Chronic Myofascial Pain and Central Sensitization:
Enter the Matrix of Pain Mechanisms, Objective Physical Findings and Treatment Strategies

Jay P. Shah, MD

Lecture Description

This comprehensive lecture will integrate the fascinating and clinically impactful knowledge emerging from the pain sciences in order to analyze the dynamic role that myofascial trigger points (MTrPs), central sensitization, limbic system dysfunction, and objective physical findings and outcome measures play in the evaluation and management of myofascial pain syndrome (MPS).

Research supports the emerging view that what has long been considered a "local" pain syndrome actually has a broader impact beyond the active (i.e., spontaneously painful) MTrP and has significant associations with mood, health-related quality of life and function. In fact, recent findings compel us to look at the phenomena of MPS and MTrPs as a type of spectrum disorder of sensitization that manifests clinically by varying symptoms and signs.

Central sensitization is responsible for aberrant pain perception. Ongoing muscle nociception can initiate, amplify and perpetuate central sensitization and may lead to functional and connectivity changes (via alterations in gene expression, somatosensory processing, and synaptic connections) in central nervous system structures.

Spinal segmental sensitization (SSS) is a hyperactive state of the dorsal horn caused by bombardment of nociceptive impulses. Active MTrPs are a very common source of persistent nociception and sensitization that often results in SSS, facilitated segments, somato-visceral effects, and chronic myofascial pain.

In addition, viscero-somatic convergence may not only provide the means for pain referral to somatic structures, but may also govern the

reflex that induces muscle spasm and the eventual formation of MTrPs. Active MTrPs, in turn, may serve as an additional source of nociceptive input, and become a key component of a chronic visceral condition. Apropos, their deactivation through a targeted intervention may be a critical aspect to reversing central sensitization and improving pain associated with an underlying visceral disorder.

Conversely, maladaptive changes in subcortical structures and dysfunctional descending inhibition may create somatic tissue abnormalities (e.g., tissue texture changes, tenderness, etc.) in addition to adversely impacting mood, affect and sleep.

Either way, typical manifestations of the sensitized spinal segment include dermatomal allodynia/hyperalgesia, tenderness of sclerotomal structures (e.g., ligaments, bursa, and tendons) and MTrPs within the affected myotomes. These objective and reproducible findings allow the clinician and patient to identify the affected spinal segment(s) that should be treated.

In summary, we will explore the enigma of MPS and central sensitization including important challenges and questions that need to be answered; how recent studies provide key insights into the mechanisms of chronic myofascial pain and the underlying pathogenesis and pathophysiology of MTrPs; and how to clinically integrate this scientific information to better evaluate and treat patients with chronic musculoskeletal pain.

Learning Objectives

Upon completion of this lecture, participants will be able to:

1) Examine the unique neurobiology of muscle pain and the dynamic interplay of muscle nociceptors and endogenous biochemicals in the initiation, amplification and perpetuation of peripheral and central sensitization

2) Distinguish active (i.e., spontaneously painful) MTrPs from latent MTrPs and identify the referred pain patterns commonly encountered in clinical practice

3) Demonstrate that active MTrPs have elevated levels of inflammatory mediators, neuropeptides, catecholamines and cytokines – substances known to be associated with inflammation, sensitization, inter-cellular communication and persistent pain states

4) Understand the complex, pivotal and fascinating roles that nociceptive "afferent bombardment, neurogenic inflammation, wide dynamic range neurons, subcortical structures (e.g., the limbic system) and dysfunctional descending inhibition play in muscle sensitization, pain chronification, somato-visceral interactions and the objective, reproducible physical findings of allodynia, hyperalgesia and referred pain patterns commonly observed in MPS

5) Outline an Integrated Hypothesis for myofascial pain as a complex state of Neuro-muscular Dysfunction involving both peripheral and central factors

6) Introduce novel applications of ultrasound imaging to visualize MTrPs and measure their stiffness properties and local blood flow

7) Demonstrate that MTrPs in the upper trapezius are stiffer than surrounding tissue and that active MTrPs can be distinguished from

latent MTrPs by their high-resistance blood flow and greater surface area

8) Demonstrate that dry needling of painful MTrPs leads to a significant decrease in muscle stiffness and how ultrasound can be used as an objective and repeatable outcome measure for dry needling.

9) Discuss how muscle pain preferentially activates limbic system structures, providing a neuro-physiological basis for increased anxiety, fear and stress

10) Discuss the dynamic interplay of somato-visceral/viscero-somatic integration and spinal facilitation in the dorsal horn

11) Identify the reproducible physical manifestations of spinal segmental sensitization (involving dermatomes, myotomes and sclerotomes) commonly observed in chronic myofascial pain

12) Design an appropriate treatment algorithm (e.g., dry needling, electrical stimulation techniques, etc.) to desensitize the involved segments, eliminate chronic MTrPs and alleviate chronic myofascial pain and dysfunction