Editorial

Psychosocial Factors in Diffuse Upper Limb Disorder

Regional and generalized musculoskeletal pain is common, particularly between the ages of 35 and 55. Sometimes a specific condition can be diagnosed, but often the condition has to be labelled "nonspecific." Agreed diagnostic criteria for both specific and nonspecific disorders have recently been published[1,2], but neither provide a satisfactory criteria set for nonspecific upper limb disorder. As the affected population is of working age, it is not surprising that people should ascribe the onset to work — reflected in the extreme example of an epidemic of work related upper limb disorders that occurred in Australia in the 1980s[3]. The legacy of that epidemic still pervades our practice, and the courts, in the pejorative term "repetitive strain injury." In the United Kingdom, in the last few years, there has been a steady rise in claims for compensation for upper limb disorder and, after a number of well publicized rejections, successful claims are now occurring[4]. The courts have had difficulty with 2 things: the diagnostic label of nonspecific upper limb disorder and the attribution of this disorder to work.

The diagnosis of nonspecific upper limb disorder has caused problems because it does not (yet) fit the medical model of disease. The pathophysiology is unknown and in the absence of overt, specific tissue abnormalities many are reluctant to make a diagnosis[5]. Indeed, the agreed criteria developed in Birmingham[1] emphasized that nonspecific upper limb disorder is a condition diagnosed only when all other conditions have been excluded. Couple this with the finding, in cross-sectional studies, of a high prevalence of psychological abnormalities in this group and the claim that these disorders are "all in the mind" is inevitable[3,6]. However, I have argued that we must at this stage accept the diagnosis of nonspecific upper limb disorder on a phenomenological basis, just as we do low back pain[7]. By accepting the existence of the condition, further studies can be undertaken to define clinical criteria and to elucidate pathophysiology with the ultimate aim of finding both an appropriate biologic marker and a rational treatment.

Can work cause nonspecific upper limb disorder? The writings of Nortin Hadler will be familiar to many[8]. Hadler contends that pain in the arm is part of the human condition and studies purporting to show an association between the pain and work are flawed mainly because of inadequate study design. Cross-sectional studies cannot in general imply causation. Yet a metaanalysis of these studies is strongly indicative of an association between arm pain and work[9,10], and the courts have affirmed that a sudden change in working practice (and hence physical load) can be associated with an upsurge in workforce symptomatology[3].

Many now accept a multifactorial model for (work related) upper limb disorders, which owes its origins to the biopsychosocial model of disease[11]. Some of the features of this approach have been synthesized in a conceptual model by Armstrong, *et al*[12] (Figure 1), which allows for external factors such as repetition and force but also incorporates internal elements such as phenotype and personality. Clinical and psychological observations in these chronic, painful disorders can be assimilated, and more recent observations concerning cortical sensitization[13] can easily be incorporated into this model. Like any model it serves not only to explain the known facts but also to generate hypotheses for further, suitably designed studies to examine[14]. Of particular relevance would be the relative strengths of the factors that may contribute to pathogenesis, and the consistency of these factors across populations. In terms of suitability the most useful studies have prospective case control design with sufficient numbers and duration of followup to make inferences about the variable under study.

Does carpal tunnel syndrome fit this model in relation to occupational disease? Carpal tunnel syndrome is the archetypal upper limb disorder and the one to which all other disorders aspire. This is because there is a plausible pathogenesis (swelling of the contents of the narrow carpal tunnel), a biologic marker (electrodiagnostic testing), and a curative treatment (surgical decompression). However, in relation to occupational carpal tunnel syndrome the pathogenesis is less clear and surgical decompression often does not provide complete relief of symptoms. A number of explanations may account for this. Cross-sectional studies that have established an association between certain occupations and carpal tunnel syn-

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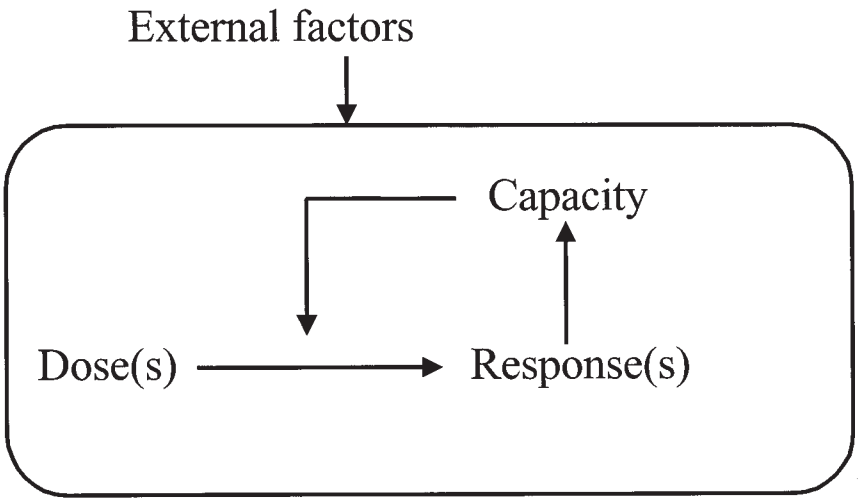

Figure 1. External factors might include prolonged, abnormal working posture or an adverse workplace environment where the worker perceives a lack of control over working practice. *Doses* imply the internal response to these external factors and might, for example, refer to muscle fatigue in response to external loads. *Responses*, in this hypothetical case, would refer to pain felt in the muscles of the arm, whereas *Capacity* refers to internal characteristics of the person that might enhance or modify the response. Such characteristics might be phenotypic or might include such factors as prior knowledge or experience causing fear and anxiety. Based on Armstrong, *et al*[12] and from Helliwell PS, Baillieres Best Pract Res Clin Rheumatol 1999;13:311-28, with permission.

drome may have been subject to methodological errors[8,15], and electrodiagnostic testing in this select group may be simply recording the delay in conduction across the canal, a delay that may be found in up to 16% of a random population sample[16]. Further, the recent imaging studies of Greening and Lynn suggest that, in work related conditions, reduced mobility of the median nerve, possibly due to tethering, may be the primary pathology, rather than compression within the canal[17].

Given these considerations, how does the article by White, *et al*, reported in this issue of *The Journal*[18] contribute to this debate? Their study was predicated on the assumption of nonspecific upper limb disorder as a disorder of unknown etiology, indistinct diagnostically, and having a particularly high prevalence of psychiatric morbidity. Since the psychiatric morbidity may precede the onset of this condition, the authors hypothesized that psychiatric abnormalities in this group would exceed those in a comparison group with a painful condition of the upper limb where psychological factors have not been implicated in the pathogenesis — carpal tunnel syndrome. Psychiatric morbidity was assessed by interview, using a trained inteviewer rather than a psychiatrist. Secondary outcomes included a number of validated psychological self-rating scales and a separate measure of trunk and limb movements. The main finding of the study was that there was no difference in psychiatric morbidity between the groups, so they concluded that nonspecific upper limb disorder is no more psychiatric, psychological, behavioral, or related to personality than a similarly chronic and painful condition of known pathology.

What are the major limitations of this study? First, this was a cross-sectional study and is therefore unable to make any meaningful observations on causation. Second, given this study design we have to examine the participants as closely as allowed (and if unhappy with the information it provides we enter into correspondence via the pages of *The Journal*!). The 37 subjects with nonspecific upper limb disorder were recruited consecutively (the authors do not tell us whether these cases were also selected prospectively) from secondary and tertiary care clinics and they all fulfilled the "Birmingham" criteria[19] such that other specific soft tissue syndromes were not identified. In all these cases their pain had arisen in the workplace. The control group were identified from orthopedic and rheumatology clinics in a teaching hospital, and they also fulfilled the Birmingham criteria for this condition. Since the authors do not say so, and because of the chronic nature of these cases, I assume that these control cases were also identified retrospectively. It is important to note that the cases with carpal tunnel syndrome had a median duration of disease of 60 months, 23/36 were on the waiting list for decompression, 3/36 had already undergone unsuccessful decompression surgery, and 34/36 had received some form of "symptomatic" treatment. We are not informed how many of the control cases attributed their symptoms to work, only that they were not selected for this attribute. Therefore, despite being selected as cases of known pathology (and with a supposedly curative treatment) the control cases had a lengthy history and a background of unsuccessful conservative, and in some cases surgical, treatment. It follows that they were not an ideal control

group but a group of people with a chronic painful condition in whom treatment had been unsuccessful and who were likely to share the same uncertainties about their condition, and their future, as people with nonspecific upper limb disorder.

Third, there is concern that this is a false negative result. Their power calculations were based on a likely prevalence of psychiatric morbidity of 60% in the nonspecific upper limb disorder group and 30% in the carpal tunnel syndrome group. In fact the observed differences were 38% and 22%. This is not just "nit picking" — their figure of 60% for psychiatric morbidity in nonspecific upper limb disorder is at the upper end of the quoted figures from the literature and is unlikely to reflect their local situation. If the observed figures were properly used as pilot data for a prospective study, the power calculations would require a sample size of 130 per group.

In summary, the results of this study should be interpreted with caution because of the above methodological considerations. The study was otherwise well designed and well executed and we should look forward to hearing more from this group, particularly the prospective study that they plan as the next stage. For the moment the model for pathogenesis must remain unchanged and the entity we call nonspecific upper limb disorder requires further study. Linking the disorder to work and to premorbid factors requires good quality prospective studies that are methodologically challenging because of the need to find a large stable workforce where ergonomic and work organizational changes have not yet been implemented. Indeed the chances of finding such an environment are receding, as many workplaces are now adopting such changes as a result of efforts by such bodies as the Health and Safety Executive in the UK[20] and the European Agency for Safety and Health at Work[21].

The parallel between nonspecific mechanical low back pain and nonspecific upper limb disorder has been highlighted[7]. Perhaps, in the absence of more complete data relating to upper limb disorders, we should apply what we know from the low back pain evidence. For example, in low back pain we know that psychosocial factors and workplace organizational factors are more important in symptom presentation than physical factors, although physical factors play a part[22]. Further studies of nonspecific upper limb disorder may only serve to confirm what we already know from this alternative paradigm.

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