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 at menopause, contrary to young women'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 the present study, however, ICTP does not seem to be a sensitive marker of bone formation.

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**Rheumatoid arthritis**

This article discusses the role of cytokines in the development of rheumatoid arthritis (RA). The authors review the evidence implicating cytokines in the pathogenesis of RA, focusing on the role of tumour necrosis factor (TNF) and interleukin (IL)-6. The article also highlights the potential therapeutic implications of targeting these cytokines, including the use of biologic agents and small-molecule inhibitors. The authors emphasize the importance of understanding the molecular mechanisms underlying RA for the development of novel therapeutic strategies.

**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 experiences. We suggest that Dr Reilly might have consulted this problem before he had read, or even 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 disorientation of views and discussion in favour of unfounded 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-social causation was effective in reducing the apparent incidence and in influencing the outcome of fibromyalgia 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 frank statement prior to each use for the work of elucidation; 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 'sclowness to roots' concept. That is, literally, this term suggests pain in the fibrous tissue and muscles. The reality is that the cervico-brachial disorder involves not only pain but also paraesthesia, sensory impairments, sympathetic hyperactivity, autonomic dysfunctions, 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 day to day, and virtually all musculoskeletal tissues. 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 is misleading.

As stated by Anderson, 1, 2 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, psychological and psychosocial factors. For example, the presence 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 cervico-brachial disorders. Peripheral nerve dysfunction includes well defined entrapment neuropathies, increased mechanosensitivity of peripheral nerves including brachial plexus, and disordered C fibre function as identified in research with capsaicin. 1 The most striking features on examination are allodynia and hyperalgesia extensively in the soft tissues of the neck and arm, implying central sensitisation of nociceptive C fibres. 1 This is consistent with these observations, research with cerebral event-related responses induced by carbon dioxide laser stimulation and with electrocutaneous stimulation 2 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. 3 These considerations establish a plausible case for a relation between persistently painful activities and chronic pain states. Because the evaluation of neuropathic mechanisms in these workers has not been well established in clinical practice, when doctors find insufficient pathology to account for a disorder, psychosocial inter-pretations 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 the physique is a complex entity and must be demonstrated, however, in the study by Moulton and Spence. 4

Reilly refers to the 'unproved concept of pain being due to an injury caused by the strain of repetitive movement'. We 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 causative psychosocial mechanism 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 consequences of the repetitive work and the 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. For these reasons, it is not necessary to 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 work and case studies 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 paradigm is that the occupational risk 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, however, in which alternative interpretations have been reasonably excluded, a medicolegal conclusion that the work was a causal or aggravating factor can be reached with confidence. The traditional—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 mindset is that the neurophysiological and neurochemical bases of chronic pain. In the meantime we should observe with enlightenment and 'quizzical regard', 12 apply our current knowledge of chronic musculoskeletal disorders to help 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, who state that the demonstrable pathology does not mean no disorder or disability. The investigators and commentators who have focused on the epidemiological aspects of neck-arm pain and arm pain (which I have not 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 rheumatological 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 editorial1 and for once again documenting their passionately held and well informed views on the contentious subject of the related upper limb pain syndrome. 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 own comments 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 media publicity, judicious 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, conditions of both were attributed to the introduction of new technology (steel-nibbed pens and the telegraph respectively) and both were felt to be related to anxiety, neurotism, 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).

I have been an ardent supporter of the concept of RSI, and have advocated a 'cervicobrachial deconstruction of 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 terms, 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.