these clinical situations than during HRT. This suggests that ICTP might be a less sensitive marker for changes in bone resorption than expected.

In summary, PICP decreases and ICTP does not change during one 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 resorption.

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Fibromyalgia in the workplace

Sir: In his leader 'Fibromyalgia in the workplace: a "management" problem'[1] 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 psychosocial causation was effective in reducing the apparent incidence and in influencing the outcome of workers' compensation 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 uninformed discussion of pain pathophysiology, which ignores essential concepts of hyperalgesia; the omission of any framework 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 'slowness to recognise this concept'. Taken literally, this term suggests pain in the fibrous tissue and muscles. The reality is that the cervicobrachial disorder involves not only pain but also paraesthesiae, sensory impairments, sympathetic dysfunction, dystonic and antalgic impairment of motor function, and secondary hyperalgesia, all phenomena which require explanation. Of these, the central concept is secondary hyperalgesia. The latter can be elicited frequently from skin and virtually all musculoskeletal tissues.[2] It is true that the neck and arm disorder acquired in the workplace shares many clinical features with the 'fibromyalgia syndrome', but to direct us into this maze is a retrograde step. As stated by Anderson,[3] '... 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 disorder as a source of nociceptor 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 these cervicobrachial disorders. Peripheral nerve dysfunction includes well defined entrapment neuropathies, increased mechanosensitivity of peripheral nerves including brachial plexus,[4] and disordered C fibre function as identified in research with capsaicin.[5 6] The most striking features on examination are allodynia and hyperalgesia extensively in the soft tissues of the neck and arm, implying central sensitisation of nociception.[2] Consistent with these observations, research with cerebral event-related responses induced by carbon dioxide laser stimulation[7] and with electrocutaneous stimulation[8] has supported the concept of central sensitisation. It seems likely that altered central processing is maintained dynamically by peripheral nociceptive input along the lines presented by Gracely *et al.*[9] These considerations establish a prima facie case for a relation between persistently painful activities and chronic pain states. Because the evaluation of neuropathic mechanisms in chronic pain disorders has not been well established in clinical practice, when doctors find insufficient pathology to account for a disorder, psychosocial interpretations are sought. There is no scientifically sound empirical evidence for primary (and causative) psychosocial mechanisms in these disorders, nor are such concepts readily testable. The interplay of the psyche and reflex variations in muscle tone is well demonstrated, however, in the study by Moulton and Spence.[10]

Reilly refers to 'the unproved concept of pain being due to an injury caused by the strain of repetitive movements'. We would agree that the term 'injury' is not appropriate in this context and that proof in an individual patient is a difficult concept. It is acknowledged that the biological plausibility of the causal connection between repetitive work and the chronic pain state is hampered by incomplete knowledge of pathogenesis. There is however a well established temporal, ergonomic, and epidemiological link between repetitive work and neck-arm pain disorders. The symptomatic worker and his/her advisers present a retrodictive causal proposition that the conditions of work resulted in the genesis and persistence of symptoms. The nature and conditions of the work, involving repetitive use of the upper limbs and sustained arm forward postures (unsupported) and repetitive neck movements and sustained neck postures, are risk factors for neck or arm disorders, or both. They are not a necessary cause (the pain syndrome is not specific to repetitive work) nor a sufficient cause (not all workers performing this or similar work acquire the disorder). Representative epidemiological data[11] indicate a relative risk of at least 3 to 4 for a neck-arm disorder from numerous categories of repetitive work compared with the risk associated with light domestic or sedentary activities. The real meaning of a retrodictive causal proposition depends on 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,[2] however, in which alternative interpretations have been reasonably excluded, a medicolegal conclusion that the work was a causal or aggravating factor can be reached based on the balance of probabilities. In strictly 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 on 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 paradigm is the neurophysiology of chronic pain. In the meantime we should observe with enlightenment and 'quizzical regard',[13] apply our current knowledge of chronic musculoskeletal pain control, and support 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 should keep in mind that no demonstrable pathology does not mean no disorder or disability.

The investigators and commentators who have focused on the epidemiological aspects of neck and arm pains in the workforce have failed to account for the clinical features exhibited by individual patients which are similar across various occupational groups.

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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 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[1] and for once again documenting their passionately held and well informed views on the contentious subject of work related upper limb pain. 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 publications 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 medicolegal judgments, 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' 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, both 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 occur less frequently than new terminology, 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 disease come to assume the importance of specific entities, whereas they are for the most part no more than insecure and therefore temporary conceptions'.[2]

The majority do not now believe that fibromyalgia is due exclusively to pain in fibrous tissue and muscles, any more than Sir William 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 modification 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 and his team in Melbourne, I am well aware of his work on chronic pain, and have myself presented data on augmented neurogenic inflammation in fibromyalgia.[5] This followed up previous work by Helme and colleagues on fibromyalgia and regional pain problems.[6] The key term used by Champion *et al* is 'central sensitisation of nociception', and this occurs reflexly through sympathetic and higher cerebral 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 remains putative only, 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 offices, states, countries, and 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 subtle, 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 patients' 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*[1] 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 constituted 11% of their scleroderma population that this represented a significant association of macrovascular disease with scleroderma.

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