Fibromyalgia in the workplace

Sirs: In his leader "Fibromyalgia in the workplace: a "management" problem?" Paul Reilly asks what lessons Britain might learn from the Australian experiences. We suggest that Dr Reilly might have assisted this process had he more adequately considered Australian publications and also the papers describing earlier epidemics in Scandinavia and Japan. He has continued the theme of opinion papers which in Australia led to a distortion of views and discussion in favour of unproved psychological mechanisms and away from scientific research. Presumably because of the important implications for economics and industrial relations, and in the absence of adequate knowledge, the unsubstantiated opinions of authorities were given more prominence in Australia than is usual in medical matters. The emphasis on psycho-somatic causation was effective in reducing the apparent incidence and in influencing the outcome of litigation against the year's HRT. PICP seems to be a reliable marker of bone formation. In the present study, however, ICTP does not seem to be a sensitive marker of bone formation. In summary, PICP decreases and ICTP does not, not differentiating ongoing year's HRT. PICP seems to be a reliable marker of bone formation. In the present study, however, ICTP does not seem to be a sensitive marker of bone formation.

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Doctors such as ourselves who have treated individual patients and performed research on representative samples have been thought to have inadequate explanations for the epidemiological phenomena. Unfortunately, in Australia there have been no concerted national mechanisms implemented with a view to resolving these important issues. Perhaps that is the most important lesson to be learnt.

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AUTHOR’S REPLY: I am grateful to Drs Champion, Cohen, and Quintner for their interest in my recent editorial and for once again documenting their passionately held and well informed views on the contentious subjective physiological and related upper limb pain problem. From previous meetings and exchanges of correspondence I am aware that their approach is fundamentally different from my own and, indeed, from many of their rheumatology colleagues in Australia.

Having been an ARC research fellow in Melbourne looking into soft tissue rheumatism, I am in fact very familiar with Australian RSI. My views on ‘repetitive strain injury’ (RSI). Whatever its cause and pathogenesis it seems clear that the incidence and prevalence of ‘RSI’ have declined since the mid-1980s, just as similar epidemics subsided in Japan and Scandinavia. As yet it is unclear whether this decrease was due to improved workplace ergonomics, adverse anthropological changes in the attitude of society, or to altered beliefs in individuals about the nature of arm pain and fatigue as a result of normal work practices.

The nomenclature of many rheumatological problems is unsatisfactory. Today’s ‘RSI’ is yesterday’s ‘writers’ cramp’ and ‘telegraphists’ cramp,’ both of which were major public health problems in the past, but neither of which is diagnosed commonly in the 1990s. Of interest, certain conditions were attributed to the introduction of new technology (steel-nibbed pens and the telexograph respectively) and both were felt to be related to anxiety, neuroticism, and a temperamental predisposition. As for ‘fibromyalgia’, it is as good as bad an example of ‘invalidated terminology’ as lupus (a wolf), anklylosis (a bend), and rheumatic (pertaining to evil humours escaping from the nose and mouth). New diseases just for the sake of it, a terminological point, a point made eloquently by the great Sir Thomas Lewis in 1944: ‘Diagnosis is a system of more or less accurate guessing, in which the end point is a name. These names applied to the disease should assume the importance of specific entities, whereas they are for the most part no more than insinuate and therefore temporary conceptions.’

The majority do not now believe that fibromyalgia is the cause of pain in fibrous tissue and muscles, any more than Sir Thomas Gowers believed, when he invented the term, that ‘fibrositis’ was due to infiltration of fibrous tissue. However, the term allows for the investigation of consistent features in exploring the spectrum of chronic pain syndromes, and a neurogenic basis is not only suggested but also based on hard scientific fact (for reviews see references 3 and 4).

According to Champion et al the pain of ‘RSI’ has its origin in peripheral nociceptive inputs, but these are themselves considerably influenced by noopeptide down regulation in the spinal cord and by sympathetic efferent activity. Further production by higher cortical centres is amply illustrated by the influence on pain of anxiety, stress, guided imagery, and the distraction of battles and sporting events.

Having been a research associate of Professor Helme in Melbourne, I am well aware of his work on chronic pain, and have myself presented data on augmented neurogenic infiltration in fibromyalgia. This followed previous work by Helme and colleagues on fibromyalgia and regional pain problems. The key term used by Champion et al is ‘central sensitisation of nociception’, and this occurs reflexly through sympathetically maintained function. Do we not sweat when anxious, does our hair not stand erect when frightened, and do our muscles not tense at times of danger? These phenomena cannot occur if sensory inputs fail to register in higher centres. In the same way pain is the way in which an individual brain interprets an unpleasant sensation, with this interpretation being dependent on numerous internal and external variables.

I disagree entirely with the statement that there is a ‘well established temporal, ergonomic, and epidemiological link between repetitive work and neck-arm pain disorders’. Such a link is not found, and the observed epidemiology is indeed largely at variance with this hypothesis. I certainly believe that patients with ‘RSI’ present with genuine pain problems, but deny that terms such as functional, psychosomatic, and psychogenic mean that I am adopting a tongue-in-cheek attitude. In attempting to explain the neck-arm pain in a worker one has to accept that the nervous system is plastic and not hard-wired, that pain does not exist in a vacuum. How else can one account for the very different incidence of ‘RSI’ in different offices, at different time periods, or the low incidence in self employed workers? Travers has recently reported that 50% of employees examined in one Australian bank are still performing the same job 10 years after presenting with disabling ‘RSI’ (Travers R, 18th ILAR Congress, Barcelona, July 1993). The implication is that the medical and legal professions must try harder to differentiate pain, injury, illness, disease, and disability. These are not, but vitally important, distinctions are impossible to make solely on the basis of molecular neurophysiology, and easier to explain in the context of mass hysteria, anxiety, and unrealistic expectations. Pain and fatigue are the necessary results of motor function, and their significance must be explained to those individuals sufficiently worried to present with such complaints attributed to normal activity. The goal of a good doctor is to always put the patient’s long term interest above all else. The Australian, and increasingly the British, experience is that such a noble aim is difficult when the patient is simultaneously seen as employee, client, claimant, and victim.

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Large vessel disease in CREST

SIR: We read with interest the report by Youssef et al of four cases of large vessel occlusive disease associated with the CREST Syndrome and scleroderma. The authors described three cases of limited and one case of diffuse scleroderma in which the patients developed symptoms, signs, and angiographic evidence of large vessel occlusive disease. Furthermore, the authors suggested in the discussion that as these four patients were in the age group with the highest population that this represented a significant association of macrovascular disease with scleroderma.