

# The Management of Myofascial Pain Syndromes

Robert D. Gerwin

**SUMMARY. Objective:** To present an approach to the management of the Myofascial Pain Syndrome [MPS] based on clinical experience, addressing the problems posed by the specific nature of primary myofascial trigger point pain, and the problem of chronic myofascial pain.

**Results:** Specific trigger point therapy is designed to inactivate the myofascial trigger point by means of manual techniques, intermittent cold and stretch, and invasive trigger point needling or injection with a local anesthetic. Management of recurrent MPS requires addressing the perpetuating factors of mechanical imbalances [structural, postural, compressive] and systemic abnormalities which interfere with the ability of muscle to recover or which continuously stress muscle reactivating the trigger point. The common systemic factors associated with MPS are hypothyroidism, folic acid insufficiency and iron insufficiency. The relationship of many of the perpetuating factors to MPS is clinically apparent, but has yet to be established firmly by statistically rigorous clinical studies. Corrective measures to prevent the reactivation of trigger points, physical reconditioning and psychological re-education help maintain improvement.

**Conclusion:** MPS is a condition which is treatable by eliminating the specific trigger points that are the immediate cause of pain, and correcting those factors that predispose to recurrence.

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### *INTRODUCTION*

Management of the *MYOFASCIAL PAIN SYNDROME* [MPS] requires both general measures for treatment of the structural and systemic perpetuating factors, and specific measures for treatment of the *Myofascial Trigger Point* [MTrP] itself. Treatment is based on the concept that the pain in MPS, as well as the taut band, local twitch response, referred pain, restricted motion, and weakness and autonomic phenomena, is caused by the trigger point within the muscle. The goal of treatment is to inactivate the MTrP, and to prevent its reoccurrence. The specific measures used to inactivate the MTrP are intermittent cold and stretch, and Trigger point [TrP] injections. The general measures are directed towards the perpetuating factors to correct the structural asymmetries and imbalances, and the systemic disorders which lead to muscle stress or overuse, and which perpetuate MTrPs and the MPS. Finally, corrective action must be taken to reduce the risk of reactivation of MTrPs and recurrence of MPS.

### *DIAGNOSIS*

The first task is to make an accurate and complete diagnosis of MPS and of any pertinent associated condition such as cervical spondylosis, fibromyalgia, or specific nutritional or hormonal insufficiency state. The physical examination for MTrPs should determine if the MPS is localized to one regional functional group of muscles which work together as synergists or antagonists, or if it is widespread, affecting many functional muscle groups. The more chronic MPSs tend to spread through the development of *secondary* TrPs within the functional group, or through the development of *satellite* TrPs within zones of referred pain at distant sites (1, see Vol.1: 12-17).

### *PERPETUATING FACTORS*

Factors that maintain chronic MTrPs can be divided into Mechanical factors and Systemic factors. These should be corrected insofar as possible prior to treating the MTrP specifically. If left uncorrected, reactivation of the MTrP is likely.

### *Mechanical Perpetuating Factors*

The mechanical factors include structural inadequacies, (1, see Vol.1: 41-63), postural stresses and muscle compression. The most common structural inadequacies are the short leg syndrome or leg-length discrepancy, the small hemipelvis, which produces the same effect as the short leg syndrome, the long second metatarsal bone, and short upper arms.

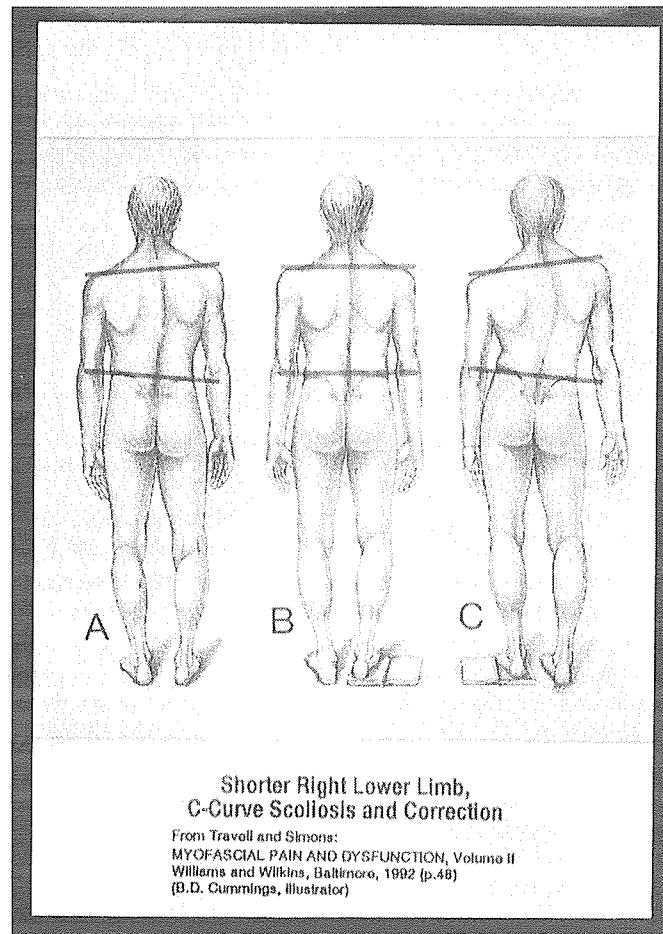
*The short leg syndrome [Figure 1].* The most common structural inadequacy is lower limb inequality. As with other mechanical stresses, this is usually well tolerated until an injury or illness destabilizes previous compensatory mechanisms. When one leg is shorter than the other, the pelvis tilts, causing the spine to tilt towards the short leg side. The spine then curves to right the head and level the eyes. If the inequality is slight, less than 1/2", a C-shaped scoliosis develops, causing the shoulder to rise on the side of the short leg. With a greater inequality, the scoliosis is often S-shaped, and the low shoulder may be on the side of the short leg. In the case of C-shaped scoliosis caused by leg-length discrepancy, a heel lift can level the pelvis and the shoulders, relieving the stress on the lower and upper back muscles, the trapezius, the levator scapulae, scaleni, and sternocleidomastoid muscles.

*The small hemipelvis.* This condition, caused by an asymmetry in the height of the two halves of the pelvis, causes a functional scoliosis when sitting that is similar to that described for the short leg syndrome. The stress on muscle is the same as in the short leg syndrome, as the head is maintained in the erect posture. An ischial or butt lift under the ischial tuberosity will level the pelvis and provide a similar correction as a heel lift.

*The long second metatarsal bone (2).* The first metatarsal bone is commonly longer than the second, allowing a stable tripod support of the foot by the heel and the heads of the first two metatarsal bones. When the second metatarsal bone is longer, the foot balances along a line from the heel to the second metatarsal head, creating an unstable knife-edge effect. As weight is transferred from the second to the first metatarsal head, the foot pronates, internally rotating the leg at the knee and hip. This causes MTrPs to develop and persist in the peroneus longus [ankle pain], the vastus medialis [knee pain], and the gluteus medius [low back pain]. Secondary TrPs in the gluteus minimis refer pain to the posterior thigh and calf. The condition can be corrected by an orthotic selectively supporting the first metatarsal head, thereby recreating the usual tripod support (1, see Vol.1: 110-112; Vol.2: 381-392).

*Short upper arms.* Persons with relatively short upper arms experience postural stresses on shoulder girdle muscles when sitting in chairs which

FIGURE 1. Lower limb inequality with a short right leg. A. Uncorrected, the right iliac crest is lower than the left, tilting the spine to the right. Compensatory contraction of the left quadratus lumborum muscle brings the left rib cage downward towards the left iliac crest, curving the thoracic spine back to the left and dropping the left shoulder. The right lateral cervical muscles right the head, leveling the eyes. B. Corrected with a lift under the right foot, leveling the pelvis and shoulder axes, relieving the stress on the quadratus lumborum, levator scalenus, trapezius, scaleni and sternocleidomastoid muscles. C. Incorrect placement of a lift under the longer left leg, accentuating the scoliosis. (From Travell JG, and Simons DG: *Myofascial Pain and Dysfunction: The Trigger Point Manual*, volume 2. Williams & Wilkins, Baltimore, 1992. Used with permission.)



do not give adequate arm support. Leaning to one side or slouching activates MTrPs in the quadratus lumborum causing low back pain. Inadequate shoulder support maintains MTrPs in the trapezius muscles. Adjustment of armrest height or desk height, or slanting the work surface, can alleviate this condition.

Other postural stresses that occur at work or at home can be discovered through a detailed history, or by examining photographs of the individual at different activities.

Chronic compression of muscle can cause MTrPs to persist. Tight brassiere straps, tight collars, and compression of the hamstring muscles by a seat edge are examples of readily correctable causes of muscle compression.

### *Systemic Perpetuating Factors*

Metabolic, endocrine, toxic, inflammatory, and other systemic disorders can stress muscle, and impair its ability to heal. The most common systemic factors that we encounter among persons with MPS are hypothyroidism, folic acid inadequacy and iron insufficiency. The relationship of these factors to persistent MPS seems clinically evident, but has not been established by statistically rigorous studies. Among systemic illnesses, fibromyalgia deserves special mention because it causes widespread muscle discomfort, and may be confused with MPS.

Subclinical or marginal *hypothyroidism* (3) is often overlooked. Symptoms, which can be subtle, include widespread MTrPs, cold intolerance, fatigue, and constipation. Serum cholesterol can be elevated. Hyperactivity is an unexpected sign, caused by constant body movement in an attempt to generate heat. Muscle cramps, stiffness and pain occur as a result of muscle overactivity. The most useful test is the highly sensitive thyroid stimulating hormone assay. A level in the upper range of normal and a low or low normal T4 should lead to further investigation and consideration of a trial of thyroid hormone replacement therapy. Previously unresponsive MPS may improve when marginal hypothyroidism is corrected.

Nutritional inadequacy states must be considered in terms of optimum function of enzyme systems, rather than absolute deficiency states (4). Vitamins act as cofactors in different enzyme systems that may be functioning at different rates at any one time. The optimum level of a vitamin is that which permits maximum function for each enzyme for which it is an essential cofactor. The vitamin requirements therefore change with time and circumstances. The daily vitamin intake should thus support optimum function. The daily requirement is therefore affected by host factors such as smoking or by competitive inhibition from drugs.

The most common vitamin inadequacy in persons with MPS in my experience is that of *folic acid* (5). Persons with folic acid levels in the lower quartile of normal often feel cold, but tend to have a low cholesterol level in contrast to hypothyroidism. They have diarrhea, rather than constipation as seen in vitamin B12 inadequacy. The fast-twitch type 2 muscle fibers of the upper body are more likely to develop MTrPs. Headache, disturbed sleep and restless legs can be seen. Folate is present in food as reduced polyglutamates. The stable inactive, oxidized form pteroylglutamate used pharmacologically must be reduced to the active form tetrahydrofolate by dihydrofolate reductase. A vitamin B12 dependent enzyme transport system brings folate into bone marrow, where it can bind to a red cell surface membrane receptor. Measurement of serum folate, serum vitamin B12 and red blood cell folate gives the most complete assessment of folate status. Impairment of DNA synthesis by either vitamin B12 or folic acid deficiency leads to megaloblastic anemia. In persons with intrinsic factor deficiency parenteral B12 is administered. In persons with restricted intake of animal food products, vitamin B12 supplementation is essential, since it is available only from animal sources.

*Iron* inadequacy (6) is also frequently seen in persons with MPS, usually premenopausal women who have inadequate iron intake to replace menstrual blood loss. Persons chronically taking NSAIDs can also have microscopic gastrointestinal blood loss, leading to depletion of iron stores. Unusual fatigue, exercise-induced muscle cramps, and cold intolerance are characteristic symptoms. Tissue iron stores are best assayed by measuring serum ferritin levels. Iron is important in heme-enzyme functions such as the cytochrome-oxidase system essential for oxidative phosphorylation.

Many persons with MPS are cold or cold intolerant. Inadequate levels of thyroid hormone, folic acid or tissue iron (7) can be associated with this feeling. Iron is essential for the conversion of T4 to the active form of T3, which may be one link between these conditions.

Rheumatoid arthritis, gout, polymyalgia rheumatica, recurrent *Candida albicans* infections, other nutritional insufficiency states, and psychological stresses are among the many other medical conditions that may perpetuate MTrPs.

Fibromyalgia [FM] (8,9) causes generalized muscle pain, fatigue and an alpha-delta sleep disorder. Muscle involvement in FM is homogeneous, affecting muscles in all body areas uniformly. Muscle involvement in MPS is heterogeneous, affecting different muscle groups, and affecting muscle fibers differently within each muscle. MTrPs can occur in FM, creating an

overlap condition. When this happens, the MTrPs are treated in the same manner as usual.

### *MTrP INACTIVATION*

The specific treatment of MPS is the inactivation of the MTrP, most commonly by intermittent cold and stretch (10,11), and by TrP needling or injection.

Before initiating treatment, the patient must first be made comfortable and relaxed [Figure 2]. Functional scoliosis is corrected and arm and leg support is provided to prevent tension on the shoulder and hip muscles. The patient is often treated lying down to avoid postural hypotension which can accompany injection therapy.

*Intermittent cold and stretch (1, Vol.1: 63-74) [Figure 3].* Vapocoolant spray or ice accompanied by stretch utilizes a tactile and thermal stimulus to inhibit spinal cord mediated reflex muscle contraction as muscle is

FIGURE 2. Positioning the patient in the lateral decubitus position in preparation for examination and treatment with either intermittent cold and spray or trigger point injections. Pillows are placed to support the head and the uppermost arm and leg. The room is kept warm and glare is avoided.

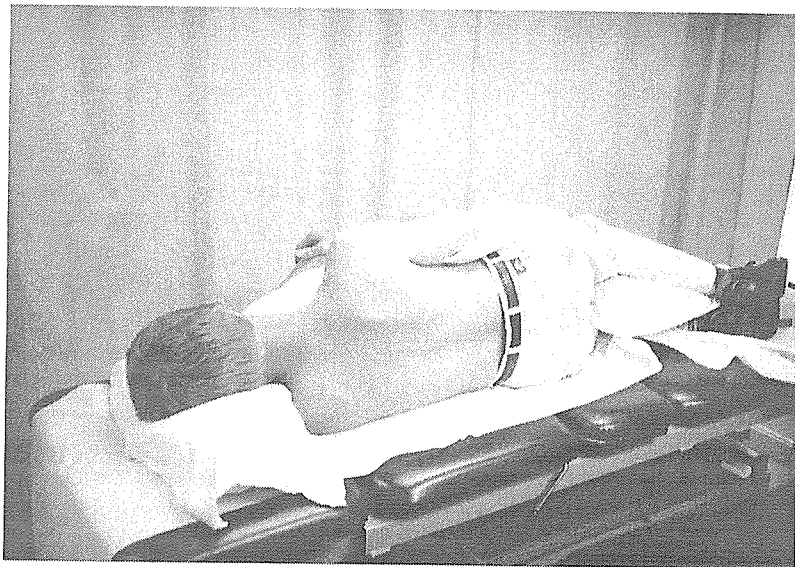
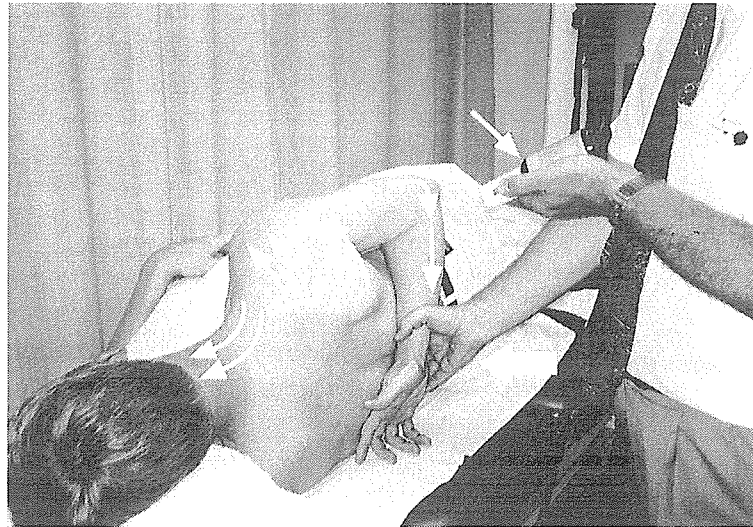


FIGURE 3. Intermittent cold and stretch. The vapocoolant used in this case is held about 40-50 cm from the muscle to be treated. The spray is applied at an acute angle to the skin, moving at 10 cm per second from the trigger point area over the muscle towards and over the zone of referred pain. Stretch is applied as the skin is sprayed. Moist heat is applied after the spray and stretch is completed, following which active and passive range of motion is performed to the part treated. X's mark common trigger point sites in the infraspinatus muscle. The arrows show the direction of spray over the muscle and into the zone of referred pain, including the arm and hand. The large short arrow shows the angle of application of the vapocoolant. The small short arrow indicates the stretch that is the essential action that inactivates the trigger point.



stretched. The combination of cold and the accompanying tactile stimulation produced by the fine jet spray or the touch of the ice produces a noxious stimulus which may excite the off-cells that suppress nociceptor transmission in the spinal cord. In this way, intermittent cold applied while stretching a muscle may inhibit a pain-initiated spasm or contraction. Stretching to the full length of the muscle is the essential action that inactivates the TrP. The cold stimulus is applied in parallel sweeps from the TrP over the functional muscle group to the zone of referred pain. There may be a diagnostic as well as therapeutic decrease in pain and increase in range of motion. Each muscle has a specific direction of action; the movement applied to stretch a muscle is opposite the muscle's direc-

tion of action. After stretching, the muscle is rewarmed with moist heat. This technique is particularly suitable for treating large areas of the body, when MTrPs involve many muscles.

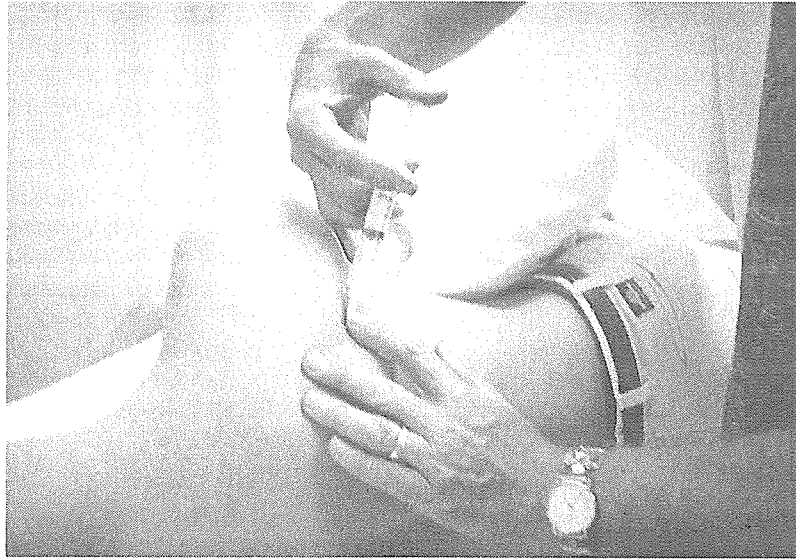
*Trigger point injection (1, Vol.1: 74-86) [Figure 4].* Dry needling a MTrP or injecting it with a local anesthetic is a highly effective way of inactivating a TrP. The procedure may act by disrupting the sarcolemmal membrane causing a change in intra- and extra-cellular calcium concentrations. Inactivation of the MTrP by injection is reversed by intravenous naloxone (12), suggesting that the procedure modifies the endogenous opioid system in the central nervous system. Dilute procaine is the preferred anesthetic (13), as it has low myotoxicity, a short half-life, and is metabolized peripherally by procaine esterase. An inadvertent nerve block is quite transient. Bacteriostatic saline can be used for those allergic to procaine.

The MTrP is located by palpation and fixed between the fingers. A 25 gauge needle is inserted at an oblique angle into the TrP. A local twitch response and transient pain may be elicited. Referred pain and transient

FIGURE 4. A and B. The needle is inserted obliquely through the skin after fixing the trigger point between the palpating fingers. Trigger points frequently occur in clusters, so that the muscle must be probed in a circular manner in order to identify and inactivate them all. The muscle is treated with intermittent spray and stretch after the injections are completed.



FIGURE 4 (continued)



dysesthesia of short duration can be felt. MTrPs often occur in clusters and all must be treated. Satellite TrPs need to be treated either by injection or by stretch. Intermittent cold and stretch followed by moist heat is performed after the injections.

TrP injection is an invasive procedure with the risk of hemorrhage, pneumothorax, allergic reactions and vasodepressive syncope. Emergency care should be available in case of a complication. Failure of TrP injections to succeed can be due to injecting an inactive, not active, MTrP; to injecting the taut band, not the TrP; missing the TrPs in related muscles, to inadequate hemostasis, and to failure to actively stretch the muscle after injection.

*Manual myofascial release techniques.* In addition to ultrasound and electrical stimulation, other manual techniques have been effective in inactivating the TrP. Ischemic compression of a MTrP for 30-60 seconds can relax a TrP, presumably by depriving the muscle of oxygen and glucose through compression of capillary vessels. Rhythmic percussion of the TrP at about 2 second intervals will often inactivate a TrP, the percussion acting as a counterirritant in a manner similar to electrical stimulation or the application of heat or cold, possibly stimulating endorphin production

or activating the off-cells that suppress nociceptive transmission centrally. Post-isometric relaxation (14), isometrically contracting a stretched muscle against resistance, relaxing it and then stretching it can be taught to the patient. Rhythmic stabilization utilizing reciprocal inhibition through alternating contraction of agonist and antagonist muscles against resistance will gradually lengthen muscle and increase range of motion. Other techniques such as friction massage can be effective in skilled hands.

*Drug therapy.* There is no specific drug therapy for MPS. Sleep disturbance can be corrected pharmacologically. Analgesia may be provided by aspirin, acetaminophen or by NSAIDs. Use of tricyclic antidepressants are well established in management of chronic pain states, and can also help lessen insomnia.

Finally, the patient must be taught to recognize those activities that aggravate the pain, and to understand the distribution of pain from affected muscles. Corrective activities must be learned, and a home exercise program is prescribed to first stretch and then strengthen muscle. When muscle is injured, the body learns adaptive, protective behavior. Psychological reeducation and muscle conditioning is essential for long term recovery.

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