The World Health Organization classifies rheumatic disorders into four main groups: back pain, periarticular disorders, osteoarthritis and related disorders, and the inflammatory arthropathies [1]. For the purposes of this series of articles, musculoskeletal disorders such as upper limb pain have been included. These clinical conditions are responsible for a large proportion of ill health in society, and are a major cost to industry and the State. For example, the National Back Pain Association estimates that 60% of adults suffer back pain annually. Half of these individuals are chronic sufferers, leading to 93 million certified days of incapacity per year, costing over £5 billion in lost industrial production.

Back pain features prominently in self-reported musculoskeletal ill health from the Health & Safety Executives Labour Force Survey [2]. The survey suggests that 880 000 individuals believe that their musculoskeletal disorders are caused or made worse by work. A further 74 000 complained of ‘RSI’ (repetitive strain injury) with the vast majority of cases confined to the upper limb and most of those to the hand, wrist or forearm. RSI is, however, a misleading term, but included under this group of work-related upper limb disorders are some supposedly well-defined conditions such as carpal tunnel syndrome, tendosynovitis and epicondylitis, as well as some less-defined syndromes such as shoulder girdle problems.

Additionally, new evidence has been emerging recently that some of the common sites for osteoarthritis, such as the knee and hip, may have significant work-related aetiologies. The picture thus begins to develop that general practitioners and rheumatologists may well be confronted with a growing number of patients with these disorders, and that a sizeable proportion of these cases will have work as a causative or exacerbatory factor.

Whether these conditions are work related or not, the clinician has a responsibility to consider the work capacity of the patient [3]. Complaints about neck or back pain may appear to be relatively minor, but the investigating physician should closely question the patient about the exact physical requirements of their job. In modern industry, there is little scope for suggesting a period of ‘light work’ as production line industries such as vehicle manufacture require considerable speed and agility. If a rheumatologist had time, it would be enlightening to visit some of these work places in order to see what the real impact has been on the shop floor from such overused terms as downsizing, delayering and multi-skilling! Ergonomics clearly play an important part in preventing some of these disorders and, in some cases, the problems can be engineered out of the process. This is certainly true of the office environment, whilst in food processing and heavy industry mechanical lifting aids can bring great benefits. However, the largest employer of labour in most developed countries these days is the health service. In this case, the load to be lifted is a human being and this presents unique manual handling problems. Patients tend to be heavy, they are insufficiently shaped for lifting and lack convenient hand holds. Furthermore, they are unpredictable and may suddenly collapse or resist movement. They may also require manual handling on an irregular or even on an emergency basis [4].

So far as work-related upper limb disorders are concerned, there is much evidence in the scientific literature to suggest that ‘job-relatedness’ is a major factor [5–7], yet most of the ‘positive’ studies are cross-sectional in design, exposures are often inadequately assessed, and little attempt has been made to integrate the physical, psychosocial and ergonomic aspects in a coherent and logical way. Even the use of diagnostic labels is unresolved. Considerable difference of opinion exists between, for example, hand surgeons and the ‘ergonomic–epidemiologist’ on the definition of these apparently clear-cut conditions as tendosynovitis. Intervention studies are almost non-existent. Much more needs to be learnt about the natural history of these conditions. As many of them have pain as an important symptom, little has been done to date to incorporate the concept of central sensitization to pain into the equation [8], whilst the assumptions about long-term damage to muscle or tendon from work-related activity are not borne out by experimental studies of repair mechanisms in these tissues [9].

The recent rise in disability and work absence due to occupationally related back pain is a cause for considerable concern. Conventional views of the causes of the epidemic tend to polarize into one of three camps: the clinical view of pain due to injury or psychosomatic conditions, the biomechanical exposure view which sees these conditions as ergonomic and preventable, and the perverse incentives view which suggests that work dissatisfaction and accessible disability benefits are the spur to complaints. These divergent and divisive views are unhelpful in achieving progress and, in a thoughtful review of the issues, Frank and his colleagues [10] propose a radical re-think of attitudes and propose a new multidisciplinary research programme to resolve the divisions.

So far as management of back pain is concerned, this is clearly inadequate or there would not be a growing number of individuals seeking help from a variety of conventional and alternative medical practitioners.
Whether psychosocial factors play a part in the initial development of pain, they are bound to come into play as the acute condition becomes chronic in the face of what the sufferer at least sees as a lack of concern on the part of conventional medicine, unsatisfactory or inadequate diagnostic tests, and a failure of many treatments to ameliorate the condition. Loss of employment frequently adds to the problem as the quality of life for the patient deteriorates. These conditions may not be life threatening, but they exert a seriously adverse effect on quality of life which can ruin a patient’s life just as effectively. Given the real costs to people, industry and the State of such a burdensome complaint, it is surprising, if not reprehensible, that more has not been done to pool knowledge and co-ordinate research effort.

On the face of it, osteoarticular disorders appear to be a much clearer picture for review. Indeed, for the knee and hip, surgical intervention has revolutionized the treatment of these painful and disabling conditions. Yet recent research has suggested that the role of occupation has been underestimated. A recent review of the literature suggests that evidence is growing that jobs involving kneeling or squatting at work are an important risk factor for knee osteoarthritis [11] with risk ratios as high as 4-fold for some jobs. Although anecdotal evidence exists to link meniscal lesions and chondromalacia with work, the published literature does not provide a sufficiently well-documented case for occupational exposure. Heavy physical work has been so linked and similar evidence is accruing for osteoarthritis of the hip, where studies suggest that farming is one occupation of particular relevance [12].

In short, rheumatic disorders are a major component of ill health and disability in human populations. Evidence for the role of work as a cause of these conditions or as an exacerbatory factor is growing. Occupationally induced disorders are, by definition, preventable. If the toll of rheumatic disorders is to be diminished by both primary and secondary prevention, one area in need of development is the liaison between the rheumatologist and the occupational physician.

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REFERENCES

VASOMOTOR REACTIONS WITH GOLD

Forester pioneered the use of gold in rheumatoid arthritis in 1929 [1] and, since then, gold compounds have become an important treatment in rheumatology. In the UK, gold is available as sodium aurothiomalate (GSTM) or myocrisin in an aqueous solution for i.m. use, and auranofin or RiDaura, a triethyl phosphine gold compound for oral use. In addition, in some other countries such as the USA, Australia and Germany, i.m. gold is also available as sodium aurothioglucose (GSTG) or Solganal in an oily suspension.

The dermatological, oral, pulmonary and gastrointestinal side-effects of gold therapy [2, 3] are widely recognized, with the renal and haematological complications being particularly important with regard to regular monitoring.

Nitritoid or vasomotor reactions are also a well-recognized side-effect of gold therapy. They are termed nitritoid because the clinical effects resemble those of the nitrate compounds used in ischaemic heart disease. Characteristically, they are transient and occur
within minutes of drug administration, with symptoms including flushing, sweating, dizziness, nausea, malaise and weakness. There is often an associated hypotension and patients feel faint, the features being those of abnormal vasodilatation. Although the reactions may be mild, they are better remembered for being frightening and dramatic, sometimes with a sense of impending doom. Nitritoid reactions have been reported as having an incidence of 4.7% with myocrisin, 2.8% of patients requiring a change of treatment due to recurrent reactions [4]. These vasomotor reactions are usually self-limiting and some patients describe a single non-recurrent event, whilst others experience it after a few doses or every dose [3, 4]. They usually occur with an early (but often not the first) dose of gold, but may occur for the first time in patients long established on gold therapy [5]. Tachyphylaxis usually occurs to these reactions, as it does to the effect of nitrates but, paradoxically, in other cases the severity of reaction increases with repeated doses. Such reactions may not, however, always be benign and there have been reports of myocardial infarctions [6, 7], stroke [8], transient ischaemic attack [9] and transient monocular visual loss [10] complicating such vasomotor reactions. These complications are presumably the result of severe hypotension, are unpredictable and may occur as the initial or first reaction, and also in patients with no history of atherosclerotic vascular disease, although it is tempting to assume that such patients must have had undetected underlying vascular disease and, indeed, in one case with no previous history of cardiovascular disease, the patient was found at autopsy to have occlusion of the left anterior descending branch of the left coronary artery [7].

The choice of gold compound can reduce the incidence of vasomotor reactions. Most are associated with GSTM, but they have also been reported with auranofin [11] and GSTG [10]. Both GSTM and GSTG contain 50% elemental gold by weight, whilst auranofin contains 29% gold. GSTM, being water soluble, is rapidly absorbed after i.m. injection and reaches peak plasma concentration between 2 and 6 h after injection. The plasma half-life is ~5–7 days. GSTG is absorbed more slowly and lower peak gold concentrations are achieved. Absorption of auranofin is rapid, but only ~25% of the administered dose is absorbed. It must be assumed that it is the gold compounds, rather than the vehicle or base in which they are made, that are responsible for these vasomotor reactions since they are the only common constituent of these three preparations. The increased incidence with GSTM is likely to be a reflection of its pharmacokinetics, with its more rapid absorption and higher peak concentration. This would imply a pharmacological effect of gold in a small number of susceptible individuals. However, the description of the nitritoid reaction is strikingly similar to that given for a so-called anaphylactic reaction to GSTM [12] and raises the question as to whether this was true anaphylaxis or a misclassified nitritoid reaction. A report of anaphylaxis, this time occurring in a patient taking a concomitant ACE inhibitor who after unremarkable treatment with GSTM received a single dose of GSTM, described the features of cardiovascular collapse accompanied by bronchospasm with the GSTM, making a true allergic reaction more likely [13]. In another description of anaphylaxis, angioedema and bronchospasm occurred without any hypotension, and clearly this did represent a true anaphylactic reaction [14]. Anaphylaxis to gold has recently become topical with the recommendation by the manufacturers of myocrisin that patients are observed for 30 min following each injection. Apparently, the basis for this recommendation is three reports of life-threatening anaphylaxis. In each case, this was associated with a darkening of the myocrisin solution [15].

What can be done for patients who experience a nitritoid reaction? Firstly, such reactions should be distinguished from true anaphylactic reactions. Most patients develop tolerance or tachyphylaxis to this particular event, and are able to continue the drug. Perhaps this should initially be in a lower dose, increasing gradually over subsequent weeks. Patients who have experienced nitritoid reaction should receive the gold in the recumbent position. In the USA, rheumatologists have the option of substituting GSTG for GSTM in patients who have had a significant benefit with gold injections. However, for marketing reasons, GSTG is not readily available in the UK, although it can be obtained by special arrangement with the manufacturer (Schering Plough). Although stroke and myocardial infarction have been reported in association with the nitritoid reaction, this should be viewed in perspective. We were able to find only one case of established stroke and two of myocardial infarction apparently resulting from nitritoid reaction in the literature over the past 20 yr. Furthermore, nitrate vasodilatory reactions, including ‘GTN syncope’, are common in patients receiving nitrates. The patients for whom such drugs are prescribed are much more likely than the rheumatoid population to have critical narrowing of blood vessels, rendering the appropriate organ susceptible to a sudden fall in perfusion pressure; however, no special precautions are taken or advice given to such patients.

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