EDITORIALS

OCCUPATIONAL RHEUMATOLOGY: ARE WE USING THE WRONG MODEL?

How much are rheumatologists concerned with occupational disorders? The latest edition of Copeman [1] states that 'these conditions have remained largely within the province of practitioners of occupational medicine, and rheumatologists have tended to ignore the problems'. Yet surely this is something that concerns us all. In a recent survey of referrals to rheumatology out-patients in Leeds, 26% of patients had back or neck pain and 8% localized soft tissue lesions; with an average age of 48 and a female to male ratio of 2, occupational factors were important in both aetiology and treatment. Back, neck and soft tissue problems in particular are a major cause of sickness incapacity. One per cent of the population experiences a spell of certified sickness due to low back pain [2] and tenosynovitis ranks second (behind dermatitis) for the number of people receiving injury and disablement benefit under the Industrial Injuries Benefit scheme [3]. Since, as clinical rheumatologists, we are expected to see these cases, it is worth considering them as paradigms with which to examine the usefulness of the medical model.

The medical model proposes that, following an injury, damage to a specific tissue occurs so that a correct anatomical and pathological diagnosis can be made leading to appropriate pharmacological and physical treatment with a subsequent cure. What evidence is there to support the components of this model in regard to low back pain (LBP) and work-related upper limb disorder (WRULD)? A relationship between physical load and LBP in industry was documented by Lawrence [4] who found that miners had twice the rate of LBP than clerical workers. In a recent community survey conducted in Southampton, heavy lifting at work was strongly associated with LBP (relative risk 2.0 [5]. Chaffin [6] was able to make much more specific observations; in a prospective study of LBP he measured lifting capacity and job-specific lifting requirements, finding twice as many episodes of LBP occurring where mismatch occurred. WRULD occurs in association with both excessive cumulative static loads and repetitive low magnitude dynamic loads. Often, as in the case of keyboard operators, both risk factors may be operative [7]. Clearly then, in both LBP and WRULD physical factors are involved in the aetiology.

The published data on diagnosis and treatment of these disorders suggest that the medical model is inadequate. In many cases of LBP and WRULD no apparent physical or pathological abnormality can be found clinically or on investigation. With the advent of MRI it was hoped that at last the soft tissue responsible for pain in some cases of chronic LBP would be identifiable but, as yet, the situation is still unresolved. In WRULD, although specific identifiable lesions can occur (e.g. de Quervain's tenosynovitis, carpal tunnel syndrome, tennis elbow) often there is little to find apart from tenderness and swelling which cannot be localized to a particular tissue. Yet there is no doubt that genuine symptoms, resulting in dysfunction of the part and disability, are experienced in both these conditions. Not surprisingly, in the absence of a clear diagnosis, treatment is found to be unsatisfactory. The wide range of unproven pharmacological and physical treatments available for LBP was highlighted by the Quebec task force [8] and although physical treatment and splinting are thought to be helpful in WRULD, the lack of prospective controlled trials has been emphasized [9].

Waddell and co-workers [10] have suggested that physical factors account for about 40% of the variance in a predicted model of LBP disability. Therefore an alternative model is necessary, encompassing other dimensions of illness including social, psychological and work place factors. There is ample evidence to support the alternative model in both conditions under discussion. A prospective study at the Boeing company in Seattle found that episodes of sickness leave for LBP were related first to a history of LBP but second to perceived work place environment and psychological factors [11]. In a recent study of medical secretaries, neck and shoulder pain was related both to working practice and to a poorly experienced psychosocial work environment [12]. In our own unit we have recently studied a group of employees in light industry in whom the prevalence of WRULD was 81%. scores on psychometric variables such as anxiety, depression and the tendency to report somatic symptoms were higher in employees with a history of pain (unpublished data).

In chronic painful conditions it is always difficult to decide how much the psychological distress is a result of the pain rather than a contributory factor. However, the data from the Boeing study, which was collected prospectively, would suggest the existence of a vulnerable personality. Our data would also support this notion: in the study quoted above, the highest scores for anxiety and depression were related to perceived disability and not to severity of the illness. In this way, following an initial physical insult, both the presentation and subsequent dysfunction would be modified by pre-existing psychological factors and perceived work place environmental factors.

In practice we do not need the non-organic physical signs of Waddell [10] nor do we need formal psychometric assessment since we are all capable of identifying patients in whom these factors are playing a part in the presentation and continuation of the illness. The psychological distress indicated by Waddell's non-organic signs does not mean the patient is malingering but that there are non-physical factors which need to be
The most abundant integrin on hepatocytes was found to be the heterodimeric fibronectin receptor \((\alpha_5\beta_1)\). During liver regeneration the expression of the \(\alpha_5\beta_1\) receptor is transiently increased by 5-10 fold with a gradual return to normal levels after 9-10 days. Hepatocytes are surrounded by extracellular fibronectin and cell-cell contacts with fibronectin become reinforced during regeneration when extensive turnover of the pericellular matrix accompanies cell division. Indirect immunohistochemical staining of liver sections revealed \(\beta_1\) and fibronectin mainly in the sinusoidal region of the hepatocyte plasma membrane. It is likely that the temporal and spatial distribution of these molecules help to maintain the unique architecture of the liver parenchyma. Stamatoglou and Hughes [2] have recently isolated a novel glycoprotein (AGp110). It is a monomeric integral membrane protein extensively glycosylated with sialylated O-glycans and 1,2-N-glycan chains. In developing liver, AGp110 appears later than fibronectin and integrin and it is initially distributed in a non-polarized fashion. Perinatally, during liver plate formation its distribution is progressively restricted to the apical (canalicular) membrane. In vitro both receptors (\(\alpha_5\beta_1\) and AGp110), co-localize in cell-substratum adhesion contacts, and both synergistically affect spreading of hepatocytes on fibronectin. In vivo stable linkage of AGp110 to actin filaments of microvilli at the canalicular domain coincides temporarily with assembly of intercellular junctions. In chemically induced hepatomas, expression of integrin \(\alpha_5\beta_1\), AGp110 and fibronectin is significantly reduced on both hyperplastic and poorly differentiated carcinomas. AGp110 is developmentally regulated and its stable expression correlates with the state of differentiation of the hepatic parenchymal cell. Heparan sulphate proteoglycans have been implicated in a number of fundamental cell processes such as control of cell growth and cell-cell matrix adhesion. Lyon and Gallagher [3] have succeeded for the first time in purifying, in quantity, the cell surface HSPG of liver. Upon deglycosylation the preparation yields three core proteins, the major species having an \(M_c\) of 49 kDa. All of the core proteins were hydrophobic and are probably integral plasma membrane molecules. Structural analysis of the disaccharides from the liver HSPG showed a shift in the sulphation pattern compared to HSPG from the other tissues.