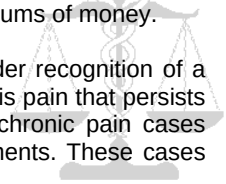
Introduction

RSI came to prominence in Australia in the 1980s. At that time it was tentatively described as a pain syndrome associated with repetitive movements. Usually there was no firm, specific diagnosis and there was considerable uncertainty as to causation and exposure to hazard.

It is now widely recognised that the sudden changes in the numbers of reports of upper limb pain in that decade were associated more strongly with social dynamics than with exposure to any physical hazard. There was also a considerable delay in the definition of an authoritative position on the diagnosis and cause of this reported pain syndrome, a delay that cost compensators considerable sums of money.



However, the Australian and subsequent international phenomenon lead to the wider recognition of a small subset of people with pain who go on to develop chronic pain syndrome. That is pain that persists long after any original cause has been eliminated or healed. A subset of these chronic pain cases attributes their plight to some tortious exposure, usually involving repetitive movements. These cases are referred to here as cases of Diffuse RSI (DRSI).

A detailed state-of-knowledge review of DRSI has been undertaken during the year 2000. This review is selectively summarised here.

This summary also makes repeated comparison with a review of the state-of-knowledge for Carpal Tunnel Syndrome (CTS). CTS is a useful reference point for DRSI as it is more fully understood and less contentious. Occasional comparison will also be made with Fibromyalgia, a diagnosis which to date has much in common with DRSI.

Each section of the summary also draws attention to current areas of uncertainty. These areas of uncertainty will provide a focus for the monthly monitoring and reporting work.

Further information on the state-of-knowledge for CTS, DRSI and Fibromyalgia can be obtained from the LPC Centre for Risk Sciences, see contact details on the cover page.

Diagnosis

Over the past two decades, there has been considerable revision of the meaning of the term RSI. In many specific cases a more accurate diagnosis, such as tenosynovitis, epicondylitis and so on could be found. In these cases RSI was used as a term to suggest a cause or hazard rather than the resulting condition. In those cases where there was no specific pathology, the term RSI was usually used to indicate the presence of aches and pains, attributed to some activity. This usage of the term has now been widely discredited.

Under normal circumstances, a firm diagnosis is an essential component of any successful claim for compensation. Without a recognised diagnosis it may not be possible to establish that harm has occurred. A recognised diagnosis for physical injury should normally have an agreed organic pathology, a unique objectively determined sign or set of signs (consistent with the pathology) and specific, consistent symptoms.

For example, cases of Carpal Tunnel Syndrome have been recognised as industrial injuries by authorities around the world. Whilst there is no unique method for making a diagnosis, specialists usually have little difficulty in agreeing a diagnosis in each case. There is a widely agreed pathology.

On the other hand, the current method of diagnosis for Fibromyalgia (FM) was created by a committee, which was endeavoring to define a sensitive and specific combination of symptoms that could be tested for accuracy by community trials. Such community trials have not yet been definitively performed. In spite of this, a diagnosis of FM has become increasingly common. It appears to be in use as a label to describe the plight of patients with a wide range of painful conditions. The value of FM as a diagnosis is hotly disputed.

At this time, FM and DRSI have in common that there is no agreed underlying pathology, no unique objective sign or combination of signs, no properly tested history and no specific symptomatology.

In spite of this, the decision of the Court of Appeal in Alexander and others –v- Midland Bank July 1999 seems to set a precedent allowing an unclear diagnosis to succeed as the basis for a claim for compensation. The evidence of the claimants' principal medical expert relied on making a reasonable inference, from a combination of examination and common sense, that the claimants had all suffered from a physical injury. In our view the circumstances of this case were unusually compelling (in the

context of UK culture), and it would have been surprising if the trial judge had not accepted the non-specific medical evidence called on behalf of the claimants.

This leaves largely unanswered the question of whether the harm in general cases of DRSI is psychological, psychosomatic or organic. There is a reasonable body evidence that points to the conclusion that DRSI is a syndrome that is neither purely organic nor purely psychological in origin or manifestation. This overlap creates considerable problems for civil law and is the inspiration for a great deal of research work. The Alexander case has for the time being apparently shifted the emphasis onto the defendant to establish positively that the condition was non-organic.

Some of this research work is centred on establishing the presence or absence of pathology or physical signs. However, it should be borne in mind that even the presence of physical signs is not a guarantee that there was an underlying physical cause. For example, a leading contender for a specific physical sign is vasoconstriction in the region where pain is felt (that is, where the blood supply is restricted by the contraction of muscles in the vessel wall). The control of such muscles is normally involuntary or, at least, not conscious. However, it is consistent with theory and with laboratory evidence that these muscles can in effect be trained to respond to stimuli that involve patient perception and that their contraction can be sensitised to low levels of physical stimulus. The resulting pain may be very real and medium-term physical changes may be found while this persists. The effect of pain is then to reinforce the conditions that lead to the development of sensitivity.

Other contenders for objective physical signs include: reduced mobility of the median nerve at the wrist, abnormal flare response, raised thresholds for vibration sensitivity and, fall of hand skin temperature following a familiar activity. However each of these effects *can* be explained by a mind/body interaction or by confounding factors in experimental conditions. As yet, they have not been shown to be specific to DRSI or entirely objective.

The distinction between DRSI and cramp of the hand or forearm (PDA4) is highly uncertain. It is plausible that PDA4 represents a subset of DRSI.

Any diagnostic regime which relies on the absence of positive signs of other conditions should continue to be unlikely to withstand close examination.

Causation

An understanding of causation usually involves assessment of evidence under the headings of aetiology, pathogenesis and natural history. Much of the scientific evidence of causation is based on epidemiological studies (which attempt to identify risk factors), the efficacy of treatment or other interventions and, clinical experience. The assessment of causation is particularly complicated in those cases where one condition evolves into another.

This point is particularly apposite for conditions that involve pain – which may occasionally develop into chronic pain syndrome.

In the case of CTS, if the patient is otherwise healthy and has no predisposing condition (e.g. diabetes, pregnancy) an episode of CTS will be usually be short-lived, mild and unlikely to recur. However, some of those people with an apparently favourable prognosis, and complete protection from likely physical hazards develop chronic pain conditions and long term disability.

Studies of the cause of CTS must therefore distinguish between acute and chronic conditions if they are to accurately identify and classify causal factors. Such distinction in scientific reports is very rare. There is a strong body of evidence that supports the view that, frequently repeated or prolonged awkward postures involving high force, are risk factors for *acute* symptoms of CTS. These risk factors do not appear to be significant predictors of the persistence of CTS symptoms for more than a month. Significant, but not yet fully accepted, predictors for chronicity are pain severity and catastrophising, both of which are subjective and may have little relationship to the degree of any organic harm.

What is of interest is the possibility that DRSI like chronic CTS originates from some mild condition that somehow becomes a chronic pain condition. For example, If DRSI originated as CTS then it would share the same physical risk factors in its early stages and may share the same risk factors for progression to a chronic pain condition. Unfortunately we were not able to find studies that could specifically resolve this possibility. Studies that link DRSI with a specific pain condition would provide a significant change to the state-of-knowledge.

So far, no specific pathology or objective sign for DRSI has been found. This makes the likelihood of defining its cause somewhat remote. In particular, there are few, if any, prospective epidemiological

studies that measure DRSI as an outcome. Most studies measure acute pain (which may, or may not, be a precursor to DRSI).

The most consistent theoretical model for the cause of DRSI is, in outline, as follows:

- In the first instance there is a muscle activity that induces muscle fatigue, all else is normal. Fatigue can arise from many causes including overuse, unusual use, lack of rest before or after activity, ill health, poor sleep and so on.
  Fatigue gives rise to chemical stimuli perceived as pain. There is no need for diagnosable inflammation or tissue damage to initiate this process.
- In some circumstances, rest (cessation of conscious/deliberate muscle activity) does not resolve fatigue. This could be because of lack of sleep, habitual tension in the affected muscles or ill health for example.
- If there is continuous and/or repeated pain, and lack of recovery, the spinal cord adapts so as to increase sensitisation (possibly mediated by the sympathetic nervous system, the system that controls vasoconstriction). The normal, so-called 'gate closing' mechanisms begin to be overridden. Stimulation of pain becomes more facile with lower and lower levels of fatigue.
- Higher brain centres become involved if the above steps are repeated, especially if the activity is associated with distress or there is some psychiatric instability, reducing the spinal cord's ability to change back to a normal state. Secondary effects such as behaviour changes, e.g. avoiding the activity and anticipatory adaptations e.g. pre tensing, become established. Opportunities for resolution of spinal cord adaptation and prevention/resolution of fatigue are gradually lost.
- Hypersensitivity becomes established. Other secondary pain phenomena become established. Any muscle tension induces pain. Habitual muscle tensing becomes the norm or is readily provoked.

Evidence to support this hypothesis:
It is consistent with the widely accepted (but not objectively assessed) natural history.
It is consistent with symptoms and signs. (Pain, response to cold (localised muscle tensing), cramps, pins and needles, depression, anxiety, cold hands….)
It is consistent with established, pain mechanisms.
It is consistent with the finding that psychological distress predicts chronicity.
It supports the finding that the guilty activity need not be repetitious (e.g. finger tapping, screwing) but that is must be, or perceived to be, cyclical (e.g. daily). This would explain why cases are not activity specific.

This hypothesis is concerned with pain and may not be entirely consistent with the posture specific cramping (dystonia) associated with writer's cramp. In this case, the act of picking up a pen causes cramping to occur, with the wrist and fingers locked in the usual writing position. It is not clear whether or not posture specific cramping requires a different hypothesis or is just a subset of the above hypothesis (e.g. an extreme case of pre-tensing to avoid pain).

Contribution - DRSI
Whether or not the above hypothesis proves to be correct, the evidence so far points away from physical / ergonomic causes and more towards issues of perception and belief. In our view it is as yet highly unlikely that a case could be made for a tortious contribution to these factors at work. In principle, a hasty diagnosis or excessively protective response at work might go a long way towards convincing a patient that they should fear the worst.

Aggravation - DRSI
So far, there is no convincing evidence that DRSI can be made more likely by exposure to physical factors at work. Indeed, the strongest evidence so far is that physical factors are not significant.

This should be contrasted with CTS where it is plausible that predisposition can be aggravated by physical factors at work. So far, no definitive work has been done to support this, but in practice this hypothesis often goes unchallenged.

Foreseeability

Only when causation is established and defined is it reasonable to define foreseeability.

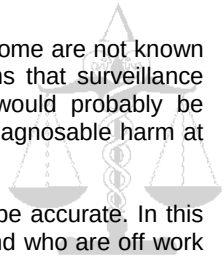
At present, there are no pre-placement individual signs or job design factors that allow reasonable foreseeability of DRSI.

However, if a competent diagnosis of work-related non-specific chronic pain syndrome is made, this would provide sufficient notice of an individual vulnerability. The probability that this outturn then indicates significant risk for other people employed in the same work is not known but, if the preferred aetiological hypothesis is correct, this probability should prove to be very small indeed.

Absence from harm after 5 to 8 years would suggest that harm will not occur/recur but this cannot be guaranteed.

The popular trend is toward allowing any cause of discomfort (aches and pains) at work to be considered sufficient notice of potential harm. In our view, this approach is more precautionary than it is reasonable. Social trends are moving towards a preference for a precautionary approach.

Surveillance
Given that clear risk factors for DRSI or work-related non-specific chronic pain syndrome are not known and given a trend towards a precautionary approach to risk management it seems that surveillance would be the preferred method for preventing diagnosable harm. However, it would probably be unreasonable to allow that reports of aches and pains provide sufficient notice of diagnosable harm at some unspecified time in the future.



An analogy with another chronic pain syndrome, back pain, may one day prove to be accurate. In this case, the proportion of people who take time off work with non specific back pain and who are off work with back pain a year later is less than 5%. If you accepted that taking a day of work with back pain made it foreseeable that long term disability would occur, you would be wrong on at least 95% of occasions. More accurate surveillance techniques would be required before they could be considered reasonable.

Duty of care

There remains considerable uncertainty about the variables required for the cause of DRSI. Repeated fatigue, depressed mental state, catastrophising and lack of rest seem to be likely requirements at some point in the pathogenesis of DRSI, but the relative timing of these and whether one causes the other is unclear. Which if any, of these should or could be controlled by employers is not entirely clear.

Postulated ergonomic risk factors for the initiation and perpetuation of DRSI do not appear to have the support of the scientific literature.

Given these uncertainties it is not possible to provide accurate assessment of the reasonable requirements of a duty of care to prevent exposure to significantly increased risk.

Regulatory duties which, it could be argued, purport to address these health risks are specified under the Management of Health and Safety at Work Regulations (1999 and 1992), Display Screen Equipment Regulations (1992), Workplace Regulations (1992) and, the Manual Handling Regulations (1992), and associated guidance. These standards are currently widely accepted in the UK.

In the absence of reasonably justifiable/quantifiable protective measures it seems likely that regulators will be attracted to surveillance as a method of health risk management.

It may also be found to be appropriate to employ a system that combines the identification of new cases of competently diagnosed occupational injury with known risk factors. However, as yet there is no agreement on the definition of risk factors for DRSI that may fall properly within the control of an employer.
Risk factors for aches and pains include overwork, unaccustomed work, high force and extreme postures. Aches and pains are not injuries. Risk factors for progression from aches and pains to any specific ULD and that should properly fall within the duty of the employer are not specifically known.

A well-meaning employer might choose to respond to reports of aches and pains by reassessing risk factors like those listed above but if they do take this course of action, they should be aware of the risk of appearing to over-react. Such action may reinforce or create inappropriate beliefs that are conducive to the development of chronic pain syndrome. There is no Official guidance on the psychology of intervention.

Given these uncertainties and a trend towards a precautionary approach, it seems likely that Regulators will endeavour to ensure that aches and pains should be prevented and that they will use Health and Safety legislation to that end. I.e. they are likely to substitute diagnosable harm for aches and pains.

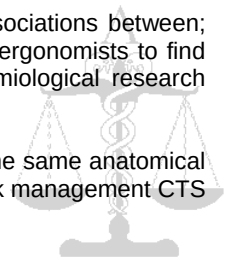
Risk factors for acute CTS are more clearly defined and include:

Posture
There is growing acceptance that work that requires extreme wrist flexion and extension should be avoided. However, intervention studies have yet to demonstrate the effects (good or bad) of reducing

flexion and extension. Epidemiological evidence for an association between wrist posture and risk of CTS is weak.

Repetition
There is growing acceptance of the need to reduce repetitive flexion/extension/force cycles at work. It is unlikely that a standard for repetition could be developed in isolation from other factors.

Force
Force on the fingers and hand is transmitted in some way by the wrist. Clear associations between; force (grip and load bearing) and, pressure in the carpal tunnel, have encouraged ergonomists to find suitable exposure standards. Such moves are supported by findings from epidemiological research which links force to symptoms.



If DRSI and CTS are to be considered 'harm of a kind' (since at least they occupy the same anatomical region and both involve pain to some degree) then the duty and standards for the risk management CTS may be argued by some to be relevant for DRSI. This remains to be seen.

Prognosis

The ultimate outcome for diagnosed cases of CTS varies with time of diagnosis and treatment. Early intervention (in the absence of systemic causes) should prove effective.

Prognostic indicators for chronicity are not established to the standard accepted by evidence based medical practice, but they are likely to include: pain severity and catastrophising.

By definition, a diagnosis of DRSI would be made (by a specialist) after several months. Chronic pain conditions generally have a small probability of spontaneous recovery within a year of diagnosis.

Rehabilitation -DRSI

Unremarkable aches, pains and fatigue, and other normal (but sometimes named) effects of excessive physical work or unaccustomed activities are usually self treated by a combination of pacing, rest, training, work adaptation and working through.

Moving up a level from self-care, by referral to some other intervention requires careful thought to be given to gate keeping. Unfortunately there does not appear to be a consensus on appropriate gate-keeping criteria nor is there a scientifically defined natural history of DRSI.

A reasonable gate-keeping criterion would be:

- the point where symptoms persist several hours after beginning to rest

and

- there is disturbed sleep attributed to unusual sensations in the affected region of the arm

and

- symptoms return as soon as activity begins again.

An intervention at this point should allow rapid recovery from the above symptoms and prevent degeneration into a chronic pain syndrome. Interventions should address psychosocial yellow flags, provide reassurance and avoid the development of pain behaviour. It may be appropriate to make temporary modifications to work; such modifications should be made on a consensual basis as far as is reasonable.

There is no agreement on the most effective treatment for chronic pain syndromes. What seems to be agreed is that up to now, treatment is usually ineffective. Various forms of cognitive behaviour therapy would seem to be consistent with the state-of-knowledge of chronic pain syndromes and their efficacy has some support from clinical research.