

Fibrosis May Be Related to Chronic Pain

Warren Hammer, MS, DC, DABCO

A frequent visitor to a chiropractor's office is the patient who explains that for more than a year, they have experienced intermittent lower back pain. They usually have a tough time arising from bed in the morning and feel pain during the day when getting up after prolonged sitting. Often they will state something to the effect of, "I played tennis last week and the pain has been more severe than usual." When evaluating these types of patients, from a soft-tissue point of view, you will find many significant areas of connective tissue and muscle restrictions up and down the kinetic chain. Researchers have a difficult time determining the source of chronic back pain because the anatomical findings found on X-ray and MRI such as disc bulging, annular tears and herniated discs often do not correlate with the patient's symptoms. Recent research on chronic pain has incriminated increased neuronal activation or brain hypersensitivity as the perpetuator of the pain.^{1,2}

Most authorities feel that chronic pain is a multifactorial problem. But one thing is certain: Stiffness or fibrosis is frequently present in various areas of the body. Recently, a landmark paper was written by Langevin and Sherman.³ Dr. Langevin is a medical doctor and prominent researcher in connective tissue from the University of Vermont. These researchers hypothesized that associated with acute or subacute low back pain are psychosocial factors such as job dissatisfaction, poor social support and emotional stress, leading to a cycle of decreased movement due to fear of pain. According to Langevin and Sherman, evidence supports the fact that among chronic low back patients, pain affects how they move, resulting in abnormal trunk muscle activity during postural perturbation, impaired control of trunk and hip during arm movements and abnormal postural compensation for respiration. As a result of emotional, behavioral and motor dysfunction, abnormal connective tissue remodeling, inflammation, nervous system sensitization and further decreased mobility occurs, creating a vicious cycle.

Langevin and Sherman stressed the association of abnormal connective tissue with the nervous system: "Both increased stress due to overuse, repetitive movement and/or hypermobility, and decreased stress due to immobilization or hypomobility can cause changes in connective tissue." Both hyper- and hypomobility can result in either atrophy or fibrosis. Inflammation, tissue hypoxxygenation and cytokines such as TGF-1 will promote fibrosis. Whether it is the presence of trigger points within the fascia causing painful muscle contraction, microinjury, inflammation (release of inflammatory mediators [prostaglandins, bradykinin]), growth factors or abnormal biomechanics, there will be an increase in fibrosis, leading to increased tissue stiffness and further loss of motion.

So, the patient with an increase in chronic, intermittent pain since playing tennis the previous week is the accident waiting to happen. They already have fibrosed, disorganized connective tissue associated with poor blood and lymphatic flow. Langevin and Sherman write that within the research world, not enough attention has been paid to connective tissue in relation to low back pain, especially the loose connective tissue and fasciae compared with specialized connective tissues such as cartilage. They hypothesize that "connective tissue remodeling may play an important role in the pathophysiology of low back pain because plasticity in response to changing

mechanical loads is one of connective tissue's fundamental properties, and pathological remodeling (fibrosis) due to changes in tissue movement is well-documented in other types of connective tissues (e.g., ligaments, joint capsules)." They also state that research into this area will help explain why treatments such as physical therapy, massage, chiropractic manipulation, acupuncture, movement therapies and yoga may be valuable. We already know anecdotally and with early research that directed mechanical load using Graston technique, active release, friction massage and other techniques are helping the chronic patient. There is no question more research is needed to prove the positive results we experience in our offices on a daily basis.

References

1. Giesecke T, Gracely RH, Grant MAB, Nachemson A, et al. Evidence of augmented central pain processing in idiopathic chronic low back pain. *Arthritis & Rheumatism* 2004;50(2),613-623.
2. Apkarian AV, Sosa Y, Sonty S, Levy RM. Chronic back pain is associated with decreased prefrontal and thalamic gray matter density. *J of Neuroscience* 2004;24(16),L10410-10415.
3. Langevin HM, Sherman KJ. Pathophysiological model for chronic low back pain integrating connective tissue and nervous system mechanisms. *Medical Hypotheses* 2006; article in press.

DECEMBER 2006