

FEATURE

The Muscle Spindle Trigger Point Model of Chronic Pain

Richard Gevirtz, PhD, BCIA-C

California School of Professional Psychology at Alliant International University, San Diego, CA

Keywords: trigger points, muscle spindle, chronic pain, sympathetic innervation

An explanatory model for chronic muscular pain is presented that incorporates recent findings in fields such as neuromuscular physiology, psychophysiology, and treatment outcome studies. Based on a sympathetically innervated muscle spindle feedback loop, the model has proved useful in constructing treatment and prevention protocols.

Introduction

The author and his colleagues recently completed a study (Oliveira, Gevirtz, & Hubbard, in press) that produced a remarkable result. One hundred twenty-six acute whiplash patients were randomly assigned to see a 12-minute video or to treatment as usual (TAU) in the emergency department (ED) or urgent care center of a number of Orange County, California, facilities and followed for 6 months. The video was designed to show the patient that the natural course of a whiplash injury often involves pain that persists beyond the 24- to 72-hour inflammatory stage and that this pain is often caused by trigger points (TPs) and can be sustained and aggravated by emotional tension or stress. The results were quite remarkable and beyond our most optimistic expectations. The video group, beginning at 1 month post-ED visit and continuing to the 6-month mark, had dramatically less pain, used less narcotic medications (36% for TAU, 2% for the video), did not seek surgical consultations, did not return to the ED, and so forth. The TAU group reported much more continued pain, use of narcotics, and consultations. We have just completed a replication of this study with patients with chronic (vs acute) low back pain and found a similar but less dramatic pattern (K. Gabriele, R. N. Gevirtz, & D. Hubbard, unpublished data).

These results have reinforced our belief that most chronic pain is muscular in nature and is caused by an area of local tenderness in the muscle that Janet Travell (who was John F. Kennedy's back physician) called a trigger point (Travell & Simons, 1983). But what exactly are TPs and how do psychological factors influence them?

In this article, I will trace the history of our work in this area over almost 20 years, which has led us to design treatments such as the video mentioned above.

Historical Perspective

The story starts in the 1960s with my colleague David Hubbard and me following quite disparate but converging paths. David was a philosophy major at Yale when he became fascinated by the mind/body problem. I was a psychology major at Wisconsin (philosophy minor) also fascinated by the age-old enigmas of how mind and body influence each other. David chose to pursue the problem by first studying counseling at Stanford and then medicine (neurology) and I by studying psychophysiology. He applied his training to the treatment of headache (especially tension headache), and I began using biofeedback for headache treatment as well as other "mind/body" disorders. We were both disturbed by the following anomaly: No credible mechanism for chronic head pain had been accepted in either the medical or psychological literatures. I had seen this in using surface electromyography (SEMG) in muscle pain syndromes. In the 1970s, I was fortunate to have taken a short course with Tom Budzinski, so using SEMG seemed a logical and effective manner of treating muscle pain. And though the treatment seemed to work, I was always puzzled by the lack of correspondence between pain, stiffness, and hardness of the muscles and the SEMG readings. The literature seemed to corroborate my observations. I can distinctly remember reading an article by Ken Holyroyd and colleagues in the *Journal of Behavioral Medicine* in 1980, in which the authors reported that raising forehead tension had the same effect as lowering it and that both biofeedback groups did better than the no-treatment controls (Holyroyd, Andrasik, & Noble, 1980). In countless instances, I had palpated a painful trapezius muscle finding it stiff to the touch with a "ropey nodularity," yet the SEMG registered levels as low as 1.5 μ V.

At the same time, David had been scouring the medical literature for physiological models that would

Needle EMG Activity

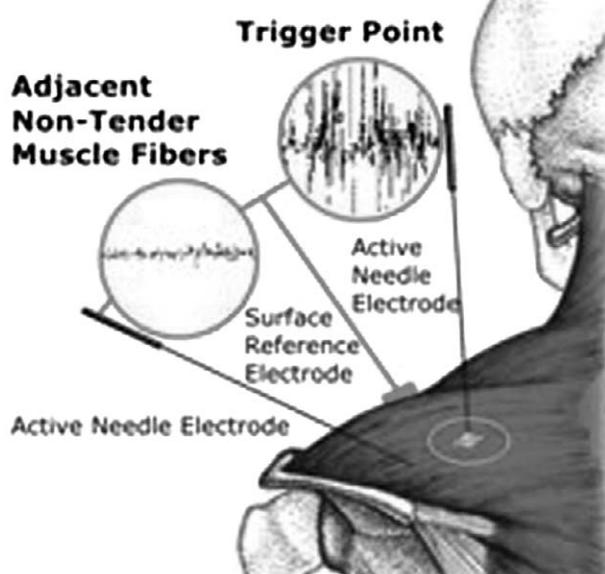


Figure 1. Needle electromyography paradigm. Left circle shows activity in the nontender site, right circle for the trigger point.

explain the chronicity of the pain and its apparent connection to emotional factors. Although there was good epidemiological data to show a relationship between stress and pain, there were no physiological models to explain this. Interestingly, neither of us could accept purely psychological models (“all in your head” models). In those models, the pain is seen as centrally mediated with no real peripheral mechanism involved.

In 1983, our paths converged (at a temporomandibular joint study group). After a short conversation, we realized that we were seeking answers to similar phenomena. This was the “start of a beautiful friendship” and collaboration. We began considering how we might tackle the problem by meeting study-group style at his headache treatment center. Also present were occupational therapists, Greg Berkoff (a chiropractor with great knowledge of muscle anatomy and physiology), and others. After some interesting discussions, we decided that we should be looking for autonomic nervous system pathways capable of creating pain afferents in the muscle. The sympathetic nervous system (SNS) seemed to be the most logical candidate, but the only pathways mentioned in the traditional texts were to the vasculature of the muscle system. One day, David came beaming into the room with a discovery. He searched Medline under SNS and muscle and found a series of elegant animal

studies from Italy that showed quite clearly that sympathetic pathways innervate the muscle spindle in several species (Grassi, Deriu, Artusio, & Passatore, 1993; Grassi, Deriu, & Passatore, 1993; Grassi et al., 1996; Grassi & Passatore, 1987, 1988, 1990).

The muscle spindles are small capsular structures embedded among the much larger extrafusal fibers that are responsible for all of the movement in the body. Their primary role is as a stretch receptor feedback system to maintain proper muscle length. The capsule is filled with an acid found in many joints and has its own much smaller fibers called bag and chain intrafusal fibers. It is known to have pressure sensors, substance P, and unmyelinated C-fibers, all involved in afferent pain pathways. This would make it a likely candidate for the mind/muscle connection we were seeking.

Finding the Trigger Point

We then began to search for a way to study this connection in humans. Greg and David attempted shocking the stellate ganglion while measuring EMG activity. This proved unworkable (and quite painful). We made several attempts to detect the signal with surface electrodes, but at least in the trapezius, we were not successful. (I have also tried two small surface electrodes over the brachial radialis and have detected some unusual muscle action potentials, but this remains to be studied.) Needle electrodes were then proposed, but where should the needle be inserted in the muscle? A medical student (Dawn Bravata) working with us on various projects suggested a trigger point in the trapezius muscle. At that time, Janet Travell’s work was not well known nor accepted in medical circles (Travell & Simons, 1983), so we were quite skeptical at this suggestion. But in a rare fit of open-mindedness, we tried inserting the needle into a tender nodule in the trapezius and—*eureka*—the needle EMG (nEMG) monitor lit up at a very specific locale. A second needle nearby in nontender tissue remained electrically silent. Figure 1 shows a typical finding.

This method was then perfected and the first systematic study run and published (Hubbard & Berkoff, 1993). Almost all of the subsequent studies used this methodology. We estimate that we have tested more than 300 patients in this manner. With rare exception, we get the same pattern.

A series of pharmacological studies followed (Hubbard, 1996) that showed that the nEMG activity in the TP was unaffected by curare (a powerful cholinergic blocker that

blocks all motor neuron activity) but dampened by phentolamine (an alpha-sympathetic blocker; see Figure 2). Thus far, the data seemed consistent with our idea that TPs were sympathetically mediated spindles. They were unaffected by acetylcholine, the usual motor neuron neurotransmitter, but blocked by a sympathetic blocker.

We then began a series of psychophysiological studies. My student, Walt McNulty, gathered patients with palpable TPs and found that the TP, but not the adjacent site, responded vigorously to psychological stress (McNulty, Gevirtz, Hubbard, & Berkoff, 1994). As our theory predicted, the nontender site remained silent during the stress. It seems that the muscle contraction we see in the clinic during stress can be inhibited with instruction (and perhaps the fear of moving a needle in the muscle). Another student, Sonia Banks (Banks, Jacobs, Gevirtz, & Hubbard, 1998) replicated the McNulty paradigm but added a relaxation component. TPs were clearly activated during stress, but activation decreased during autogenic relaxation. Carole Lewis found that the same relationships existed in pain patients (Lewis & Gevirtz, 1994).

Because we were unsure of the nature of the psychological stimuli that would invoke a TP response, we then undertook a series of studies to elucidate the emotional stimuli. Rick Gadler used interview techniques to try to elicit TP responses (Gadler & Gevirtz, 1997) and found that during recall of an emotional event, very high nEMG activity was produced (up to 120 μ V). Professional actors were used by Toni Cafaro (Cafaro, Gevirtz, Hubbard, & Harvey, 2001) to see if dramatically expressed or inhibited emotions would produce higher TP activity. They didn't; it seems that recall of an emotional event drives the TPs at about the same rate as an angry or volatile outburst.

During this time, we started a clinical pain treatment program at Sharp Memorial Hospital in San Diego. Based on our clinical experience, we theorized that the stimulus that would produce severe chronic pain would have to be prolonged in nature. Because we noticed that most of our patients were internalizers (anxious, perfectionistic, hard on themselves, etc.), Janeen Armm (Armm, Gevirtz, Hubbard, & Harpin, 1999) conducted a study with about 80 of our own graduate students. We measured their TPs with an Algometer (a device used to quantify pressure pain thresholds for TPs) and gave a series of paper-and-pencil tests in September prior to the semester's beginning and then again in December just before final exams. As predicted, those students with traits such as high worry, fear of criticism, or low

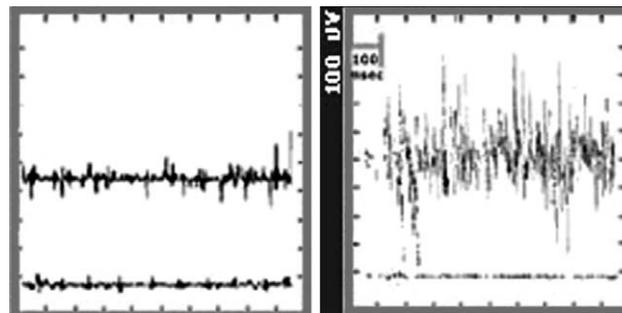


Figure 2. The left panel shows the needle electromyographic (nEMG) action potentials for Curare, a potent cholinergic blocker. The right panel shows the nEMG response to phentolamine, an alpha sympathetic blocker. In both panels the upper trace is the trigger point needle and the lower trace is the non-tender adjacent needle.

assertiveness showed more pain increment in TPs than did those low on these traits.

In the meantime, we were observing that treatments based on this model appeared very successful (Gevirtz, Hubbard, & Harpin, 1996). By using psychophysiological education models, group formats, biofeedback, and physical TP management, patients seem to experience a shift in their attributional beliefs about the pain. Typically, this attributional shift moves the patient from an expectation that medical interventions will “fix” a problem and reduce pain to a belief that the patient can learn skills that will manage and reduce the pain. From a cognitive point of view, the patient moves toward self-efficacy. The whiplash and low back pain studies offer further evidence of the power of this attribution shift.

Conclusion

The journey described here has been an exciting one, but it is far from complete. The exact pathways we have discovered are still poorly understood. For example, we know that TP activity correlates poorly with other SNS measures such as skin conductance or the ratios of low-frequency heart rate variability (HRV) to high-frequency HRV. Many aspects of the TP morphology are not well understood and are—some think—not consistent with the spindle hypothesis (Hong & Simons, 1998; Simons, Hong, & Simons, 2002). Furthermore, we have not broken down the treatment components to see which are necessary for effective treatment. We use many modalities to dampen SNS activity but have not determined if they are all equally effective (SEMG, HRV biofeedback, respiratory training, stretch techniques, CBT, etc.).

Despite these limitations, we think that we have discovered a direction for the explanation and treatment of muscle pain disorders. We hope to continue this journey

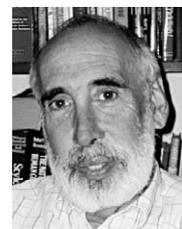
of discovery so that the suffering and financial burden that afflicts millions of people can be minimized.

Acknowledgment

The work related here is the product of a great team of researchers, clinicians, and students. None of it would have happened without them. I would especially like to thank David Hubbard, MD, for the years of collaboration and stimulation he has provided.

References

- Armm, J., Gevirtz, R., Hubbard, D., & Harpin, E. (1999). The relationship between personality characteristics and local muscle tenderness development in first year psychology graduate students: A prospective study [Abstract]. *Applied Psychophysiology and Biofeedback*, 24, 125.
- Banks, S., Jacobs, D., Gevirtz, R., & Hubbard, D. (1998). Effects of autogenic relaxation training on electromyographic activity in active myofascial trigger points. *Journal of Musculoskeletal Pain*, 64(4), 23–32.
- Cafaro, T. A., Gevirtz, R. N., Hubbard, D., & Harvey, M. (2001). The exploration of trigger point and heart rate variability excitation and recovery patterns in actors performing anger inhibition and anger expression [Abstract]. *Applied Psychophysiology and Biofeedback*, 26, 236.
- Gadler, R., & Gevirtz, R. N. (1997). Evaluation of needle electromyographic response to emotional stimuli [Abstract]. *Applied Psychophysiology and Biofeedback*, 22, 137.
- Gevirtz, R., Hubbard, D., & Harpin, E. (1996). Psychophysiological treatment of chronic low back pain. *Professional Psychology: Research and Practice*, 27, 561–566.
- Grassi, C., Deriu, F., Artusio, E., & Passatore, M. (1993). Modulation of the jaw jerk reflex by the sympathetic nervous system. *Archives Italiennes de Biologie*, 131, 213–226.
- Grassi, C., Deriu, F., & Passatore, M. (1993). Effect of sympathetic nervous system activation on the tonic vibration reflex in rabbit jaw closing muscles. *Journal of Physiology*, 469, 601–613.
- Grassi, C., Deriu, F., Roatta, S., Santarelli, R., Azzena, G. B., & Passatore, M. (1996). Sympathetic control of skeletal muscle function: Possible co-operation between noradrenaline and neuropeptide Y in rabbit jaw muscles. *Neuroscience Letters*, 212, 204–208.
- Grassi, C., & Passatore, M. (1987). Effect of cervical sympathetic nerve stimulation on nociceptive jaw opening reflex in the cat. *Functional Neurology*, 2, 547–552.
- Grassi, C., & Passatore, M. (1988). Action of the sympathetic system on skeletal muscle. *Italian Journal of Neurological Sciences*, 9(1), 23–28.
- Grassi, C., & Passatore, M. (1990). Spontaneous sympathetic command to skeletal muscles: Functional implications. *Functional Neurology*, 5, 227–232.
- Holroyd, K. A., Andrasik, F., & Noble, J. (1980). A comparison of EMG biofeedback and a credible pseudotherapy in treating tension headache. *Journal of Behavioral Medicine*, 3(1), 29–39.
- Hong, C. Z., & Simons, D. G. (1998). Pathophysiologic and electrophysiologic mechanisms of myofascial trigger points. *Archives of Physical Medicine and Rehabilitation*, 79, 863–872.
- Hubbard, D. (1996). Chronic and recurrent muscle pain: Pathophysiology and treatment, a review of pharmacologic studies. *Journal of Musculoskeletal Pain*, 4, 123–143.
- Hubbard, D. R., & Berkoff, G. M. (1993). Myofascial trigger points show spontaneous needle EMG activity. *Spine*, 18, 1803–1807.
- Lewis, C., & Gevirtz, R. N. (1994). Needle trigger point and surface EMG measurements of psychophysiological responses in tension-type headache patients [Abstract]. *Biofeedback and Self-Regulation*, 19, 274–275.
- McNulty, W. H., Gevirtz, R. N., Hubbard, D. R., & Berkoff, G. M. (1994). Needle electromyographic evaluation of trigger point response to a psychological stressor. *Psychophysiology*, 31, 313–316.
- Oliveira, A., Gevirtz, R. N., & Hubbard, D. (in press). A psycho-educational video used in the emergency department provides effective treatment for whiplash injuries. *Spine*.
- Simons, D. G., Hong, C. Z., & Simons, L. S. (2002). Endplate potentials are common to midfiber myofascial trigger points. *American Journal of Physical Medicine and Rehabilitation*, 81, 212–222.
- Travell, J., & Simons, R. (1983). *Myofascial pain syndrome: The trigger point manual*. Baltimore: Williams and Wilkins.



Richard Gevirtz

Correspondence: Richard Gevirtz, PhD, 104 Daley Hall, 10455 Pomerado Road, San Diego, CA 92131, e-mail: rgvirtz@alliant.edu.