fibromyalgia in the workplace

Sir: In his leader "Fibromyalgia in the workplace: a "management" problem?" Paul Reilly asks what lessons Britain might learn from the Australian experience. We suggest that Dr Reilly might have consulted this protocol had it been 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 economic 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-

social causation was effective in reducing the apparent incidence and in influencing the outcome of both litigation and professional litigation, but at a cost of considerable distress to many affected workers. It is to be hoped that henceforth editors will insist on clearer distinctions between knowledge and opinions.

The outstanding shortcomings in this paper are inadequacy of the clinical description; the use of invalidated terminology; an uninforme discussed discussion of pain pathophysiology, which ignores essential concepts of hyperalgesia; the omission in any fraction of work for the elucidation of pathogenesis; and the essentially negative approach to the treatment of affected subjects.

Not unreasonably, Dr Reilly suggests that a descriptive diagnosis be applied—for example, 'upper limb regional chronic pain syndrome'. Why then the bizarre leap to 'fibromyalgia' terminology? We suggest that British rheumatologists maintain their 'skepticism towards this concept'.

As stated by Anderson,7 "...terms such as "indeterminate pains" or "pains of undetermined origin", though unsatisfactory to both clinician and epidemiologist, will probably lead to less confusion in the long run than the use of labels which sound scientific but which are not accurately defined".

In assessing a chronic pain syndrome comprehensively, it is necessary to consider somatic tissue and muscles. The concept of nociception, referred pain stimulus, ectopic impulse generation from peripheral nerves or nerve roots, central sensitisation of nociception, and psychosocial factors. No significant somatic abnormality has been identified in the fibrovascular syndromes. Peripheral nerve dysfunction is a common feature of these syndromes and may be a necessary condition for the construction of an alternative world—would the disorder have developed had he/she not done this work, an inherently unobservable condition. We can never know for certain in the individual. In the typical case,7 however, in which alternative interpretations have been reasonably excluded, a medicolegal conclusion that the work was a causal or aggravating factor can be reached on the balance of probabilities.

We should be clear that diagnostic criteria and strict scientific terms we should, at this stage of knowledge, go no further than recognising the work as a risk factor.

Reilly focused more on prevention than treatment. That is appropriate because more can be done to prevent the disorder than to cure it. He stated that unproved remedies should be avoided. Unfortunately that does not help the patient or doctor much as the only treatment demonstrated by controlled trial to help (a little) is cognitive-behavioural intervention.12 We need the guidance provided by studies into the pathogenesis of these perplexing disorders. The appropriate mind is the neurophysiology of chronic pain. In the meantime we should observe with enlightenment and 'quizzical regard', apply our current knowledge of chronic musculoskeletal pain, and work to ensure that the workers as our patients in their difficult interactions with their employers and with the medicolegal system. Those who act as agents for insurance companies or in the demonstrable pathology does not mean no disorder or disability.

The investigators and commentators who have focused on the epidemiological aspects of repetitive work and arm pain in general have failed to account for the clinical features exhibited by individual patients which are similar across various occupational groups.
Doctors such as ourselves who have treated individual patients and performed research on representative samples have been thought to have inadequate explanations for 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 subject of 'RSI'. I hope 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 publicity, judicious use of the term 'RSI' 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 ill-informed today. Today 'RSI' was 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, control conditions were attributed to the introduction of new technology (steel-nibbed pens and the telegraph respectively) and both were felt to be related to anxiety, neuroticism, and a temperamental predisposition. As for 'fibromyalgia', it is as good or bad an example of 'invalidated terminology' as lupus (a wolf), ankylosis (a bend), and rheumatic (pertaining to evil humours exiting from the nose and mouth).

New diseases just found to be different than new terminologies, 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 symptoms assume the importance of specific entities, whereas they are for the most part no more than insecure and therefore temporary concepts.'

The majority do not now believe that fibromyalgia is 'a syndrome of pain in fibrous tissue and muscles, any more than Sir Thomas Gowers believed, when he invented the term, that 'fibrositis' was due to inflammation of fibrous tissue. However, the term allows for the investigation of useful concepts 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 neuropeptide down regulation in the spinal cord and by sympathetic efferent activity. Further progression by higher cortical centres is additionally influenced 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 inflammation in fibromyalgia. This followed up 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 sympathetic efferent 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 very tenuous, 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 upper limb 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 work forces, over 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 hard to differentiate pain, injury, illness, disease, and disability. These so-called biologically important, distinctive indications 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 tell the patient the true 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 belong to the older age group which constitute a population that expressed a significant association of macrovascular disease with scleroderma.

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