

Connective tissue contractility is the central mediating factor in myofascial pain syndrome: A Fasciagenic Pain Model.

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BACKGROUND Myofascial pain (MP) may be the most common human physical complaint, yet its pathogenesis remains unproven. Explanations for the chronicity of MP include the integrated trigger point hypothesis, the central sensitization theory, and more recently, the “barrier-dam” theory. Others contend that MP is due to central and peripheral neuropathies. Trigger points, taut bands and faulty nerves are believed to be the source of the pain. But what makes the muscles lumpy and ropy? Why do the nerves misfire? The answers may lie in a growing appreciation of fascial plasticity.

HYPOTHESIS Connective tissue contractility is the central mediating factor in myofascial pain syndrome (MPS). Fascial remodeling occurs to protect the integrity of vulnerable structures that the deep fasciae ensheathes. Fascia changes in response to a real or imagined threat to the organism; to sudden, prolonged or repetitive tensional demands; during tissue repair; following viral infections; or for idiopathic reasons. Fascial remodeling can occur rapidly or gradually as determined by the sum of proprioceptive, mechanoreceptive, chemoreceptive, thermoreceptive, nociceptive, and psycho-emotional input to the central nervous system. Fascia thickens and stiffens as it contracts, exerting pressure on muscles, nerves, and blood vessels. If the pressure is unremitting, metabolic and functional deficits can occur in these structures, creating the symptoms of MPS.

EVALUATION OF THE HYPOTHESIS Evidence for a fasciagenic pain model is presented as an historical examination of research in the fields of MP and connective tissue physiology. Proof of pathological connective tissue changes in MPS has existed for over 30 years, but has been overlooked in favor of muscular and neural interpretations. Recent studies of the effects of acupuncture and Roling® lend credence to the idea that all manual therapies are merely making mechanical suggestions to the higher brain centers to promote fascial remodeling. Testing the hypothesis is predicted to be straightforward. Ultrasound elastography, algometric readings, range of motion testing, and subjective reports from the clinician and the subject make it possible to document fascial changes during manual therapies.

Contracted fascia is palpable in the form of thickened endomysium (taut bands) and thickened perimysium (taut bundles) and can be clinically assessed as limitations in passive muscle mobility, passive joint mobility, and dermal-fascial mobility. The author names the manual therapy recommended for testing the hypothesis as Fascial Facilitation. This method is amenable to testing as it quickly detects contracted fascia and initiates beneficial changes that are palpable, observable, and measurable. Testing is predicted to demonstrate that contracted fascia is always present in MPS and that remission of symptoms is a consequence of fascial relaxation.

CONCLUSION Confirmation of the hypothesis will contribute to a greater understanding of fascial plasticity and the process of mechanotransduction. Clinical acceptance of the hypothesis will shape future treatment strategies for relieving MP.