wilkins, Baltimore, 1983, 1992.
4. Simons DG: Clinical and etiological update of myofascial pain from trigger points. *J Musculoske Pain* 4(1/2):97-125, 1996.
5. Gerwin RD: The management of myofascial pain syndromes. *J Musculoske Pain* 1(3/4):83-94, 1993.
6. Chu J: Dry needling (intramuscular stimulation) in myofascial pain related to lumbosacral radiculopathy. *Eur J Phys Med Rehabil* 5(4):106-121, 1995.
7. Rachlin ES: Myofascial Pain and Fibromyalgia, Trigger Point Management. St. Louis, Mosby, 1994.
8. Fischer AA (ed): Myofascial Pain-Update in Diagnosis and Treatment. *Physical Medicine and Rehabilitation Clinics of North America* 8(1):1-230, 1997.
9. Kimura J: Electrodiagnosis in Diseases of Nerve and Muscle, Vol. 2. F.A. Davis, Philadelphia, 1989.
10. Johnson EW, Pease WS: Practical Electromyography, ed 3. Williams & Wilkins, Baltimore, 1997.
11. Fischer AA: New approaches in the treatment of myofascial pain. In: Myofascial Pain-Update in Diagnosis and Treatment, edited by AA Fischer. *Physical Medicine and Rehabilitation Clinics of North America* 8(1):153-169, 1997.
12. Simons DG: Myofascial pain syndrome: One term but two concepts: A new understanding. [Editorial] *J Musculoske Pain* 3(1):7-13, 1995.
13. Wu C-M, Chen H-H, Hong C-Z: Inactivation of myofascial trigger points associated with lumbar radiculopathy: Surgery versus physical therapy. *Arch Phys Med Rehabil* 78:1040-1041, 1997. (Abstr.)
14. Hong C-Z: Lidocaine injection versus dry needling to myofascial trigger point: the importance of the local twitch response. *Am J Phys Med Rehabil* 73:256-263, 1994.
15. Leonard JP, Salpeter MM: Agonist-induced myopathy at the neuromuscular junction is mediated by calcium. *J Cell Biology* 82:811-819, 1979.

Reply to Dr. Simons

C. Chan Gunn

Simons has posed a large number of questions that can only be given concise answers in this response. For a more comprehensive explanation of intramuscular stimulation [IMS] and radiculopathic pain, he should refer to *The Gunn Approach to the Treatment of Chronic Pain* (1).

It is important to note that Gunn's Radiculopathic Pain (2) is a review of my previously published papers which pioneered the concept of radiculopathic pain; most of these papers preceded by many years the publications from the various authors quoted by Simons. For instance, Chu is a staunch follower of the radiculopathy model; Hong's work supports Gunn's model; and it was Fischer [who now regularly examines the nerve root] that had suggested the title of the review article.

Simons: What is the relation between myofascial pain caused by trigger points and myofascial pain caused by neuropathy?

The answer to the question will become apparent when this response has been read in full. Effectively, the trigger point [TrP] is part of myofascial pain, but myofascial pain is, itself, part of a dysfunction in the peripheral nervous system [PNS], specifically, radiculopathy [i.e., peripheral neuropathy at the nerve root]. The radiculopathy model explains how apparently different and unrelated musculoskeletal pain conditions—from headache to low back pain—can belong in one classification. The key to successful management of this widespread and important category of chronic pain is to recognize it in its many guises.

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Simons: What research questions or ideas did Gunn have in mind?

The Radiculopathy Model article was submitted to the *Journal* as a review, but the Editor had placed it [appropriately] in the Research Ideas section because the model challenges the present limited understanding of myofascial pain as a localized entity within muscle. Any future research on myofascial pain, that is undertaken without a broad understanding of dysfunction in the PNS, will be wasted effort.

Persistent pain that follows gross nerve injury is well known, but the many and diverse effects of *subtle* dysfunction in the PNS are recent concerns (3,4,5). Gunn introduced the concept of radiculopathic pain in his paper on "Prespondylosis" (3), and also initiated a system of dryneedling based on the radiculopathy model (6). Neuropathic pain is now firmly established: it was incorporated in Bonica's textbook *The Management of Pain*, which also featured the physical signs of neuropathy in the evaluation of the patient with pain (7). J. Loeser, editor of the next edition, is including a chapter on Neuropathic Myofascial Pain Syndromes.

Simons: Gunn is proposing to add a third definition of myofascial pain.

Simons has complained that the subject of musculoskeletal pain originating from muscle has been plagued by multiple terms that emphasized various aspects of basically the same phenomenon. An obvious reason for the confusion is the vague definitions presently used: general—*any* regional musculoskeletal pain syndrome; and specific—myofascial TrPs. These are imprecise and non-descriptive terms. By "any," does Simons include *any* type of pain, regardless of source or cause [nociception, inflammation or neuropathy]? And *syndrome* merely alludes to a collection of symptoms. The radiculopathy model does not add a new definition—it adds a new dimension.

Another reason for confusion is that musculoskeletal pain crosses into the territories of several disciplines: not unexpectedly, each discipline will insist on interpreting the subject from its own point of view. Imagine inspecting a scene from opposite ends of a telescope: the two images will appear different, and both will differ from the image that is seen with the naked eye. Of course, any opinion that is derived from only one point of view will miss out on the full picture. The following paragraphs will discuss the three approaches.

1. The "let's get closer" approach—myofascial pain caused by trigger points

Simons, by assiduously pursuing just one small aspect of myofascial pain—the TrP—is responsible for adding to the confusion. He uses the telescope to bring the quarry closer and closer, and he zooms in on the myofas-

cial TrP, ignoring the full clinical picture. To prove his notion that musculoskeletal pain originates from taut muscle bands, he proposes to locate an "active locus" by its "spontaneous electromyographic activity," biopsy it, and announce that contraction knots contain motor endplates.

Can examination of the *structure* of a muscle explain pain which is likely the result of a *functional* disturbance? No, no more than the presence of Purkinje cells in brain tissue explain how we think. Motor endplates are naturally abundant in a muscle's zone of innervation. Finding them in a shortened sarcomere merely shows that they are there as part of the muscle contraction apparatus, but their presence does not prove that they are the primary or sole cause of muscle pain. Musculoskeletal pain is a physiologic disorder and cannot be explained from an anatomic angle. Looking through a microscope to examine a piece of lifeless muscle tissue will not help.

A wider perspective is needed. The frog who surveys the sky from the bottom of a well can never have a complete understanding of the universe.

2. The "Hands-Off" approach: Myofascial pain and fibromyalgia

When "fibrositis" became "fibromyalgia," emphasis moved from the palpable nodule to tender points. The American College of Rheumatology [ACR] criteria for the classification of fibromyalgia [FMS] no longer require careful palpation of individual muscles for increased tone, muscle shortening, enthesopathic tendons, restricted joint range and other signs. The ACR diagnosis relies on a count of standard tender points. Simons maintains that FMS is an entirely different condition from myofascial pain syndrome, but, is it a distinct syndrome? Both FMS and myofascial pain syndrome have features of a peripheral neuropathic disorder [such as, widespread aching point tenderness, skin fold tenderness, articular pain, swelling of the hands or knees, numbness or coldness of the extremities, reticular skin discoloration, irritable bowel and trophedema]. Far from being a distinct syndrome, FMS represents the most extreme and extensive of the mundane aches and pains that we all have in various degrees at one time or other. Mildly tender points are not unusual in asymptomatic individuals especially after strenuous physical activity, and moderately tender points are not exceptional in those who have a history of a "vulnerable" spine (8). Myofascial pain syndrome patients also have multiple tender points. Even in localized conditions, such as lateral epicondylitis, examination will reveal numerous tender sites scattered throughout the body (9).

Those FMS proponents who prefer the "hands-off" approach and limit their physical examination to a tender point count, are using the wrong end of the telescope. They distance themselves from the patient, and view musculoskeletal pain from afar. They, too, will miss out on the full picture.

3. The "Overall" view—the radiculopathy model

The radiculopathy model describes a large category of pathophysiologic conditions that occur when there is some functional disturbances or pathological changes in the PNS, i.e., peripheral neuropathy. The model explains the TrP and its associated myofascial pain phenomena as secondary hyperalgesia of peripheral neural origin.

Simons: How does a mild peripheral neuropathy produced by only a modestly severe radiculopathy cause the development of trigger points?

"Radiculopathy" equals "peripheral neuropathy occurring at the nerve root." They are identical pathophysiological conditions.

Pain is a singular experience that depends on activity in specific receptors, neurons and interneuronal circuits—there is not a simple one-to-one relationship between activity in the primary afferent nociceptor and the perceptual experience (16). In peripheral neuropathy, supersensitive structures can respond excessively to low intensity stimuli and give rise to the perception of allodynia, and pain—sensitivity has been shown to increase by 1000 times (10). Supersensitivity can cause muscle to become tender and TrPs to develop. Low-intensity stimulation can be perceived as painful mechanical allodynia when there is supersensitivity in A β fibers. High intensity stimulation, which is normally painful, can lead to exaggerated pain response or hyperalgesia when there is supersensitivity in C and A δ fibers (11,12).

Simons: How commonly do trigger points develop without any evidence of neuropathy?

Spondylosis is near-universal, and it is by far the most common cause of radiculopathy. Ordinarily, spondylosis follows a gradual, relapsing, and remitting course that is silent, unless and until symptoms are precipitated by an incident, often so minor, that it passes unnoticed by the patient. All gradations of spondylosis exist, but early or incipient spondylotic changes, even when unsuspected, can cause radiculopathy.

The manifestations of neuropathic/radiculopathic dysfunction are motor, sensory and autonomic. In our studies, early and subtle signs of peripheral neuropathy were found in a significant number of young [under 30 years], apparently normal, and asymptomatic individuals (3,8,9). Motor manifestations are the first to occur and radiculopathy can occur without pain. Muscle-shortening is an early feature of radiculopathy because large diameter nerve fibers at the nerve root, axons of motoneurons and myelinated primary afferents [muscle proprioceptors] are the first to suffer physically. Painless, tight muscle knots can be felt in most individuals, and not uncommonly, even in

toddlers. Pain is not a feature of radiculopathy unless nociceptive pathways are involved. Many neuropathies are pain-free, such as sudomotor hyperactivity in hyperhidrosis, and muscle weakness in ventral root disease.

Simons: Gunn's article raises a number of questions that deserve competent research investigation

The radiculopathy model has evolved from clinical observations and competent clinical research on chronic pain carried out over a period of more than 20 years. In 1973, frustrated by the generally unsatisfactory results of conventional physical therapies for chronic pain, patients who had chronic back pain but no obvious signs of ongoing nociception were carefully examined. The significant finding was that tenderness in muscles in affected myotomes were sensitive indicators of radicular involvement and differentiated a simple mechanical low back strain from one with neural involvement (8).

A study of patients with "tennis elbow" [and a similar study on shoulder pain] showed that tender points in the upper limb were related to cervical spondylosis and radiculopathy. Treating the neck, but not the arm provided relief (9).

A pattern began to emerge—patients who have pain but no obvious signs of injury, generally have subtle but discernible signs of peripheral nerve involvement affecting all components of the PNS—motor, sensory, autonomic and trophic. This is an important observation because there is no satisfactory laboratory or imaging test for early neural dysfunction.

Dryneedling was tested for low back pain in a randomized clinical trial: the group treated with needling was found to be significantly better than the control group. [These papers were determined as significant studies by the 1979 Volvo Competition Awards Committee.]

Simons: The rationale by which disuse supersensitivity and peripheral neuropathy can cause a shortened muscle syndrome was unexplained.

The largest component of the PNS is the musculoskeletal system, therefore, the impact of neuropathy is most apparent in muscle.

Neuropathic muscle is almost always shortened from contracture. Simon and Travell have proposed a hypothesis to explain the absence of action potentials, localized tenderness, and the prompt release of the taut band by inactivation of the TrP (13). They proposed that contracture was associated with a local energy crisis in the muscle produced by increased metabolic demand of contracted sarcomeres in the face of ischaemia-induced hypoxia which resulted from the vigorous sustained contraction. Their hypothesis remains unproven and does not explain the physiology of contracture. Features such as hypoxia, myogelosis, contraction knots are the products of

muscle shortening. Muscle shortening is not caused by palpable bands—on the contrary, a muscle shortens into palpable bands.

Classic contracture, the evoked shortening of a muscle fiber in the absence of action potentials, requires no conjured-up hypothesis. Physiologists explain it with Cannon and Rosenblueth's law of denervation which applies notably to skeletal muscle. Four types of increased sensitivity can occur: lessened stimuli which do not have to exceed a threshold produce responses of normal amplitude [increased susceptibility]; the threshold of the stimulating agent is lower than normal [hyperexcitability]; the capacity of the muscle to respond is augmented [super-reactivity]; and the amplitude of response is unchanged but its time-course is prolonged [super-duration of response].

Supersensitive skeletal muscle fibers can overreact to a wide variety of chemical and physical inputs including stretch and pressure. They have a lowered threshold to acetylcholine [which may be increased from reduced levels of acetylcholine esterase]. Acetylcholine slowly depolarizes supersensitive muscle membrane and this induces electromechanical coupling with the consequent slow development of tension *without* action potentials (14).

Furthermore, in normal muscle, acetylcholine acts only at receptors that are situated in the narrow zone of innervation. But in neuropathy, acetylcholine acts at newly formed extrajunctional receptors ["hotspots"] that are present throughout the muscle (1,10) [Figure 1].

Simons: The main difference between dryneedling and injecting an anesthetic is that the anesthetic relieves much of postinjection soreness which often appears the next few days.

Intramuscular Stimulation has many advantages over injection techniques and it avoids the iatrogenic effects of injected medications. To therapists who are familiar with both techniques, IMS is the preferred method as it can be used for diagnosis and localization of the contracture. For example, when paraspinal muscles at consecutive segmental levels are needled, resistance to needle penetration is substantially increased at the involved segmental level[s] as compared to the levels above and below. The fine, solid, needle [borrowed from acupuncture], accurately discerns the position and status of deep muscles [3 inch needles are commonly used]. The pointed needle causes minimal tissue trauma, unlike the cutting edge of a hollow needle, and post-needling soreness is minimal. When correctly used, IMS permits extensive needling of many muscles; sometimes, up to a hundred needle insertions may be given in one session.

A great advantage of using the whippy acupuncture needle is to elicit the Deqi sensation and needle grasp. It is not possible to understand neuropathic pain without having personally experienced the Deqi phenomenon. Neuropathic pain has a peculiar, cramp-like quality which is referred to in acupuncture

FIGURE 1a

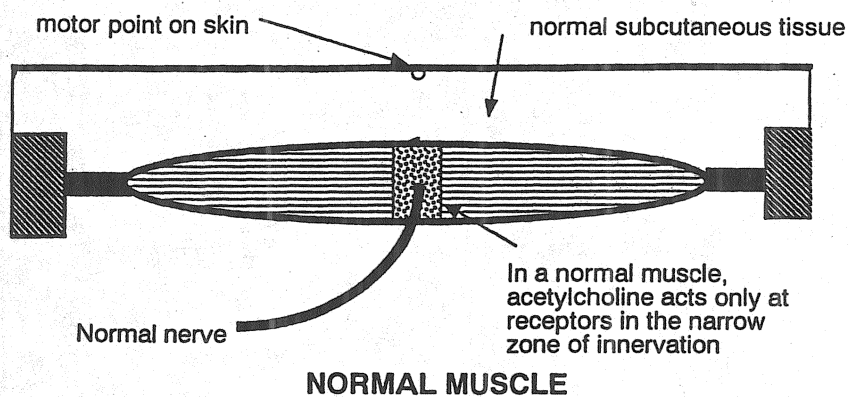
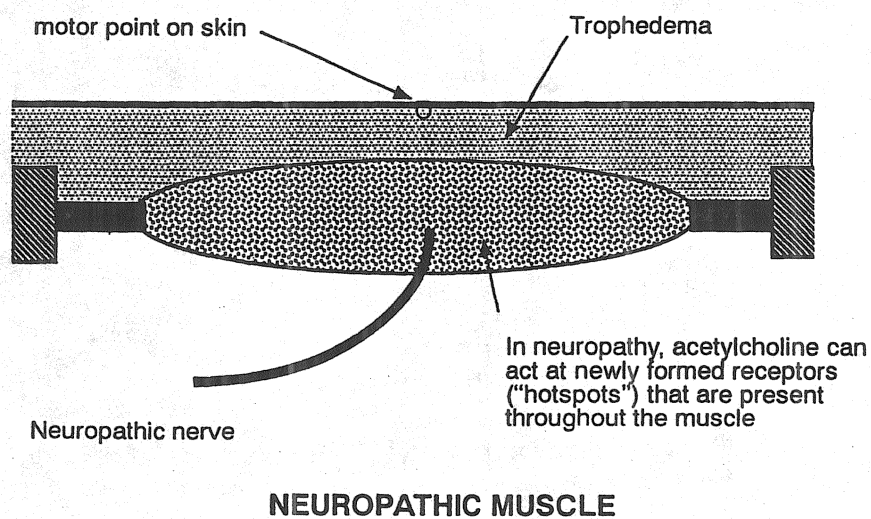


FIGURE 1b



Reproduced with kind permission from *The Gunn Approach to the Treatment of Chronic Pain*, C. Chan Gunn, Churchill Livingstone, London, 1996.

ture literature as the Deqi Phenomenon (15). According to Fields (16), this strange quality of neuropathic pain probably results from disruption of the sensory apparatus so that a normal pattern of neural activity is no longer transmitted to the perceptual centers. Neuropathic pain is due, in part, to the activation of nociceptive neurons [because the message that reaches these centers is clearly unpleasant] and patients recognize that the sensations are not "normal" pain sensations. The Deqi sensation is closely associated with receptors that sense muscle shortening [proprioceptors]. It is important to differentiate between pain that has the Deqi [neuropathic] response, and pain that does not [nociceptive]. This distinction is crucial because of the difference in the nature and treatment of the two types of pains.

Simons: The statement by Gunn that "persistent nociception is not a common cause of chronic pain" flies in the face of the last five years of pain neurophysiological research.

Our studies have not shown ongoing nociception [that is, the constant agitation of nociceptors by tissue damaging impulses, such as, an unhealed fracture] to be a common cause of persistent pain.

Contrary to Simons' statement, Gunn, over 20 years ago, proposed that pain can be produced by the development of supersensitive receptor organs and hyperreactive control systems (3). Radiculopathy leads to a large number of neuropathic disorders: it is important to realize that radiculopathy can masquerade as any one of many seemingly unlike syndromes—myofascial pain is only one of them.

Simons: Clearly muscle overload plays an important role.

Normal muscle is very resilient, but in neuropathy the abnormal, shortened muscle is made up of collagen that has been weakened by neuropathic degradation (17). Muscle overload can then be an important factor.

Simons: Ischemia . . . is the result of mechanical compression of circulation due to muscle fiber contracture . . .

Other decentralized and supersensitive structures in neuropathy include smooth muscle, sympathetic ganglia, spinal neurones and associated spinal reflexes. Neuropathic pain can be most daunting when it is dominated by vasomotor changes, e.g., complex regional pain syndrome [viz, reflex sympathetic dystrophy, causalgia]. Vasomotor, sudomotor and pilomotor changes are also important epiphenomena of radiculopathy and Simons misunderstands the role of vasoconstriction in myofascial pain when he attributes ischaemia simply to muscle fiber contracture. The sympatholytic effect of

acupuncture and IMS generally occurs within minutes, is clinically evident, can be recorded by thermography, and is well known to experienced needle practitioners (18).

Simons: Gunn proposed that insertion potentials represent an outburst of injury potentials . . .

According to Johnson (19), insertional activity is a much-misinterpreted step in the needle examination. By definition, it results from moving the electromyographic needle briskly through the muscle and mechanically discharging some muscle fibers resulting in a burst of injury potentials. These potentials are single muscle fiber discharges resulting from disruption of the muscle cell membrane by the tip of the needle. The character of the insertional activity may be described as increased when the muscle cell membranes are extraordinarily hyperirritable.

The electrical discharges can be seen in the electromyograph to coincide with the release of muscle shortening: they produce a potent reflex stimulation into the entire segment (1). For example, releasing paraspinal contracture in the lumbar back can release muscle shortening and disperse trigger points in the leg. [Also see letter from Chu (20).]

SUMMARY

Simons' enquiries demonstrate his great interest and experience in myofascial pain. Gunn approached musculoskeletal pain from a different direction—examination of the disabled workman. During his examination of the *whole* individual, Gunn discovered the TrP as only one part of the total clinical picture of neuropathic pain. Intramuscular stimulation was developed from studying traditional acupuncture-tender points in muscles had been described in Chinese literature [as Ah-Shi points] in the eighth century.

Intramuscular stimulation has been in use since 1976; its effectiveness has been proven and published. The beneficial effects of IMS on musculoskeletal pain are self-evident.

REFERENCES

1. Gunn CC: The Gunn Approach to the Treatment of Chronic Pain—Intramuscular Stimulation for Myofascial Pain of Radiculopathic Origin. Churchill Livingstone, London, 1996.
2. Gunn CC: Radiculopathic pain: diagnosis and treatment of segmental irritation or sensitization. *J Musculoske Pain* 5(4): 119-134, 1997.

3. Gunn CC: "Prespondylosis" and some pain syndromes following denervation supersensitivity. *Spine* 5(2):185-192, 1978.
4. Gunn CC: Neuropathic pain: a new theory for chronic pain of intrinsic origin. *Annals of The Royal College of Physicians and Surgeons of Canada* 22(5):327-330, 1989.
5. Thomas PK: Symptomatology and differential diagnosis of peripheral neuropathy: clinical features and differential diagnosis. In: *Peripheral Neuropathy*. Edited by Dyck PJ, Thomas PK, Lambert EH, Bunge R. WB Saunders, Philadelphia, 1984, Vol. II, pp. 1169-1190.
6. Gunn CC, Milbrandt WE: Dry needling of muscle motor points for chronic low-back pain; a randomized clinical trial with long-term follow-up. *Spine* 5(3):279-291, 1980.
7. Bonica JJ: *The Management of Pain*. Lea & Febiger, Philadelphia 1990, pp. 389-392, 1432-1433, 1454, 1500.
8. Gunn CC, Milbrandt WE: Tenderness at motor points—a diagnostic and prognostic aid for low back injury. *J Bone Joint Surg* 58:815-825, 1976.
9. Gunn CC, Milbrandt WE: Tennis elbow and the cervical spine. *CMA Journal* 114:803-809, 1976.
10. Lomo T: The role of activity in the control of membrane and contractile properties of skeletal muscle. In: *Motor Innervation of Muscle*. Edited by Thesleff S. Academic Press, New York, 1976, pp. 289-315.
11. Munglani R, Hunt SP, Jones JG: The spinal cord and chronic pain. In: *Anaesthesia Review* 12. Edited by Kaufman L and Ginsburg R. pp. 53-75.
12. Woolf CJ: The dorsal horn: state dependent sensory processing and the generation of pain. In: *Textbook of Pain* (3rd edition). Edited by Melzack R, Wall PD. Churchill Livingstone, Edinburgh, 1994, pp. 101-112.
13. Simons DG, Travel JG: Myofascial trigger points, a possible explanation. *Pain* 10:106-109, 1981.
14. Eyzaguirre C, Fidone S: *Physiology of the Nervous System*. Year Book Medical Publishers, Chicago, 1977, pp. 375-382.
15. Gunn CC: Transcutaneous neural stimulation, needle acupuncture and 'Teh Ch'i' Phenomenon. *Am J Acupuncture* 4(4):317-322, 1976.
16. Fields HL: *Pain*. McGraw-Hill, New York, 1987, pp. 133-169.
17. Klein L, Dawson MH, Heiple KG: Turnover of collagen in the adult rat after denervation. *J Bone Joint Surg* 59A:1065-1067, 1977.
18. Liao SJ, Lee MHM, Ng LKY: *Principles and Practice of Contemporary Acupuncture*. Marcel Dekker, Inc, New York, 1994.
19. Johnson EW, Pease WS: *Practical Electromyography*, ed 3. Williams & Wilkins, Baltimore, 1997.
20. Chu J: Comment on the Simons Literature Review Column, "myofascial pain syndrome-trigger points." *J Muskuloske Pain* 5(1):133-135, 1997.

Myofascial Pain– A Radiculopathy Model [Comment on Gunn Research Ideas Article]

Peter Baldry

The concept put forward by Dr. Chan Gunn (1) that myofascial trigger point [MTrP] pain is neuropathic is, in my view, open to challenge.

This is because there are grounds for believing the MTrP pain, which is of a diffuse, dull aching type and quite unlike the burning or electric shock-like pain that develops when there is damage to the peripheral or central nervous system, most commonly arises as a result of trauma-induced activation and sensitization of nociceptors at MTrP sites. And is, therefore, primarily nocigenic.

Admittedly, nerve entrapment pain such as that, for example, which may be brought about by a ruptured intervertebral disc, gives rise to muscle spasm. And that as a result of the muscle ischemia created by this spasm, nociceptors at MTrP sites may become activated with, as a consequence, the development of MTrP pain. Despite this latter type of pain, therefore, having developed as a result of radiculopathy it is still, nevertheless, nocigenic.

Also, although MTrP nociceptor activity gives rise not only to pain but also to muscle shortening, and the latter in certain parts of the body leads to compression of a nerve with, as a consequence, the development of a sensory and/or a motor deficit, the MTrP pain is nocigenic.

The views of others concerning this subject would be of interest.

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REFERENCE

1. Gunn CC: Radiculopathic Pain: Diagnosis and treatment of segmental irritation or sensitization. *J Musculoske Pain* 5(4): 119-134, 1997.

Reply to Dr. Baldry

C. Chan. Gunn

Baldry has challenged Gunn's concept that myofascial trigger point [MTrP] pain is neuropathic (1). He maintains that:

1. . . . MTrP pain, which is of a diffuse, dull aching type and quite unlike the burning or electric shock-like pain that develops when there is damage to the peripheral or central nervous system, most commonly arises as a result of trauma-induced activation and sensitization of nociceptors at MTrP sites. And is, therefore, primarily nocigenic.

Baldry follows traditional medical wisdom in that he presumes pain to be a signal of tissue injury, conveyed to the central nervous system via a healthy nervous system. He holds on, perhaps too tightly, to the definition of pain according to The International Association for the Study of Pain: "an unpleasant sensory and emotional experience associated with actual or potential tissue damage, or described by the patient in terms of such damage."

It is well known, however, that pain need not be "trauma-induced." Injury does not always generate pain, nor does pain always signal injury. Pain perception can arise from non-noxious input, and spurious pain can arise from within the body when there is some functional abnormality in the nervous system, e.g., neuropathic pain (1-3).

"Nocigenic" simply means "pain-causing." It does not describe a physiological process. Baldry had probably intended to signify "nociceptive." Nociception involves the stimulation of nociceptors—primary afferent nerves [A δ mechanosensitive/mechanothermal and C-polymodal] that can transduce specific forms of energy [mechanical, thermal or chemical] into electrochemical nerve impulses and transmitting them to the central nervous system.

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Patients with neuropathic pain typically show no evidence of tissue injury, instead, pain is caused by abnormal function in the nervous system. Gunn had proposed that following damage to a peripheral nerve, abnormally sensitive receptor organs and hyperreactive control systems can develop (2,3). It is now known that irritation of a nerve root by spondylosis or inflammation [e.g., in arthritis] can lead to a cascade of molecular events in the nerve and spinal cord [such as, activation of the N-methyl-D-aspartic acid channel, increase in intracellular Ca^{2+} , wind-up wide dynamic range neuron sensitization and other phenomena]; these can supersensitize the pain sensory system (4,5). A supersensitive pain sensory system can respond inappropriately and overwhelmingly to non-noxious stimuli to give rise to pain. Lomo has shown sensitivity to increase by 1000 times (6). Supersensitivity can cause muscle to become tender and TrPs to develop. Low-intensity stimulation can be perceived as painful mechanical allodynia when there is supersensitivity in A β fibers. And high intensity stimulation, which is normally painful, can lead to exaggerated pain response or hyperalgesia when there is supersensitivity in C and A δ fibers.

2. Baldry: Nerve entrapment pain, such as that, for example, which may be brought about by a ruptured intervertebral disc, gives rise to muscle spasm. And that as a result of the muscle ischemia created by this spasm, nociceptors at MTrP sites may become activated with, as a consequence, the development of MTrP pain. Despite this latter type of pain, therefore, having developed as a result of a radiculopathy it is still, nevertheless, nocigenic.

Pressure on a nerve root can cause local tissue damage, but since the nerve root is not well provided with nociceptors, any pain that may arise, will not be from nociception at the entrapment site.

Baldry has suggested a roundabout route for pain to materialize from radiculopathy, but any pain from abnormal nerve function, whatever its cause, is neuropathic pain.

3. Baldry: MTrP pain, which is of a diffuse, dull aching type . . .

Myofascial muscle pain is not merely dull and aching: it has a peculiar, cramp-like quality which is associated with muscle tenderness and shortening (7,8). As an experienced acupuncturist, Baldry should be aware of this distinctive sensation [which is referred to in acupuncture literature as the Deqi or Teh Ch'i Phenomenon]. The classical acupuncturist painstakingly differentiates between pain that has the Deqi [neuropathic] response, and pain that does not [nociceptive]. This distinction is important because of the difference in the nature and treatment of the two pains.

According to Fields (9), the strange quality of neuropathic pain probably results from disruption of the sensory apparatus so that a normal pattern of neural activity is no longer transmitted to the perceptual centers. He accepts that neuropathic pain probably activates nociceptive neurons [because the message that gets through to the perceptual centers is clearly unpleasant], but comments that patients recognize these peculiar sensations are not "normal" pain sensations (8).

Deqi pain sensations are not "normal" because they are associated with receptors that sense muscle shortening [proprioceptors]. The classical acupuncturist demonstrates this by the "needle-grasp" occurring at the site of penetration when a neuropathic muscle is needled. Needling is usually pain-free when an acupuncture needle enters a normal muscle, but when the needle pierces a shortened muscle, it produces a cramp-like sensation, and the needle is observed to be firmly grasped by the shortened muscle. The intensity of the needle grasp parallels the degree of muscle shortening (8,10), and it gradually eases off during treatment as muscle shortening is released [usually within minutes]. Because muscle pain eases simultaneously with the release of the needle grasp, patients soon become aware of the importance of eliciting the Deqi sensation during treatment. It is true to say that a therapist must personally experience the Deqi sensation in order to fully appreciate the quality of neuropathic pain.

For needling to be fully effective, the needle must penetrate the shortened muscle—whether it is superficial or deep. Superficial dry-needling into skin and subcutaneous tissues, without penetrating muscle (11), cannot yield the Deqi response nor its beneficial effect.

COMMENTS

It is only through hands-on examination of patients, explicitly searching for neuropathic signs, that one is able to gain proficiency with this, the largest and most important, category of chronic musculoskeletal pain. It is a most convincing experience, to diagnose neuropathic pain by finding its unmistakable physical signs (12,13), then to treat the patient and witness these signs disappear, usually within minutes. Baldry is always welcome at the Multidisciplinary Pain Center, or The Institute for the Study and Treatment of Pain.

REFERENCES

1. Gunn CC: Radiculopathic pain: diagnosis and treatment of segmental irritation or sensitization. *J Musculoske Pain* 5(4):119-134, 1997.
2. Gunn CC: "Prespondylosis" and some pain syndromes following denervation supersensitivity. *Spine* 5(2):185-192, 1978.

3. Cannon WB, Rosenblueth A: The supersensitivity of denervated structures, a law of denervation. New York: The MacMillan Company, 1949.
4. Munglani R, Hunt SP, Jones JG: The spinal cord and chronic pain. In: Anaesthesia Review 12. Edited by Kaufman L and Ginsburg R. pp. 53-75.
5. Woolf CJ: The dorsal horn: state dependent sensory processing and the generation of pain. In: Textbook of Pain (3rd edition). Edited by Melzack R, Wall PD. Churchill Livingstone, Edinburgh, 1994, pp. 101-112.
6. Lomo T: The role of activity in the control of membrane and contractile properties of skeletal muscle. In: Motor Innervation of Muscle. Edited by Thesleff S. Academic Press, New York 1976, pp. 289-315.
7. Gunn CC: Transcutaneous neural stimulation, needle acupuncture and 'Teh Ch'i' Phenomenon. Am J Acupuncture 4(4):317-322, 1976.
8. Chapman CR, Gunn CC: Acupuncture (dry-needling), Chapter 90. In: The Management of Pain. Edited by Bonica JJ. Lea & Febiger, 1989.
9. Fields HL: Pain. McGraw-Hill, New York, 1987, pp. 215-216.
10. Gunn CC: The Gunn Approach to the Treatment of Chronic Pain, Intramuscular Stimulation for Myofascial Pain of Radiculopathic Origin. Churchill & Livingstone, 1997.
11. Baldry P: Superficial dry needling at myofascial trigger point sites. J Musculoske Pain 3(3):117-126, 1995.
12. Bonica, JJ, Loeser, JD: Medical evaluation of the patient with pain. Chapter 30. In: The Management of Pain. Edited by Bonica JJ. Lea & Febiger, 1989.
13. Gunn CC: Mechanical manifestations of neuropathic pain. Annals of Sports Medicine 5(3):138-141, 1990.

LETTERS

Comment on Gunn's "Radiculopathy Model of Myofascial Trigger Points"

Chang-Zern Hong

Gunn has proposed a radiculopathy model for the development of myofascial trigger points [MTrPs] (1-3). The concept that MTrPs are exclusively neurogenic [initiated or caused by radiculopathy] has been challenged by Baldry (4) and Simons (5).

In the reply letter to Simons (3), Gunn has listed two of his previous important studies (6,7) to support his "radiculopathy model." I totally agree with the conclusion that radiculopathy *can* cause MTrPs in the distribution of corresponding myotome. However, just based on these two studies, it is unrealistic to reach the conclusion that *all* MTrPs are initiated or caused by radiculopathy. The "early and subtle signs" of radiculopathy are actually also MTrP signs [tenderness and taut bands]. In fact Gunn has stated that "... This is an important observation because there is no satisfactory laboratory or imaging test for early neural dysfunction" (3). He did not prove the existence of radiculopathy in all myofascial pain syndrome patients. It appears that Simons' questions were not adequately answered.

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There is evidence that does not support the concept that "all myofascial pain, including that of MTrPs, originates as a radiculopathic neuropathy."

1. Myofascial trigger points can cause neuropathy and the neuropathy can be cured after MTrPs are effectively treated. This has been well documented in Travell and Simons' Trigger Point Manual (8,9). A brachial plexus lesion due to pectoralis minor syndrome can be cured by treating pectoralis minor MTrPs (10).
2. In clinical practice, MTrPs caused by [or related to] traumatic injury [but not degenerative lesions] of tendon/ligament can be cured completely by effectively treating the associated tendon/ligament lesions (11).

Gunn's hypothesis would be more acceptable if it were applied to the etiology of latent MTrPs rather than active ones. Latent MTrPs may be formed due to subclinical or mild peripheral neuropathic or radiculopathic process. Then Gunn's concept does not contradict to the concept that a latent MTrP can be activated by either a neuropathic or a non-neuropathic lesion. The nature of the interaction between the dysfunctional endplate and its anterior horn cell [motoneuron] raises many important questions that must be resolved by research experiments.

Gunn has disputed Simons' "energy crisis theory." He has described the "slow depolarization without action potentials" of the supersensitive muscle membrane by acetylcholine, which is present throughout the entire muscle (3). In such case, all sarcomeres in an affected muscle fiber are shortened due to contracture. However, the contraction knots can be microscopically observed in the endplate zone, but not the whole muscle fiber (8). Up to date, I can not find any scientific evidences that are directly against Simons' "energy crisis theory." In fact, Gunn has ignored the studies that have strongly supported this theory (8).

Gunn has also criticized rheumatologists concerning the fibromyalgia syndrome [FMS]. He stated that "Both FMS and myofascial pain syndrome have features of a peripheral neuropathic disorder" (3). How can he explain the increased substance P and reduced serotonin level in cerebrospinal fluid of FMS patients as demonstrated by Russell (12)?

In conclusion, Gunn's theory may be more acceptable if it were applied on latent MTrPs [but not all active MTrPs]. On the other hand, Simons' theory has still not been disproved.

REFERENCES

1. Gunn CC: "Prespondylosis" and some pain syndrome following denervation supersensitivity. *Spine* 5(2):185-192, 1978.
2. Gunn CC: Radiculopathic pain: Diagnosis and treatment of segmental irritation or sensitization. *J Musculoske Pain* 5(4):119-134, 1997.
3. Gunn CC: Reply to Dr. Simons [Letters to the Editor]. *J Musculoske Pain* 6(4):129-138, 1998.
4. Baldry P: Myofascial pain—a radiculopathy model [comment on Gunn Research ideas article]. *J Musculoske Pain* 6(4):117-118, 1998.
5. Simons DG: Experimental resolution of the myofascial pain enigma [comment on Gunn Research ideas article]. *J Musculoske Pain* 6(4):123-127, 1998.
6. Gunn CC, Milbrandt WE: Tenderness at motor points—a diagnostic and prognostic aid for low back injury. *J Bone Joint Surg* 58:815-825, 1976.
7. Gunn CC, Milbrandt WE: Tennis elbow and the cervical spine. *CMC Journal* 114:803-809, 1976.
8. Simons DG, Travell JG, Simons LS: Travell & Simons's Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol. 1, second edition, Williams & Wilkins, Baltimore, 1999.
9. Travell JG, Simons DG: Myofascial Pain and Dysfunction: The Trigger Point Manual, Vol. 2, Baltimore: Williams & Wilkins, 1992.
10. Hong C-Z, Simons DG: Response to standard treatment for pectoralis minor myofascial pain syndrome after whiplash. *J Musculoske Pain* 1(1):89-131, 1993.
11. Hong C-Z: Consideration and recommendation of myofascial trigger point injection. *J Musculoske Pain* 2(1):29-59, 1994.
12. Russell IJ. Neurochemical pathogenesis of fibromyalgia syndrome. *J Musculoske Pain* 4(1/2):61-92, 1996.

Reply to Chang-Zern Hong

C. Chan Gunn

Hong: “. . . Gunn did not prove the existence of radiculopathy in all MPS patients.”

Although Hong totally agrees with the Gunn Radiculopathy Model that radiculopathy can cause myofascial trigger points [MTrPs] in the distribution of its corresponding myotome, he states that it is unrealistic, based on only two studies, to reach the conclusion that all MTrPs are initiated or caused by radiculopathy. That was also our opinion in 1976, when we published our study on patients with elbow pain and cervical radiculopathy. At that time, we had recognized that it was “obviously not possible to draw definite conclusions from this small series . . . yet the findings challenge current concepts” (1).

However, signs of radiculopathy are, by now, well known—Bonica had recommended them for the evaluation of the patient with pain (2). We have, since 1976, found that tenderness and other signs of peripheral nerve involvement are virtually always present in patients with myofascial pain. Physicians and therapists at the Multidisciplinary Pain Center and the Institute for the Study and Treatment of Pain [ISTOP] routinely examine for and regularly find these signs, readily validating the radiculopathy model. For example, from September 1, 1997 to September 1, 1998, the Institute admitted 1,021 myofascial pain patients for treatment—almost every patient demonstrated radiculopathic signs.

Hong: “MTrPs can cause neuropathy and the neuropathy can be cured after MTrPs are effectively treated . . . ”

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A peripheral neuropathy may be defined as a disease which causes disordered structure and/or function of the peripheral nerve. The causes of peripheral neuropathy include several hundred diseases—trauma, infection, metabolic disease, old age, vitamin deficiencies, toxins, hereditary, carcinoma and tumor, collagen-vascular, radiation, tropical disease and idiopathic (3). The MTrP is not usually considered as a cause of neuropathy. On the contrary, the MTrP represents mechanical allodynia located in a palpable contracture band and is, therefore, an outcome of neuropathy.

Hong: “. . . a brachial plexus lesion due to the pectoralis minor syndrome can be cured by treating pectoralis minor MTrPs.”

Hong is right. The “pectoralis minor syndrome” is produced by shortening in that muscle entrapping the brachial plexus. Treating the shortened pectoralis minor muscle releases the contracture and relieves the entrapment.

Hong: “. . . MTrPs caused by [or related to] traumatic injury [but not degenerative lesions] of tendon/ligament can be cured completely by effectively treating the associated tendon/ligament lesions.”

The MTrP is not an entity within muscle created by traumatic injury. It is, instead, a manifestation of radiculopathy resulting from trauma to the nerve. Neuropathic pain does not result from tissue injury, but from faulty processing in the nervous system which causes supersensitivity in receptors, neurons and spinal cord interneuronal circuits. Myofascial trigger points can occur at the musculotendinous junction but not within tendon or ligament, as these are not appropriately innervated.

Hong declares that a MTrP “can be cured completely,” but a patient is not “cured” unless all physical evidence of radiculopathy has been found, and eliminated with treatment. When an entrapped nerve root is released, all manifestations of radiculopathy—sensory, motor and autonomic—including TrPS, will disappear together. It is a gratifying experience, to diagnose neuropathic pain by finding its unmistakable physical signs, then to treat the patient with intramuscular stimulation and witness the signs disappear, often within minutes. Findings that

can be objectively verified are important in these days of evidence-based medicine.

Hong: “. . . a latent MTrP can be activated by either a neuropathic or non-neuropathic lesion . . . ”

Whether a MTrP is “latent” or “active” depends on the condition of the nerve. In early spondylosis, the large-diameter nerve fibers—axons of motoneurons and myelinated primary afferents [muscle proprioceptors]—are the first to suffer physically. Motor manifestations are therefore the first to occur, and muscle shortening is usually the first feature of radiculopathy. Painless, tight muscle knots can be felt in most individuals, and they *precede* allodynia and pain. A “latent” MTrP becomes “active” when there is peripheral and central sensitization. Pain becomes a feature when low-intensity stimulation [transmitted via A-beta fibers] is perceived as painful mechanical allodynia [tenderness], and high-intensity stimulation, which is normally painful [transmitted via C and A-delta fibers], causes the exaggerated pain-response of hyperalgesia.

Hong: “. . . Gunn has disputed Simon’s ‘energy crisis theory’ ”

Simon’s hypothesis proposed that contracture is associated with a local energy crisis in muscle, produced by increased metabolic demand of contracted sarcomeres, in the face of ischemia-induced hypoxia which result from vigorous sustained contraction. Gunn prefers the generally accepted physiological explanation for a contracture as the exaggerated and protracted response of supersensitive skeletal muscle fibers to chemical and physical input (4). In denervation, shortened sarcomeres can be present throughout the entire muscle—released acetylcholine now acts not only at motor end-plates, but also at newly formed clusters of extrajunctional receptors [“hotspots”] spread out in the muscle (5). However, Hong’s statement that shortened sarcomeres can affect some muscle fibers only in the end-plate zone may also be valid when there is partial denervation.

Hong: “. . . how can he explain the increased substance P and reduced serotonin level in cerebrospinal fluid of FMS patients as demonstrated by Russell?”

Russell reported a wide range of complex biochemical abnormalities in fibromyalgia [FMS], including increased substance P [SP] and

reduced serotonin level in the cerebrospinal fluid. He noted that SP can also be elevated in other painful conditions, such as osteoarthritis in the hip, and it can decrease after hip replacement and pain relief. The changes described by Russell are probably related to those found in central sensitization, which follows peripheral sensitization, when tissue inflammation and nerve injury has caused the nerve to respond much more vigorously to stimulation. These changes cause dramatic and long lasting changes in the spinal cord, including the release of neurotransmitters, such as SP, glutamate, neurokinin A, and calcitonin gene-related peptide.

In neuropathic pain, large-diameter primary afferent neurons that normally transmit non-noxious stimuli now start to express SP [which is normally associated with small-diameter C fibers that transmit pain and temperature]. The exact significance of this phenotypic switch by large-diameter fibers is not known, but it may explain how light touch and proprioceptive information [that are carried by A-Beta fibers] may be misinterpreted as pain by the spinal cord. Tissue inflammation and nerve injury also activates the N-methyl-D-aspartate receptor and cause changes in neuropeptide levels (6,7).

Hong: “. . . *Gunn's hypothesis would be more acceptable if it were applied to the etiology of latent MTrPs rather than active ones.*”

The radiculopathy model is not a hypothesis. It is a description of clinical findings that can be found by anyone who examines a patient for radiculopathy. The signs are consistent and not exceptional. The model explains how assorted pain syndromes, such as tennis elbow or low back pain, can belong in one classification; and the key to successful treatment is to recognize radiculopathy in its many disguises.

Both FMS and myofascial pain syndrome have features of a peripheral neuropathic disorder. Are they separate entities, or varying degrees of one condition? At the Pain Center and ISTOP, FMS is rarely an admitting diagnosis. Typically, a tentative diagnosis of radiculopathic pain is made when sensory, motor and autonomic features of radiculopathy are found in the dermatomal, myotomal, and sclerotomal structures of both rami of the segmental nerve. All of these must be confirmed, especially the motor manifestation of neuropathy. [Examination for radiculopathic signs is essential, but the technique needs to be learned.]

A deep contracture can *only* be demonstrated by needle exploration. It has been previously explained that the fine, flexible acupuncture needle used in intramuscular stimulation is a unique diagnostic tool for detecting deep contractures. When a needle pierces a contracture, it stimulates the muscle spindle and evokes the stretch or myotatic reflex. The muscle contracts, grasps the needle and causes the patient to experience the cramp-like Deqi phenomenon (8). The Deqi is a significant finding because the nature and treatment of neuropathic pain are different from those of nociceptive pain. Neuropathic pain yields the Deqi sensation and needle grasp, but nociceptive pain does not. Neuropathic pain responds to needle stimulation, but nociceptive pain does not.

How effective is needle stimulation? This depends on many factors such as the general health and age of the patient; the type and reversibility of the neuropathy [most neuropathies are of mixed pathology occurring at varying degrees together], but the most important factor is the experience and skill of the therapist.

Before labeling FMS as a distinct syndrome, it is important to recall that neuropathy is a condition for which there are several hundred possible causes (9). When neuropathy occurs at the nerve root, spondylosis is the most common cause. Muscle shortening, TrPS, and pain [which is not always present] are but epiphenomena of radiculopathy. When a patient does not demonstrate the Deqi response [which is rare], the cause of the pain has yet to be determined. The "FMS" label unfortunately does not add to our understanding of the condition.

REFERENCES

1. Gunn CC, Milbrandt WE: Tennis elbow and the cervical spine. *CMA Journal* 114:803-809, 1976.
2. Bonica JJ: The management of pain. Lea & Febiger, Philadelphia 1990, pp. 389-392, 1432-1433, 1454, 1500.
3. Thomas PK: Symptomatology and differential diagnosis of peripheral neuropathy: clinical features and differential diagnosis. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. *Peripheral neuropathy*. W.B. Saunders, Philadelphia 1984, Vol. II, pp. 1169-1190.
4. Eyzaguirre C, Fidone SJ: Physiology of the Nervous System. Year Book Medical Publishers, Chicago, 1975, pp. 375-382.
5. Lomo T: The role of activity in the control of membrane and contractile properties of skeletal muscle. In: Thesleff S, ed. *Motor innervation of muscle*. Academic Press, New York 1976, pp. 289-315.

6. Munglani R, Hunt SP, Jones JG: The spinal cord and chronic pain. In *Anaesthesia Review 12*. Edited by Kaufman L and Ginsburg R. pp. 53-75.
7. Woolf CJ: The dorsal horn: state dependent sensory processing and the generation of pain. In: Melzack R, Wall PD, Eds, *Textbook of pain* (3rd edition). Churchill Livingstone, Edinburgh, 1994, pp. 101-112.
8. Gunn CC: Transcutaneous neural stimulation, needle acupuncture and 'Teh Ch'i' Phenomenon. *Am J Acupuncture* 4 (4):317-322, 1976.
9. Thomas PK: Symptomatology and differential diagnosis of peripheral neuropathy: clinical features and differential diagnosis. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. *Peripheral neuropathy*. W.B. Saunders, Philadelphia 1984, Vol. II, pp. 1169-1190.