Travell & Simons'

Myofascial Pain and Dysfunction:

The Trigger Point Manual

VOLUME 1. Upper Half of Body

Second Edition

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Table 2.6. ***The American College of Rheumatology 1990 Criteria for the Classification of Fibromyalgia****

1. History of widespread pain.
   Definition. Pain is considered widespread when all of the following are present: pain in the left side of the body, pain in the right side of the body, pain above the waist, and pain below the waist. In addition, axial skeletal pain (cervical spine or anterior chest or thoracic spine or low back) must be present. In this definition, shoulder and buttock pain are considered as pain for each involved side. "Low back" pain is considered lower segment pain.
2. Pain in 11 of 18 tender point sites on digital palpation.
   Definition. Pain on digital palpation must be present in at least 11 of the following 18 tender point sites:
   Occiput: bilateral, at the suboccipital muscle insertions.
   Low cervical: bilateral, at the anterior aspects of the intertransverse spaces at C5-C7.
   Trapezius: bilateral, at the midpoint of the upper border.
   Supraspinatus: bilateral, at origins, above the scapular spine near the medial border.
   Second rib: bilateral, at the second costochondral junctions, just lateral to the junctions on upper surfaces.
   Lateral epicondyle: bilateral, 2 cm distal to the epicondyles.
   Gluteal: bilateral, in upper outer quadrants of buttocks in anterior fold of muscle.
   Greater trochanter: bilateral, posterior to the trochanteric prominence.
   Knee: bilateral, at the medial fat pad proximal to the joint line.

   Digital palpation should be performed with an approximate force of 4 kg.

   For a tender point to be considered "positive" the subject must state that the palpation was painful. "Tender" is not to be considered "painful."

Note: For classification purposes, patients are said to have fibromyalgia if both criteria are satisfied. Widespread pain must have been present for at least 3 months. The presence of a second clinical disorder does not exclude the diagnosis of fibromyalgia.

*Reprinted by permission from Wolfe F, Smythe HA, Yunus MB, *et al.* The American College of Rheumatology 1990 criteria for the classification of fibromyalgia: Report of the Multicenter Criteria Committee. *Arthritis Rheum* 1990; 33:160-170.

what they found to be the 19 most discriminating points. The tender sites selected as diagnostic criteria are quite arbitrary, but adequately representative of the patient's total-body, physiologically enhanced sensitivity to pain.

Fibromyalgia can be thought of as a set of core features and two types of ancillary features. The core features are generalized pain and tenderness over 11 of 18 prescribed anatomical sites. Characteristic ancillary features occur in over three-quarters of individuals: fatigue, nonrestorative sleep, and morning stiffness. Less common findings, in perhaps 25% of cases, include: irritable bowel syndrome, Raynaud's phenomenon, headache, subjective swelling, nondermatomal paresthesia, psychological stress, and marked functional disability. Patients with fibromyalgia experience at least as much pain as those with other painful disease states.[163] Even though fibromyalgia was at first thought to originate in skeletal muscles, a careful histological and ultrastructural study has shown no abnormality of skeletal muscles that was sufficiently common for that to be considered the cause of fibromyalgia.[16, 224]

On the other hand, the etiology of myofascial TrPs is clearly a focal muscular dysfunction which can exert a strong influence on all major parts of the nervous system, and can lead to spinal level neuroplastic changes that help to convert an acute pain problem into a chronic one.

There is strong research support for a systemic, metabolic/neurochemical pathogenesis of fibromyalgia. Fibromyalgia is considered an upward modulation of pain sensitivity throughout the body. Extensive research in recent years has led to the "serotonin deficiency hypothesis"[224] that involves measurable disturbance in nociception, including serotonin regulation of

(Myogelosen) (muscle indurations or myogelosis) in human subjects.

In 1960, Miehlke, *et al.*[193] reported *"bauchige Anschwellungen"* (bulging swellings) of muscle fibers in longitudinal sections, and also much variable width and staining intensity in cross sections of muscle fibers, in biopsies taken from regions of *Muskelhärten* (muscle indurations or nodules) in patients with *Fibrositissyndrom* (fibrositis).

In 1976, Simons and Stolov[253] used TrP criteria to examine canine muscles for a tender spot in a palpable taut band comparable to that observed in human patients. With animals under anesthesia, the same location in the muscle was identified by palpation and widely biopsied. Some isolated, large, round muscle fibers and some groups of these darkly staining, enlarged, round muscle fibers appeared in cross sections (Fig. 2.23). In longitudinal sections, the corresponding feature was a number of contraction knots. An individual knot appeared as a segment of muscle fiber with extremely contracted sarcomeres. This contractured segment showed a corresponding increase in diameter of the muscle fiber, as illustrated in Figure 2.24.

The structural features of contraction knots, one of which is illustrated in Figure 2.24, are portrayed schematically in the lower half of Figure 2.25. This figure presents a likely explanation for the palpable nodules and the taut bands associated with TrPs. The inset below in Figure 2.25B shows three single contraction knots scattered among normal muscle fibers. Figures 2.24 and 2.25B illustrate that beyond the thickened segment of contractured muscle fiber at the contraction knot, the muscle fiber becomes markedly thinned and consists of stretched sarcomeres to compensate for the contractured ones in the knot segment. In addition, a pair of contraction knots separated by empty sarcolemma are illustrated in the upper right of the inset (part B) of Figure 2.25. This feature[96, 253] may represent one of the first irreversible complications that result from the continued presence of the contraction knot.

The muscle fibers containing contraction knots are clearly under increased tension both at the contraction knot and beyond. The total muscle schematic in Figure 2.25A illustrates that this sustained tension could produce local mechanical overload of the connective tissue attachment structures in the vicinity where the taut band fibers attach. This sustained tissue distress could be expected to induce the release of sensitizing agents that would sensitize local nociceptors, producing local tenderness and the characteristics of an attachment TrP.

In 1996, Reitinger, *et al.*[214] biopsied in fresh cadavers the still-palpable nodules of myogelosis that were located in the gluteus medius muscle where trigger point 1 and trigger point 2 are found as described by Travell and Simons.[280] Cross sections showed the previously described, large, rounded, darkly staining muscle fibers and a statistically significant increase in the average diameter of muscle fibers in the myogelosis biopsies compared to nonmyogelotic control biopsies from the same muscle. Electron microscopic cross sections showed an excess of the A-Band and lack of the I-Band configuration. Exclusive presence of A-Band in the absence of I-Band occurs only in fully contracted sarcomeres.[15] It is highly likely that this fully-

Figure 2.23. Giant round muscle fiber in the center of the figure is surrounded by open space that may have resulted from a local severe energy crisis. This space may contain substances that could sensitize adjacent nociceptive nerve fibers. In addition to the normal-size irregularly shaped muscle fibers surrounding the giant fiber, there are four abnormally small fibers, two above to the right, and two below to the left, that may be the segments of muscle fibers which are narrowed because of a contraction knot elsewhere in that fiber.

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5. FUNCTIONAL (MYOTATIC) UNIT

The **quadratus plantae, flexors digitorum longus and brevis, lumbricals,** and **interossei** function as a team to flex the four lesser toes and to control their extension. Their antagonists are the extensors digitorum longus and brevis.

The **flexor digiti minimi brevis,** abductor digiti minimi, the **fourth lumbrical,** and the **third plantar interosseus** muscles function together to flex the fifth toe. They are opposed by the tendon slips of the extensors digitorum longus and brevis that attach to the fifth toe.

The **adductor hallucis** and **flexor hallucis brevis** form a functional unit to control the positioning of, and the force exerted by, the great toe.

The dorsal and plantar interossei together with the lumbricals control abduction and adduction efforts of the four lesser toes.

6. SYMPTOMS

Patients with TrPs in the deep intrinsic foot muscles are likely to present with marked limitation of walking due to pain, and they may complain of numbness of the foot and a feeling that it is swollen. The altered sensation usually includes the entire distal end of the foot and is not limited to only one toe. This altered sensation is especially likely to arise from TrPs in the flexor digiti minimi brevis, flexor hallucis brevis, or adductor hallucis muscles. Patients with TrPs in these muscles often have tried orthoses inserted in the shoes, but usually quickly remove them because of intolerably greater pain from the increased pressure on the TrPs and tender reference zones.

Muscular imbalances and articular dysfunctions of the foot may lead to problems in any proximal segment of the body, including the knee, hip, pelvis, and spine.

The pain complaints of patients with involvement of the deep foot intrinsic muscles are often combined with myofascial patterns of TrPs in other muscles that refer pain to the foot.

Active or latent TrPs in the dorsal interosseus muscles can be associated with **hammer toes.** The deformation of the toes may disappear after inactivation of these TrPs, especially in younger patients.

DIFFERENTIAL DIAGNOSIS

Other Myofascial Pain Syndromes

Because patients often have active TrPs in several foot and leg muscles at the same time, one sees many combinations of pain referral patterns.

Quadratus Plantae. The quadratus plantae TrPs refer pain and tenderness to the bottom of the heel (Fig. 27.1), whereas both the gastrocnemius TrP_1 (*see* Fig. 21.1) and flexor digitorum longus TrPs (*see* Fig. 25.1) refer pain and tenderness to the instep, anterior to the heel. The heel referral pattern of soleus TrP_1 (*see* Fig. 22.1) is more extensive than that of the quadratus plantae. The soleus TrP referral covers not only the plantar surface of the heel, but usually extends over the back of the heel and part of the way up the Achilles tendon. The pattern of the tibialis posterior TrPs (*see* Fig. 23.1) may spill over to the heel, but focuses primarily on the Achilles tendon above the heel. Pain and tenderness referred from the abductor hallucis muscle (*see* Fig. 26.2) concentrates along only the medial border of the heel, whereas the quadratus plantae referral pattern covers the plantar surface of the heel.

Adductor Hallucis. The adductor hallucis refers pain and tenderness to the plantar surface of much of the forefoot (Fig. 27.2*A*), but gastrocnemius TrP_1 (*see* Fig. 21.1) usually refers more proximally to the instep. Distinguishing the more restricted pain and tenderness of interosseous TrPs (that usually include a strong pattern to one toe) ordinarily is not much of a problem. Both the flexor digitorum longus (*see* Fig. 25.1) and the flexor digitorum brevis (*see* Fig. 26.3*B*) refer pain and tenderness to the plantar surface of the forefoot in an area that could easily be confused with the pattern of the adductor hallucis. When the pain complaint includes the plantar surface of the forefoot, the former two muscles and the adductor hallucis should be examined.

Flexor Hallucis Brevis. Flexor hallucis brevis TrPs refer pain and tenderness mainly to the region of the head of the first metatarsal with only a spillover pat-

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PART 3

sential to know the precise location of the pain and to know which specific muscles can refer pain to that location. Muscles that could be causing the pain are tested for restriction of *passive* stretch range of motion and for pain at the shortened end of *active* range of motion, as compared with uninvolved contralateral muscles. Suspected muscles are also tested for mild to moderate weakness either by conventional isometric strength testing or during a lengthening contraction. Such weakness is not associated with atrophy of the muscle.

The muscles showing abnormalities in these tests are the ones most likely to have the taut bands and spot tenderness of the TrP. The taut bands are located by palpation and then tested for a local twitch response and reproduction of the patient's pain complaint by digital pressure on the TrP. One must try to distinguish active TrPs from latent ones, which can also respond positively to the tests described but are not responsible for a pain complaint. Active TrPs are more irritable than latent TrPs and show greater responses on examination. If inactivation of the suspected TrP does not relieve the pain, it may either have been a latent TrP or it may *not* have been the *only* active TrP referring pain to that area.

Examination for mechanical perpetuating factors requires careful observation of the patient's postures, body symmetry, and movement patterns. A recent review[47] lists many of these factors that need to be considered; they are discussed in detail on pages 104–114 in Volume 1[35] and in Section 7 (**Activation and Perpetuation of Trigger Points**) of the muscle chapters in both volumes of THE TRIGGER POINT MANUAL. Common mechanical factors that can influence many muscles are the round-shouldered, head-forward posture with loss of normal lumbar lordosis, and body asymmetries including a lower limb-length inequality and a small hemipelvis. The postural factors are discussed in the following section on treatment, in Chapter 2 of this volume, and, as appropriate, in individual muscle chapters. Body asymmetries are presented in detail in Chapter 4 of this volume. Tightness of the iliopsoas and hamstring muscles can also seriously disrupt balanced posture.

3. DIFFERENTIAL DIAGNOSIS

Two variants of myofascial pain syndromes should be recognized: the myofascial pain modulation disorder, which leads to diagnostic confusion, and the post-traumatic hyperirritability syndrome, which complicates management. In addition, either fibromyalgia or articular dysfunction can confusingly mimic a chronic myofascial pain syndrome. Each requires an additional specific examination technique and its own treatment approach.

To help a patient with chronic enigmatic pain, the examiner must find sources of pain that have been overlooked, and that means conducting examinations that were not previously performed. After the history, the first order of business is to conduct a time-consuming, detailed, complete physical examination looking for well-known causes of pain that were missed.[21,34] Such an examination is rarely performed when the examiner expects to find that the patient's pain is "all in the head."

Myofascial Pain Modulation Disorder

The term "myofascial pain modulation disorder",[45] adapted from a term used by Moldofsky,[35] identifies a relatively small group of myofascial pain patients who show a remarkable distortion of their pain referral patterns. Instead of each active TrP projecting pain to its expected location (reference zone), the referred pain and tenderness from all TrPs in a region converge on one common location. This location may not be the expected zone of pain reference for any of the involved muscles. Characteristically, the convergent focus is the site of previous trauma or intense pain prior to onset of the pain modulation disorder. These features resemble the experimental observations of Reynolds and Hutchins.[36]

It appears that the aberrant referral patterns are caused by a distortion of sensory modulation in the central nervous system. Many of these patients had previously experienced trauma or painful impact at the focus of pain, but often not of such severity that it would be expected to cause structural damage to the central nervous system. The mechanism behind

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