

Hyaluronic Acid and the Myofascial Pain Syndrome

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"Myofascial pain syndromes (MPS) are among the most frequent pain conditions encountered in the general population. They are also the most often under-diagnosed or misdiagnosed condition."¹ MPS, previously called fibrositis or myofibrositis, could be the underlying etiology of even nonspecific back pain. It is estimated that 30 percent of patients with regional pain complaints seen in primary care clinics have myofascial pain and 85 percent of patients presenting to specialized pain management centers have myofascial pain.²⁻³ A major problem with this diagnosis is that there has been "no standard, universally accepted biochemical, electro-diagnostic, diagnostic-imaging or physical examination criteria existing for a diagnosis of MPS."⁴

A major question with regard to all types of manual load-type treatments including Graston, ART, fascial manipulation, deep massage, Rolfing, etc., is why these treatments are successful for MPS. Another question to eventually be answered is why they are sometimes unsuccessful. There may be a common underlying reason that answers both questions related to hyaluronic acid (HA)

In a previous article in *Dynamic Chiropractic*,⁵ I shared that it is very possible HA may be the principal substance causing soft-tissue restriction, and is the substance responding to mechanical load that allows a decrease in viscosity and thereby a normalization of densified tissue. HA (sodium hyaluronate) injections into the glenohumeral joint for primary adhesive capsulitis has proved to be very successful.⁶ It has also been proven beneficial in the treatment of knee osteoarthritis and in reducing postsurgical tendon adhesions.⁷⁻⁸

In healthy tissue, HA supports normal homeostasis and suppresses cell proliferation, angiogenesis, inflammation and immunogenicity. HA also lessens the proinflammatory mediators and pain-producing neuropeptides released by activated synovial cells.

A [recent paper](#) on this subject by Stecco A, et al. (2013)⁹ explains how densified tissue (increased viscosity) can be responsible for MPS. Densification results in abnormal sliding between the overlying deep fascia on the muscle and within the epimysium and perimysium layers within the muscles. Changes in viscoelasticity of fascia can modify activation of the nerve receptors within fascia. Within fascia are mechanoreceptors that can send pain messages when stretching occurs within densified fascia. Muscle spindle cells are within the epi and peri layers; if embedded in fascia, not allowing the full stretch of the spindle cell during muscle contraction, there will be improper feedback to the CNS, resulting in muscle incoordination. Increased concentration and size of HA chains entangle into complex groupings, changing hydrodynamic properties and thereby altering normal viscoelastic properties.

Abnormal HA fragmentation can be reversed by increased temperature, local alkalization, deep massage or physical therapies "that are able to cause disaggregation of the pathologic chain-chain

(HA) aggregations."⁹ Densification occurs within the loose connective-tissue layers inside and around the fascia. Within loose connective tissue are water, ions and other substances that also can affect the biomechanical properties of this connective tissue. Sliding between fascia and muscles occurs at the loose connective-tissue level.

Increased acidity is a major factor causing increased HA viscosity. A pH of 6.6 (increased lactic acid) increases the viscosity of HA in the endomysium and perimysium of muscles. This accounts for possible stiffness after activity and the eventual normalization that occurs after degradation of lactic acid. But it is possible that before, let's say, an athletic event, overwork or trauma, there are already areas of densifications in individuals that cannot be restored to normal viscosity. These areas may react as MPS "trigger points" or densifications and when stressed, result in pain.

As is often said, most patients are accidents waiting to happen. Could it be that the fascial densifications due to HA accumulation and the abnormal proprioceptive effects are responsible for so many myofascial problems, especially MPS?

Notice the use of the word *densification* instead of *fibrosis* or *scars*. A fibrosis / scar results from the biological process of wound repair in the skin and other tissues of the body. Scarring is therefore a natural part of the healing process. With the exception of very minor lesions, every wound (e.g., after accident, disease or surgery) results in some degree of scarring.

Abnormal fibrosis (collagen cross-links) forms a pronounced alignment in a single direction. This collagen scar-tissue alignment is usually of inferior functional quality to the normal collagen randomized alignment. In other words, an injury is necessary to form scar tissue. Most of the areas we palpate are not scars or fibrotic tissue, but are considered densifications. When fibrosis does happen, it occurs in the dense connective tissue.

Dysfunction or "densification" of fascia occurs in the loose connective tissue containing adipose cells, GAGs and HA. Alterations of the contents of the loose connective tissue can be the result of a combination of any of its elements, especially the HA. This will result in increased viscosity and functional involvement of the fascial and muscular component.

Since these densifications may be palpated throughout the body, random treatment of these areas may not necessarily prove to help the patient. A reason for treatment failure using manual load may be due to the fact that the correct points / areas are not treated and not enough time is spent to truly eliminate the densification. Patients will point to the site of their pain, but on palpation, there is no palpable densification.

Fascial manipulation teaches the practitioner to choose the particular sequence of densifications based on planes (acupuncture meridian, fascial expansions, fascial planes) that are principally involved. It is necessary to decide in advance what plane is the most significant and to choose the points you intend to treat in advance.

It has been found that a certain amount of time and pressure is necessary to free the abnormal viscous point and eliminate the HA entanglements (usually about two minutes or more). With experience, using a compression / friction on these points should allow the practitioner to palpate a "melting" of the area. If the right area or areas are dissipated, then a functional test that expressed the patient's pain should retest normally.

In treating these densifications, once you decide to treat an area, the treatment should continue on the area until palpation reveals that the area has dissipated and no longer exists. Sometimes due to patient tenderness you have to treat an area of density above or below the area to free up the plane

of fascia you have chosen to treat. After treating a proximal or distal location, you will find that the painful density has markedly diminished and can now be disposed of in short order. It's like releasing a tense rubber band that could extend in some cases from the feet to the neck.

The coupling of mechanical load on tissue, with its inherent mechanoreceptors, and the realization that fascia is considered a sensory organ and not just a "protective covering," as it has been referred to over the years, opens up the door to a healing modality dealing with actual causation rather than the traditional approach of just symptomatic treatment.

References

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