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Work Related Musculoskeletal Pain and It's Management

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<http://dx.doi.org/10.5772/51706>

1. Introduction

This chapter reviews current best evidence in the identification and management of work related factors causing musculoskeletal pain and discomfort.

Work related MSDs of the low back and upper extremity are an important cause of morbidity and have a high economic cost to society, which in 2001 was estimated to cost the United States \$54 billion per annum[1]. The cause of the morbidity is pain and discomfort caused by a mis-match between the physical and physiological factors of tissue load and tissue tolerance which causes a combination of structural damage and metabolic waste accumulation. Some of these strains and sprains are acute overexertion, where there may be direct damage to the muscle fibres, the tendons or the ligaments, with either micro-tears or more gross structural damage. This in turn leads to activation of peripheral nociceptors (type II and IV pain fibres) by direct mechanical or chemical action. This may, in turn, result in central sensitization.

A subacute mechanism may also occur whereby there is a gradual onset of tissue damage over time. Chronic MSDs are also associated with pain syndromes or the 'overuse' syndromes. The combination of peripheral and central sensitization has led to investigation into how psychosocial factors modify the experience of pain. There are social and cultural associations with these chronic pain syndromes which have resulted in 'epidemics' of non-specific musculoskeletal symptoms, for example repetition strain injury (RSI) in Australia[2]. Patients may lie at the extremes of this spectrum of disorder, some have acute pathology readily amenable to intervention while others are disabled by 'chronic pain syndromes.' Many do however lie between these extremes and there is now more general agreement that work related MSDs have a multifactorial aetiology.

When attempting to manage any of these conditions it is therefore essential to recognize when work related factors are part of the aetiology because there is a greater likelihood of

successful treatment and rehabilitation if these are successfully identified and dealt with. The important work related factors which must be assessed include force, posture and repetition, factors which have an important bearing on muscle loading and the ability to recover. Once an MSD has occurred, early identification and reporting will ensure that interventions can be applied and in planning these there are work related and organizational factors which must be understood. Chief amongst these is the ability to modify the work to avoid or reduce the effect of precipitating factors. Organisational factors encompass the availability of medical resources, but also include the environment in which recovery takes place. The psychosocial climate of the workplace, for example whether co-workers and management are perceived as being supportive or not, is acknowledged to be important[1,3].

It is essential in dealing with workplaces and employees that the doctor recognizes their role. If they are acting for a third party such as an employer or an insurance organization (the 'double agent' scenario)[4] there is a fundamental change in the nature of the clinical relationship. It may not be a traditional 'doctor patient' relationship but may become a 'doctor client' relationship. The role of the doctor is to be impartial and honest with all of the agents and it is essential to be explicit about this point with the client. This may be perceived as running contrary to the Hippocratic principle of 'do no harm' because the consultation may not always achieve outcomes perceived by the 'patient' as being in their best interest, for example cessation of a benefit and returning to work with low back pain. The philosophical framework within which we make our judgments is in fact complex[5], and an area of practice well worthy of further study. The ethical implications of our work become clearer with experience. For this reason the practitioner is urged to reflect on the effects of the decisions that they make. Opening up an ethical dialogue with the client will help in this matter

2. The epidemiology of work related MSDs

There are significant difficulties in implementing studies which examine the relationships between work related factors and MSD outcomes. Prospective studies are best suited to clarifying causal issues, but the dynamic nature of MSDs, with a tendency towards recurrence, causes difficulties in identifying incident cases. Case identification is usually based on measures of frequency, duration, severity or disability. If meaningful comparisons are to be made between studies consistent case definitions are required, which is often not the case. This means that the results do not lend themselves to inclusion in meta-analysis, thus losing opportunities for more powerful estimation of the magnitude of the risks. Many studies also rely on self-reported symptoms as an outcome measure, without the additional benefit of clinical examination or investigation. The exposures, for example work related physical factors, have the multiple domains of force, posture and repetition. All are subject to misclassification errors, as is the classification into exposure groups. Lastly, there are the complicating factors of the epidemiological confounders, not least of which are psychological factors. Despite these problems, many studies on occupational MSDs have been carried out and the relationships with work are becoming clearer.

3. MSD classification

A convenient classification of work related MSDs is into the 'axial' anatomical areas of the neck, neck/shoulder and back and the 'peripheral' areas of the shoulder, elbow and hand-wrist. Both low back pain (LBP) and neck pain (NP) are amongst the commonest MSDs, with over 80% of individuals experiencing some degree of discomfort in the area over a life-time: over 80% in the low back[6] and 67% in the neck[7]. It has been suggested that the most important causes of LBP are genetic, this influence causing a tendency towards spinal degeneration[8]. The reality is however more complex because the normal biological pain experience is modulated by psychological and social factors, the biopsychosocial model providing a heuristic framework for understanding the pain process[9]. There is evidence that occupational factors play a significant role within this model. As the most prevalent MSDs, LBP and NP provide a useful framework for understanding the other MSDs: the aetiological factors have the same nature and the investigation and management follow similar principles.

4. Axial pain: Acute low back pain

If LBP is of acute or subacute onset, of less than three months duration and has neither radiation suggestive of radicular involvement nor 'red flags' (features of cauda equina syndrome; severe worsening pain, especially at night or when lying down; significant trauma; weight loss; history of cancer; fever; use of intravenous drugs or steroids and patients over 50 years old) suggesting other pathology it can be considered 'non specific' or mechanical in nature.

There are many risk factors that have been associated with LBP and they have opportunities to act at various points during the complex 'natural history' of the disorder, affecting incidence, prevalence, recurrence, the chronic pain state, the transition to a chronic pain state, sickness absence and disability. The risk factors themselves can be classified into work related factors, being the physical nature of the work and the psychosocial work climate, along with the personal factors of physical and psychological attributes.

4.1. Work related physical factors in LBP

There have been many epidemiological studies of the relationship between the physical attributes of work and MSDs in general. Well designed studies are necessary to understand exactly how these attributes, including the main occupational factors of force, posture and repetition, contribute to the development of musculoskeletal pain and ill health. The quality of some epidemiological studies has been criticized[10], the problems already having been mentioned.

One of first major reviews of the physical causes of MSDs, including LBP, was carried out by the National Institute of Occupational Safety and Health (NIOSH)[3]. The factors that the review team looked at were force, leading to muscular stress; repetition, which increases the

cumulative loading; awkward postures which put the tissues at a mechanical disadvantage and vibration which causes adverse effects on blood vessels and nerves. The conditions for which the review panel found 'strong evidence' for an association with physical workplace factors were neck pain with posture; a combination of force, posture and repetition with elbow tendonitis, carpal tunnel syndrome and hand/wrist tendonitis and lifting/forceful movement with LBP. The factors where reviewers considered that there was 'evidence' of an association were neck pain with repetition and force; shoulder with posture and repetition; elbow with force; hand/wrist with repetition, force and vibration; tendinitis with repetition, force and posture and back pain with heavy physical work and awkward posture.

A more recent look at the evidence mandated by NIOSH and the National Institutes of Health emphasised the complex interrelationships involved in the causation of MSDs. The panel on musculoskeletal disorders and the workplace[1] (panel on MSDs), approached the problem (p.2) "...from a whole-person perspective, that is, from a point of view that does not isolate disorders of the low back and upper extremities from physical and psychosocial factors in the workplace, from the context of the overall texture of the worker's life, including social support systems and physical and psychosocial stresses outside the workplace, or from personal responses to pain and individual coping mechanisms." Their synthesis of the evidence was that the pattern of evidence for both the low back and upper extremity (p.7) "supports an important role for physical factors".

Systematic reviews provide important contributions to understanding occupational MSDs. Important points about these reviews is that they follow strict rules by providing explicit information about the quality classification of studies that they include in the first place. They then use similarly explicit criteria to assess the quality of the evidence, for example the number of studies showing a positive association, the consistency of the results and the strengths of the associations. There are then many methods for presenting the evidence base, for example support for the relationship between exposure and outcome is usually expressed qualitatively as, for example, 'strong; limited; insufficient or no evidence' or 'strong, moderate or insufficient' evidence.

An alternative approach, the 'narrative review', an expert consensus which follows fewer rules but sometimes gives a broader perspective, can give evidence which differs from that given by systematic reviews. This contrast is illustrated by the differences between the panel review support for a relationship between physical factors and MSDs and a more recent systematic review which found 'conflicting' evidence about the relationship between force, in terms of heavy or physically demanding work, and the incidence of LBP[11]. It also illustrates some of the pitfalls because this review group were criticized for the way in which they categorised the evidence. They were taken to task by their definition of 'conflicting' as "inconsistent evidence in the available studies" by Takala (p.E282)[12] and also (p.E1011) for "mechanistic interpretation of statistical testing", the latter further elaborated by Olsen[13] (pE576) as "counting and comparing the number of statistically significant and nonsignificant associations". These commentaries suggest that the 'conflicting' classification is misleading. The 'mechanistic' criticism implies that some

systematic reviews are prone to the effects of rather arbitrary choices between 'numbers of counts' in deciding when a relationship is significant or not. Systematic reviews do involve qualitative (in the research sense) decisions. Such decisions have in fact been common practice in occupational epidemiology as the decision to choose a 'cut point' between exposure categories can also be somewhat arbitrary. Meta-analyses avoid this particular problem.

One should not of course ignore the results of individual good quality studies, for example well powered prospective cohort studies. A good example is a large Danish cohort study[14] which concluded (p.1) that "Uncomfortable working positions, lifting or carrying loads, and pushing or pulling loads increased the risk of onset of long term sickness absence".

As regards posture, there is little evidence that sitting is a risk factor, and strong evidence that standing and walking has no association[11].

Whole body vibration (WBV) is a prevalent exposure for workers who drive or operate plant and machinery. Vibration acceleration is measured in three standard axes, X, fore and aft; Y, lateral; and Z, vertical. Acceleration acting in the Z axis is likely to place significant dynamic compressive loads on the spine. Exposure, like noise, is rarely steady and there are two main acceleration exposure indices, the 'equal energy' root mean square (r.m.s.) acceleration measured in ms^{-2} , or a measure of vibration dose, the vibration dose value (VDV) measured in $\text{ms}^{-1.75}$. The latter gives more weighting to 'shock' vibration, a quality which will have been apparent to those who have travelled 'cross country' in an all terrain vehicle or '4 wheel drive'.

There are standards for such exposure[15], probably the most useful values to remember being the European 'exposure action value' of 0.5 ms^{-2} which requires employers to activate a programme to reduce the exposures and a value of 1.5 ms^{-2} as the 'exposure limit value' above which workers should not be exposed.

The panel for MSDs[1] was of the opinion that WBV was an important factor for LBP, with (p.99) 16 out of 17 studies reporting a positive association, risk factors lying between 1.3 and 9.0. This is an example of when systematic reviews give a different perspective. Taking the quality of studies into account Bakker et al.[11] were able to include only 6 studies in a review, reporting (p.E284) "conflicting evidence for whole-body vibration as a risk factor for LBP". Although it is always difficult to interpret how epidemiological studies text should influence clinical decision making when dealing with individual cases, the European (or other) limits should be borne in mind when managing LBP in those who may be subject to WBV.

4.2. The psychosocial work climate in LBP

The psychosocial work climate has been difficult to characterize and therefore to study, but three systematic reviews summarise the evidence[1618]. Hartvigsen et al.[16], in a review of prospective studies, provided a useful categorisation of work related factors: perceptions of work (e.g. job satisfaction, feelings toward work); organisational aspects of work (job security, job demands); social support at work (recognition and respect, social support) and

stress at work (stress, job strain). This review considered both LBP and the consequences of LBP, but after assessing 40 prospective studies (p.3) “no clear picture of the relation between work related psychosocial factors and LBP emerges”. Hoogendorn et al.[17], assessing cohort and case-control studies, said that (p.2122) “comparing the results of this and other reviews on psychosocial factors showed that although there was evidence for the effect of some psychosocial work characteristics in all reviews, the results were rather heterogenous”. Linton[19], in his review of prospective studies, did find significant associations. These were ‘strong’ for job satisfaction; monotonous work; work relations and perceived demands/load, ‘moderate’ for job control and pace and ‘insufficient’ for job content. The apparent disparity in these findings once again reflects the quality criteria applied in selection of the studies and how the evaluation of strength of association was carried out.

4.3. Individual demographic factors in LBP

Examining basic demographic factors and the association with low back pain highlights the heterogeneity of MSD outcomes of interest. Age has been shown to have a strong association with duration of sick leave[20]; an association with poor recovery[21]; an inconsistent relationship with work disability[22]; and no relation with either to return to work[23] or chronic disability[24]. The review showing a strong association with age and duration of sick leave[20] looked specifically at factors predicting duration of sick leave in workers in the early stages of an LBP related sick leave episode. On the other hand that showing no association between age and sickness absence[23] was specifically aimed at patients with non-specific low back pain and distinguishing predictors for either reporting sick (absence threshold) or to returning to work (return to work threshold). The concepts of absence threshold and return to work threshold are part of a sickness absence model first described by Steers and Rhodes[25] but adapted by Allegro and Veerman[26], which is, as usual, multifactorial. The model has a number of complex decision points for the patient with multiple opportunities for the psychosocial environment to exert an influence on their choices. The environment includes the economic and socio-political environment with factors such as the availability of alternative employment, sick leave and ill health retirement. These factors are all plausible reasons for the differences between studies.

4.4. Individual psychological factors

In 2002 Pincus et al.[27] carried out a review of psychological factors and their relationship with chronicity and disability in relation to low back and neck pain. The authors acknowledged the epidemiological uncertainties introduced by the characteristics of the specific populations in question and the measurement tools applied. The measurement tools, to be specific using questionnaires designed to measure psychological attributes in psychiatric patients (in contrast to MSD patients), proved to be a limiting factor. Because of the shortcomings in psychological instruments this review group could not differentiate between psychological distress, depressive symptoms and depressive mood, so grouped them together as ‘distress’. They did however find that distress was a significant predictor

and, acknowledging the importance of the population from which the sample is drawn, that this was especially so in the primary care setting. Linton[19], in examining low back and neck pain, also reported a 'clear link' between distress, as well as anxiety; stress; mood and emotions; cognitive functioning and pain behaviours. Cognitive variables such as fear avoidance beliefs have been associated as (Linton, p.1152) "a stable factor" for both LBP and neck pain, as have expectations of recovery. Linton also gives a particular mention as to how the person experiencing the pain perceives that the problem will develop, saying that (p.1152) "This anticipated outcome may be of special importance in assessing risk factors given the simplicity in obtaining such information".

4.5. Clinical assessment

The history should include aspects surrounding the onset of the pain and whether there is any history of radiation or neurological disturbance. It is also important to ask about previous episodes and any disability that resulted, as well as current limitations. In the absence of red flags as discussed in section 4, page 3, further investigation, at least in the first 6 weeks, is not indicated.

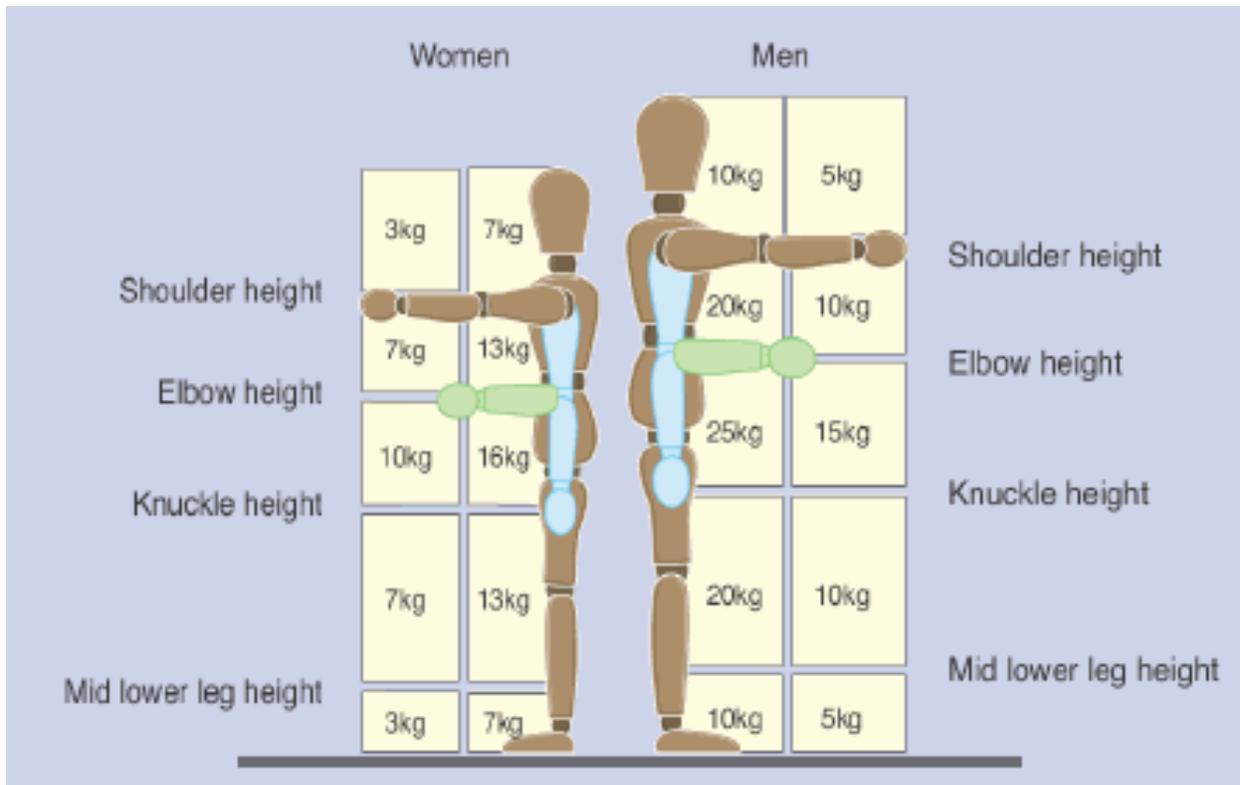
In the clinical assessment of LBP, as in all occupational MSDs, a thorough occupational history must be elicited. The history of the events preceding the acute episode is important, but many episodes occur spontaneously.

Occupationally, it is important to identify activities that may have caused the pain, or may be associated with limitations. This part of the history should include a review of the job in question in terms of occupational tasks and activities, best described from start to finish of each task or activity, working through them in a temporal or other logical sequence. Each task will require a description of the relevant work place physical factors of force, posture and repetition. As regards force and LBP there is no absolute 'cut-off' in magnitude of weight that it is safe or unsafe to lift, but carried close to the body, with arms flexed at the elbow and shoulders not extended, 16 kg for females and 25kg for males is a recommended maximum[28]. Postural factors modify this, reducing acceptable force to less than half if there is a mechanical disadvantage, for example with shoulders or elbows extended (figure 1). Twisting moments are thought to place additional biomechanical strain on the spine.

In terms of repetition there are no strict definitions, but a cycle of tasks occurring more frequently every 30 seconds should be classified as high.

There is no substitute for observing the person at work, actually performing the job tasks, however regular workplace visits may not be logistically practicable for many practitioners. If dealing with referrals from a workplace on a routine basis, it is however well worthwhile to carry out a workplace familiarisation visit during which relevant ergonomic factors can be assessed.

Any sporting activities in which the patient participates should also be assessed in much the same way as the occupational assessment, along with activities of daily living.



Source: HSE Leflet, Getting to Grips with Manual Handling

Figure 1.

There are a number of detailed assessment tools which are available to help in the assessment of the risk of MSDs. The Rapid Upper Limb Assessment (RULA) is an example, giving algorithms which may be used in the workplace setting[29].

4.6. Management of LBP

The management of LBP has been the subject of many reviews and much debate. In common with many areas of medical treatment, a sound evidence base, in the form of high quality randomised controlled trials (RCTs) is often lacking. The Cochrane Back Review Group[30] is instructive in the matter of treatment for LBP.

Adequate analgesia is necessary, paracetamol being a good initial choice. Non steroidal anti inflammatory drugs (NSAIDs) are also effective in acute and chronic LBP without sciatica, however a Cochrane review group found that effect sizes were small, that NSAIDs had more potential for adverse effects and that no one NSAID was more effective than another[31].

'Physiotherapy' includes many modalities of treatment including exercise therapy, but spinal manipulative therapy (SMT) and mobilisation (MOB) are common techniques. Other healthcare providers such as osteopaths and chiropractors also carry out these techniques.

A review of the evidence for their effectiveness[32], reported that, for acute LBP, (p.339) "There is moderate evidence that SMT has better short-term efficacy than spinal mobilization and detuned diathermy. There is limited evidence that SMT has better short-term efficacy than a combination of diathermy, exercise and ergonomic instruction."

When there was a mix of acute and chronic LBP[32], as is commonly the case, SMT/MOB provided (p.332) "either similar or better pain outcomes in the short and long term when compared with placebo and with other treatments, such as McKenzie therapy, medical care, management by physical therapists, soft tissue treatment and back school."

For chronic LBP, Bronfort et al.[32] reported that SMT/MOB was effective compared with a placebo, general practitioner care or other physical therapy and similarly effective as NSAIDs. However, a later review of SMT (which in this case included both manipulation and mobilisation techniques)[33], reported that (p.3) "SMT appears to be as effective as other common therapies prescribed for chronic low back pain such as exercise therapy, standard medical care or physical therapy."

4.7. Intervention strategies

Good quality interventions can only be designed on the basis of RCTs, which have proved a challenge to implement, follow up being difficult in the modern more dynamic workforce. In keeping with the multifactorial aetiology of MSDs, and knowledge about injury reduction interventions, it is becoming clear that single faceted approaches to primary and secondary prevention are not effective[34]. An example of a single faceted approach is an ergonomic intervention in which work-stations are physically changed. A multi faceted approach would include additional educational programmes and training.

As noted by Boocock et al.[35], Westgaard and Winkel[36] provided a useful construct for understanding ergonomic or 'mechanical' exposures and how the 'confounders' of other physical and psychosocial factors can be influenced by intervention strategies. They identified three main groups of intervention: mechanical exposure, production systems/organisational culture and modifier interventions. The first two are designed to change job exposures. Mechanical exposure interventions are aimed at improving the design of the work to reduce mechanical stresses. Production systems interventions change the exposure by changing the systems and culture of a company with focus on how the work is done and participation, for example using teams instead of individuals, influencing product flow and work technique training. Modifier interventions are not designed to change the exposure but to put the worker in a better position to deal with demands such as providing micro-breaks and pauses in work. These intervention strategies are considered further in section 6.2.

4.8. Rehabilitation

A fundamental tenet of occupational rehabilitation for all MSDs is that the patient should stay at work if possible, as physical activity will benefit most patients and protect against the

adoption of a 'sick role'. There is evidence that if a worker is on sick leave for 4-12 weeks they have a 10-40% risk of still being off work at one year[37].

This tenet must however be tempered with the knowledge gained from a review of the job, and will require that the demands of the job are balanced with the abilities of the individual. Functional capacity of the individual client can be assessed, on a pragmatic basis, by asking them which tasks are giving difficulty, if (and with what degree of difficulty) they can be carried out, for how long, if there are any alternative tasks in the workplace that they could manage and if, in their opinion, the job or tasks could be modified. In general, task modification can be by means of hardware or software. Hardware in this context refers to the interface between the person and the work. A regards prevention of LBP loads should be presented to the worker so that they can perform manual handling in as 'near neutral' a posture as possible. Transfer points, for example the ends of conveyors, should therefore be adjustable to avoid trunk flexion. Software interventions include administrative arrangements which facilitate job rotation. This can shorten intensive job cycles and bring into play different muscle groups. Another strategy to reduce musculoskeletal stress is to provide adequate pauses and breaks.

This is a good point at which to consider the ethical implications of any recommendations. A return to work is undoubtedly good therapy for those who have an MSD, but the work context must be taken into account. One should consider whether the work, for example whole body shock vibration in a bulldozer driver, will cause excessive discomfort. The effect on the work should also be assessed, another good illustration being the effect of discomfort on cognitive performance in safety critical jobs.

The knowledge gained from this overall assessment should form the basis of a return to work contract between patient and treating practitioner. This must be communicated to the workplace in as specific and detailed a manner as possible in terms of the tasks that can be carried out, along with any limitations such as restrictions in the force or weight that can be borne, tasks that cannot be undertaken or that require modification and restrictions in 'duty cycle' (the frequency and or duration of tasks) that may be necessary. Objective assessment and agreement between practitioner and patient is essential, in particular the time frames that can be expected for a return to normal work, for which there are guidelines: the normal expectation should be of recovery from LBP within two to four weeks. The process from here will be a matter of adjustment and matching between the physical capabilities of the individual and the demands of the workplace.

4.9. Barriers to adjustment and return to work

A number of psychological factors have been associated with the prevalence of MSDs, their chronicity and poorer outcomes. These have been described as 'yellow flags'[38]. They will not be present in every patient, but barriers should be suspected if recovery is delayed beyond the usual 2-4 weeks. These can be broadly classified into personal and work related factors. The point at which they act, either in aggravation of the experience of acute pain or

in the transition from acute to chronic pain cannot be identified with any certainty. They do however need to be identified and are discussed in the next section on chronic LBP. The New Zealand Guidelines Group provide a practical guide to assessing 'yellow flags', in the form of a screening questionnaire and clinical guide-line[39].

5. Chronic low back pain

Chronic low back pain can be classified by the frequency, duration, and intensity of the pain that is experienced, by the degree of disability that results, by the amount of sick leave that is taken or a combination of these indicators. All may be variously defined, which does give rise to some difficulty in making comparisons.

A number of variables seem to be important in the transition between acute and chronic LBP, these include biological, cognitive, affective and behavioural elements. It is very important to bear in mind that research into these factors has been conducted in many settings, be it presenting for treatment in primary care, in a workplace, a specialist referral centre or for disability benefit assessment. The setting will influence how the risk factor influences both the prevalence and strength of association. When identifying the significance of risk factors one must therefore examine carefully the population from which the study is drawn. With psychological variables it must be clear what instruments were used to measure the psychological constructs and how outcome measures, in terms of 'unfavourable outcomes', were defined.

It has been suggested that cognitive and behavioural factors might play a part in the transition from acute to chronic LBP. A review by Ramond et al.[40] looked at the problem prospectively in the primary care setting. Coping strategies were evaluated in four studies and fear-avoidance beliefs in seven, with statistical associations in about half of these. The authors specifically mention high pain related fear as being a powerful predictor in a Dutch study[41].

Presence of a belief about future harm from, or being disabled by, LBP has been shown to result in adverse outcomes in a number of studies. It is relatively simple to gain information in this respect[19], so it is an area where reassurance may be helpful.

In people with both non-chronic[42, 43] and chronic LBP[23] more positive expectations of recovery have been associated with better sick leave related outcomes.

6. Axial pain: Neck pain

Shoulder/neck disorders are an important 'cross-over' region. There is debate about 'specific' shoulder pain, discussed below, and 'non specific' shoulder pain in the cervico-thoracic area. The latter seems to have few occupational risk factors and thus seemingly spontaneous onset, for example 'tension neck syndrome'. A considerable range of clinical conditions is therefore represented. Epidemiologically, this can give rise to classification problems, many studies using self reported neck pain as an outcome rather than the result of a clinical assessment.

There is no doubt that neck pain is common, The annual prevalence for neck pain has been reported to range between 16.7% to 75.1%, with a mean of 37.2%, similar figures to LBP[44].

Diagnostically, there is a great deal of variation in the terminology used to describe neck pain and the clinical diagnostic protocols involved. Larsson et al.[45] make a good point in saying that (p.455) "The basis for the diagnostic criteria of neck and shoulder myalgia is relatively vague, and the diagnostic terminology and methods for assessment of neck and upper-limb musculoskeletal disorders are variable." They introduce a standardized clinical examination protocol as a diagnostic aide for neck and upper extremity disorders, which includes standardised questions on pain, tiredness and stiffness on the day of examination, with physical tests including range of motion and 'tightness' of muscles, pain threshold and sensitivity, muscle strength, and palpation of tender points, to which should be added tests of sensation.

A broad and functional diagnostic classification would distinguish between muscular and vertebral dysfunction. Acute muscular dysfunction is recognisable as torticollis, with acute or subacute onset and severe muscle spasm and limitation of movement. It is usually idiopathic however if there has been a motor vehicle crash or other incident involving sudden neck acceleration it may be classified as a 'whiplash' injury. Vertebral dysfunction arises because of facet joint, intervertebral disc or spondylitic problems. Nerve root problems will be suggested by the presence of cervical nerve root symptoms including dermatomal radiation but especially if sensorimotor symptoms are present. Cervical outlet syndrome will be suggested by symptoms and signs in the ulnar nerve distribution.

Non-specific neck pain, a diagnosis of exclusion, lies in a continuum. It may only involve the neck muscles, but may also spread beyond that boundary, seldom strictly classified but would include the middle and lower fibres of the trapezius. If tender points are palpable in the trapezius area, some would advocate a diagnosis of 'trapezius myalgia', which may occur at the more chronic limit of the spectrum.

As with LBP, it is essential to rule out 'red flag' disorders.

Of the risk factors, sex does appear to play a significant role in neck pain, females having a higher prevalence than males [44]. There is no ready explanation for this.

6.1. Interventions in neck and upper extremity pain

As for LBP, adequate analgesia is necessary in the acute phase, along with an explanation that the discomfort will settle.

Conservative treatments, including physiotherapy, have played a major role in treatment and intervention in these conditions, however there is not a good evidence base for the efficacy of such treatment, partly because there have been fewer high quality intervention studies. A Cochrane review was carried out by Verhagen et al. in 2006[46]. Although subsequently withdrawn for reasons of being 'out of date' (the new protocol is however

available[47]) it summarised the evidence available at the time. The reviewers examined the role of physiotherapy and workplace ergonomic interventions for adults suffering from 'complaints of the arm, neck and/or shoulder' or CANS. The results, in terms of quality of the studies, was disappointing. From 126 references selected for full text retrieval only 21 trials were included in the review. The reviewers concluded that (p.4) "...there is limited evidence about the positive effectiveness of exercises when compared to massage; adding breaks during computer work; massage as add-on treatment on manual therapy, manual therapy as add-on treatment on exercises; and some keyboards in people with carpal tunnel syndrome when compared to placebo. There is conflicting evidence concerning the efficacy of exercises over no treatment or as an add-on treatment, and no differences between various kinds of exercises can be found yet. At the moment there is also conflicting evidence about the effectiveness of ergonomic programs over no treatment."

6.2. Workplace interventions in neck pain

Boocock and colleagues[35] carried out a review specifically based on the classification of Westgaard and Winkel[36]. Once again, although 451 studies were suitable for review, only 31 were selected for full assessment. Of these 31 (p.293) "Ten were classified as mechanical exposure interventions, two as production systems/organisational culture interventions and 19 as modifier interventions." Of the 10 mechanical exposure interventions, four looked at work environment/workstation adjustments. These showed some evidence of positive health effects. Three studies looked at changes to workstation equipment (keyboards and mouse design) in visual display unit workers with upper extremity symptoms and there was moderate evidence to support this intervention. Two of three studies examining the introduction of ergonomic equipment concerned vibration reduction, and although reporting positive health outcomes, were of low quality and gave insufficient evidence of benefit. Two low quality studies did not find improvements after organisational and work-task design changes in office workers and manufacturing assembly workers therefore there was not sufficient evidence to support these interventions.

Nineteen studies focussed on modifier interventions. Three medium-quality studies, including one randomised controlled trial, in workers with neck/upper extremity conditions (not including fibromyalgia) investigated exercise such as strengthening, flexibility and coordination and provided some evidence of the effectiveness of this type of intervention.

Another four studies looked at the benefits of this type of intervention in fibromyalgia sufferers, with some evidence in support of exercise regimes.

The effects of multiple modifier interventions including exercise were examined in eight studies, four that excluded fibromyalgia and four that included the condition. These also gave some evidence that such regimes were beneficial.

Lastly, one RCT examined multiple modifier interventions excluding exercise but including cognitive behavioural training and education from which non fibromyalgia patients showed positive benefits. However, there was insufficient evidence to support this approach in patients with fibromyalgia.

Boocock et al. specifically compare their results with that of Verhagen et al.[46] who carried out the Cochrane review. The results for the mechanical intervention group were similar, bearing in mind that the 'limited evidence' category of the earlier review compares with the 'some evidence' category of the latter. Both reviews agreed that production systems interventions showed little benefit. However, one significant difference was that the Boocock et al. review showed that there was some evidence that modifier intervention would provide benefit.

As regards overall recommendations, there does seem to be benefit in introducing mechanical interventions. The majority of the studies in this category in the Boocock et al. review were of office workers using computers and either involved either work environment change (primarily lighting) and/or adjustment to office layout or equipment or looked at new keyboards and introduction of adjustable equipment. Fewer studies focussed on manufacturing, where force and repetition would be most prevalent and where one would expect most benefit in terms of mechanical interventions to reduce demand. Two studies did however look at hand-arm vibration reduction in a manufacturing setting again showing showing some benefit.

7. Peripheral pain

The acronyms associated with MSDs have already been introduced. The term RSI has been used as a broad term to describe conditions of the upper limb that have an association with force, posture and repetition, also known as cumulative trauma disorder in the US, with the term occupational overuse syndrome used in Australia and New Zealand emphasising the occupational associations.

There is good epidemiological evidence that peripheral MSDs have specific occupational associations. There is a much greater level of evidence than is the case with LBP and NP that the physical factors of force, posture and repetition play a significant role in these conditions, for example the postural factor of working above head height is associated with shoulder disorders[3]. As a general rule if a combination of these factors is present they should be considered significant.

An example of a population based study supporting the role of work comes from Roquelaire et al.[48] who carried out a population based cross sectional study over 3 years, taking a random sample from workers undergoing mandatory health screening by 83 occupational physicians. All the patients underwent a standardised clinical examination. Occupational risk factors were assessed by questionnaire. From the total of 3,710 workers a total of 472 had at least one MSD. Rotator cuff syndrome was, with 274

cases (7.4%) the most prevalent condition, followed by 113 cases (3.1%) CTS; 90 (2.6%) lateral epicondylitis; 45 (1.2%) De Quervain's disease; 30 (2.6%) ulnar tunnel syndrome and 29 (0.8%) wrist tendinitis. The risk of an MSD increased with age, those aged 50-54 having a higher risk (in comparison to those aged less than 30, an Odds Ratio (OR) of 4.9, 95% confidence interval (CI) 2.7-8.6 for men and an OR of 5.0, 95% CI 2.7-9.3 for women. A final multivariate logistic model showed an increased risk with age (all age groups compared to those less than 30 years; obesity (OR 1.6, 95%CI 1.1-2.4) and a prior history of upper extremity MSD (OR 3.3, 95% CI 2.6-4.2);

The associated physical factors were high repetitiveness (OR 1.6, 95% CI 1.2-2.0); arms at or above shoulder level (OR 1.5, 95% CI 1.1-2.0) and wrist bending in extreme postures (OR 1.5, 95% CI 1.2-2.0). The only work-related psychosocial factor remaining in the final model was low supervisor support (OR 1.3, 95% CI 1.1-1.7).

7.1. Shoulder pain

Occupational shoulder pain does have specific workplace associations with forceful repetitive work especially when the work is carried out above shoulder height[3]. Such work provides a biomechanically plausible mechanism for many of these injuries, but especially for rotator cuff syndrome (supraspinatus, infraspinatus, or bicipital tendinitis) where increased pressure may impair the microcirculation and cause microtrauma.

Rotator cuff syndrome is a common condition, and the most common source of shoulder pain in those over the age of 35, the prevalence rising with age. In those over the age of 70, tears may be found in more than 50% of those presenting with shoulder pain[39].

The pathological changes in rotator cuff are becoming clearer with time, but involve either tendinosis or a tendon tear, either full or part thickness. Tendinosis may occur due to the changes outlined above with microtrauma, but degenerative changes also occur due to age and there is an association with systemic conditions including diabetes. Tears occur with more forceful activity or where tendons are weakened due to degenerative changes. These may be classified as partial, full thickness or massive (involving more than one rotator cuff tendon).

The history should include the mode of onset of the pain, the location (typically at the point of the shoulder), whether the onset was acute or chronic and whether night pain is present. A detailed occupational history should include the factors of force, posture and repetition, and a sporting and recreational history is essential. The occupations which involve work at or above shoulder height include automotive engineers, builders, electricians and assembly line workers.

There is no single clinical finding that is pathognomonic of the condition, but a 'painful arc' is highly suggestive, and weakness in shoulder abduction may indicate the presence of a tear.

If a tear is suspected, diagnostic ultrasound of the shoulder should be carried out: this is a valid test for full thickness tears but the validity for detecting tendinosis or in differentiating other causes of painful shoulder has not been demonstrated[39].

Differential diagnoses include subacromial bursitis and acromioclavicular joint problems. The clinical findings (location of the pain and aggravating movements) may be sufficient to distinguish between them. Plain X-ray of the shoulder may exclude osteoarthritis in the older patient.

7.1.1. Interventions in shoulder pain

Treatment is aimed at pain relief and restoration of function.

The initial approach should include an adequate dose of NSAIDs. The occupational review should identify the activities giving rise to discomfort, and if these are causing significant pain the particular task or activity should be avoided or modified.

Corticosteroid injections are quite often used as an adjunct treatment for tendinopathy if NSAIDs have been given an adequate trial. For rotator cuff syndrome, the injection may be given either sub-acromially or intra-articularly. A Cochrane review of sub-acromial injections showed that they had a small benefit over placebo in some trials with generally short term benefit. Only 3 trials comparing steroid to NSAIDs were available for comparison, and there were however no significant benefit of injection.[49]

Physiotherapy is commonly used for shoulder conditions and Green et al.[50] (p.2) say that "There is some evidence from methodologically weak trials to indicate that some physiotherapy interventions are effective for some specific shoulder disorders. For example, the review group found that mobilisation plus exercise was better than exercise alone in people with rotator cuff disease.

Surgery has been advocated for rotator cuff syndrome, particularly in young and active patients. A variety of open and arthroscopic techniques has been developed, including subacromial decompression, debridement and repair of tendons, with multiple techniques often being used. A Cochrane review[51] summarised the evidence. Only two trials of rotator cuff tear repair met the inclusion criteria and neither had advantageous outcomes. The review group concluded that (p.9) "Based upon our review of 14 trials, all highly susceptible to bias, we cannot draw firm conclusions about the efficacy or safety of surgery for rotator cuff disease".

7.2. Elbow

The classical forms of elbow tendinitis are tennis (lateral) and golfers (medial) epicondylitis. These are, apart from the sporting connotations, well recognised occupational entities.

Lateral epicondylitis is caused by injury to the extensor tendons of the forearm caused by repeated resisted contraction of the extensor muscles, the force coming classically from

resistance to the action in the backhand swing in tennis or the more prevalent action of carrying heavy shopping or suitcases. The pathology is thought to be small tears or granulation tissue occurring at the attachment of the common extensor origin at the lateral side of the elbow. The pain originates here and may radiate to the forearm or wrist.

Occupationally the pain is caused by repetitive wrist extension and supination against resistance.

Medial epicondylitis is much less common and is caused by wrist flexion and pronation, or the forces acting against this motion, classically a golf swing.

There is strong evidence that a combination of the risk factors of force, posture and repetition are associated with both of these conditions. The occupational history should include the nature of the tasks undertaken, and observation, as always, is useful. Workers involved in assembly or construction work using screwdrivers are at particular risk, as are those using air or electrically driven screwdrivers or torque wrenches especially if the torque is not controlled adequately as tightening occurs. Hammering, and the repetitive motions of meat packing may also be causative. If a tool is being used, the grip should be of the right size- neither too large nor small.

The history should also include any sporting activities that are undertaken.

On examination in lateral epicondylitis, pain will be present distal to the insertion of the extensor carpi radialis brevis tendon at the elbow, and increased by wrist extension, especially with a pronated, radially deviated wrist in elbow extension. Classically, an introductory hand shake with the patient will exacerbate the discomfort.

In medial epicondylitis, the pain occurs with resisted wrist flexion and resisted forearm pronation.

The main differential diagnosis in the occupational setting will be non-specific forearm pain. This is more likely if the pain is diffuse, there is no clear history of force, posture or repetition in the job, no clear precipitating factors and the pain is atypical with a burning or other dysaesthetic quality.

Investigations are seldom needed in the presence of a typical history.

Treatment is by the avoidance of the provoking activities and maintenance and improvement of strength and range of motion by exercises which can be provided by a physiotherapist. It may be difficult to achieve the desired degree of rest in the occupations which give rise to epicondylitis, as they are often truly forceful and repetitive. If the treating physician is not familiar with the workplace, a task analysis will be necessary. This can often be carried out by an occupational therapist or physiotherapist who has training in, and experience of, workplace assessment. There may be ways in which the work can be changed, for example job rotation, to reduce cumulative loading on the individual. Individual technique and the design of any tools used also requires assessment. Bissett et al.[52] comment upon interventions for tennis elbow with outcomes including pain relief, functional improvement and 'global improvement', the

latter including return to work, or normal activities, or both. Corticosteroid injections seemed more effective than placebo or no intervention at increasing global improvement at 6 weeks, but not at one year. The authors comment that corticosteroid injection may however increase the recurrence rate. Autologous blood injection is a relatively new technique, with the benefits unknown at present, but case series have reported significant improvements[53]. Compared with placebo, it seems that topical NSAIDs reduce pain at 4 weeks but may not give functional improvement[52].

7.3. Hand/wrist pain

The classical hand-wrist occupational disorder is carpal tunnel syndrome, entrapment of the median nerve at the wrist. Ulnar neuropathy at the elbow is less common.

De Quervains tendonitis and wrist tendonitis also have occupational associations.

The occupations involved will generally be those involving significant forces or effort in performing the job, for example those carrying in assembly work, for example in the automotive industries, and those involved in meat processing, for example butchering or packing.

7.4. Carpal tunnel syndrome (CTS)

CTS is a condition which results from pressure on the median nerve at the wrist where it passes under the transverse carpal ligament. The pressure in this space may arise due to either tendinopathy or by enlargement of the other wrist structures. The symptoms are sensory changes, tingling and pain, in the distribution of the median nerve at the wrist. The motor branches may also be affected, with wasting of the thenar eminence. The prevalence in the general population[54] depends on the depth of screening, with 354 individuals (14.4%) of 2466 responders in a general population survey reporting symptoms, 94 (3.8%) confirmed 'clinically certain', 168 (6.8%) 'clinically uncertain' and 120 (4.9%) from both groups having neurophysiological tests reported as positive.

CTS is associated with endocrine disorders (diabetes and thyroid disorders) and pregnancy and there seems to be a familial tendency[55].

The diagnosis is primarily by the history of tingling and numbness, with loss of manual dexterity supporting the diagnosis. The pain is classically present at night, waking the patient, and may radiate either proximally or distally.

The symptoms typically localise to the palmar aspect of the first (thumb) to fourth (ring) fingers and the distal palm, being the sensory innervation of the median nerve at the wrist.

Numbness predominantly in the fifth finger or extending to the thenar eminence or dorsum of the hand should suggest other diagnoses. The pain may be atypical with more generalised radiation. This may indicate autonomic fibre involvement and does not exclude CTS from the diagnosis. There may also be a loss of motor function: the median nerve also

supplies motor fibres to the thenar muscles and the lateral two lumbricals. Apart from the 'hands falling asleep' the deficit in motor function may cause loss of dexterity and grip strength. The functional loss may cause the patient to drop things.

Investigations should include inflammatory indices and thyroid function.

Nerve conduction studies (NCS) are often advocated in the diagnosis of CTS. Using the results of such diagnostic tests in diagnostic algorithms does however require insight as to the properties of such a screening test. One must firstly bear in mind that there are many modalities of NCS and that a specific prescription for the test is required. A New Zealand review[56] looked at (p.59) "intra-individual comparative tests of the measurable conduction of the median nerve over equally long nerve segments in the hand" The specificity (a low number of false positives, the likelihood of correctly identifying those who do not have the disorder) was reported as lying between 83 and 100%. The sensitivity (a low number of false positives, so likely to identify those who do have the disorder) was reported as being in the order of 70-80%. Relying on the results of NCS requires the application of rules. NCS is therefore good at "ruling in"[57]: if it is negative then a patient is unlikely to have the condition. It is not however good at "ruling out" because a positive test does not necessarily mean that the patient has the condition. CTS is therefore a very good example of a condition which has a complex diagnostic algorithm.

Ultrasound and MRI scanning can also demonstrate compression, but are not superior to NCS[56].

Bearing in mind the prevalence of CTS, the occupational associations have been questioned. A review by Palmer et al.[58] found reasonable evidence that regular and prolonged use of hand-held vibratory tools increased the risk of CTS by a factor of 2 (a prevalence Odds Ratio of 2 or more) and also "found substantial evidence for similar or even higher risks from prolonged and highly repetitious flexion and extension of the wrist, especially when allied with a forceful grip" (p.57). The studies reviewed did have limitations, many being cross sectional in nature, but the clinical advice given in this paper is sound. The authors comment that highly repetitive gripping should be avoided by ergonomic design of tasks and tools, and by scheduling appropriate rest breaks. They defined repetitive wrist movements as flexion/extension every 30 seconds or more often. A detailed job assessment should be carried out in all cases of suspected occupational CTS.

Treatment is, in the first instance, expectant. A wrist splint, worn especially at night (as with De Quervains tenosynovitis, may improve the nocturnal symptoms and local corticosteroid injection may also be helpful in the short term. Definitive treatment is by surgical division of the retinaculum, which can be performed endoscopically.

7.5. Ulnar neuropathy

Ulnar neuropathy at the elbow (UNE) is, after CTS, the second most common upper limb compression neuropathy. The nerve is in a superficial position at the medial epicondyle of the elbow and this, along with the arrangement of the muscular septae in this area, means

that it is prone to ischaemia and traction injuries. Compression also occurs in the Guyon canal at the wrist. It has proved difficult to estimate incidence and prevalence, but between 1995 and 2000 Juratli et al.[59]. reported that 2,863 cases were diagnosed by the Washington State workers compensation system. It seems that the condition is expensive: this group reported (abstract) that of 250 surgical cases selected at random "The mean wage replacement and medical benefits paid per case were \$19,100 and \$15,200, respectively."

The diagnosis is suggested by pain and dysaesthesiae in the ring and little fingers of the hand. As in CTS this may be noticed at night and there may also be discomfort at the elbows. The sensory changes usually precede any motor loss, which involves the ulnar innervated small muscles of the hand. It is possible to have concurrent CTS and UNE in which case the clinical picture can be complex. Ulnar nerve conduction studies can be performed to help confirm the diagnosis, but there is little information on the validity of such testing. X-ray of the elbow and wrist may also be indicated if there are anatomical abnormalities or a history of trauma.

The work relatedness of the condition appears not to have been widely studied. Descatha and colleagues[60] describe a longitudinal study in which they were able to follow 598 workers with 15 incident cases. The variables most strongly associated with UNE were (p.237) "holding a tool in position"; BMI and another work related MSD. This does suggest that occupational activities may be important, the most likely suspects being those that are associated with epicondylitis and CTS. Direct pressure on the elbow (as in driving or leaning on the elbows at a work bench) and trauma around the elbow are also associated with UNE.

Treatment guidelines e.g. those of the Washington State Department of Labor and Industries[61], suggest that conservative treatment should be tried for 6 weeks. Surgery is usually reserved for cases that limit activities (i.e. with motor involvement). A Cochrane review group[62] could not recommend a single best treatment or when to treat a patient conservatively or surgically. Of the two main surgical options, decompression or transposition, both seemed equally effective.

7.6. De Quervains tenosynovitis

De Quervains tenosynovitis is caused by entrapment of the tendons of the abductor pollicis longus (APL) and the extensor pollicis brevis (EPB) tendons where the extensor retinaculum binds the tendon sheaths at the radial styloid process. The pathophysiological process is thought to be the same as with rotator cuff syndrome, with repetitive microtrauma giving rise to impaired circulation and granulation tissue.

Clinically the condition is characterised by wrist pain at the anatomical snuffbox that may radiate proximally or distally. There may also be localized swelling and crepitus on thumb and wrist movement.

There will be localized pain on palpation over the APL and EPB tendons, and discomfort on resisted abduction or extension of the thumb.

The original NIOSH panel was of the opinion that there was 'strong' evidence that a combination of force, posture and repetition (in other words highly repetitive, forceful hand/wrist exertion) was associated with hand wrist tendinitis[3], which includes De Quervains. The populations studied have included meat workers and assembly line workers, the wrist motions involved being complex in nature, but involving a strong grip.

The treatment involves avoiding the provoking activities, and wrist splinting (at least in daytime) for a period of two to six weeks may be helpful. Splinting should be applied in wrist extension of 10° and first carpo-metacarpal and metacarpo-phalangeal joints also both splinted in slight extension. If there is no improvement in four to six weeks, corticosteroid injection may be tried, as may adjunct NSAIDs. The evidence of effectiveness of the latter two, as in other MSDs, is uncertain.

7.7. Wrist tendonitis

Tenosynovitis of the wrist is a disorder of the tendon sheaths at the wrist, localised to the involved tendons, with symptoms on wrist movement. The prevalence in the general population is thought to be about 1% in men and 2% in women[63]. It is higher in working populations. A study of 5,338 workers in the Henan province of China[64] found the prevalence of self reported wrist discomfort to be 33.5% and associated with physical job characteristics: prolonged wrist bending or twisting; exerting great forces with arms or hands and using vibrating tools. In a study of garment workers[65], 146 out of 520 individuals (35.6%) reported hand/wrist pain for at least two days in the past month. Fifteen of the 374 who were pain free had one or more physical signs at the wrist and only 21 of the 146 reporting pain had one or more sign on examination. Tendinopathy examination findings were recorded as flexor carpi radialis (2.5%) or flexor carpi ulnaris (1.1%) tendinopathies; digital flexor tendonitis (0.9%) and extensor tendonitis (2.7%). Only about half of those diagnosed with signs reported symptoms. With the wrist stabilised, pain should be exacerbated by resisted movement of the affected tendon.

Conservative treatments include rest, NSAIDs physiotherapy and workplace interventions [66]. A review of conservative treatments[67] did however identify that there was no evidence to support or refute these strategies.

A systematic review of the efficacy of corticosteroid and other injections for the management of tendinopathies[68] concluding (p.1762) that "We have shown strong evidence that corticosteroid injection is beneficial in the short term for treatment of tendinopathy, but is worse than are other treatment options in the intermediate and long terms."

7.8. Hand arm vibration syndrome (HAVS)

HAVS is characterised by disorders of the intrinsic muscles, nerves, bone, joints and circulatory systems and is caused by vibration being transmitted to the hands by a vibrating tool or a work piece. Vibration White Finger (VWF) is the most common syndrome in the

UK and in other countries with similar climates, but the vascular changes are not so prevalent in more temperate climates.

This condition occurs following exposure to hand vibration, the first symptom being vascular, manifest as blanching of the fingers. The attacks usually occur on exposure to cold (and not directly after the vibration exposure): touching a cold surface, being exposed to a cold damp environment, or immersing the hands in cold water (cold water provocation, one of the diagnostic tests) will precipitate the blanching sign. Attacks are therefore more common in the winter.

There is a gradual progression of symptoms: the finger tips are first to be affected, but with increasing vibration dose the middle and proximal digits are affected. This blanching lasts for a period of minutes to hours, and ends with a reactive hyperaemia with pins and needles or frank discomfort or pain.

VWF also has a neurological component, noticed as tingling, numbness and a loss of manual dexterity. At first this is most noticeable after the vibration exposure stops and it recovers after exposure, but it may get worse with continued exposure and become permanent.

The two main mechanisms, vascular and neurological, have separate mechanisms. The vascular component is caused by ischaemia, and interest has focused on direct damage to the vessel walls and also the sympathetic neural network which regulates the arterioles. Neural changes seem to progress independently of the vascular damage, and result in the loss of sensation and motor function.

Other possible changes seen in the musculoskeletal system of the hand, wrist and arm in vibration exposed workers have been muscle fatigue, callus formation in palms and digits, enlargement of metacarpophalangeal joints, bone changes and alteration in grip force, but these have been difficult to quantify.

It is also difficult to differentiate between these effects caused by vibration and the effects of heavy physical work on the hand. Repetitive flexion and extension of the wrist, which action is also prevalent in some of these occupations, may lead to CTS and this may complicate the clinical picture.

7.9. Diagnosis

The clinical assessment is based on the extent of involvement according to the vascular and neurological components separately according to the 'Stockholm classification' (table 1) [69].

It is more usual for the vascular signs and symptoms to occur first, in other words the vibration white finger. The 1987 Stockholm classification of the vascular component mentioned above gives some guidance as to the onset. It is more usual for the blanching to affect the tips only of one or more fingers. The vibration exposure itself does not usually cause white finger, it is exposure to cold and wet conditions that trigger the vascular spasm, which is most commonly followed by hyperaemia.

Vascular component		
Stage	Grade	Description
1	Mild	Occasional blanching attacks affecting tips of one or more fingers
2	Moderate	Occasional attacks affecting distal and middle phalanges of one or more fingers
3	Severe	Frequent attacks affecting all phalanges of most fingers
4	Very severe	As in 3, with trophic skin changes (tips)
Neurological component		
Stage	Description	
0 _{SN}	no symptoms	
1 _{SN}	Intermittent or persistent numbness with or without tingling	
2 _{SN}	As in 1 _{SN} with reduced sensory perception	
3 _{SN}	As in 2 _{SN} with reduced tactile discrimination and manipulative dexterity	

Table 1. Stockholm Classification (1987)

The onset is usually gradually progressive with cumulative exposure, spreading to more fingers and farther down the fingers.

There are many occupations which involve exposure to vibration. Some of the highest exposures are to be found in the silviculture, construction and metalworking industries.

Primary and secondary Raynauds phenomenon must be ruled out.

Secondary Raynauds phenomenon (including vibration) may complicate other conditions, the most frequent of these being connective tissue disorders. Blood indices and inflammatory markers are required. A cervical rib will be excluded radiologically.

Routine laboratory investigations should therefore include rheumatoid factor, antinuclear serum antibodies, possibly cryoglobulins and serum protein electrophoresis. Scleroderma must be excluded.

There are a number of objective tests described to assess HAVS: vascular (cold provocation), the neurological component includes tactile, vibrotactile, and thermal threshold impairment (detected by aesthesiometry and altered vibration perception threshold). Musculoskeletal testing is sometimes carried out (dynamometry, pinch test and

pick-up test), but not all clinicians agree on what should be present in the test battery. Single tests such as the Allen test are of very limited validity.

Primary prevention of HAVs should be aimed at reducing exposures to a 'safe' level. The exposure action value¹ is an r.m.s acceleration value of 2.5 ms^{-2} , and the exposure limit value 5 ms^{-2} [15]. Exposure at 2.5 ms^{-2} will result in HAVS in 10% of persons after 12 years of exposure, and at 5 ms^{-2} after 5.8 years. Many vibrating tools, for example chainsaws and construction equipment, frequently exceed this value.

There is no specific treatment for HAVS, but with cessation of exposure the condition should remain static.

8. Summary

There is no clinical hallmark with which to distinguish occupational MSDs from those due to any other agency apart from the evidence gained from the occupational history. The astute clinician will always ask about the occupation of the patient during a consultation, knowing that important opportunities for disease prevention and treatment will otherwise be missed. In the clinical interpretation of epidemiological data it is as well to bear in mind that there are many methodological pitfalls in the study of MSDs. Not least of these is the dynamic nature of these disorders in that they tend towards recurrence. It can be therefore be difficult to distinguish between 'new' incident cases and those which are in fact recurrent or persistent. This misclassification of outcome will distort the relationship with risk factors. There are also problems with case definition either in terms of the self reported outcome measures of frequency, duration and severity or on the other hand clinical case identification. Exposure assessment and classification is also difficult, particularly assessing the elements of force, posture and repetition. There are also many important psychosocial confounders in the personal and work domains, the importance of which are becoming clearer. It can therefore be difficult to demonstrate a causal relationship when studying MSDs, for example the temporal relationship is often uncertain; the strength of the relationship can be diluted by confounding or misclassification and, as it is technically very demanding to measure physical exposures, dose response information is often lacking. Although the work-relatedness of some MSDs has been questioned, it is nevertheless of vital importance to understand the role of the work place in both causation and rehabilitation. The authors cannot do better than quote the words of Ramazzini, the 'father of occupational medicine', whose advice given to the profession was[70] (page 13) "There are many things that a doctor, on his first visit to a patient, ought to find out from the patient or from those present. For so runs the oracle of our inspired teacher: "when you come to a patients house, you should ask him what sort of pains he has, what caused them, how many days he has been ill whether the bowels are working and what sort of food he eats" So says Hippocrates in his work *Affections*. I may venture to add one more question: what occupation does he follow?"

¹ For definitions see section 4.1 (page 65)

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