

Differential Diagnosis of Myofascial Pain Syndrome and Fibromyalgia

Robert D. Gerwin

SUMMARY. Objectives: This review examines diagnostic factors of myofascial pain syndrome [MPS] and fibromyalgia [FMS], and their differential diagnosis.

Findings: A base of electrophysiologic and anatomic data exists that supports the clinical identification of myofascial trigger point [TrP] features. Included are electrical characteristics of the TrP, visualization of the taut band and strong support for a model of the TrP. The widespread nature of chronic MPS mimics FMS. Fibromyalgia has been found to be associated with a host of clinical and biochemical markers. Objective markers besides widespread tenderness are needed to distinguish FMS from other conditions. It is not clear if either response to electrical stimulation of the parietal tissues or the skin rolling test will provide the necessary specificity. Biochemical markers such as substance P elevation in spinal fluid may be sensitive for FMS, but the specificity of the finding is unknown. The possibility is raised that some findings may be related to the severity of chronic widespread pain, and non-specific.

Conclusion: A variety of clinical tests are available to assess both MPS and FMS. However, tests that are based primarily on the identifica-

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tion of widespread tenderness will over-diagnose FM, and under-diagnose MPS, and may miss other causes of chronic myalgia. [Article copies available for a fee from The Haworth Document Delivery Service: 1-800-342-9678. E-mail address: getinfo@haworthpressinc.com]

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Myofascial pain syndrome [MPS] and fibromyalgia [FMS] are now better characterized and distinguished in years past. In part this reflects new data on the pathophysiology of MPS and a better characterization of FMS.

The myofascial trigger point. Myofascial pain syndrome is associated with Taut band [TB] in muscle. In symptomatic trigger points [TrPs], the TB is tender to pressure. The anatomic correlate of the TB is the contraction knot (1,2). In brief, it is an intense contraction of muscle sarcomeres that causes a shortening of the muscle fibrils and an increase in their diameter. Multiple contraction nodules could produce the palpable nodularity of the trigger zone.

Trigger point electrical activity. The electromyographic [EMG] activity (1-4) is most consistent with activity generated by a dysfunctional motor end-plate. This abnormal endplate activity is modulated by sympathetic nerve activity (5).

The local twitch of the TB (6-11) is not usually cited as a diagnostic tool, but in fact it is a unique objective finding. It is not a neuropathic fasciculation, nor is it found in FMS unless there is a coincidental TrP. The local twitch-related EMG discharge is correlated with the contraction of the TB as seen by high resolution ultrasound (Gerwin RD, unpublished data). The simultaneous EMG discharge with contraction of the stimulated TB is objective confirmation of these two TrP features. In a symptomatic TrP, activation of the TB is invariably and uniquely accompanied by referred pain, recognition of "usual" pain, and elimination of pain.

Algometry. Algometry and tissue compliance measurements quantify tenderness and muscle tone, respectively (12). They are useful tools for objective documentation of decreased pressure pain thresholds and increased tone. They are specific and sensitive for differentiation of the TrP from adjacent uninvolved muscle. Algometry is accurate for distinguishing tender points [TeP] from control points in FMS. There is widespread tenderness in patients with FMS, not confined to TePs. The clinical usefulness of this fact is not generally stressed. Algometry does not by itself identify a point that relates to the patient's pain syndrome. It has utility in documenting levels and

changes in tenderness, but lacks specificity in distinguishing one cause of tenderness from another. In the new epidemic of myalgia caused by the statin family of cholesterol lowering drugs, algometry would be expected to show the same findings as in FMS, although this has not been studied to my knowledge.

Trigger point examination reliability. The interrater reliability of TrP examination had been a problem in the literature prior to 1997. Gerwin et al. (13) showed that it is possible to have a high degree of interrater reliability if care is taken to define the elements of the examination and if the examination is performed uniformly. The twitch response remains the most difficult of the clinical features to elicit. Referred pain takes time to develop, as much as 10-15 seconds of continued pressure. A hurried examination will, therefore, miss it. Reproduction of pain requires an educated subject. Some subjects will respond only about present pain, others will tell you about pain they have at any time, and others discount any pain that is not their more severe pain. Hence, the subject must be instructed as to what is to be considered usual pain. Less variable is the identification of the TB and tenderness; with experience, an examiner will recognize subtle degrees of increased tissue resistance as the TB. The degree of tenderness may vary among patients according to their perception of pain, a finding that we have encountered in our algometry studies.

The trigger point and pain. Tender TBs may be widespread in adults, symptomatic or not (13, Gerwin RD, Dommerholt J, unpublished data). The distinguishing feature of a symptomatic TrP is the reproduction of present pain. The TB is the initial identifiable response to stressors that produce a TrP. The development of pain is a separate step involving activation of peripheral nociceptive receptors. The anatomic change may be present without the sensory change. An analogy is osteoarthritis, where there is an anatomic alteration in the joint, but not necessarily pain unless there is physical stress.

Regional and widespread myofascial pain syndrome. Myofascial pain syndrome is often presented as single muscle syndromes for the purpose of instruction (14). Functional muscle groupings indicate inter-relationships individual muscles have with each other, and help explain the widespread nature of chronic MPS (14, chapter 28, pp. 541-51). Muscles work in functional groups. The smallest functional unit is a pair of muscles acting in a reciprocal agonist/antagonist relationship. A TrP results in the loss of the normal reciprocal inhibition of agonist/antagonist muscles. Therefore, muscles that are normally inhibited may be chronically contracted and fatigued. The spread of TrPs through dysfunctional muscle units is very common as pain syndromes become chronic. Functional relationships between muscles are different at the two ends of a muscle. The functional unit of the

biceps at the shoulder includes the pectoral and deltoid muscles, whereas at the elbow it includes the triceps, supinator and brachioradialis muscles. Spread of painful TrPs in axial and postural dysfunction through dysfunctional muscle units crosses the midline to involve both sides, as well as spreading through upper and lower halves of the body through muscles that span the trunk in whole or in part, like the latissimus dorsi, or the longissimus muscles. Nevertheless, the FMS literature remains replete with references to MPS as a regional syndrome in contrast to FMS as a widespread syndrome. This is a particularly dangerous concept in chronic pain, where MPS is more likely to be generalized. A new survey of patients with mostly chronic MPS of diverse causes and duration updates previous data, and shows that about 45% of subjects have generalized three or four quadrant trigger point pain (15, Gerwin R, unpublished data). It is simplistic and inappropriate to use widespread pain as a feature that distinguishes FMS from MPS.

Myofascial pain syndrome is often co-morbid with other conditions. It is present in 100% of cases of chronic whiplash pain, including those with facet joint injury and discogenic pain (Gerwin and Dommerholt, unpublished data). It is a common cause of post-laminectomy pain. It coexists with arthritis and with spondylosis. In each of these conditions, it is a treatable, even when the underlying condition is not. It is often associated with biomechanical and medical perpetuating factors that influence outcome.

Fibromyalgia. Fibromyalgia is a chronic pain syndrome (16), associated with a number of phenomena such as high spinal fluid substance P (17), a deficiency of serotonin (18) and generalized hypersensitivity, as well as sleep disturbance and unusual fatigue. The diagnosis of FMS is described as a simple two-step process (19) that requires identification of muscle as the source of pain and the identification of widespread pain with about 11 of 18 TePs [the number is not absolute for clinical practice]. No other studies are necessary to make a diagnosis of FMS—as it is not a diagnosis of exclusion (19). However, there is no unique marker for FMS, and particular care must be taken to exclude other conditions of widespread myalgia. The established criteria for the diagnosis of FMS do not do that.

The paradigm for the study that became the American College of Rheumatology guidelines for the diagnosis of FMS (20) was the selection of a cohort of individuals who experts in FMS considered to have FMS. Another group of doctors examined them, and found that only tenderness in 11 of 18 specified sites [i.e., widespread tenderness] was statistically significant in distinguishing FMS subjects from control subjects. If the original selection for FMS was made because the individuals had widespread tenderness [and that criterion was known], then it should not be surprising that widespread tenderness was a distinguishing characteristic of FMS in that study. What is surprising is that there was no other feature that was as statistically signifi-

cant. This diagnostic circular reasoning has been the target of criticism. Fibromyalgia is a legitimate chronic pain syndrome. However, there is a need for an objective diagnostic test that does not depend only on tenderness, but that correlates well with it. One would anticipate that the biochemical markers might fill this need, if they can be shown to be both sensitive and specific to FMS. Studies are being published that use sleep disturbance, fatigue and fibro-fog [a cognitive disorder] as criteria to distinguish FMS from MPS and other conditions. Care must be taken to distinguish a finding present in FMS from one that is specific and sensitive for FMS.

Vecchiet and coworkers approached the problem of tenderness in a unique and elegant way, using electrical stimulation of skin, subcutis and muscle to examine differential responses in different musculoskeletal pain conditions (21). Hyperalgesia of all three tissues was found in painful and nonpainful areas in FMS, muscle hyperalgesia alone in painful and nonpainful areas of chronic fatigue syndrome, and hyperalgesia in all three tissue layers at the TrP zone in MPS, of muscle in the referred pain zone, and no hyperalgesia in nonpainful areas.

Skin-rolling test. Zohn and Clauw (22) have examined skin rolling as a manifestation of tissue plane stickiness and found that decreased skin rolling, or increased tissue stickiness, correlates well with the TeP count, and is therefore potentially useful as a diagnostic tool.

Phenomena versus epiphenomena. Hypersensitivity (23-25) to many different stimuli (24,25) and biochemical abnormalities in FMS such as substance P elevation in spinal fluid (26) could potentially become useful markers for FMS. Sensitivity to a variety of stimuli was greatest for the FMS group, intermediate for the arthritis group, and lowest for the pain-free control group in each study. The comparison and control groups used in the studies are critical. They are often another rheumatologic illness such as rheumatoid arthritis and normal pain-free subjects. Hawley and Wolfe (27) showed that on self rating scales of pain [VAS] and on scores for global severity, FMS patients rated themselves as having pain greater than any other group. Patients with rheumatoid and other forms of arthritis had low pain ratings. It may be that these phenomena are related to severity of pain, but not related to FMS per se. Control groups of matched pain severity to that of FMS are needed.

Myoadenylate deaminase deficiency. A final issue is the specificity of tenderness, the distinguishing characteristic of FMS. Tenderness alone does not distinguish FMS from MPS. The myalgia of cholesterol-lowering drugs has already been mentioned. Hypothyroidism and amoebic infestation can cause it. Myoadenylate deaminase deficiency is another condition that needs further study, as it may mimic FMS, or may be the cause of FMS in at least a subset of patients. The clinical presentation is highly variable, ranging from

entirely asymptomatic to having post-exercise myalgia, fatigue or myalgia beginning in adulthood (28,29).

Conclusion. Fibromyalgia and MPS share muscle pain as a symptom and tenderness as a clinical sign. This has resulted in many persons with bilateral or widespread muscle pain being diagnosed as having FMS when in fact they have MPS, or other types of myalgia.

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