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Medical Progress

Fibrositis: Misnomer for a Common Rheumatic Disorder

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Fibrositis is a misnomer for a very common form of nonarticular rheumatism. The name implies an inflammatory process in fibroconnective tissue which has never been verified. The symptoms of fibrositis are ill-defined musculoskeletal pain made worse by stress, cold, noise and unaccustomed exercise; there is usually a significant element of depression, nonrestorative sleep, chronic fatigue and early morning stiffness. Results of physical examination are strikingly normal, apart from painful tender spots which are remarkably consistent in location from patient to patient. It is important to realize that fibrositis can complicate diseases such as rheumatoid arthritis and systemic lupus erythematosus, where its prompt recognition is essential in averting inappropriate medication. Drug therapy alone is seldom effective in alleviating symptoms; a carefully planned education program is necessary to readjust both psyche and soma.

FIBROSITIS is generally used to describe a type of nonarticular rheumatism characterized by aching and stiffness in the presence of focal tender points.[1,2] It is a poorly defined and controversial term that implies some knowledge of a specific underlying pathological conditon (that is, inflammation of fibroconnective tissue) which has never been proved to exist. The absence of a well-defined pathological lesion, normal laboratory test results and an apparently inappropriate degree of pain and misery have led many physicians to regard this condition as purely psychogenic in origin or a form of malingering. This reluctance to make a diagnosis of fibrositis has led to the use of other terms (Table 1) or the elaboration of nonscientific explanations for this symptom complex. *Fibrositis* as now generally used is descriptive of a common clinical syndrome without any implication as to pathogenesis. Recent clinical studies have led to a sharper definition of fibrositis. The rapidly expanding areas of sleep research, neurotransmission and pain perception have provided some intriguing clues which may be germane to the symptom complex of fibrositis. The purpose of this review is to present these

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TABLE 1.—*Terminology of Fibrositis*

Fibrositis	Myofasciitis
Fibromyositis	Myositis
Myofascial pain syndrome	Strain
Muscular rheumatism	Sprain
Fasciitis	Trigger point
Myalgia	Psychogenic rheumatism
Nonrestorative sleep syndrome	Pain amplification syndrome

newer ideas and, it is hoped, stimulate a heightened awareness and more scientific approach to this common medical problem.

Historical Aspects

The term fibrositis was coined by Gowers in 1904 in a paper in the *British Medical Journal* on lumbago that also mentioned fibrositis of the arm.[3] He hypothesized that both conditions were due to inflammation of the fibrous tissue of muscle; no pathological verification was provided. In the same year Stockman reported findings from seven biopsy studies of "fibrositic nodules" in a group of patients complaining of stiffness, aching and painful muscular movement.[4] The nodules that were excised showed a low-grade inflammatory reaction which he ascribed to "small colonies of microbes invading the tissues and causing a reaction which comparatively rapidly destroys the invader." However, his cultures remained sterile. Subsequent workers, such as Abel, Sibert and Earp from St. Louis, Missouri,[5] and Collins[6] and Slocumb[7] both from the Mayo Clinic, have failed to find any evidence of a low-grade inflammatory response in biopsy specimens of muscle.

The concept of fibrositis was popularized by Llewellyn and Jones of Bath, England, in a book published in 1915 entitled *Fibrositis*.[8] They defined myofibrositis as "inflammatory change in the interstitial fibrous tissues of a striated or voluntary muscle." Stockman further reinforced this unfounded concept in a chapter in his own book *Rheumatism and Arthritis*.[9] A different view was taken by Elliott in 1944.[10] On the basis of electromyographic studies he suggested that fibrositic nodules were the result of localized muscle spasm. Further support of the muscle spasm concept was provided by the studies of Kelly, who showed that there was a temporary relief of symptoms in 30 percent to 40 percent of cases after the use of local procaine injections into the primary myalgic spot, rather than the area of referred pain.[11] This he attributed to an interruption of a reflex arc. At about the same time Copeman and Ackerman published two convincing studies which indicated that fat herniation and peduncu-lation through adjacent fascial planes were responsible for the painful "trigger points."[12,13] In 1968 Kraft, Johnson and LaBan defined the "fibrositis syndrome" as having four essential features: point tenderness of muscle, a localized induration of muscle, dermatographia and a reduction of pain with ethylchoride spray.[14] In electromyographic studies of 29 patients they found no evidence to indicate that muscle spasm was a cause of the localized muscle induration.

Thus there has been continuing point and counterpoint in the saga of fibrositis. It would seem that a major impetus to further study has been the consistent finding of the enigmatic "fibrositic nodule." Even here the occasional dissenting voice is heard; Hench remarked that they were "only accessible to the finger of faith."[15] However, most workers have not denied the existence of these tender spots and indeed the first description goes back to 1843 and is accredited to the German physician Froriep.[16] In fact there is an extensive German language literature, attesting to the reality of these tender spots, which has recently been reviewed by Simons.[17] Given this historical background it is hardly surprising that the whole area of muscular rheumatism and fibrositis is often treated with some skepticism.

Diagnostic Evaluation

Patients with fibrositis complain of musculoskeletal pain and soreness of a predominantly proximal distribution. The pain can seldom be accurately localized and there is no history of joint swelling. It is inconsistent in location with a tendency to shift in position and vary in intensity. Muscle weakness is often claimed, but on closer questioning this is invariably a secondary response to pain and true weakness is not a problem. Morning stiffness and easy fatigability are usually prominent symptoms and mimic those seen in association with the classical inflammatory rheumatic disorders. Such patients are naturally concerned that they may be afflicted with a potentially crippling and incurable arthritic disease.

There are certain clues in the history which should suggest a possible diagnosis of fibrositis; these revolve around those factors that exert an exacerbating or ameliorating influence. Most of the patients will volunteer that their symptoms are aggravated by one or more of the following:

emotional stress, chronic fatigue, unaccustomed exertion, noise, bright lights, exposure to dampness or chilling, and unresolved conflicts or decisions. Improvement can occur with a hot bath or shower, massage, local application of heat, a holiday, gentle exercise and other diversionary activities. The history should always include information about potential initiating factors either real or imagined. It is not uncommon for the onset of fibrositis to be associated with an automobile or work-related accident. The perpetuation of symptoms by chronic anxiety, often in association with considerations of secondary gain, is difficult to evaluate and poses sensitive medicolegal issues.

It is important to gain some understanding of the effect of this disease on the patient's ability to work, perform the usual chores of daily living and pursue preferred recreational activities. An ability to make light of the symptoms and the continuation of a normal living pattern augur well for a good prognosis. Conversely a history of many days lost from work and a major disruption of the previous life-style usually indicate that effective treatment will be difficult.

Patients with fibrositis are often depressed, and specific questions and observations should be directed toward the following points: decreased energy, miserable or troubled facies, ponderous thinking, indecisiveness, loss of sexual interest, reduced appetite, constipation, feelings of inadequacy, suicidal ideas and inadequate sleep. Indeed, a nonrestorative sleep pattern may be a major contributing factor to the symptomatology of fibrositis (see section on pathophysiology).

The physical examination of a patient with fibrositis is notable for the lack of abnormal findings in contradistinction to the patient's history of pain and misery. Such patients usually relax poorly and complain of pain during the examination, but no specific changes are found in the joints and there is a well-preserved musculature. Such unrewarding examination findings in the context of the history should always suggest a diagnosis of fibrositis and direct attention to areas not routinely examined. Such areas are the so-called trigger points or tender spots. They are at locations over muscles and ligamentous bony insertions that are often tender but not painful in healthy persons. These areas should be firmly palpated by the examiner's thumb in a systematic manner while observing the patient's reaction. Symptomatic tender spots will usually elicit verbal expression of pain or a withdrawal response. There is often a reproduction of the patient's symptoms in a referred distribution. Smythe has described 14 typical sites of deep tenderness.[1,2] Other areas have often been noted and some 25 tender spots are shown in Figure 1.

One further finding should be sought, namely skin-fold tenderness over the upper scapular region. Rolling the skin between the thumb and index finger in this area is excruciatingly painful

TABLE 2.—*Diagnostic Guidelines*

History

Ill-defined musculoskeletal pain, often shifting in location.

Exacerbation of symptoms by stress, noise, cold, unaccustomed exercise and chronic fatigue.

Temporary relief of symptoms by heat and massage.

Prominent morning stiffness.

Chronic fatigue and poor work tolerance.

Nonrestorative sleep.

Initiation of symptoms by emotional or physical trauma.

Examination

Normal findings on joint examination.

Normal muscle mass and strength.

Tenderness over the "tender points." (See Figure 1.)

Skin-roll tenderness over upper scapular region.

Depressed affect.

Temporary relief of referred pain by infiltration of the trigger point with local anesthetic.

Normal ESR, SGOT, muscle enzymes, rheumatoid factor, ANF and appropriate findings on skeletal x-ray studies.*

ANF = antinuclear factor
ESR = erthrocyte sedimentation rate
SGOT = serum aspartate aminotransferase (serum glutamic oxaloacetic transaminase)

*When fibrositis is secondary, there will, of course, be abnormalities shown on x-ray studies.

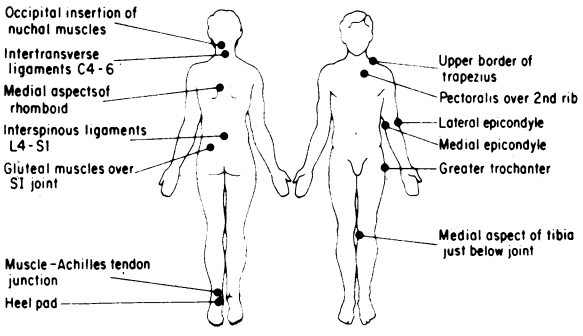

Figure 1.—Areas where pain is commonly found in patients with fibrositis. Palpations should be firm and the specificity of tenderness confirmed by palpation of an adjacent area. There are 25 representative points shown, making up 12 paired areas and one central area.

in patients with fibrositis and is often followed by a pronounced reactive hyperemia of the overlying skin.

Positive observations in the aforementioned areas should be controlled by equally firm palpation of adjacent areas.

Smythe has proposed a rigorous definition of fibrositis which would appear useful in controlled clinical studies[1]: (1) widespread aching for longer than three months; (2) local tenderness at 12 of the 14 specified sites; (3) skin-roll tenderness over the upper scapular region; (4) disturbed sleep with morning fatigue and stiffness, and (5) no abnormalities of erythrocyte sedimentation rate, serum aspartate aminotranferase (SGOT), rheumatoid factor, antinuclear factor and muscle enzymes, or on sacroiliac films.

These criteria are too strict for many patients who undoubtedly have fibrositis, and it is suggested that they be modified for general clinical use by altering item 2 to "local tenderness at 10 of the 25 specified sites" (see Figure 1). Furthermore, other investigations, in addition to those given in item 5, are sometimes indicated (see the section on differential diagnosis, which follows). One useful additional diagnostic maneuver is the local infiltration of a major tender spot with local anesthetic. This results in a temporary alleviation of the pain in the referred area and also serves as a convincing demonstration in explaining the nature of the disease to the patient. These diagnostic considerations are summarized in Table 2.

Differential Diagnosis

Psychogenic Rheumatism

The occurrence of persistent and often disabling symptoms, in the face of an ostensibly normal physical examination and normal laboratory studies, usually suggests major psychogenic factors. In this respect the seeking of painful tender spots and skin-fold tenderness is of paramount significance in differentiating fibrositis from true psychogenic rheumatism. This latter term is in itself imprecise as it encompasses a variety of nonorganic pain syndromes. Classical hysteria occurs in an attention-seeking, emotionally unstable egocentric personality, usually in response to some distressing experience, and offers little problem in diagnosis. Willful malingering is rare and can only be diagnosed with confidence in some self-mutilating patients or picturesque pathological liars, such as seen in the Münchausen syndrome. Nonwillful malingering is probably a very common occurrence in association with many musculoskeletal disorders and attests to the remarkable powers of human self-deception which persuade a patient of the validity of his own symptoms. Such patients invariably stand to gain some secondary benefit from the continued *illness:* by avoiding the tensions of ordinary life, by getting more love and attention than previously, by getting a pension or an insurance settlement. This is by no means uncommon in bona fide fibrositis patients and seems to be particularly common in middle-aged manual laborers with fibrositis; such patients cannot tolerate the idea of continuing hard work into their latter years and see a disability settlement as their only escape.

A final consideration concerning psychogenic rheumatism versus fibrositis is the concept of the unconscious symbolic use of pain. Such pain tends to be localized to a single region, which usually has emotional rather than segmental boundaries, such as the back, the neck, the hand, the heart or the chest. Descriptions of the pain are usually narrated in colorful and metaphorical language, often accompanied by dramatizing facial expressions and hand movements. A paradoxical association of numbness and tenderness is not uncommon. Such patients may have an underlying mental disease such as schizophrenia, psychopathic personality, organic psychosis, depression or various psychoneuroses. The diagnosis of "symbolic pain" does not rule out an underlying organic disease, but makes its diagnosis and management exceedingly difficult.

Secondary Fibrositis

Fibrositis not infrequently exists in conjunction with another disease or is secondary to some other condition. Some physicians consider that fibrositis is always a secondary phenomenon and that a diagnosis of primary fibrositis merely implies a lack of recognition of the underlying cause.

Generally speaking diseases that seem to predispose to fibrositis are those conditions which in themselves have fatigue as a prominent manifestation, such as rheumatoid arthritis, systemic lupus erythematosus, polymyalgia rheumatica, viral hepatitis, influenza, hypothyroidism and the like. It is certainly important to recognize these predisposing causes as some are self-limiting (such as viral hepatitis) while in others (such as sys-

temic lupus erythematosus) the profound fatigue may be ameliorated by low doses of prednisone (7.5 to 10 mg per day) with concomitant improvement in the fibrositis. It is the anecdotal experience of many rheumatologists that primary fibrositis is not benefited by prednisone administration; indeed a beneficial response should lead to reassessment of the diagnosis.

On the other hand fibrositis may complicate a well-developed rheumatic illness such as ankylosing spondylitis, rheumatoid arthritis, systemic lupus erythematosus or primary Sjögren syndrome. It is most important to recognize this occurrence because increased therapeutic efforts to control the primary disease are seldom effective in treating the fibrositis and may lead to unnecessary and undesirable side effects.

Similar But Distinct Pain Syndromes

The occurrence of pain over the medial and lateral condyles, exacerbated by gripping, is a common occurrence, as an isolated problem, and is referred to colloquially as golfer's elbow and tennis elbow respectively. Patients with fibrositis are seldom aware of tenderness in these situations until they are palpated; this is in contradistinction to patients afflicted with tennis or golfer's elbow.

Pain over the heel and Achilles tendon is a cardinal feature of Reiter disease and related seronegative spondyloarthropathies. However, such pain is virtually never seen in the absence of an oligoarticular arthritis or significant sacroiliitis. In such conditions the Achilles tendon is usually thickened, whereas it is of normal size and contour in fibrositis. Pain in these areas also occurs in joggers and may develop in other sites such as the patellotibial junction and over the greater trochanter. Ossification occurs at bone-tendon junctions in elderly patients with a distinctive band of ossification running down the right side of several contiguous thoracic vertebrae. This was originally referred to as Forestier disease[18] and is now more commonly referred to as diffuse idiopathic skeletal hyperostosis (DISH).[19] These patients often have pain at the back of the occiput, the greater trochanter, ischia, ilia and calcanei.

Patients who have been receiving corticosteroids for long periods often complain of vague arthralgias and myalgias on attempted dose reduction. This has been referred to as "steroid pseudorheumatism."[20] Furthermore they often complain of a pronounced tenderness of the muscles of the extremities and have extreme sensitivity to pressure over the shins.

TABLE 3.—*Partial Differential Diagnosis of Fibrositis*

Secondary to Rheumatic Diseases
Rheumatoid arthritis
Ankylosing spondylitis
Systemic lupus erythematosus
Polymyalgia rheumatica
Primary Sjögren syndrome
Polymyositis
Osteoarthritis of the spine
Secondary to Fatiguing Illnesses
Influenza
Viral hepatitis
Infectious mononucleosis
Subacute bacterial endocarditis
Brucellosis
Inflammatory bowel disease
Endogenous depression
Mimicking Diseases
Seronegative spondyloarthropathies
Athletic overuse syndromes
Forestier disease-DISH
The enthesopathies (tennis elbow, etc.)
Hypothyroidism
Osteopenia/osteomalacia
Psychogenic syndromes
Steroid pseudorheumatism
Narcotic withdrawal syndrome
Polymyalgia rheumatica

DISH = diffuse idiopathic skeletal hyperostosis

Elderly postmenopausal women with a combination of osteoporosis and osteomalacia not infrequently complain of total body pain and there is tenderness to pressure over both bone and muscle. They do not usually have the characteristic tender spots of fibrositis, although this can occur as a secondary phenomenon.

Hypothyroidism with myxedematous infiltration of muscle occasionally presents with muscular aching and soreness.[21] The histological finding of interfascicular mucopolysaccharides is strikingly similar to that found in fibrositic nodules (see section on pathophysiology). These differential diagnostic considerations are summarized in Table 3.

Some Pathophysiologic Considerations

At present there are no generally accepted pathognomonic changes in muscle histology, electromyography, blood tests or psychophysiologic testing that can be claimed to characterize fibrositis. However, there have been some intriguing advances in the pathophysiology and biochemistry of pain, sleep disturbances and muscle histology,

which may be of some relevance to unraveling the enigma of fibrositis.

Sleep Disturbance and Central Nervous System Serotonin Metabolism

Moldofsky and colleagues[22] have made the interesting observation that patients with fibrositis have a reduced amount of non-rapid eye movement sleep (non-REM sleep). This seems a particularly pertinent observation as a complaint of nonrestorative sleep is commonplace in fibrositic patients. Furthermore, they have induced musculoskeletal pain, similar to fibrositis, in healthy volunteers by depriving them of non-REM sleep.[23] Of particular interest was the finding, in a pilot study, that in very fit persons (those running two to seven miles every day) fibrositic symptoms did not develop.

A recent study has shown that physically fit persons have a higher level of non-REM sleep than unfit subjects and that following exercise the amount of non-REM sleep increased in fit, but remained unchanged in unfit subjects.[24]

There is increasing evidence in animals and humans that serotonergic neurotransmission plays a central role in the regulation of non-REM sleep, pain sensitivity and affective states.[25] Moldofsky has proposed that a disturbance of central nervous system serotonin metabolism may underlie the non-REM sleep, the heightened pain perception and depressive symptoms in patients with fibrositis.[22] Indeed in a small study involving eight fibrositic patients he showed an inverse relationship between plasma-free tryptophan and subjective pain.[26]

Enkephalins, Substance P and the Gate Theory of Pain

It is becoming increasingly evident that there exists a set of powerful endogenous mechanisms of pain control.[27] The amelioration of pain by acupuncture, massage and transcutaneous nerve stimulation may well be due to activation of these endogenous antinociceptive mechanisms.[28] The endogenous morphine-like substances, the enkephalins and endorphins are generally considered to be the prime mediators of this phenomenon.[29] On the other hand it appears that *substance P* (initially discovered by Von Euler in 1931), a small peptide of 11 amino acid residues, may be the major neurotransmitter for pain signals.[30] It has been shown that substance P is present in the small diameter nerve fibers of the dorsal horn, which have traditionally been considered to be pain fibers. Particularly high concentrations of substance P are found in the substantia gelatinosa, that region of the dorsal horn that has been implicated in the *gate theory* of pain modulation.[31] The synthetic enkephalin analogue (Met-enkephalin) blocks the release of substance P and this effect is blocked by naloxone. Descending neurons utilize serotonin as a neurotransmitter and are thought to modulate pain perception through the gate control mechanism at the level of the substantia gelatinosa.[32]

It is now firmly established that stimulation of the brain activates descending efferent fibers which in turn influence afferent conduction of pain and other sensations.[33] In this way it is possible for cerebral functions subserving emotion, attention and memories of past experience to exert control over sensory input. For example, men wounded in battle may feel no pain from their wound, because favorable descending impulses block pain perception—they survived the battle and for this they are grateful. On the other hand they may complain vociferously about an inept venipuncture.[34]

In what manner these newer ideas on pain may be applicable to fibrositis can only be speculative at this time. The traditional emphasis on pain fiber stimulation via tissue disease, with a relative neglect of the motivational and cognitive contributions, has probably sidetracked us from the core of the issue.

The Fibrositic Nodule

Although Hench considered fibrositic nodules "only accessible to the finger of faith,"[15] it is generally accepted that band-like or nodular thickenings are a commonly encountered finding in fibrositis.[17] Their presence poses two, as yet unanswered, questions: what is their nature and what role, if any, do they play in the pathogenesis of symptoms?

It has been the experience of most investigators that muscle biopsy of fibrositic patients is unrewarding in terms of conventional staining techniques and that the inflammatory changes found by Stockman[4] are not present. However, there are two independent reports on biopsies of actual fibrositic nodules that indicate that the palpable induration may be due to local edema and the interfascicular deposition of acid mucopolysaccharides.[35,36] Electromyography, with monopolar needle insertion directly into the fibrositic nodule,

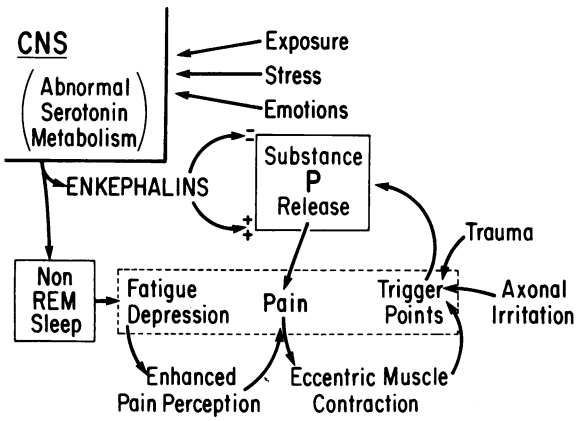

Figure 2.—A schematic representation of some of the factors that are possibly relevant to the psychopathophysiology of fibrositis. The features within the dotted rectangle are the common clinical findings in fibrositis. CNS = central nervous system. REM = rapid eye movement (sleep).

showed polyphasic motor unit action potentials in six of ten cases.[36] The fact that fibrositic nodules are still palpable in curarised patients and persist after death, until obscured by rigor mortis[37] is strong evidence against their being primarily local muscle spasm. The inconsistent muscle hyperreactivity, found on electromyography, may well be a fluctuating response to a painful trigger point and account for the shifting nature of fibrositic pain and its amelioration by activities which promote muscular relaxation.

Deep Hyperalgesia

The pain experienced in fibrositis is referred pain of deep origin and does not correspond to a dermatomal distribution. Such pain is poorly localized and is often associated with feelings of heaviness, a tingling sensation or a swollen feeling. Most deep pain is referred distally often at some distance from the site of origin; the distribution patterns of such pain are well described in Kellgren's pioneering studies of this subject.[38]

It is generally assumed that the fibrositic nodule or the tender spot at bone-tendon insertion is a trigger point for the referred pain in fibrositis. The fact that local anesthetic infiltration of such an area usually affords some temporary relief of pain supports this hypothesis. The existence of the fibrositic nodule is often used as evidence for fibrositis being an organic disease of muscle. However, it seems more likely that it is an inconsistent phenomenon secondary to the previously described neuropsychiatric changes, but when present may well play a role in perpetuating the pain syndrome. Figure 2 is an attempt to bring together some of these facts into one central hypothesis.

Treatment

Fibrositis is an extraordinarily dfficult condition to treat effectively, indeed most physicians are more successful at treating a complicated case of systemic lupus erythematosus than fibrositis. This most surely stems from an undue reliance on medications and inadequate time given to explanation to patients. Patients with fibrositis are naturally concerned that their musculoskeletal pain is the harbinger of a serious illness or crippling arthritis. They feel threatened by their prospects for the future, the effect of fatigue upon their work and recreational activities, and the lack of effectiveness of the prescribed medications. This latter point is especially important in those who have long-standing fibrositis; they invariably resort to doctor shopping and paramedical treatment. Not unnaturally, they become disenchanted and skeptical of the medical profession.

First and foremost in treatment is a thorough history and physical examination along with some carefully chosen investigations (albeit elaborate investigational studies are seldom indicated). This is a prerequisite to gaining the patient's confidence as well as gaining important insight into provocative factors and an understanding of what effect the disease is having on the patient's life. A careful explanation of the nature of fibrositis is then called for, emphasizing the following points: (1) lack of crippling or serious illness, (2) the concept of referred pain and trigger points (can often be dramatically conveyed by local anesthetic injection), (3) the nonrestorative sleep disturbance and its relationship to fatigue, (4) the role of aggravating and ameliorating factors on the fluctuation of symptoms and (5) a heightened self-awareness of the patient's own attitudes and expectations. The intellectual level of the discussion must be carefully adjusted to the patient's own level of intelligence. In particular it is most important to avoid the impression that the symptoms are primarily of emotional origin; this will be taken by most patients to imply that they are imagining their pain or malingering. With time and an increased understanding of the disease process, most patients come to appreciate the importance of the underlying emotional currents in their own case. This realization should come about gently and naturally without overt prodding

by the physician. However, where there are obvious gains to be derived from the illness or the patient is a nonwillful malingerer, an early but sympathetic exposure is mandatory, if further progress is to be made.

Most patients will already have tried aspirin or similar medications; in most cases they are of minimal help, and there is little use in prescribing a long line of nonsteroidal anti-inflammatory agents; in fact, there is every reason not to, in terms of professional credibility. Many patients will be helped by a tricyclic antidepressant at night. This appears to improve the restorative quality of sleep and patients report feeling less fatigued and more able to cope with their pain. They are often less tolerant of such medications than most patients and it is best to start at a relatively low dose (for example, 10 to 25 mg of amitriptyline). Tranquilizers seem to be of little benefit in most cases and there seems to be an aversion for such medications in the personality make up of many fibrositic patients. Cyclobenzaprine, a centrally acting muscle relaxant, is sometimes of some benefit; it is given in a dosage of 10 mg in the early evening and before retiring—drowsiness usually precludes its use during the day. Injection of trigger points with a local anesthetic, with or without hydrocortisone, is only a temporizing measure and its frequent usage fosters undue dependency on the physician. Repeated injections in the same area may well produce a focus of irritation, but this is not proved.

Some patients are benefited by relaxation classes, but in the absence of a good understanding of their disease, this form of therapy is only of temporary benefit; the same can be said for physical therapy, heat applications, massage and acupuncture.

Many of the problems encountered in treating fibrositis seem to originate from the adoption of the expected stereotyped roles of patient and doctor. Talcott Parsons, a Harvard sociologist, has defined the sick role under four headings: (1) the sick person is exempted from some or all of his normal activities and duties, (2) the sick person cannot help being ill and cannot get well by willpower alone, (3) the sick person is expected to want to get well as soon as possible and (4) the sick person is expected to seek appropriate help and cooperate in an effort to get well.[39]

The patient with fibrositis seems to act out this sick role par excellence; the physician's role is best served by placing the burden of improvement on the patient and not on drugs. It is a delicate and precarious dance of readjusting both psyche and soma; a true test of both the art and science of medicine.

Conclusions

The fibrositic misnomer has been a disservice to the widespread recognition and research of an ubiquitous affliction. Moldofsky has proposed the term "nonrestorative sleep syndrome," in recognition of one important aspect of its psychopathophysiology. Smythe regards it as a disorder of pain modulation and uses the term "pain amplification syndrome."[40] These terms are preferable to fibrositis but do not explain such somatic elements as the "fibrositic nodule." For the time being a noncommittal term as regards pathogenesis, such as the old term *muscular rheumatism,* would be preferable. It is to be hoped that the burgeoning increase of knowledge of the psychopathophysiology of pain will lead to a better understanding of the fibrositis syndrome. Whatever we call it, it will not go away just because we may choose to ignore it.[41]

REFERENCES

1. Smythe HA: Non-articular rheumatism and psychogenic musculoskeletal syndromes, *In* McCarty DJ (Ed): Arthritis and Allied Conditions, 9th Ed. Philadelphia, Lea & Febiger, 1979, pp 881-891
2. Smythe HA, Moldofsky H: Two contributions to understanding the "fibrositis syndrome." Bull Rheum Dis 28:928-931, 1978
3. Gowers WR: Lumbago: Its lessons and analogues. Br Med J 1:117-121, 1904
4. Stockman R: The causes, pathology and treatment of chronic rheumatism. Edinburgh Med J 15:107-116, 223-235, 1904
5. Abel O Jr, Siebert AJ, Earp R: Fibrositis. J Missouri Med Ass 36:435-437, 1939
6. Collins DH: Fibrositis and infection. Am Rheum Dis 2: 114-126, 1940
7. Slocumb CH: Fibrositis. Clinics 2:169-178, 1943
8. Llewellyn LJ, Jones AB: Fibrositis. London, Heinemann, 1915
9. Stockman R: Rheumatism and Arthritis. Edinburgh, W Green, 1920
10. Elliott FA: Tender muscles in sciatica: Electromyographic studies. Lancet 2:47-49, 1944
11. Kelly M: The nature of fibrositis. Am Rheum Dis 5:69-77, 1946
12. Copeman WSC, Ackerman WL: Fibrositis of the back. Q J Med 13:37-51, 1944
13. Copeman WSC, Ackerman WL: Edema or herniations of fat lobules as a cause of lumbar and gluteal "fibrositis." Arch Intern Med 79:22-35, 1947
14. Kraft GH, Johnson EW, LaBan MM: The fibrositis syndrome. Arch Phys Med 49:155-162, 1968
15. Hench PS, Bauer W, Fletcher AA, et al: Fibrositis in "Review of the American Literature for 1935." Ann Intern Med 10: 878-884, 1936
16. Froriep H: Ein Beitrag zur Pathologie und Therapie des Rheumatismus. Weimar, 1843
17. Simons DG: Muscle pain syndromes, Part 1. Am J Phys Med 54:289-311, 1975
18. Forestier J, Rotes-Querol J: Senile ankylosing hyperostosis of the spine. Ann Rheum Dis 9:321, 1950
19. Resnick D, Niwayama C: Radiographic and pathological features of spinal involvement in diffuse idiopathic skeletal hyperostosis (DISH). Radiology 119:559-568, 1976
20. Rotstein J, Good BA: Steroid pseudorheumatism. Arch Intern Med 99:545-555, 1957

21. Golding DN: Hypothyroidism presenting with musculoskeletal symptoms. Ann Rheum Dis 27:10-14, 1970

22. Moldofsky H, Scarisbrick P, England BA, et al: Musculoskeletal symptoms and non-REM sleep disturbance in patients with "fibrositis syndrome" and healthy subjects. Psychosomatic Med 37:341-351, 1975

23. Moldofsky H, Scarisbrick P: Induction of neurasthenic musculoskeletal pain syndrome by selective sleep stage deprivation. Psychosomatic Med 38:35-44, 1976

24. Griffin SJ, Trinder J: Physical fitness, exercise and human sleep. Psychophysiology 15:447-450, 1978

25. Chase TN, Murphy DI: Serotonin and central nervous system function. Ann Rev Pharmacol 13:181-197, 1973

26. Moldofsky H, Warsh JJ: Plasma tryptophan and musculoskeletal pain in nonarticular rheumatism ("fibrositis syndrome"). Pain 5:65-71, 1978

27. Liebeskind JC, Paul LA: Psychological and physiological mechanisms of pain. Ann Rev Psychol 28:41-60, 1977

28. Mayer D, Price DD, Rafii A: Antagonism of acupuncture analgesia in man by the narcotic antagonist naloxone. Brain Dis 121:368-372, 1977

29. Terenius L: Endogenous peptides and analgesia. Ann Rev Pharmacol 18:189-205, 1978

30. Marx JL: Brain peptides: Is substance "P" a transmitter of pain signals? Science 205:886-889, 1979

31. Melzack R, Wall PD: Pain mechanisms: A new theory. Science 150:971-979, 1965

32. Besson JM, Guilbond G, Le Bars D: Descending inhibitory influences exterted by the brainstem upon the activities of dorsal horn lamina—V. Cells induced by intra-arterial injection of bradykinin into the limbs. J Physiol 248:725-739, 1975

33. Del Bianco DL, Del Bene E, Sicuteri F: Heart pain, *In* Bonica JJ (Ed): Advances in Neurology, Vol 4. New York, Raven Press, 1974, pp 375-381

34. Abraham VC: An induction of prolonged change in the functional state of the spinal cord, *In* Bonica JJ (Ed): Advances in Neurology, Vol 4. New York, Raven Press, 1974, p 243

35. Brendstrup P, Jespersen K, Asboe-Hansen G: Morphological and chemical connective tissue changes in fibrositic muscles. Ann Rheum Dis 16:438-440, 1957

36. Awad E: Interstitial myofibrositis: Hypothesis of the mechanism. Arch Phys Med Rehabil, 54:449-453, 1973

37. Schade H: Beitrage zur Umgrenzung and Klarung einer Lehre von der Erkaltung. Z Ges Ech Med 7:275-374, 1919, cited in Simons DG[17]

38. Kellgren JA: Observation on referred pain arising from muscle. Clin Sci 3:175-190, 1938

39. Parsons T: The Social System. New York, Free Press, 1964

40. Smythe HA: "Fibrositis" as a disorder of pain modulation, *In* Dixon AS (Ed): Clinics in Rheumatic Diseases, Vol 5. Philadelphia, WB Saunders Company, 1979, pp 823-832

41. Rosenbaum EE: Rheumatology. New York, Medical Examination Publishing Co Inc, 1980, p 219

Mammography and Breast Disease

I THINK MAMMOGRAPHY is a very important advance in the management of patients with breast disease. The pendulum swings back and forth, of course. I consider it an important adjunct to the physical examination. It is an examination like anything else and must be looked upon as that. In any patient who has a lump, a mass that the examiner feels is discrete, a biopsy must be done regardless of what mammography shows. And in fact, I do not do mammography in patients who have a mass—I do a biopsy. Mammography is to look for nonpalpable disease. When a patient has a mass that turns out to be carcinoma on biopsy, I then do a mammogram primarily to look at the other breast and to look at this breast to see if there is any other lesion and for subsequent follow-up of the other breast, which I then do once a year for any treatment of primary breast cancer. Patients with disease that is not clearly discernible as a mass, but you are concerned about what you're feeling in the breast, that is where mammography can be very helpful. It is helpful because it can find things that you cannot feel or help push you to carry out a biopsy when you cannot feel anything wrong. I think the biggest problem in mammography is that in young women it is not very useful, because the density of the breast is such that it does not allow itself to show other disease through the dense normal breast tissue. Under the age of 35, I do not believe very many people should have mammographies, just women in whom breast cancer has been diagnosed. High-risk factors, other malignant conditions that might predispose a patient to breast cancer development and certain other factors may justify doing mammography in patients younger than 35, but very few people need it. From 35 to 50, I think women with genetic risk factors should probably have a mammogram once a year or at least every two years. And over the age 50, I think any woman that has any breast abnormality should have a mammogram. . . . Now, if the mammogram shows abnormalities which the mammographer is concerned about, in the form of stippled calcification and the formation of a mass lesion which you cannot feel, you are obligated to do a biopsy. Likewise, failure to take a biopsy specimen of a lesion when the mammogram does not show anything is also a very dangerous procedure. So I think mammograms and physical examinations complement each other. They will both identify disease and they should both be used appropriately.

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