Whiplash associated disorders

Michele Sterling

INTRODUCTION

Whiplash associated disorders are common, disabling, and costly conditions that occur as a consequence of a motor vehicle crash (MVC). Recent data indicate that rapid improvement in levels of pain and disability occur in the first three months post injury with little if any change after this period and that the majority of injured people will not fully recover (Kamper et al 2008). The associated cost secondary to whiplash injury, including medical care, disability, lost work productivity, as well as personal costs is substantial (Crouch et al 2006, MAIC 2004).

Whiplash is a recalcitrant condition for some individuals. Management options for both the acute and chronic stages of whiplash are not straightforward and whilst offering some improvements in pain and disability are far from being a panacea. Trials of treatment for acute whiplash have not demonstrated efficacy in terms of decreasing the incidence of those who develop persistent symptoms (Provinciali et al 1996, Borchgrevink et al 1998, Rosenfeld et al 2000, 2003). Whilst these trials have provided evidence to show that maintenance of activity is superior to rest and prescription of a collar for most whiplash injured people (Scholten-Peeters et al 2002), significant numbers of patients still transition to chronicity. Further, trials of treatment for the chronic stage of the condition, including various exercise forms have offered only modest effects with only 10–20% of patients having a completely successful outcome, that is minimal or no disability at the 12-month follow-up (Jull et al 2007, Stewart et al 2007).

Recent research findings show that whiplash is a remarkably complex and heterogeneous condition and this heterogeneity may explain the modest effects of treatment strategies investigated to date. Most trials have previously investigated relatively non-specific approaches to treatment without targeting interventions toward specific physical or psychological characteristics of the condition. However recent investigations have begun to provide insight into the characteristics of the whiplash condition – both physical and psychological – that allows speculation on the potential underlying mechanisms.

This chapter will outline the whiplash condition and classification before discussing the burgeoning knowledge
of physical and psychological manifestations of the condition and the implications for clinical practice.

### THE WHIPLASH CONDITION

A motor vehicle crash can lead to bony or soft-tissue damage, which in turn may result in a variety of clinical manifestations called whiplash-associated disorders (WAD). The primary symptom is neck pain, although headache, arm pain, paraesthesia, dizziness and cognitive difficulties are also frequently reported (Spitzer et al 1995).

It is conceivable that virtually any cervical spine structure may sustain injury following whiplash. Bioengineering studies where cadavers were subjected to simulated rear end crashes have demonstrated perturbations in segmental movement including inter-segmental hyperextension, S-curve formation and differential acceleration of the upper cervical spine (Cusick et al 2001). This together with evidence from autopsy (Taylor & Taylor 1996) and animal studies (Winkelstein et al 2000) indicates that lesions may occur to cervical structures including bony elements, intervertebral discs and zygapophyseal joints, ligaments, muscles and nerve tissues. Unfortunately, in vivo identification of structural pathology has proved difficult probably due to the insensitivity of current radiological diagnostic imaging (Davis et al 1991, Ulhrenholt et al 2002), although recent studies begin to provide hope that this situation may change in the future for at least some structures (Krakenes & Kaale 2006). It is also generally accepted that in some persons with chronic whiplash, the zygapophyseal joint is a symptomatic structure, demonstrated by placebo controlled nerve blocks to these joints (Lord et al 1996).

Whilst it could be argued to be beneficial if specific structural lesion(s) could be identified in whiplash injured persons, it has more recently been promoted that the identification of the patho-anatomical source of symptoms provides little basis for appropriate management of musculoskeletal pain disorders and emphasis should instead be placed on treatment approaches directed toward mechanisms and processes underlying the painful condition (Jensen & Baron 2003). With respect to WAD, there is evidence that a variety of physical and psychological impairments characterize the condition.

### CLASSIFICATION OF WHIPLASH INJURY

Classification systems have been proposed in order to assist in the early assessment, prognosis and management of whiplash. The most commonly used classification system is the Quebec Task Force (QTF) system (Spitzer et al 1995). It broadly defines the condition into four groups as WAD I (neck complaint, without musculoskeletal signs); WAD II (with musculoskeletal signs); WAD III (with neurological deficits) and WAD IV (with a fracture or dislocation). While this system provides some necessary information related to condition classification, a major systematic flaw exists as the majority of whiplash injured people are grouped within one classification (WAD II), which falsely assumes homogeneity of the most common complaints within this group (Sterling 2004). The QTF Classification system has received some criticism in the past for the lack of scientific validation (Hartling et al 2001), failure to accommodate the heterogeneity of the WAD presentation (Sterling 2004) and its lack of prognostic capacity (Kivioja et al 2008).

To date, neither the QTF classification system for WAD nor trials investigating various management approaches for this condition have considered both the physical (biological) and psychological factors that are emerging as playing a role in the pain and disability of whiplash. It is becoming apparent that whiplash is a heterogeneous condition and more complex than previously assumed. Furthermore, it is emerging that whiplash is in some ways different from neck pain conditions of a non-traumatic nature. In particular chronic whiplash shows marked sensory features indicative of central nervous system hyperexcitability that have now consistently been shown not to be a feature of chronic non-traumatic neck pain (Chien et al 2010, Elliott et al 2008, Scott et al 2005).

### PHYSICAL AND PSYCHOLOGICAL CHARACTERISTICS OF THE WHIPLASH CONDITION

Historically much past research and certainly the clinical assessment of spinal pain conditions, including whiplash has aimed to identify the patho-anatomical source(s) of the patient’s reported symptoms. This approach has had limited success as a patho-anatomical diagnosis is not possible in the vast majority of patients with common musculoskeletal pain conditions nor does such a diagnosis necessarily shed light on the most optimal intervention for a specific condition or patient. As a consequence, the focus has shifted in recent years more towards attempting to identify the underlying mechanisms or processes of the patient’s pain syndrome (Max 2000). The purpose of this more specific diagnosis and classification of musculoskeletal pain syndromes is to help tailor interventions toward identifiable underlying processes to try to improve treatment success, particularly in some of the more recalcitrant conditions. Of all neck and upper quadrant conditions, there is arguably the most data for motor, sensory and psychological characteristics available for whiplash. One reason for this may be due to the easily defined onset of injury (MVC) versus a more insidious onset that often occurs with other conditions.
Motor and sensori-motor control dysfunction

One of the most common clinical characteristics of patients with WAD is that of movement loss or decreased cervical range of movement (Heikikila & Wenngren 1998, Dall’Alba et al 2001). Most prospective studies have shown that all whiplash injured subjects have a loss of cervical active range of movement from soon after injury (Radanov et al 1995, Kasch et al 2001b, Sterling et al 2004). Kasch et al (2001b) reported that restoration of movement loss occurred in all individuals by three months post-injury, irrespective of recovery or non-recovery. However if whiplash subjects are classified more precisely, it can be seen that those with persistent moderate/severe levels of pain and disability (measured with NDI) continue to display active movement loss several years post injury (Sterling et al 2006). In contrast, participants who had recovered or reported lesser (but still significant) pain and disability showed restoration of movement loss within 2–3 months of injury (Sterling et al 2003b), similar to that seen by Kasch et al (2001b). This demonstrates the importance of differentiating individuals with whiplash based on pain and disability levels.

Altered patterns of muscle recruitment in both the cervical spine and shoulder girdle regions have been clearly shown to be features of chronic WAD (Nederhand et al 2002, Jull et al 2004). Longitudinal data demonstrate that these changes are apparent from very soon after injury (Nederhand et al 2002, Sterling et al 2003b) with greater deficits in those reporting higher levels of pain and disability (Sterling et al 2003b). Sterling et al (2003b, 2006) observed that the disturbed motor patterns persisted, not only in those with ongoing chronic symptoms, but also in those with milder pain and disability and those who reported full recovery with this phenomena occurring at significant time periods post-injury – up to 2 years. These persisting deficits in muscle control may leave recovered individuals more vulnerable to future episodes of neck pain but this proposal needs to be substantiated with further investigation (Sterling et al 2006).

Altered patterns of muscle recruitment are not unique to whiplash and identical changes have also been observed in neck pain of insidious onset (idiopathic neck pain) (Nederhand et al 2002, Jull et al 2004, Woodhouse & Vasseljen 2008). These findings suggest that the driver of such motor changes may be more due to the nociceptive input rather than the injury mechanism itself.

Recent investigation, using MRI, has shown marked morphological changes to cervical spine muscles in people with chronic whiplash. Elliott et al (2006, 2010) demonstrated the presence of fatty infiltrate in both deep and superficial cervical extensor and flexor muscles in WAD patients compared to an asymptomatic control group. Although the fatty infiltrate was generally higher in all muscles investigated for the patient group, it was highest in the deeper muscles, the rectus capitis minor/major and multifidi (Elliott et al 2006). In contrast to muscle recruitment changes, preliminary data indicate that similar morphological changes are not apparent in individuals with chronic idiopathic neck pain (Elliott et al 2008). The relevance of these findings in terms of pain, disability or functional recovery and the cause of the muscle changes are not yet known but the findings illustrate the profound disturbances in motor function present in people with chronic WAD.

Dysfunction of sensori-motor control is also a feature of both acute and chronic WAD. Greater joint re-positioning errors have been found in patients with chronic WAD and also in those within weeks of their injury, and with moderate/severe pain and disability (Sterling et al 2003b, Treleaven et al 2003). Loss of balance and disturbed neck influenced eye movement control are present in chronic WAD (Treleaven et al 2005a,b) but their presence in the acute stage of the injury are yet to be determined. It is important to note that sensori-motor disturbances seem to be greater in patients who also report dizziness in association with their neck pain (Treleaven et al 2005a,b).

Most of the documented motor deficits (movement loss, altered muscle recruitment patterns) seem to be present in whiplash injured individuals irrespective of reported pain and disability levels and rate or level of recovery (Sterling et al 2003b). Additionally, apart from cervical movement loss, motor deficits do not appear to have predictive capacity (Sterling et al 2005). Further, treatment directed at rehabilitating motor dysfunction and improving general movement shows only modest effects on reported pain and disability levels (Jull et al 2007, Stewart et al 2007). Together these findings suggest that motor deficits, although present, may not play a key role in the development and maintenance of chronic or persistent symptoms following whiplash injury. However, this is not to say that management approaches directed at improving motor dysfunction should not be provided to patients with whiplash. Rather the identification of motor deficits alone may not equip the clinician with useful information to either gauge prognosis or potential responsiveness to physical interventions.

Augmented pain processing mechanisms in whiplash

There is now considerable and consistent evidence of sensory disturbances in WAD which indicate the presence of augmented central pain processing mechanisms. Changes include both sensory hypersensitivity (or decreased pain thresholds) to numerous stimuli such as pressure, thermal, electrical stimulation and light touch in both acute and chronic WAD (Curatolo et al 2001, Sterling et al 2003a, Raak & Wallin 2006). Sensory hypersensitivity is
found not only over the cervical spine (area of injury) but also at remote uninjured areas such as the upper and lower limbs (Koelbaek-Johansen et al 1999, Sterling et al 2003a). The absence of tissue damage at the site of testing suggests that central sensitization of nociceptive pathways is the cause of the pain hypersensitivity. Recently the presence of widespread hypoesthesia (that is, elevated detection thresholds) occurring concurrently with hypersensitivity has also been found in WAD and suggests disturbances in central inhibitory processes as well (Chien et al 2008a,b).

Hypersensitivity has been shown not only to be present in testing involving a cognitive response from the participant. Facilitated flexor withdrawal reflexes in the lower limbs of participants with chronic WAD have been demonstrated following electrical stimulation of the sural nerve (Banic et al 2004, Sterling et al 2008). In this test, reflex activity of the biceps femoris was measured and evidence of spinal cord hyper-excitability (central sensitization) was provided without relying on the subject’s self reported response to the stimuli, as is required with pain threshold testing. It has also been shown that the heightened reflex responses are not associated with psychological factors such as catastrophization and distress (Sterling et al 2008).

In contrast to the apparently uniform presence of motor dysfunction, sensory disturbances seem to differentiate whiplash from less severe neck pain conditions and whiplash sub-groupings with higher or lower levels of self-reported pain and disability. Individuals with chronic WAD manifest a more complex presentation involving lowered pain thresholds to pressure, heat and cold stimuli in areas remote to the cervical spine which are not present in those with idiopathic (non-traumatic) neck pain (Scott et al 2005, Elliott et al 2008, Chien et al 2010). Similarly widespread hypoesthesia to vibration, thermal and electrical stimulation, whilst present in WAD is not a feature of idiopathic neck pain (Chien et al 2010). However the presence of central hyper-excitability is not unique to whiplash with other painful musculoskeletal conditions such as fibromyalgia, tension-type headache and migraine also manifesting such signs (Yunus 2007). With respect to the cervical spine and upper quadrant, widespread sensory hypersensitivity is a feature of cervical radiculopathy with this condition and whiplash reporting similar pain and disability levels (Chien et al 2008b) (Figs 8.1 & 8.2). This suggests that chronic whiplash and chronic cervical radiculopathy share similar underlying mechanisms but differ from idiopathic neck pain illustrating the diversity of processes involved in various neck pain conditions.

The reason as to why some whiplash injured people develop a hypersensitive state is not clear but appears to be related to levels of pain and disability (Sterling et al 2003a). Numerous cervical spine structures are implicated as possible sources of noception following whiplash injury. It is possible that injuries to deep cervical structures do not rapidly heal and thus become a nociceptive ‘driver’ of central nervous system hyper-excitability. Whilst this proposal may meet opposition from those who believe injured soft tissues are healed within several weeks, it is gaining support as a possible contributor to the development of chronic musculoskeletal pain including whiplash (Curatolo et al 2006, Vierck 2006). There is also evidence from cadaver studies that certain lesions

Fig 8.1 Pressure pain thresholds (PPTs) (mean and standard deviation) at C5/6, upper and lower limbs of patients with idiopathic (non-traumatic) neck pain, whiplash associated disorders (WAD) and cervical radiculopathy compared to asymptomatic controls. The three neck pain groups showed decreased PPTs at the neck (C5/6) and upper limbs. The WAD and radiculopathy groups also showed widespread decreased PPTs in the lower limb (*p < 0.05).
can persist unresolved in MVC survivors who die of unrelated causes some years later (Taylor & Finch 1993). Additionally, the sensory hypersensitivity seen in whiplash is often also associated with other disturbances such as impaired sympathetic vasoconstriction (Sterling 2006) and stress related factors (Sterling & Kenardy 2006). The co-occurrence of these factors suggests that a complex interplay between various mechanisms may lead to this almost systemic response in some following whiplash injury. Research is now focusing on investigating such complex models which may in the future shed light on this intriguing issue (McLean et al 2005, Passatore & Roatta 2006, Sterling & Kenardy 2006).

Psychological factors in whiplash associated disorders

There is no doubt that chronic whiplash is associated with psychological distress including affective disturbances, anxiety, depression and behavioural abnormalities such as fear of movement (Williamson et al 2008). Psychological distress is also present in the acute post-injury stage with most people showing some distress regardless of symptom levels (Sterling et al 2003c). Data from some studies indicate that the ongoing psychological distress is associated with non-resolving pain and disability. A large cross-sectional study showed an association between anxiety, depression and pain and disability in people whose accidents occurred over two years previously, but not in those with acute injury, suggesting that symptom persistence is the trigger for psychological distress (Wenzel et al 2002). Longitudinal data indicate that initially elevated levels of distress decrease in those who recover, closely paralleling decreasing levels of pain and disability (Sterling et al 2003c).

Unique psychological factors may be involved in the etiology and development of chronic whiplash (Sterling et al 2003c) when compared to other painful musculoskeletal conditions. For example, the role of fear of movement beliefs seems to be a less important factor in whiplash (Sterling et al 2005) than in low back pain (Vlaeyen et al 1995). The role of coping styles or strategies in whiplash is unclear. Some data indicate that a palliative reaction (e.g. seeking palliative relief of symptoms such as distraction, smoking or drinking) was associated with longer symptom duration (Buitenhuis et al 2003, Carroll et al 2006). In contrast Kivioja et al (2005) found no evidence that different coping styles in the early stage of injury influenced the outcome at one year post-accident. The different cohort inception times of these studies may account for the differences in findings indicating that coping strategies may vary depending on the stage of the condition and this requires further investigation.

One factor that is likely unique to WAD (when compared to other common musculoskeletal conditions), due to the mode of onset being a traumatic event, is that of post-traumatic stress. Symptoms of post-traumatic stress have been shown to be present in a proportion of people following a whiplash injury due to a MVC (Drottning et al 1995, Sterling et al 2003c, Kongsted et al 2008) and these symptoms have shown prognostic capacity for poor functional recovery at six months and two years post-MVC (Sterling et al 2005, Buitenhuis et al 2006, Sterling et al 2006). These studies mostly utilized the Impact of Events Scale (IES) (Horowitz et al 1979), an instrument that measures distress associated with a specific event (in the case of whiplash a MVC). It should be noted that a diagnosis of post-traumatic stress disorder cannot be made from IES scores. However recent data utilizing a more robust tool, The Post-traumatic Stress Diagnostic Scale (Foa et al 1997) demonstrated that 22% of a
prospective sample of 155 whiplash injured people had a probable diagnosis of PTSD at three months post-MVC with this figure dropping slightly to 17% by 12 months post-injury (Sterling et al 2009). These findings indicate the need for further psychological evaluation of these patients (Forbes et al 2007) and clinicians should be aware of this factor in their assessment of whiplash injured people.

**THE PREDICTION OF OUTCOME FOLLOWING WHIPLASH INJURY**

The capacity to predict those at risk of poor recovery following whiplash injury is important because it may allow the institution of appropriate early interventions targeted at modifiable risk factors. This could potentially reduce the transition to chronicity in those individuals deemed at risk. Numerous factors have been investigated for their prognostic capability including: sociodemographic status; crash related variables; compensation and/or litigation, psychosocial and physical factors (Radanov et al 1995, Cassidy et al 2000, Kasch et al 2001a). However, recent systematic reviews of prospective cohort studies on whiplash found that only greater initial pain intensity and greater initial disability were the most consistent predictors of delayed functional recovery (Carroll et al 2008, Kamper et al 2008, Walton 2010). Other factors reported by individual systematic reviews include post-injury psychological factors such as coping strategies (Carroll et al 2008), less than post-secondary education, female gender, history of previous neck pain (Walton 2010) and symptoms of post-traumatic stress and poor self-efficacy (Williamson et al 2008). Whilst some of these factors such as pain intensity, psychological distress may be modifiable many of the others (age, education) are not. Furthermore when potentially modifiable factors of initial pain and disability levels are considered alone, whilst having high specificity had relatively low sensitivity to predict those with ongoing moderate to severe symptoms at six months post-accident (Sterling et al 2005). Furthermore measurement of pain and disability levels alone is unlikely to assist in the direction of secondary and tertiary management stages of this condition. Nonetheless it will be important for clinicians to obtain a measure of reported pain and disability (e.g. Neck Disability Index) in the assessment of the whiplash injured.

Additional prognostic factors have emerged but require replication and validation in further studies before they are included in systematic reviews. These include physical factors of decreased range of neck movement, cold hyperalgesia or cold intolerance and impaired sympathetic vasoconstriction (Kasch et al 2005, Sterling et al 2005, 2006). The psychological domain of post-traumatic stress symptoms is emerging as a dominant factor in poor outcome following whiplash injury (Buitenhuis et al 2006, Sterling et al 2006) with the latter study demonstrating a superior predictive capacity of this variable when compared to other psychological domains. Additional psychological factors such as high catastrophising, low self efficacy and palliative coping strategies have also been identified, in some studies, as potentially influencing recovery (Buitenhuis et al 2003, Hendricks et al 2005).

The role of the controversial issue of compensation related factors is inconclusive with some studies showing it has predictive capacity (Carroll et al 2008) and others reporting no predictive capacity of this factor (Scholten-Peeters et al 2003). A recent systematic meta-review outlined the limitations of research of the influence of injury compensation on health outcomes including the low quality of primary research papers in this area, the heterogeneous nature of compensation schemes studies and the lack of use of validated health outcome measures (Spearing & Connelly 2010). These authors could find only one systematic review that could be considered both internally and externally valid and based on this, their findings were: ‘that there is evidence of no association between fault-based injury compensation and poor health outcomes among people with whiplash’ (Spearing & Connelly 2010).

**IMPLICATIONS FOR ASSESSMENT OF WHIPLASH**

It is clear that the whiplash condition involves complexities between physiological and psychological factors. Whilst the presence of high initial levels of pain and/or disability are consistent predictors of poor outcome (Scholten-Peeters et al 2003), the additional presence of sensory hypersensitivity (particularly cold hyperalgesia) and also post-traumatic stress symptoms have been shown to substantially improve predictive capacity (Sterling et al 2005). These factors have also been shown to be associated with non-responsiveness to physical interventions (Jull et al 2007). The long-term functional status following whiplash may be established within a few months of injury with little further improvement after this time (Kamper et al 2008). This reiterates the important role clinicians play in the early post-injury stage and even towards the prevention of chronicity.

The patient assessment will need to include an adequate history such as previous history of neck pain and headache as well as the possible mechanism of injury. The patient should be screened for the presence of any ‘red flag’ condition (WAD IV – fracture or dislocation). Whilst accident related features have not been shown to be consistent prognostic indicators of outcome (Scholten-Peeters et al 2003), they have shown some
predictive capacity in certain studies (Sturzenegger et al 1995). Since pain and disability levels have been repeatedly shown to be a consistent indicator of prolonged recovery (Hendricks et al 2005), it is essential that a validated questionnaire, such as the NDI, is used in the initial assessment. Certain physical factors, such as loss of neck movement, cold hyperalgesia, are predictive of poor recovery and must also be carefully assessed for. With respect to whiplash injury the psychological factor of post-traumatic stress appears to be involved in the transition from the acute to chronic stages of the condition and clinicians may want to include a measure of post-traumatic stress symptoms (e.g. Impact of Events Scale) in their assessment of the whiplash injured patient.

Recent calls have been made to direct clinical examination toward the recognition and identification of mechanisms involved in the patient’s pain syndrome (Max 2000, Treede et al 2002). At present sensory examination such as that required to detect the variety of sensory disturbances outlined above is rarely performed and if it is performed, is usually limited to rudimentary assessment of muscle power, deep tendon reflexes and light touch sensation. More detailed assessment of sensory changes in neck pain patients is necessary. The first stage of this assessment would be thorough recording of the patient’s symptoms including the nature of pain. Although the usefulness of symptom classification as a way of clarifying pain mechanisms is debatable, it is a necessary part of the patient’s assessment (Jensen & Baron, 2003). In recent times questionnaires have been developed that aim to specifically identify neuropathic like pain (Bennett et al 2007). Using the S-LANSS questionnaire (Bennett et al 2005), it has been shown that 20% of an acute whiplash cohort likely have a predominantly neuropathic pain condition with certain items being particularly associated with higher levels of pain and disability (Sterling & Pedler 2009). These were ‘electric shock’ type pain that comes in bursts, burning pain in the neck and hyperalgesia to manual pressure. Inclusion of this questionnaire with particular attention to these items may be a useful addition to the clinical assessment of acute WAD.

Quantitative sensory testing can also be used. This could include the measurement of mechanical pain thresholds with pressure algometry (Fig. 8.3) and determination of the presence of allodynia with light tactile stimulation. Cold hyperalgesia is emerging as an important factor in both the prediction of outcome (Sterling et al 2005) and for gauging treatment responsiveness (Jull et al 2007). It is more difficult to measure clinically but options may include the use of thermorollers set at predetermined temperatures (Sterling 2008) or the time taken to reach pain threshold following the application of ice (Cathcart & Pritchard 2006). However, it has been shown that there is little relationship between S-LANSS items and cold pain threshold indicating that physical measures of sensory hyper-sensitivity will also need to be included in the assessment of acute whiplash (Sterling & Pedler 2009).

How it should be noted that whilst such sensory assessments can provide useful information, at present there is no consensus about the most appropriate method to use and what to compare findings with (Jensen & Baron 2003). The development of the most appropriate sensory examination of whiplash injured patients is at an early stage and moves toward further development into clinically valid and useful measures is of vital importance. Physiotherapists routinely assess cervical range of movement and this will remain a mainstay of assessment of whiplash due to the prognostic capacity of this measure. Assessment will also need to include muscle recruitment patterns of the cervical and shoulder girdle regions. Further, the assessment of sensori-motor control is relatively simple to undertake in the clinical situation and will be particularly important in whiplash injured patients who report dizziness associated with their neck pain. Readers are referred to Jull et al (2008) for a detailed account of how to undertake these assessments.

**IMPLICATIONS FOR MANAGEMENT OF WHIPLASH ASSOCIATED DISORDERS**

Clinical guidelines for the management of acute and chronic WAD promote education, assurance to the patient, the maintenance of activity levels, general and specific exercises, simple analgesics and encouragement of coping strategies (Scholten-Peeters et al 2002, MAA 2007, TRACsa 2008). Some recent clinical guidelines have
become more cognizant of the complex presentation of some individuals with whiplash and have attempted to include recommendations for the identification of factors such as sensory disturbance and psychological distress (MAA 2007).

Whilst the resumption of activity is almost universally recommended, the case for the avoidance of pain provocation may be an important aim of any physical intervention for patients demonstrating features of sensory hypersensitivity. The application of non-judicious physical mechanically stimulating treatments may serve to maintain and prolong this hypersensitivity and have a deleterious effect on the patient’s long-term outcome (Sterling & Kenardy 2008). It is not suggested that these patients avoid activity, as the maintenance of activity levels opposed to rest and use of a collar has been shown to be important in the management of acute whiplash (Rosenfeld et al 2003). Certainly studies investigating fibromyalgia syndrome, a condition also proposed to have disturbed central pain processing mechanisms, have shown that sensory hypersensitivity increases following some forms of exercise including isometric and sub-maximal aerobic exercise (Vierck 2006).

Possible treatment modalities for central hyperexcitability in pain of musculoskeletal origin are largely unexplored. Pharmacological management approaches have been suggested (Curatolo et al 2006) but effectiveness of this form of treatment is yet to be investigated in WAD. Furthermore the side-effects of some drug therapy approaches are daunting (Vierck 2006). A predominantly medication-based approach to management, particularly in the acute stage may not be feasible. An important unanswered question is whether specific physiotherapy interventions have the capacity to modulate central hyperexcitability in WAD.

Theoretically physical interventions such as TENS and acupuncture may be useful in modulating sensory hypersensitivity and these have not been specifically investigated in WAD. Few studies have investigated the effects of psychologically based interventions in this condition. However Blanchard et al (2003) found that psychological intervention directed toward post-traumatic stress disorder in chronic whiplash, whilst influencing post-traumatic stress symptoms, did not have an effect of pain levels in this group. This is not to suggest that exercise and movement approaches are not indicated for those with a more ‘complicated’ whiplash presentation. A combined specific exercise and manual therapy approach has demonstrated efficacy in the management cervicogenic headache (idiopathic neck pain) (Jull et al 2002) and this treatment approach has shown to decrease pain and disability in patients with chronic whiplash without the presence of mechanical and cold hyperalgesia (Jull et al 2007). It may be that such an approach has a greater role in these subgroups of neck pain. However it is apparent that it will be important to identify those patients with the presence of sensory hypersensitivity (and psychological distress, particularly post-traumatic stress) such that additional management can be provided.

CONCLUSIONS

Whiplash is a complex, heterogeneous intriguing condition involving both physical (motor and sensory) disturbances and psychological distress. It is also one of the most frustrating conditions for clinicians to manage. It would appear that the quest to better understand WAD has only just begun and the results from recent research efforts have paved the way for further directions for research. As new knowledge emerges, the clinical assessment of the condition will become more informed and this will translate to improved outcome for injured people.

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