Proc. R. Coll. Physicians Edinb. 1996; **26:** 20–25

FIBROMYALGIA*

P. Croft,† School of Postgraduate Medicine, University of Keele, Staffordshire

Pain and stiffness arising in muscles or other soft tissues in the absence of any definable musculoskeletal pathology may present the clinician with difficulties in diagnosis and management. Traditionally such symptoms have often been subsumed under the general term 'rheumatism' or 'soft tissue rheumatism', a label which encompasses the disorders affecting muscles, ligaments, bursae and other connecting tissues which support and move the skeleton. The pathological basis for pain associated with acute injury to these tissues is readily understood; similarly patients with acute or chronic joint disease may experience referred 'myalgic' pain distal from the source of their pathology, but many patients with chronic non-specific 'aches and pains' have no definable underlying pathology and the source of their symptoms is not understood.

Lindstedt identified some of the clinical problems associated with the interpretation of myalgia early this century.[1] First, the margins between muscular pain and other symptoms such as stiffness and tiredness are frequently blurred. Second, myalgia is often out of proportion to any apparent local cause, radiates over a wide area and may be intense. Furthermore, myalgia may occur spontaneously in the absence of injury or apparent pathology.

There have been many attempts to identify an underlying pathological process which could explain the aetiology of spontaneously occurring myalgia. Classically there was a British–Continental divide over this. The Germans and the Scandinavians believed there were structural problems in muscle and supporting tissues—the literature is dominated by descriptions of physical lumps and bumps, of all sizes ranging from a grain of wheat to the size of an apple. The British theory that the condition was caused by inflammation was reflected in the term 'fibrositis' coined by Gowers at the turn of the century.[2]

Evidence to support this notion was reported by Stockman, Professor of pathology in Glasgow[3] who described microscopical evidence of inflammation of fibrous tissue. For the next 40 years or so this remained accepted dogma until, when Stockman's specimens were reexamined along with muscle tissue from other patients, it was found that his findings could not be confirmed.[1]

'Rheumatism' became an embarrassment, tucked away for the next few decades as a branch of rheumatoid arthritis. In classic papers in the Annals of Rheumatic Diseases in the 1940s, British clinicians, affected by their war experiences, wrote elegantly and movingly of the psychosocial context of rheumatic pain[4, 5] and the term 'psychogenic rheumatism' became popular during this time. Then from the late 1970s articles appeared in the Canadian and American literature[6] which described a clinical syndrome of chronic widespread musculoskeletal pain, termed the 'fibrositis syndrome', the unique feature of which was claimed to be the presence of multiple tender points over muscle, entheses and

*Based upon a lecture delivered at the Symposium on *Psychosomatic Syndromes* held in the College on 5th April 1995.

†Professor of Epidemiology.

soft tissue structures. The term 'fibromyalgia' to describe this syndrome of pain and tender points has since gained widespread usage.

There has been debate about the location of the tender points and the importance of associated symptoms such as fatigue, poor sleep and stiffness which are recognised as common among patients with widespread muscle pain. The final accolade of respectability came with the publication of classification criteria by the American College of Rheumatology in 1990, which considered that fibromyalgia could be diagnosed in the presence of other causes of musculoskeletal pain, such as osteoarthritis.[7] In other words it was not a diagnosis of exclusion. 'Widespread' pain was defined as pain in the axial spine together with pain in at least two contralateral quadrants of the body; 'chronic' was defined as more than three months of pain; 'high tender point count' was defined as 11 or more tender points out of a specified set of 18 points to be examined.

The story has continued. Fibromyalgia is now the second most common condition seen by rheumatologists in America and Canada. In these and in the Baltic countries it has become a key welfare and medicolegal issue, because of the high proportion of those diagnosed who receive disability payments because of the acceptance that fibromyalgia can be triggered by local injuries in the workplace. It has spawned networks, support groups, consensus conferences and continuing controversy. In the scientific field, numerous concepts have been drawn from other fields in an attempt to explain the cause of this syndrome.[8] Theories of aetiology including Lyme disease and abnormalities of growth hormone or neuropeptide secretion have all gained proponents.[9] While it is easy to be critical, a quote from 90 years ago reminds us of the suffering patient at the centre of it all:

> Although many physicians deny the existence of the muscular disease because no objective pathological findings are demonstrable ... the patients' severe symptoms and disability, and the objective signs, deserve recognition.[2]

I want now to examine this condition from the perspective of the epidemiologist. Surveys of symptoms such as joint pain, headache and fatigue in the general population confirm that they are common, affecting one-third of adults at any one time, with a peak at ages 40 to 60, and more frequently reported by women than by men. From general practice studies we know that at any one time only a small proportion of people with these complaints will consult about them, and only a minority of those will be referred to a specialist. Descriptions of fibromyalgia from specialist clinics in America and Canada suggest an eight- to nine-fold increased frequency in women, in particular relatively well-off women in their 40s and 50s, with associated multiple symptoms, long duration of symptoms and a poor prognosis. This raises the following question: to what extent is the referral process simply identifying patients with common problems who are selected with respect to the number, severity and duration of their complaints? Or are such patients representative of a condition identifiable in the general population?

The American College of Rheumatology (ACR) Criteria provide an explicit example of this concern.[7] A group of clinicians with a special interest in fibromyalgia were asked to choose patients whom they regarded clinically as having this condition, and two groups of controls, one with possible inflammatory disease and one with other soft tissue conditions. The criteria simply highlight those features which were most consistently characteristic of the patients

who had been diagnosed as having fibromyalgia. So we have a completely circular argument. A group of doctors are diagnosing a new disease. They get together and compare notes, and then describe the characteristics of this new disease for the world at large. The effects of this have been enormous. Patient literature and clinical literature all quote the magic number of 11 tender points. The ACR Criteria committee did not intend this—they wished to set up some classification standards for clinical studies, not a diagnostic algorithm—but this laudable aim inevitably has become confused with diagnosis. Ask a fibromyalgia specialist: 'What if you have a patient whom you would regard as having typical fibromyalgia but who only has 10 tender points?'. Answer: 'I would diagnose fibromyalgia'. Why?: 'Because I know from clinical experience that is what they have'.

This is not a criticism of dedicated clinical practice, but an example of the inevitable problems of dealing with uncertainty. Can the picture from the general population help? No assumptions need to be made about how pain and tender points are related, so that the two elements—chronic widespread pain and multiple tender points—can be quantified separately.

The cardinal symptom of fibromyalgia is common: the point prevalence of chronic widespread pain is about 10 per cent in the adult British population. In a population survey in Manchester adults, subjects with chronic widespread pain were more likely to report other somatic symptoms, such as stiffness, abdominal pain and swollen joints, than people with localised pain or no pain at all.[10] They were also more likely to report depressive symptoms, anxiety and difficulty with sleep. So the associations noted in specialist clinics are confirmed in the general population—they are not simply a result of self-selection of patients with multiple problems into a hospital setting. However such associations are not specific to chronic widspread pain. Studies of individual musculoskeletal syndromes (low back pain, neck pain, shoulder pain) show that such problems co-exist more often than chance or age would dictate in the general population and are associated with other somatic and psychological symptoms. In a classical paper, Dworkin and colleagues showed that the more pain areas people compalined about, the higher the score on a scale of psychological problems.[11] Widespread pain is likely to be one end of a spectrum rather than a unique entity in itself.

Tender points are also common in the general population and high counts as defined by the ACR Criteria (i.e. 11 or more) are to be found in some 15 per cent of adults at any one time. However, there is no natural cut-off between high and low, and tender point counts lie on a continuum. There is an association between tender point count and chronic widespread pain in the general population, but overall more people with high counts will be found among the 90 per cent of the population who do not have chronic widespread pain than in the 10 per cent who do have the symptom. That tender points are not simply reflecting pain is supported by the finding that in the general population the number of tender points shows a linear association with scores of depression, fatigue and poor sleep.[12] This association is independent of pain complaints, and suggests that tender points are reflecting levels of distress in general.

The likelihood is that there is no qualitative difference between pain that is widespread and pain that is restricted to one area of the musculoskeletal system. It is a matter of quantity—if a patient develops more than one regional syndrome, then there is a point at which the pain will be widespread rather than local. It is probable that a similar argument applies to tender points—if they occur in relation to regional problems like tennis elbow or shoulder pain, then more than one or two areas of involvement will result in a high tender point count.

This is surmise and needs more rigorous testing. However it leads to the conclusion that we are dealing with a spectrum of pain, distress and tenderness. At one end of this spectrum lies a group of patients to whom the label of fibromyalgia is attached on apparently arbitrary grounds rather than that there is a biologically discrete population of such individuals.

That is the rather critical perspective from population studies. How can we emerge with something more positive for the approach to individual patients?

First the interest in fibromyalgia has focused justifiable attention on the problem of persistent non-articular musculoskeletal pain. There is a huge amount of suffering and distress, doctor and patient frustration, and misdirected investigation involved in this problem, and the need to treat it seriously is paramount. If low back pain is included, this represents by far the most common rheumatological problem in the community.

Secondly, if soft tissue pain is viewed as a continuum, this means that we can research its broad aetiology. The cross-sectional linkage between pain, tender points, and somatic and emotional symptoms is open to the criticism of all cross-sectional studies: cause cannot be distinguished from effect. It is just as reasonable to suppose that persistent or intermittent pain gives rise to depression as it is to consider that psychological problems might precipitate pain or make tender points manifest. We need prospective studies to try to disentangle this, and this has started in the field of low back pain. Prior pain experience and symptoms of depression and anxiety independently predict the future onset of new episodes of low back pain.

Third are the many positive aspects in the clinical arena. Population research is all very well, but what about the patient sitting in front of you with multiple complaints of widespread pain for which you have not found any serious underlying pathological explanation? Here it becomes a question of the virtue of labelling. Does it help to use the term 'fibromyalgia'? Patients are quite clear—it does. The message from the doctor who uses the label is: 'I am taking your problem seriously'. For the doctor too it can help—not only because of the message the patient gets, but also because it provides a positive rationale for avoiding further investigation and specialist referral. But what of the other implications? Much of the literature has been gloomy about prognosis, because the early series consisted of patients with a long duration of symptoms—is it reasonable to use the label when it is only attached to the statement 'Well that's all I can do, but at least you know what it is'? Balanced against this is some very good literature on positive approaches to muscle pain, well received by the fibromyalgia patient support groups, including the need to adapt to symptoms and to consider the psychological aspects of the problem as well.

Further arguments against the use of the label include the accompanying plethora of unproven therapies, the accent on this being a clearcut condition, and the slightly uncomfortable feeling that this is all helping to perpetuate a piece of diagnostic mythology, despite the reality and severity of the patients' problems. The question then is open for discussion: to label or not to label? A member of the audience at the seminar—a thoughtful and experienced practitioner—took up the challenge. He was alarmed by the label. He was bewildered that specialists

should be moving backwards, when general practitioners have developed the art of dealing with the unclassifiable, the undiagnosable and the uncertain. He was horrified at the notion of labelling, when during his working life he had been battling against the harm done by the search for the pathological explanation and had engaged in lines of communication with his patients which did not require such black and white language.

More recent developments in neurophysiology have provided a model which may help to explain the cause of musculoskeletal tenderness. No longer do we need to propose pain messages on a one-way journey from peripheral point of injury to central point of pain perception.[8,13] The nervous system is a more active participant than this. Under various circumstances the central nervous system can remember and regenerate the pain long after the stimulus has gone. It can expand the memory and amplify it, so that it is felt over a wider area and with a greater intensity than the original peripheral injury could explain—all aspects which had puzzled the Swedish investigator, Lindstedt. The central nervous system is plastic and active in all this. Previous pain memory may be triggered by various stimuli, including new pain elsewhere, perhaps trivial; pathology in the spine, including the cervical spine where minor abnormalities might influence pain; or higher influences such as emotion, memory, anxiety, sleep. This model seems to fit well with the clinical and the epidemiological observations. The memorable image conjured up by Patrick Wall in his introduction to the latest edition of *The Textbook of Pain* is of the central nervous system functioning as a virtual reality system where pain is concerned[13]

This model may be valid or it may be just as romantic as the concept of fibromyalgia. The basic scientists may yet return to the periphery as knowledge and imaging of these tissues improves, but clearly there is the opportunity to see pain and its persistence as particularly related to the general circumstances under which peripheral painful stimuli were first experienced. The notion that distress encompasses the physical, the mental and the cultural should be grasped. We should be working towards the development of a language and a way of seeing which does not polarise us to the psyche or to the soma.

ACKNOWLEDGEMENT

My work on fibromyalgia has been supported by the Arthritis and Rheumatism Council and was carried out as part of a team in the Council's Epidemiology Research Unit in Manchester.

REFERENCES

1 Reynolds MD. The development of the concept of fibrositis. *J Hist Med Allied Sci* 1983; **38:** 5–35.
2 Gowers WR. A lecture on lumbago: its lessons and analogues. *Br Med J* 1904; **1:** 117–21.
3 Stockman R. The causes, pathology and treatment of chronic rheumatism. *Edin Med J* 1904; **15:** 107–16; 223–35.
4 Edmonds EP. Psychosomatic non-articular rheumatism. *Ann Rheum Dis* 1947; 36–49.
5 Ellman P, Shaw D. The 'chronic rheumatic' and his pains. *Ann Rheum Dis* 1950; 341–57.
6 Smythe HA, Moldofsky H. Two contributions to understanding of the 'fibrositis' syndrome. *Bull Rheum Dis* 1977–78; **28:** 928–31.
7 Wolfe F, Smythe HA, Yunus ME *et al.* The American College of Rheumatology 1990 criteria for the classification of fibromyalgia. *Arthritis Rheum* 1990; **33:** 160–73.
8 Wall PD. The mechanisms of fibromyalgia: a critical essay. In: Voeroy H, Merskey H (eds). Progress in fibromyalgia and myofascial pain. Amsterdam: Elsevier Science Publishers BV 1993, 53–9.

9 Boissevain MD, McCain GA. Toward an integrated understanding of fibromyalgia syndrome. I: Medical and pathophysiological aspects. *Pain* 1991; **45:** 227–38.
10 Croft P, Rigby AS, Boswell RF *et al.* The prevalence of chronic widespread pain in the general population. *J Rheumatol* 1993; **20:** 710–3.
11 Dworkin SF, Von Korff M, Le Resche L. Multiple pains and psychiatric disturbance: an epidemiologic investigation. *Arch Gen Psychiat* 1990; **47:** 239–44.
12 Croft P, Schollum J, Silman A. Population study of tender points and pain as evidence of fibromyalgia. *Br Med J* 1994; **309:** 696–9.
13 Wall PD. Introduction. Wall PD, Melzack R eds. Textbook of pain. 3rd ed. Edinburgh: Churchill Livingstone, 1994.

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THE GREAT AND THE UNGRACIOUS

Moses ben Maimon (Moses Maimonides) 1135–1204, was known to the Arabs as Abu Imran Musa ibn Maimun ibn Abd Allah. He wrote all his medical works in Arabic from which they were translated into Hebrew and some into Latin. One of his colleagues at the hospital in Cairo was Ibn Abi Usaiba who recorded a poem written about Maimonides by the poet Al-Said ibn Sina Almulk:

Galen's art heals only the body,
But Abu-Imran's the body and the soul.
His knowledge made him the physician of the century.
He could cure with his wisdom the disease of ignorance.
If the moon would submit to his art,
He would free her from her spots at the time of full moon,
Would relieve her of her monthly ailments
And, at the time of her conjunction,
Save her from waning.

For some this had disadvantages: another poet, Abraham Ibn Ezra, made several unsuccessful attempts to see Maimonides when he was the physician at the court of the Sultan Saladin and, in the end, wrote an epigram which translates:

I call on my lord in the morning
I am told that on horse-back he's sped,
I call once again in the evening,
And hear that his lordship's abed,
But whether his highness is riding
Or whether my lord is asleep,
I'm perfectly sure, disappointment
Is the one single fruit I shall reap.

(Friedenwald H. *The Jews and Medicine* Vol. I. Baltimore: John Hopkins Press 1944.)