Myofascial Pain Syndromes: Part I — General Characteristics And Treatment

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“The phenomena of pain belong to that borderland between the body and the soul about which it is so delightful to speculate from the comfort of an armchair, but which offers such formidable obstacles to scientific inquiry.”

With these words Kellgren opens his excellent discussion of “Deep Pain Sensibility” and it is with similar caution that the entire subject of “Myofascial Pain Syndromes” must be approached. No attempt will be made to explain the pathologic origins of the syndromes which will be discussed in this paper. Under the name of fibrositis, extra-articular rheumatism, and numerous eponymic syndromes the etiology of the myofascial pain syndromes has been attached in turn to trauma, infection (focal or otherwise), fatigue, vascular, metabolic, postural, occupational and psychogenic factors. It is most likely that any or all of these factors may be operative and an intelligent approach must take all possibilities into account. Apparently we are dealing with a pattern of non-specific shock organ response to stress.

One of the only certain facts about these syndromes is the fact that they exist. Every attempt to trace them as pathological entities has proven their elusiveness. There is at present no consistent evidence concerning the pathological changes occurring in the muscles, fascia, ligaments and periosteum which are the reputed seat of the disease. The theory of herniation of fat particles and the theories of actual inflammation of fibrous tissue structures break down under careful microscopic scrutiny which so far has turned up nothing constant.

These syndromes all combine the existence of pain with the presence of tenderness in soft tissues. The pain may appear directly at the point of tenderness, or it may appear at a distant point of reference. If the pain is distant from the point of tenderness, it is customary to call the distant area the “target” area and the tender area the “trigger” area. These are so named because pressure over the trigger area often reproduces pain in the target area—in addition to the immediate local tenderness. Infiltration of the trigger area with a local anesthetic abolishes the pain in the target area. If the patient’s pain is located primarily at the major site of tenderness, local infiltration will relieve the pain.

The syndromes to be discussed are part of a single family of clinical entities with common characteristics, including the above trigger mechanisms and responses to local anesthetic infiltration. All members of the family have a similar type of pain. The pain is aching in nature, diffuse and hard to localize, sometimes actually migrating about on an extremity or on the trunk. It is aggravated by sudden changes in the weather, and especially by cool, damp weather. It is almost invariably made worse during times of emotional stress or tension. One of the most disagreeable characteristics of the pain is its accompaniment by the “gelling” phenomenon. This phenomenon is typified by stiffness of joints in the involved area after moderate periods of inactivity. The stiffness-pain combination is relieved by movement of the affected segments.
Although trigger points may exist apparently within any mesodermal tissue between the bones and the skin, the syndromes mentioned here are assumed to have their trigger areas primarily within muscle tissue. Therefore in addition to pain and stiffness after inactivity, the patient often also has pain after prolonged activity, due to stimulation of the trigger point within the muscle. The patient therefore gives a history of stiffness at rest, aching pain after long resting (with a necessity for frequent shifts in position), followed by relief of pain on first moving about, followed in turn by a gradual return of severe aching pain and tiredness after moderate amounts of activity.

The myofascial pain syndromes may be acute, subacute, or chronic. In any phase they are differentiated from other forms of rheumatic disease and from neurologic disorders by the above typical findings and symptoms, plus the absence of significant changes in vital signs, sedimentation rate, circulating white cells, muscle strength (except as restricted by pain), and deep tendon reflexes. Hyperalgesia of the skin to pinprick over the reference or target area is not uncommon, especially in the acute stage.

The acute stage is characterized by the onset over a period of hours or days of the above-described severe, aching pain, accompanied by an increase in pain on motion. In both the trigger area and the reference area there is muscle spasm, a combination of severe tenderness and tightness of the palpated areas. There is often a persistence of skin blanching, simulating dermatographia, plus a tendency to flaring as encountered in histamine injection. In the acute stage the patient will not tolerate much palpation, and such examination should be kept at a minimum to prevent the development of further spasm and pain. This can be referred to as a “hot” muscle or trigger area, and the further evolution can be considered a cooling off of the process.

The primary weapon in treatment of the acute stage of myofascitis (if such we must call it) is reflex blockade—intense heat, directed at both the trigger and reference areas—or intense cold directed at these areas. Some authors even advocate the alternate use of these modalities. Moist heat seems empirically more effective than dry heat, so that hot packs and various forms of steam applications are most often used. Ethylchloride spray serves as a method of applying intense cold; this is a method of unusual effectiveness, considering its extreme simplicity. Each of these reflex blockade methods is assumed to act by “jamming the frequency” on which the trigger point is broadcasting in the spinal cord. The cord level of trigger and target are usually within a few vertebral segments of each other; the intense afferent bombardment from the heat or cold apparently blocks the deep pain for sufficient time to upset the primary cycle of pain-spasm-pain.*

The concomitant use of anodynes in the acute stage is usually necessary. Aspirin will often suffice in large doses, but codeine is necessary in some cases, each combined with barbiturates in anxious persons. The presence of major psychogenic factors in the etiology and continuation of these syndromes has been mentioned. The patient gains a fair amount of his relief from the reassurance by the physician that there is “nothing serious” wrong and that he does “not have arthritis.” It is certainly not

*Diathermy is empirically noted often to increase pain in early fibrositis and should not be used in acute cases.
necessary to x-ray every case of acute myofascitis, but any failure to respond in a reasonable period of time (two or three days) should prompt a vigorous diagnostic search, including appropriate x-rays. The relative lack of ultimate severity of the syndrome should not mitigate against hospitalization during the acute phase if the patient is extremely uncomfortable. Close following and dogged changing of therapy in the absence of response is one of the major keys to success in treatment of myofascitis; such a program sometimes necessitates hospitalization in any phase of the disease.

The subacute stage is entered as the extreme tenderness of the involved area cools off. In this stage the pain has subsided to a dull, but fairly constant ache and one of the major problems may be lack of sleep because of inability to get comfortable. It is in this phase that injection therapy becomes valuable. Although injection is often helpful in acute cases of back or neck sprain, it seems more irritating than useful in acute fibrositis. It is therefore reserved for use when the myofascitis (recognized clinically by tenderness) has become localized to one or a few easily accessible spots. During the subacute stage ethyl chloride spray is still a very useful agent, and of course salicylates form a foundation of the total program. This author has not used the steroids or Butazolidin in the acute stage, but has preferred to reserve their use for more refractory cases which do not respond in the subacute or chronic stages. Both of these drugs are used in combination with physical treatment methods, and their administration is continued for as short a time as possible.

In the chronic stage of myofascitis we come to the controversial subject of the “fibrositic nodule.” There exists little doubt in the minds of persons who continually are engaged in palpating muscles that nodules are present in the chronic phase of myofascial pain syndromes. The inability to demonstrate them microscopically does not prove their absence, since probably a major part of their pathophysiology is spasm. The fibrositic nodule forms at the corresponding trigger area for the given syndrome. Such a nodule is by no means always present, so that chronic trigger points can exist manifested only by tenderness and by severe pain on needling. The chronic fibrositic area when needled presents a characteristic feel as though the needle were being firmly grasped by the muscle; on some occasions a grittiness is felt during the needle passage (but nothing is found on x-ray to suggest calcium deposition!)

The treatment of the chronic trigger point or nodule is by a combination of intense surface heat, specific trigger point injection, massage and/or stretching. The heat can be applied at home by an infra-red bulb; fifteen minutes once a day will suffice. The trigger point injection can be made with any calibre needle and, mirabile dictu, can be partially effective even if a dry needling is performed. Saline injections alone will relieve a large percentage of cases; 1% procaine hydrochloride (10 cc) will relieve a still larger percentage. This injection is routinely followed in our clinic by 1 cc (25 mgm) of hydrocortisone suspension; the two agents are not mixed.

Stretching is carried out in a variety of common-sense fashions, each of which will be mentioned under the appropriate syndrome. Massage itself is indeed a form of stretching the involved area in chronic myofascitis. It is inferior in rapidity of effect to injection, but its effects are just as lasting. Because of the necessity to return for repeated, expert massage at the hands of the physical therapist the massage treatment usually becomes more expensive than injection alone. On occasion the two may
be combined, or they may be substituted one for the other in the event of treatment failure.

It is common for the patient to present himself to the physician with a history of chronic myofascitis existing many months or years. A careful history may show episodes dating from childhood and encompassing practically all of the various possible symptom combinations. Such patients are said to have a “fibrositic diathesis” or increased susceptibility of muscle as a shock organ to stress. Psychogenic factors rate high among the aggravating factors of fibrositis in such patients and the physician should not hesitate to use transference, re-assurance, and environmental manipulation in the relief of stress and tension in the patient’s life.

Also, in the chronic fibrositic an occupational posture or repetitive motion factor may be operative, so that a job change will sometimes be indicated. If job change is impossible, different work habits should be encouraged. Certain postural exercises—usually stretching—may be helpful.

In the next issue of this journal will be discussed the following family of myofascial pain syndromes: Occipitofrontal tension headaches; trapezius syndrome; scapulocostal syndrome; scapulohumeral syndrome; anterior chest wall syndrome; abdominal wall syndrome; lumbago; pelvic floor syndrome; adductor longus syndrome; scalenus anticus syndrome.

REFERENCES