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Whiplash Associated Disorders

Diploma Thesis

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Prague, 2008

1. Abstract

Title

Whiplash associated disorders

Aim of thesis

To present overviews of the whiplash phenomenon from different perspectives: epidemiological, biomechanical, biopsychosocial, and treatment.

Method

A comprehensive review

Results

There is little direct evidence for the lesion or lesions that cause whiplash (despite some promising indicators), except at the higher grades of injury. Because of this, and the apparent sensitivity of the incidence and prognosis of whiplash to non-clinical factors, explanations have been sought that lie outside the biomedical model (in which pain can be attributed to the presence of a lesion) and instead whiplash phenomena are being increasingly viewed from a biopsychosocial perspective. This perspective seeks to explain the etiology and prognosis of whiplash by encompassing biomechanical, biomedical, social and psychological factors.

Acute WAD is best treated with early physical activity and active treatments, rather than with passive treatments. For chronic WAD (i.e., cases where symptoms have persisted for more than six months), radiofrequency neurotomy is effective in cases where diagnostic blocks have indicated the presence of injury associated with the cervical zygapophyseal joints. The combination of cognitive behavioral therapy with physical therapy interventions has also been found to be effective.

Declaration:

I declare that the thesis presented below is of my own work and collaboration and where I have relied on other literature it is clearly stated according to the referencing rules.

Alexander Shaiman

I would like to thank to Doc. MUDr. František Helcl, CSc for his professional contributions and support in aiding me to complete this work.

PERMISSION:

I hereby give permission to fellow students to borrow my thesis and use as a guide for future study, and ask for proper citation of literature.

Name and Surname	Date	Note

2. Contents

1. Introduction

The condition known as whiplash injury emerged in the middle of the 20th century, coinciding with that of the motor car as the main means of transport in industrialized countries. As the number of cars per head of population has increased, so has the reported rate of whiplash injury.

Research on whiplash-associated disorders (WAD) is concerned with the incidence, treatment and prevention of the condition. Epidemiological studies seek to describe, firstly, the incidence of WAD and to unearth factors that indicate the population(s) at risk. Ultimately, the aim is to discover the causal relationships between various factors and the incidence of WAD.

There are many theories about the etiology of WAD and they range from the purely mechanistic, considering only the biomechanics of the crash event, to theories that place the WAD phenomenon solely in the realm of the psychology of secondary gain, where the patient has some interest in remaining "ill". If there are some "truths" about the incidence of WAD, then they will undoubtedly be found somewhere in the middle-ground between these views. The complexity of WAD (which is often grouped with other complex pain disorders), means that very little about the etiology is understood and, it seems, even less is agreed upon in the literature.

Whiplash associated disorder is the name given to a collection of symptoms including pain in the neck, head, shoulder and arms following a motor vehicle collision. In a collision there is a transfer of energy between the vehicles and occupants. Following this interchange there may or may not be injury. Bogduk (2003) sets out a series of stages and events in the natural history of the condition:

'The injury may or may not cause acute symptoms. Those symptoms may be contrived; they may be mild; or they may be serious. Symptoms invite diagnosis, but the techniques used to make a diagnosis may be valid or not valid. Once diagnosis is made, treatment follows. Those treatments may be ineffective or effective; but sometimes treatment may only seem to be effective, i.e., the patient recovers but not because of any specific effect of the treatment. Nevertheless, the end point of acute whiplash is that either the patient has recovered or has not.

For patients who do not recover, the cycle repeats. No recovery means that the patient has developed chronic symptoms. Those symptoms invite diagnosis that may or may not be valid.

Treatment follows, and may or may not be effective. The patient recovers or they do not. Those patients who do not recover may or may not become disaffected, and their chronic symptoms persist. They may be subjected to legal proceedings, which themselves may reinforce disaffection and chronicity.'

Traditionally, as with most health interventions, the treatment of whiplash associated disorders (WAD) has been based on the biomedical model. However, more recently there has been a move toward the use of a biopsychosocial model (e.g. Nederhand et al., 2003).

There is some controversy as to which model is the most appropriate. But with increasing evidence to suggest that other factors, besides crash-related factors, are important in determining outcomes as well as the use of multidisciplinary treatments, there appears to be increasing evidence in favour of the biopsychosocial model.

2. Aim of thesis

There is a large body of scientific and medical literature devoted to the study of whiplash and a significant proportion of this is in the form discussion, opinion, editorials and correspondence. A search of the science citation index yields over 1,100 articles published since 1980 on the topic of whiplash, covering vehicle factors, the biomechanics, pathophysiology, psycho-sociology, treatment, rehabilitation and compensation of whiplash associated disorders.

In the midst of all of this, it is often difficult to get an overview that encompasses all aspects of whiplash research.

In this thesis my aim has been:

- To provide an overview of WAD from different perspectives: epidemiological, biomechanical, biopsychosocial and therapeutic.
- To describe the incidence of WAD and to link the causal relationships between various factors and the incidence of WAD as epidemiological aspect.
- To explain the mechanism that cause WAD from biomechanical aspect.
- To illustrate biopsychosocial aspects of WAD.
- To present current evidence based management of WAD.

3. Hypotheses

- The incidence of WAD is influenced by psychosocial, socio-demographic and jurisdictional factors.
- Biomechanics explain much about the mechanism of whiplash injury, but not the extent and severity of WAD.
- The explanation of etiology and prognosis of whiplash is more perspective by encompassing biopsychosocial model than biomedical one.
- The evidence of treatment of acute and chronic WAD is controversial.

4. Definition and classification of whiplash

The definition formulated by the Quebec Task Force on whiplash-associated disorders has been used because this is, at present, the most commonly used definition:

"Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear-end or side-impact motor vehicle collisions, but can also occur during diving or other mishaps. The impact may result in bony or soft-tissue injuries (i.e., whiplash injury), that may, in turn, lead to a variety of clinical manifestations (i.e., whiplash-associated disorders) " (Spitzer et al., 1995)

The Quebec Task Force classified whiplash disorders (i.e., whiplash-associated disorder) on two axes: a clinical-anatomical axis and a time axis.

The clinical-anatomical axis has five grades of severity: from 0 to 4:

Grade	Description
0	no complaints, no physical signs
1	pain, stiffness and tenderness in the neck, but no physical signs
2	neck complaints and other musculoskeletal complaints (e.g., a decreased range of motion and tender spots
3	neck complaints and neurological signs (e.g., decreased or absent deep tendon reflexes, weakness, and sensory deficits)
4	neck complaints and fractures or dislocations

* Symptoms and disorders that can be manifested in all grades of severity include deafness, dizziness, tinnitus, headache, memory loss, dysphasia and temporomandibular pain.

The time axis has six phases:

phase 1covers the period up to four days after the whiplash;

phase 2 lasts from four days to three weeks after;

phase 3 from three to six weeks;

phase 4 from six weeks to three months;

phase 5 from three to six months;

phase 6 covers the period more than six months (chronic).

This classification is based on the physiological tissue recovery process.

The implication is that the Classification is an ordered categorical scale with each successive grade severer than the previous one (Spitzer et al., 1995, suggest that the Classification corresponds "roughly" to severity). However, within each classification,there is no provision for including any assessment of the severity of the symptom, so that someone suffering from intense pain with no other signs, might be assessed as WAD-I along side an individual with only mild pain.

Although the Québec Task Force proposed a second axis to deal with the duration of symptoms of WAD, time-to-recovery is often used as an independent outcome measure,and some research articles imply that, by definition, a useful scale that grades injury/symptomology on presentation should predict the course of the injury.

Hartling et al. (2001) evaluated the utility of the Québec Classification of WAD in correctly predicting the prognosis of patients as measured by the probability of still having WAD at 6, 12, 18 and 24 months. They conclude that the Classification system is useful in the clinical setting despite somewhat inconclusive statistics. While the odds of still having WAD appeared to increase with the Grade on presentation, the relationship was weak and it is hard to envisage the clinical utility of the Classification in predicting time-to-recovery on the basis their findings. They did, however, find that physical range-of-motion of the neck might be useful as a prognostic tool.

Suissa et al. (2001) produced a better estimate of the utility of the Québec Classification of WAD in a retrospective review of 2843 claimants from the Société de l'assuranceautomobile du Québec. The study examined the prognostic value of the initial signs and symptoms and then reclassified individuals according to the Québec Classification of WAD. Groups of individuals classified as having Grades II or III WAD had different rates of recovery from those having Grade I WAD. However, the authors found many better prognostic factors with which to classify people presenting with a whiplash injury .

4.1 Chronic whiplash injury ("late whiplash syndrome")

Chronic whiplash or "late whiplash syndrome" includes the collection of symptoms and signs that exist in a patient beyond a period in which recovery might normally be expected. These symptoms include headache, radicular deficit, cranial nerve/brainstem disturbance, cervical spine osteoarthritis, fatigue, anxiety, sleep disturbances, blurred vision, forgetfulness, illness/disability worry, and stress (Radanov et al., 1995). The transition of a minority of cases

of whiplash from an acute phase to a chronic phase is an important phenomenon that may depend on many factors, of which the initial injury is probably but one.

However, the length of time since the crash that should be used to indicate chronic whiplash injury is inconsistently defined. The Quebec Task Force nominated 6 months post-crash as defining the transition from acute to chronic injury (Spitzer et al.,1995) although one similar review used 8 weeks post crash (Ro et al., 2000).

5. Epidemiology

Epidemiological data on the incidence of whiplash are mainly derived from insurance claim numbers. Therefore, the reported annual incidence of whiplash varies widely between countries and continents: figures vary from 16 per 100,000 inhabitants each year in New Zealand to 70 per 100,000 inhabitants each year in Quebec, Canada. In the Netherlands, the number of new patients who have experienced whiplash is estimated to be 94–188 per 100,000 inhabitants each year. These figures are much higher than international estimates because they are derived from accident statistics.

The true incidence of whiplash is difficult to determine, as routine data may not adequately characterize or capture all cases of WAD. However it is possible to say that the incidence of whiplash disorders in South Australia in 2001 was greater than 300 per 100,000 populations; in 2001 approximately 4000 claims were lodged for compensation for whiplash injury with the Motor Accident Commission of South Australia (Gun et al., 2005).

This claim rate is very similar to that reported by Cassidy et al. (2000) following a change from a tort to a no-fault insurance system in Saskatchewan, Canada, even though a tort system applies in South Australia. In contrast, in New South Wales, the most recent and readily available data suggests that in that State, the claim incidence rate is around 100 per 100,000 populations per year. It should be borne in mind that these differences are just more likely to arise from differences in compensation criteria and coding than from any real underlying phenomenon.

6. Prognosis

There is no consensus in the literature on the prognosis of the consequences of whiplash. The prevalence of long-term complaints (i.e., from six months to two years) varies from 19–60%. A Canadian research group, the Quebec Task Force on whiplash-associated disorders (QTF-WAD), reported that the prognosis is favorable: around 85% of patients return to work within six months after the whiplash injury. Recently, this conclusion has been criticized because the severity and duration of the complaints may have been underestimated.

Spitzer et al. (1995) reported findings from a "best evidence synthesis" of whiplash research by the Quebec Task Force. The review covered all aspects of whiplash, including road safety, vehicle design, through to the clinical spectrum of whiplash-associated disorders. However, their findings on the epidemiology of whiplash were extremely limited.

At the time of the publication, they could only identify one population based study on the frequency of WAD, Lovsund et al. (1988), and this study did not allow the calculation any relative risks for the development of WAD. Regarding the prognosis of WAD, Spitzer et al. found no evidence for differences that could be attributed to sex, education, injury mechanism, collision fault, and time from injury to initial study examination. Furthermore, studies up to 1995 had not shown an association between compensation and legal action on the prognosis of injury. Similarly, the effect on prognosis of psychological factors was ambiguous and relevant studies lacked sufficient statistical power to make firm conclusions.

Since the report of the Québec Task Force on Whiplash-Associated Disorders (Spitzer et al.1995), there have been two systematic reviews on the prognosis of WAD: Côté et al. (2001a) and Scholten-Peeters et al. (2003).

Côté et al. (2001a) updated and extended the review of research articles by Spitzer et al. (1995), prompted in part by criticism of the methodology of the Quebec Task Force review, specifically by Freeman et al., (1998) and Teasell and Merskey (1999). Côté propose a conceptual model with which to categorize and systematically review the literature on the prognosis of acute whiplash injury.

This model categorizes studies on three axes, the first being the "Target Population and Generalisability" of the study. On this axis, a study can be categorized according to whether the subjects recruited were from a hospital emergency room, from a primary care setting, from an insurance cohort, or from the entire population, the inference being that the evidence

produced in the study is largely confined to the population from which subjects were recruited.

The second axis categorized studies according to "Phases of investigation": i.e., (in ascending order of the strength of evidence) descriptive statistics, exploratory studies, or explanatory studies, the last involving hypothesis testing on the incidence of some outcome (such as chronic whiplash disorder, chronic pain, time-to-claim settlement).

The third axis categorized each study according to whether the "Article met quality cut-point" criteria.

Côté et al. (2001a) found 13 studies since 1995 of sufficient methodological quality (positive on the third categorical axis), and 5 of these were conducted in a manner that allowed the results to be applied to the general population, although only one was conducted to test a specific hypothesis (that the system of insurance affected cessation of symptoms as defined by the closure of the claim).

The main findings of this review were that risk factors for chronicity of symptoms include older age, female sex, baseline pain and intensity of headache and baseline radicular signs and symptoms. They found that insurance and compensation systems affected the prognosis of whiplash.

Scholten-Peeters et al. (2003) conducted a review similar to that of Côté et al. (2001a). However, there were subtle but important differences in their approach which meant that they identified some studies not found by Côté, included some studies rejected by Côté and, further, they placed a different weight on the evidence produced by studies accepted by both reviews.

Scholten-Peeters sought reports on the prognosis of cohorts of subjects who were suffering acute whiplash associated disorders, and so gave less weight to (or did not consider) cohorts that recruited based on the subject having been involved in a crash rather than the subject having experienced a whiplash injury (Scholten-Peeters, 2005).

Rather than using the conceptual model suggested by Côté, Scholten-Peeters based their strength of evidence on the consistency of findings between cohorts. Studies showing strong risk ratios (>2.0) were considered as showing evidence, irrespective of the statistical significance of the risk ratio. If calculated risk ratios were less than two, this was counted as evidence against an effect (Scholten-Peeters, 2005), on the basis that this indicated that the risk factor was of no clinical relevance (the motivation for their review).

Like Côté, Scholten-Peeters applied a systematic ranking protocol to various cohort studies although the criteria were somewhat different in the two studies. Most significantly, Scholten-

Peeters only included studies that were prospective in design and that studied an inception cohort (i.e. individuals in the cohort were recruited soon after the whiplash event and the time of recruitment was consistent between individuals in the cohort.)

Scholten-Peeters's systematic review of prospective cohort studies found that only high initial pain intensity was an important predictor for delayed functional recovery with no strong or consistent evidence for factors such as age, sex and compensation factors. However a later study by the same group found that female sex, a low level of education, high initial neck pain more severe disability, higher levels of somatisation and sleep difficulties were predictive of poor outcome

6.1 Normal and delayed recovery

A distinction is made between patients who undergo normal recovery and those who undergo delayed recovery after whiplash injury. Normal recovery refers to the 'average' or 'expected' course of recovery from the consequences of whiplash. Normally, over time the patient's functions improve, the patient's levels of activity and participation increase, and the patient's pain level declines. Moreover, there is some interrelationship between impairments, disabilities and participation problems.

When recovery is delayed, it may be that the patient's functions do not improve or the patient's levels of activity and participation do not increase or the patient's pain level does not decline with time. Moreover, the interrelationship between impairments, disabilities and participation problems is less obvious. Recovery can be said to be delayed if a patient suffering the consequences of whiplash shows no progress in terms of levels of activity and participation within four weeks.

With normal recovery, activity and participation levels increase over time. This is not the case with delayed recovery.

6.2 Risk factors in the etiology and prognosis of whiplash

6.2.1 Crash factors

McLean (1974) showed that neck injury severity is associated with crash severity in rear-end collisions. Compared to drivers of vehicles with minor damage, drivers of vehicles that had severe damage had twice the odds of a neck injury complaint when assessed one week after the crash.

Krafft et al. (2000) examined the incidence of long-term disability from a whiplash injury, assessed 3 – 5 years after a rear-end collision, and found that occupants with a long-term disability were more likely to have been occupants in a car equipped with a tow bar at the time of the crash. Crash testing revealed that cars fitted with a tow bar generated higher accelerations than those without.

Later, Krafft et al., (2002) reviewed 66 rear impact crashes in which crash pulse recorders fitted to the car had measured the vehicle acceleration in the crash. While the crash pulse magnitude appeared to have some limited correlation with the duration of symptoms, and accounted for some of the variation in the duration of symptoms between cases, the pulse could not distinguish the grade of whiplash, according to the Quebec Task Force Classification. Furthermore, the direct measurement of delta-v (the change in velocity induced by the crash) bore no relationship to the duration of symptoms.

In an Adelaide study in which the crash severity was estimated in individual cases, no association between delayed recovery and crash severity could be discerned (Ryan et al., 1994). In that study, the only variable collected that was predictive of delayed outcome was lack of awareness of the impending collision. The effect of this factor on recovery was substantial (odds ratio 15.0). This study was based on persons seeking treatment for whiplash injury, not on all rear-end crashes.

Many of the vehicle interventions designed to minimize whiplash have focused on seat design. The aim has been to improve the geometry of the seat and head restraint and the response of the seat to rear impact loading. There has been some success in reducing the incidence of whiplash with "active" restraints that work by minimizing harmful motions of the neck (Farmer et al., 2003).

6.2.2 Initial signs and symptoms

Several recent studies have supported the notion that initial pain and symptoms are predictive of chronic WAD. Brison et al., (2000) found that the risk ratio was 3.3 for a continuing complaint at 6 months post injury for those reporting initial symptoms, compared with those having no initial complaint.

Berglund et al. (2000) studied a cohort of drivers in Sweden that had been involved in a rear-end collision. The cohort was followed-up 7 years after the collision to record their health status at that time. The authors compared those exposed to a rear-end crash with a whiplash injury to a matched control group who had not been exposed to a crash at all.

Similarly, those exposed to a rear-end crash without sustaining a whiplash injury were compared to a matched control group of non-exposed drivers, thus accounting for levels of pain in the general population. The results of the analysis showed that those drivers who initially reported a soft-tissue whiplash injury to the insurance company immediately after the crash were 2.7 times more likely to have neck pain after 7 years than those exposed to a rear-end crash and who did not initially report any symptoms. Furthermore, their general health was poorer 7 years after the crash (Berglund et al., 2001)

Suissa et al. (2001) examined the prognostic value of initial signs and symptoms in a cohort of insurance claimants in Québec. Factors predictive of delayed recovery were neck pain on palpation, muscle pain, pain or numbness radiating from the neck to the upper extremities and headache.

The systematic reviews of Côté and Scholten-Peeters's approach consensus on the prognostic value of initial pain. Côté found that baseline neck pain, headache and radicular signs and symptoms were predictive of delayed recovery in whiplash patients. Scholten-Peeters's also concluded that baseline pain was predictive of delayed recovery. However Scholten-Peeters's concluded that there was little prognostic value in initial headache and radicular signs.

6.2.3 Previous history of neck pain

Some writers have attempted to place whiplash disorders in the context of the prevalence of generalized pain and, more specifically, neck pain in the general community. White et al.,(1999) contend that demographic risk factors for generalized pain include being female, being aged in the forties and fifties, having relatively low income and educational status and being divorced or separated.

Bovim et al., (1994) used a randomized cross-sectional questionnaire to determine the prevalence of neck pain in the general population of Norwegian adults. The responses revealed neck pain to be common in the general population, with nearly 14 percent describing an episode of neck pain that had lasted more than 6 months in the year prior to the survey. The authors of the study argued that results of whiplash studies that show similar levels of chronic pain need to be seen in context of this background prevalence of pain.

Linton (2000) systematically reviewed literature published between 1967 and 1998 on psychological factors and back/neck pain. On the basis of 37 articles that met the inclusion criteria, a link between psychological factors and back pain was clear. However, there was no indication that there was a personality type that was "pain prone". Of interest was the conclusion that "Psychosocial variables are clearly linked to the transition from acute to chronic pain disability", supporting the use of a biopsychosocial model of back and neck pain. Linton notes that Radanov (Radanov, 1994a and 1994b) found no association between personality factors or psychoneurologic cognitive functioning of whiplash patients and the patients' prognosis. But in the study of general back and neck pain (not restricted to studies of WAD), this finding stands in some contrast to the bulk of the literature. It should be noted that in Linton's review, Radanov's et al. study was the only one to explicitly recruit whiplash patients. Nevertheless studies such as Bovim et al. (1994) and those cited in Linton (2000) underscore the lack of clear etiological pathways in chronic whiplash patients that plagues the research literature on whiplash associated disorders.

Similar methodological problems were encountered by Côté et al. (2000), who found that clear associations between headache, neck pain and general health and a neck injury sustained in a motor vehicle accident sometime in the past. In a cross-sectional study of Canadians, those people who reported an episode of neck pain (of any severity) or headache were more likely to report a history of neck injury. However, the design of the study did not allow causal links between the injury and the symptomology to be concluded.

6.2.4 Socio-demographic factors

There is some disagreement on the role of socio-demographic factors being predictive of the incidence and prognosis of WAD. In a study conducted in Adelaide, female sex was predictive of the incidence of whiplash injury (Dolinis, 1997) and a later study in Adelaide that used insurance records (Gun et al., 2005) also noted that more than half of claimants for whiplash injury were female. Dolinis did not, however, find any evidence that other socio-demographic factors such as age, occupation and educational attainment, were predictive of the incidence of whiplash injury. Côté concluded that being female and older age was associated with delayed recovery from whiplash, based on the results of two Canadian population-based studies (Harder et al., 1998; Cassidy et al., 2000) and a Japanese study (Satoh et al., 1997). McLean (1974) found a strong association between female sex and the incidence of whiplash injury in rear-collisions.

Harder et al. (1998) analyzed data on individuals who had made a claim for whiplash in the Province of Québec, Canada. They assumed that the proportion of the cohort that had recovered at a particular time could be described by an exponential function; that is, the rate of recovery was proportional to the number of individuals who had not yet recovered. They then created a model that described the influence of a number of factors on the recovery time. The factors contributing to delayed recovery were a mixture of crash, injury and socio-demographic factors: Additional injuries besides whiplash, female sex, older age, number of dependents, being involved in a severe crash, in a vehicle that was not a car, and not wearing a seatbelt were all predictive of delayed recovery. Interestingly, socio-demographic factors were more influential on recovery rate when whiplash was the only injury. Where there were other injuries, crash factors were more influential on recovery rate.

Scholten-Peeters found that the majority of cohort studies refuted the notion that the prognosis of WAD is predicted by age and sex. But it should be borne in mind that Scholten-Peeters defined positive evidence as a risk ratio of at least 2.0 and the restriction of their review to inception (rather than historical) cohorts meant that they did not consider the evidence produced by Harder et al. (1998). Some of the authors of Scholten-Peeters review have recently co-authored an original research report on the prognostic factors for poor recovery in a group of acute whiplash patients (Hendriks,et al., 2005). While the Scholten-Peeters review refuted the influence of age and sex on the prognosis of WAD, Hendriks et al. (2005) found that poor recovery was indeed related to female sex, a low level of education, high initial neck pain, more severe disability, higher levels of somatisation and sleep

difficulties. They note that their findings are consistent with Côté, but not with Scholten-Peeters.

Satoh et al. (1997) studied a cohort of 6,167 subjects in Japan who were involved in a rear-end crash and reported the accident to an insurance company, reported symptoms associated with WAD but did not have any skeletal fractures or open wounds, nor had they lost consciousness. Six months after the accident, 11.1% of the cohort was still receiving treatment. Multiple logistic regressions showed that females were more likely to still be receiving treatment after 6 months (odds ratio 1.43). Consistent with other findings, lacks of immediate symptoms also were significant prognostic factors for recovery within 6 months.

One of the studies reviewed by Scholten-Peeters (Brison et al., 2000) was of a cohort of 380 consecutive patients seeking treatment at one of two emergency departments in Kingston, Ontario, after a rear-end crash. The apparent incidence of whiplash in the population served by the emergency departments was 50 per 100,000 males per year and 110 per 100,000 females per year; females being 2.2 times more likely to seek treatment for a whiplash injury.

However, for those reporting a whiplash injury, sex was not reliably predictive of the prognosis of the injury. Older age was predictive of delayed recovery.

More recently, Berglund et al. (2003) showed that female sex was associated with a 1.2 times increase in having a whiplash injury following a motor vehicle crash and, contrary to the findings of Côté; younger age groups were more likely to have a whiplash injury.

It may be that socio-demographic factors are related to neck pain in the general population and so the same factors appear to relate to the incidence of WAD. Croft et al. (2001) conducted a multivariate analysis of results from a cross-sectional follow-up survey, in which participants who were pain-free at the inception of the study were followed up after 12 months, to ask if they had had any episodes of neck pain in the intervening period. They found that a neck injury in the past was, in itself, a risk factor for episodic neck pain, beyond social, demographic and health factors. However, factors such as marital status, the number of children, a history of lower back pain and self-assessed poor health were also predictive of an episode of neck pain in the study period.

6.2.5 Psychosocial factors

Generalizing, there would seem to be two 'camps' among researchers in the WAD field. The first of these camps can be described as being „biomedical" in its outlook and the second "biopsychosocial" in outlook. The biopsychosocial model of health was introduced by George L. Engel (Engel, 1980) to counter what he saw was a reductionist approach in the prevailing biomedical approach to medicine and psychiatry which he considered to be unhelpfully dualistic; i.e. it separated an intangible mind from the physical body (McLaren, 1998). As applied to the whiplash debate, the biomedical camp tends to view WAD patients as suffering psychological distress due to the chronic nature of a whiplash lesion, and the biopsychosocial camp view WAD chronicity as a complex interaction of biological, psychological and social factors, such that the transition from acute injury to a chronic complaint is mediated through psychological and social variables. It is common to see instances of these modes of thinking coming into conflict in the whiplash literature. For example, in a paper on the psychological profiles of patients with whiplash associated headache, Wallis et al. (1998) conclude that the differences in psychological profile between whiplash patients suffering headache, and those suffering headache of a non-traumatic origin "imply a different biological basis for these headaches and their associated psychological distress." In response, the authors were criticized for needlessly relying on a "dualistic" and "linear" view of whiplash (Kwan et al, 1998) and for restricting the interpretation of the data to preclude a biopsychosocial explanation.

Several epidemiological studies have examined the role of psychological factors in the outcome of acute whiplash. Mayou et al, (1996) followed a cohort of 63 people who had been in a motor vehicle accident, and presented with neck pain at one accident and emergency department in the UK. They followed the participants up after 3 months and 12 months. They concluded that the persistence of symptoms associated with WAD was not predicted by psychological measures made on initial presentation. However, these initial psychological measures were predictive of later social impairment, so that those who rated highly in scores that measure neuroticism and those who had a history of psychological complaints were more likely to have a poor "social outcome" after whiplash. The persistence of symptoms was predicted by the intensity of the pain upon initial presentation.

Mayou et al, (1996) concludes that the hypothesis that psychological factors are important etiologically for chronic physical complaints is not supported by their study; however,

psychological problems, when co-morbid with neck pain, reduce the capacity of people to return to normal levels of social functioning.

Scholten-Peeters concluded that psychosocial factors and neuropsychological factors were of limited prognostic value in cases of whiplash injury. However, in a recent publication (Hendriks et al., 2005) the authors of Scholten-Peeters report that psychological factors affected short and long-term recovery from whiplash injury. This was one result from their study of 125 patients who had been referred by a doctor for the treatment of a whiplash injury in the Netherlands.

Reported levels of depression, before the injury, were not predictive of chronic complaints and were similar to the Dutch general population. Yet, certain psychological factors such as sleep difficulties and somatisation (perceptions of bodily dysfunction) were predictive of lack of recovery 4 weeks after the crash and 52 weeks after the crash. The authors note that the data do not rule out the possibility that these factors are a consequence of pain rather than a component of the etiology of the pain.

6.2.6 Compensation factors

Of all factors that have been proposed to explain the prognosis of whiplash-associated disorders, the most controversial is the effect of compensation and lawsuit on outcome.

Côté (2001a) reaffirmed the findings of the Quebec Task Force (Spitzer et al., 1995) that insurance systems across jurisdictions affect the prognosis of the injury in the population. Cassidy et al., (2000) examined the effect of the change in insurance system in Saskatchewan Province in Canada. In 1995 Saskatchewan moved from a tort system to a no-fault system and no longer compensated injured car occupants for pain and suffering. There was a decrease in the number of claims and the duration of the claim period: the median time to the closure of related insurance claims was 433 days and 200 days under the old and new insurance systems. The results were such that the authors could conclude that the elimination of compensation for pain and suffering was associated with fewer claims for compensation and an improved prognosis in those who did claim.

Time-to-claim-closure is sometimes used to define recovery in studies of whiplash. Scholten-Peeters , amongst others, have criticized the study of Cassidy et al., (2000) in which time-to-claim-closure was used to indicate recovery, despite the author's claim of a correlation between these two factors. However, Côté et al., (2001b) studied the association between pain, physical functioning and depressive symptomatology with claim-closure in Saskatchewan, Canada, motivated by the use of time-to-claim-closure in many studies

(including Cassidy et al.) as a proxy for recovery. They found that a host of clinical measures of pain, physical functioning and depression were all significant predictors of claim-closure, providing some validation that claim-closure is related to the alleviation of the symptomatology of whiplash associated disorders. The clinical factors appeared to bear the same relationship to time-to-claim-closure under different systems of insurance and compensation.

Busse et al. (2004) studied a retrospective file series of 33 whiplash patients attending a single chiropractor in British Columbia, Canada. Using the self-assessment of disability due to their neck injury (the neck disability index, or NDI; Vernon et al, (1991)), the authors analyzed the files to determine the relative effects of non-injury related factors on the self-assessment of the NDI. Both female sex and retention of a lawyer was associated with an increase in the NDI, although the authors caution that the results do not demonstrate a causal relationship.

Two studies conducted in Lithuania are often used to argue that cultural expectations, and factors related to compensation and litigation, affect the reporting and treatment of whiplash symptoms. In the second of these Obelieniene et al. (1999) studied 210 victims of rear-end crashes in Kaunas, Lithuania. By comparing these cases with a matched group of randomly selected members of the general population, they found that the frequency and intensity of neck pain experienced by accident victims 12 months after the crash was indistinguishable from that experienced in the general population. Forty-seven percent of crash victims experienced acute whiplash pain and the mean duration of the pain was 3 days, and the longest duration of pain was 17 days.

The study may have been prompted, in part, by criticisms of the authors' first study on crash involved occupants in Lithuania (Schrader et al., 1996). This was a study of 202 individuals who had been in rear-ending traffic accidents. The participants were sent questionnaires in which they were asked to describe any neck pain, headache, low back pain and other symptoms. Follow up questionnaires were used to measure the type and frequency of pain, and the extent to which the complaints were disabling, and to get information on the circumstances of the crash. No differences were detected between the participants and a control group and the authors concluded that no person in the study had chronic symptoms attributable to the crash and that "Expectation of disability, a family history, and attribution of pre-existing symptoms to the trauma may be more important determinants for the evolution of the late whiplash syndrome" even though there is no actual data in their study to support the last of these statements. The study design was criticized on several grounds including the

probable inability of the sample size to detect a surplus of complaints due to chronic WAD because the cohort was not composed of those suffering from the acute injury (Freeman et al, 1996).

The Lithuanian studies underscore the lack of consistent findings on the course of whiplash injury in different communities, strongly suggesting that social factors, including the treatment and compensation of WAD, are likely to affect the course of the acute pain and injury to the neck.

In South Australia, too, there is some evidence of an association between aspects of compensation and recovery. There is a study of claimants who had suffered a whiplash injury. Even allowing for initial pain, consulting a lawyer was found to prolong treatment, time-to-claim-closure and physical functioning. The cautious conclusion is that the pursuit of compensation through a lawyer adversely affected the prognosis of the injury.

In contrast, Scholten-Peeters found only limited evidence for a compensation effect and concluded that evidence for a compensation effect was very weak and, overall, the evidence was more positive in refuting such a link. However, Scholten-Peeters do not include the findings from Saskatchewan (Cassidy et al., 2000) because the outcome measure (time-to-claim-closure) was not of direct clinical relevance. This was despite an established link between clinical signs and time-to-claim-closure.

6.2.7 Jurisdictional factors

Côté (2001) found conflicting evidence on the course of acute whiplash injuries, with the duration of symptoms reportedly varying between countries and jurisdictions in the same country.

Several factors make comparisons between countries difficult: for example, outcome measures will vary from study to study; a study may use a convenient measure that is difficult to compare to other studies: e.g. claim closure, low frequency of episodic pain, ceasing treatment etc. So in Lithuania, the median duration of neck pain was 3 days (Obelieniene et al., 1999), in Japan the median length of treatment was approximately 1.5months (Satoh et al., 1997), in Saskatchewan the median time to the closure of related insurance claims was 433 days and 200 days under two different insurance systems (Cassidy et al., 2000) and in Québec the median time to claim-closure was 31 days (Harder et al., 1998).

Notwithstanding the difficulties in comparing recovery rates in different jurisdictions, the differences in duration of symptoms and/or treatment in different countries is often cited in support of a strong psychosocial component in the etiology of chronic WAD.

7. Biomechanics of whiplash injury

7.1 Classical anatomy of the neck

The cervical spine is the upper section of the spine that supports the head and protects the spinal cord. Its articulation allows the head to move relative to the torso. The four basic motions of the head and neck are *flexion* (forward bending), *extension* (rearward bending), *lateral flexion* (sideward bending), and *axial rotation*. The bones of the neck are the seven cervical vertebrae identified as C1 to C7 (superior to inferior); these are shown in Figure 1.
The upper cervical spine consists of the *occiput*, the base of the skull commonly abbreviated to OC or C0, the *atlas* (C1), and the *axis* (C2). The occiput articulates with the atlas through the occipital condyles. The atlas has no vertebral body but consists of a bony ring with anterior and posterior arches on which the articular facets and transverse processes are located. The axis is similar in structure to the lower vertebrae, but has an additional element known as the odontoid process or *dens*, which protrudes upward from the body and acts asa pivot about which the head and atlas rotate (see Figure 1).

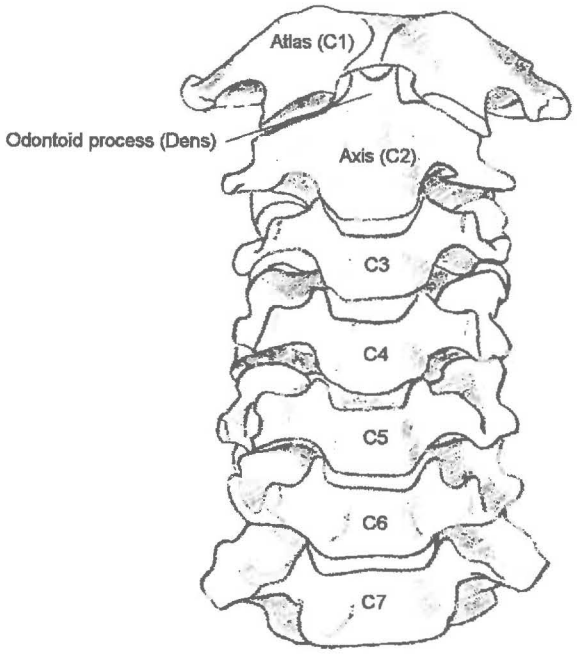

Figure .1 Anterior view of the cervical spine showing the odontoid process (dens) of the axis
Adopted from F. Netter, 1997.

The vertebrae of the lower cervical spine (C3 to C7) each consist of a cylindrical body and an arch (Figure 2). The lower end of the body (lower endplate) is concave from front to back, whereas the upper endplate is concave from side to side. The arch includes two pairs of articular facets, a spinous process and two transverse processes. The articular facets are almost flat, covered with articular cartilage and have a backward inclination of about 45° in the horizontal plane. The transverse and spinous processes are attachment points for muscles and ligaments. The arch and body enclose the vertebral foramen, which forms the spinal canal through which the spinal cord and associated structures run.

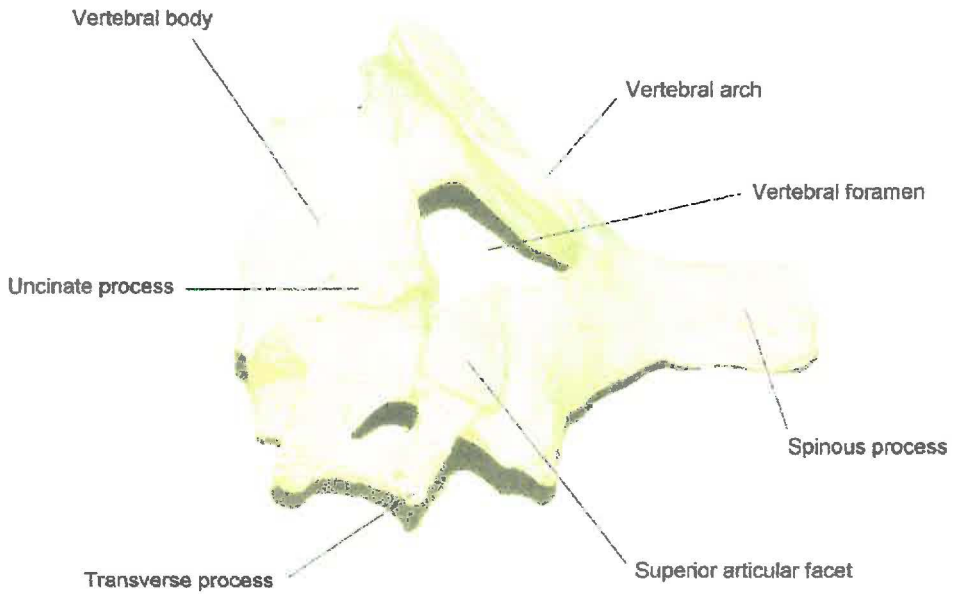

Figure .2 View of the C6 vertebra with the main sections indicated
Adopted from F. Netter, 1997

The soft-tissue linkage between two adjacent vertebrae is formed from the intervertebral disc, the facet joints and the uncovertebral joints. The disc permits motion in all directions while the uncovertebral and facet joints guide and constrain motion.

Intervertebral discs are fibrocartilaginous pads, which join adjacent vertebral endplates. Cervical discs are thicker anteriorly, giving the cervical spine a distinct curve in the sagittal plane known as the cervical lordosis. The uncovertebral joints are small synovial joints, linking the uncinate processes of the lower vertebra to the lower endplate of the upper vertebra, on either side of the disc. The facet capsular joints (FC, or zygapophysial joints) are

synovial joints formed by the corresponding articular facets of adjacent vertebrae, and are enclosed by capsular ligaments.

The major ligaments of the cervical spine include: the anterior longitudinal ligament (ALL), posterior longitudinal ligament (PLL), ligamenta flava (LF), facet capsular ligaments (FL), and the supraspinous and interspinous ligaments (SSL and ISL). These ligaments are illustrated in Figure 3.

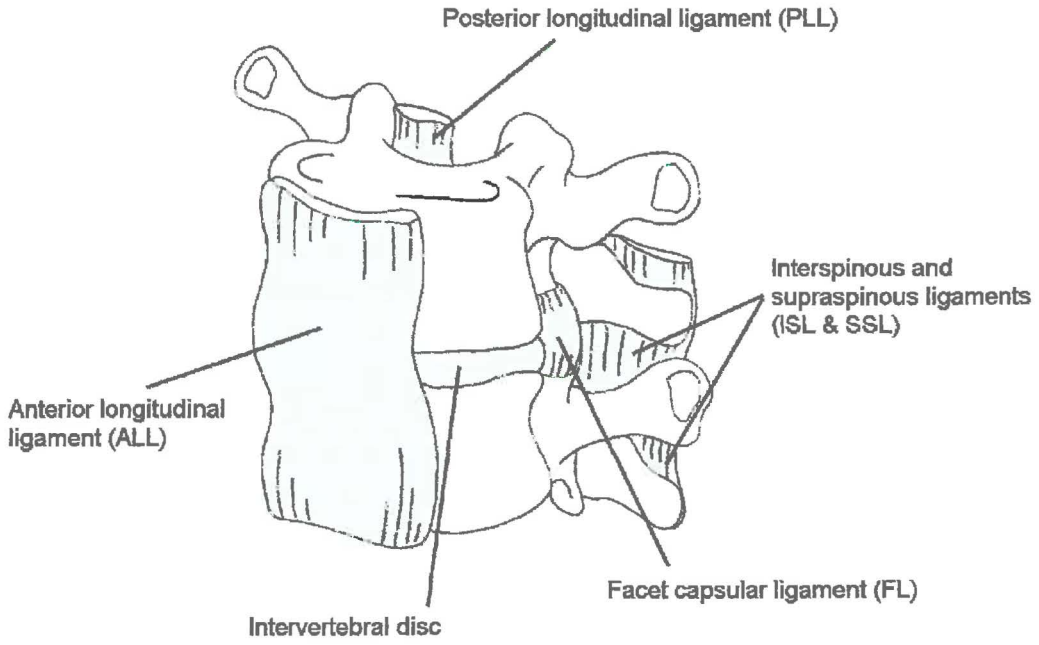

Figure. 3 Sketch illustrating a cervical spine motion segment and the major ligaments
Adopted from F. Netter, 1997.

7.2 Functional anatomy of the cervical spine

7.2.1 The rotation axes of the intervertebral joint

Bogduk et. al, (2000) describe the cervical intervertebral joints as being saddle structures. The inferior surface of the upper vertebral body is concave downwards in the sagittal plane and matches the form of the superior surface of the lower vertebral body due to the uncinate processes (Figure 4). This allows rocking motion of the superior vertebra, sliding in the sagittal plane about Axis 1, and rotation in the transverse plane about Axis 2, (Figure 5).

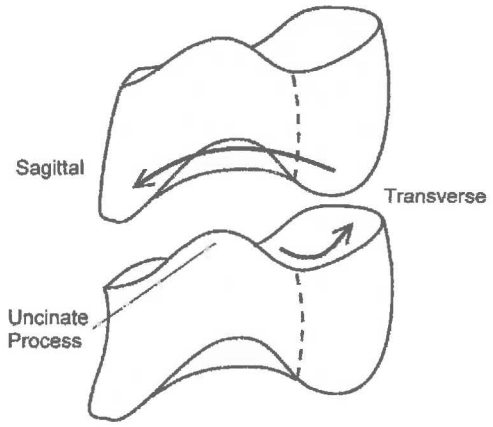

Figure 4. The cervical intervertebral joints allow sliding of the upper vertebra in the sagittal plane and rotation in the transverse plane, adapted from Bogduk and Mercer (2000)

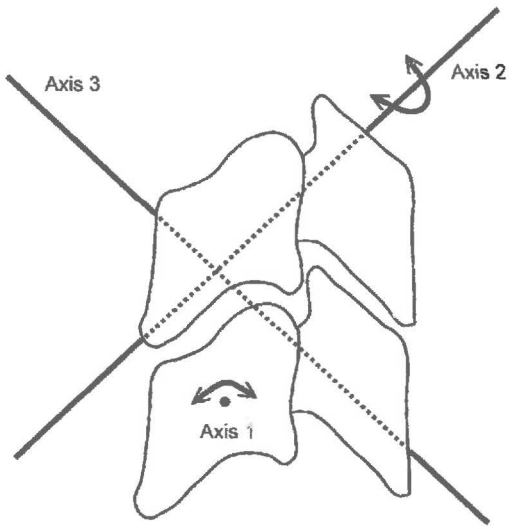

Figure 5. A sagittal section of the C5/C6 vertebra showing major axes of rotation: flexion/extension occurs about Axis 1; axial rotation may occur in the plane of the facet capsules around Axis 2; and, no motion is possible about the remaining orthogonal Axis 3, (adapted from Bogduk and Mercer (2000).

The facet capsule permits the sliding and rocking motion of the intervertebral joint in the sagittal plane, but constrains most other directions of motion. While the vertebral body is able to rotate about Axis 2, which is perpendicular to the facet plane, it cannot rotate about Axis 3 due to interference of the facet faces. Rotation in this plane may only occur if the facet face rises up the 45° slope of the opposing face. For this reason, the only pure rotation of the cervical vertebral joint is in flexion/extension, as axial rotation of the neck must be coupled with lateral flexion and vice versa.

7.2.2 The structure of the intervertebral disc

Mercer et. al, (1999) give a detailed three-dimensional description of the cervical intervertebral disc and its surrounding ligaments. The authors found that the cervical annulus fibrosis (AF) forms a crescent shaped mass of collagen: thick anteriorly and tapering laterally to the uncinate processes. The ALL covers the front of the disc, and the PLL reinforces the rear. When viewed laterally, the fibers in the anterior AF converge forward and upward towards the line of Axis 2, at approximately 45° to the plane of the intervertebral joint

7.2.3 Pain receptors in the intervertebral joint

One of the major difficulties in diagnosing, treating or preventing whiplash-associated disorders (WAD) has been the lack of any easily discernable injuries. Diagnosis has been forced to revolve around interpreting symptoms, which may have psychosomatic aspects.

Medical dictionary defines pain as an unpleasant sensation caused by noxious stimulation of the sensory nerve endings, which under normal conditions signals actual or potential tissue damage. It is a subjective feeling and the response to the cause varies amongst individuals. In the case of chronic pain, usually defined as that which continues for more than 6 months, the nervous system itself may become sensitized, and the sensation of pain may serve no useful purpose.

Cavanaugh (2000) reviewed the neurophysiology and neuroanatomy of neck pain. The specialized nerve endings for the sensation of pain are called nociceptors and, microscopically, they appear as free or finely branched nerve endings. Noxious mechanical and thermal stimuli and certain chemicals can activate nociceptive nerve endings, leading to pain. Tissue damage and inflammation can sensitize nerve endings, causing previously innocuous stimuli to be painful. Nociceptors have been shown to exist in various components of spinal tissues, namely the muscle (Bogduk et.al, 1988), disc annulus and facet joint

ligaments (McLain 1994). Consequently, injury to any of these tissues has the potential to cause neck pain.

7.3 Clinical studies of WAD

In an extensive review of whiplash injury, Barnsley et. al, (1998) concluded that the structures most likely to be injured in whiplash are the facet capsule, the intervertebral discs and the upper cervical ligaments. Injuries to other structures may occur but the available evidence appears to suggest that these are less common. The most likely injuries to be associated with whiplash, (Figure 6), were identified, and included the following:

- *Facet capsule injury* - ligament tears, cartilage damage, contusion of the intra-articular meniscus, hemarthrosis (joint hemorrhage) and possibly extending to microfractures;
- *Disc injury* - AF ligament tears, cracks in the nucleus pulposus and protrusions, and vertebral end plate avulsions;
- Major neck ligament injury - tears to the ALL.

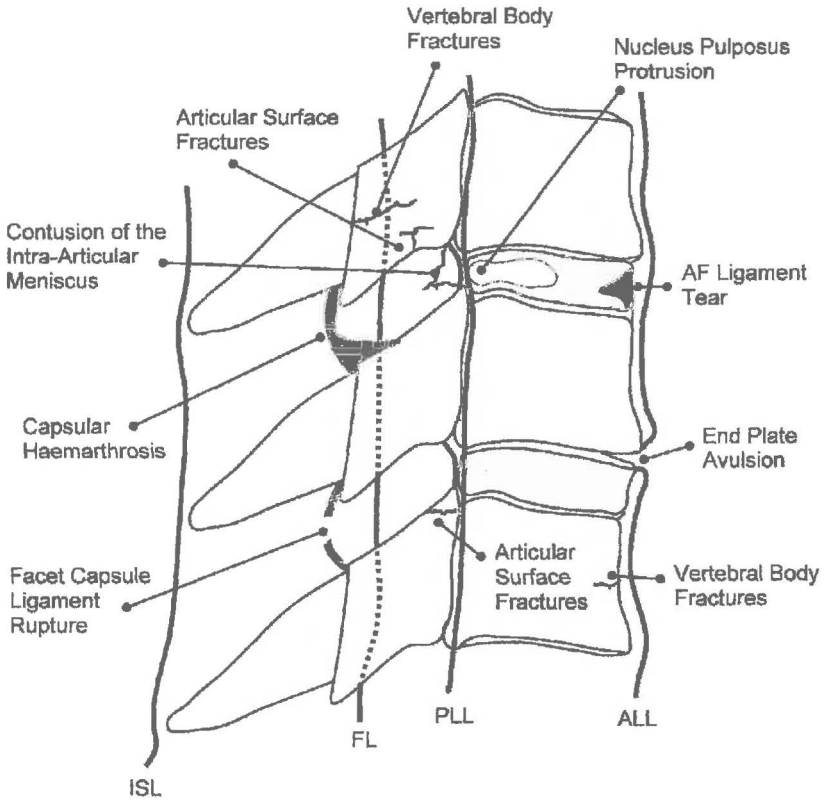

Figure 6. A lateral view of a section of the lower cervical spine showing possible whiplash associated injuries, adapted from Barnsley et al. (1995)

7.4 Experimental studies

7.4.1 Introduction

Mertz et.al (1967) tested a volunteer and several embalmed cadavers using an impacts led. They developed a method for calculating the inertia loading of the neck by the head, using a free body diagram. In a later study, Mertz et. al (1971) proposed a set of neck injury criteria, which were, until recently, used in most automotive safety evaluations.

The Mertz assessment values require that the flexion bending moment at the head/neck junction, or OC, should be less than 190 Nm and that in extension the bending moment should be less than 57 Nm. These results were obtained from multiple tests on a group of four cadavers, with no dislocations of the neck vertebrae (i.e. severe ligament damage) detectable by X-ray.

In the 1990s, a growing awareness of the increasing numbers of soft-tissue injuries and the lack of effectiveness of available head restraints led to further work in investigating the response of volunteers in rear impacts. Important among these studies were those by Ono et. al, (1993), McConnell et al. (1993 and 1995), Geigl et al. (1994), Szabo et. al (1996), Ono et al. (1997) and Siegmund et.al, (1998). Testing on human volunteer responses gives the best description of occupant kinematics in rear impact.

Volunteer testing must be strictly limited in severity for ethical reasons. As a result, cadaver testing to investigate specific injuries has also continued in various forms. Deng et al. (2000) and Geigl et al. (1994) used intact cadavers to directly investigate the transition point for injury. Yoganandan et al. (1998) and Panjabi (1998) used intact human heads and necks to demonstrate specific injury mechanisms. At the neck motion segment level, several investigators have used *in vitro* testing of excised motion segments to investigate specific injury mechanisms suggested by other studies, namely Winkelstein et al. (2000) and Siegmund et al. (2000).

7.4.2 Neck motion in a rear impact

Kaneoka et al. (2002) tested 10 volunteer subjects seated on a sled, to simulate car rear-impact acceleration (Figure 7). An impact speed of 8 km/h was used to study the head-neck-torso kinematics and cervical spine responses. The acceleration pulse generated by the sled in the 8 km/h impact speed is shown in Figure 3.8. A headrest was not used in the experiment.

The activity of the sternocleidomastoid muscle and the paravertebral muscles were measured with surface electromyography (EMG). The neck axial and shear forces, and the flexion/extension bending moments at the occipital condyle, were calculated by treating the head as a free body. The results for one of the volunteers are plotted in Figure 8.

This study has particular importance because the cervical motion was recorded by cineradiography (90 frames per second X-ray) and analysed to quantify the rotation and translation of individual cervical vertebrae resulting from the impact. This method allowed the motion patterns of cervical vertebrae in the crash motion and in normal motion to be compared.

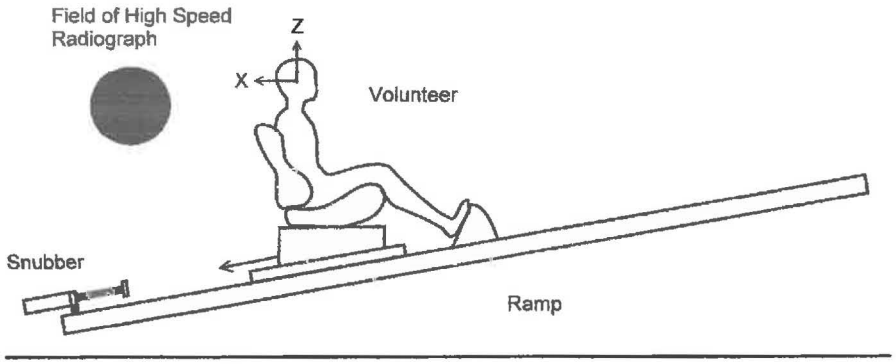

Figure 7 Volunteer seated on a sled inclined at 10°, simulating a car rear impact at 8 km/h (adapted from Kaneoka et al. 2002)

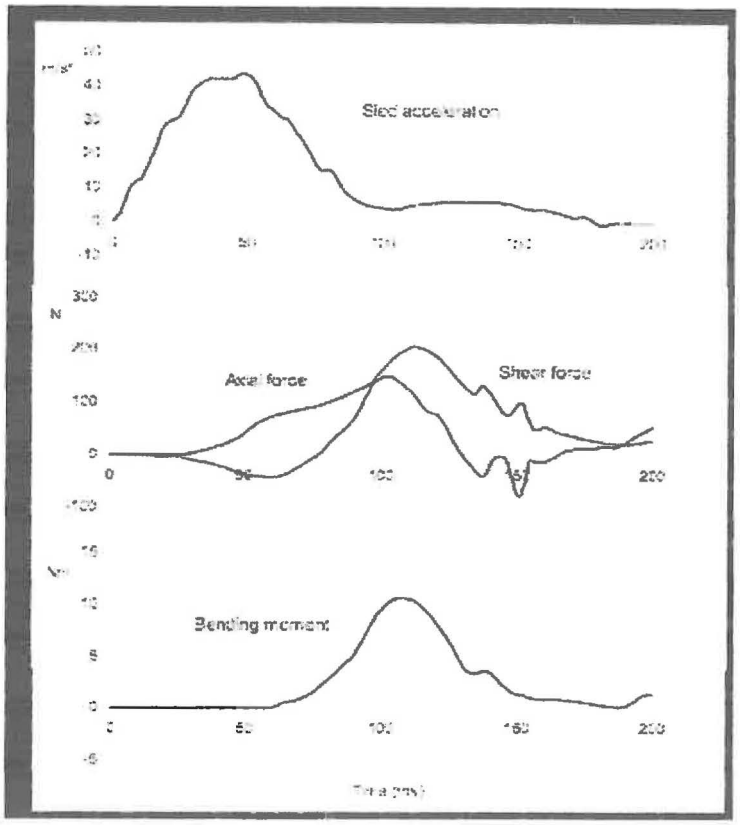

Figure 8. The acceleration pulse generated by the sled for the 8 km/h impact speed is shown along with the neck axial and shear forces and flexion/extension bending moment at the occipital condyle for one volunteer (from Kaneoka et al. 2002)

Kaneoka et. al, (1998) divided the motion and head-neck-torso responses of the test subjects into four phases (Figure 9):

PHASE 1: SLED MOTION (0–40 MS)

- The seat begins to press the back of the volunteer;
- The spine begins to straighten;
- Cervical motion has not occurred;
- No muscular response in the neck.

PHASE 2: NECK AXIAL FORCE (40–100 MS)

- The torso moves forward – pushed by the seat back;
- The torso moves upward – parallel to the seat inclination, causing axial compression of the cervical spine due to the inertia of the head, which reaches a maximum;
- The head remains stationary due to inertia, with a slight initial flexion;
- C6 rotates earlier into extension than the upper vertebral segments (C3, C4 and C5);

- The vertebra of the neck assume an 'S' shape with the upper region in flexion and the lower region in extension;
- No muscular response in the neck.

PHASE 3: AXIAL AND SHEAR FORCE (100–160 MS)

- As the sled slows the torso rebounds and moves forward with some backward rotation;
- The axial force on the neck decreases while the shear force on the neck reaches a peak at about 120 ms;
- The head begins to rotate into extension;
- The cervical spine moves into alignment in extension;
- The EMG of the sternocleidomastoid discharges from about 115 ms.

PHASE 4: FULL EXTENSION (150–220 MS)

- The torso moves forward and down;
- The head and neck rotation reaches full extension;
- Shear and axial forces in the neck decrease;
- The muscular discharge finishes by around 220 ms.

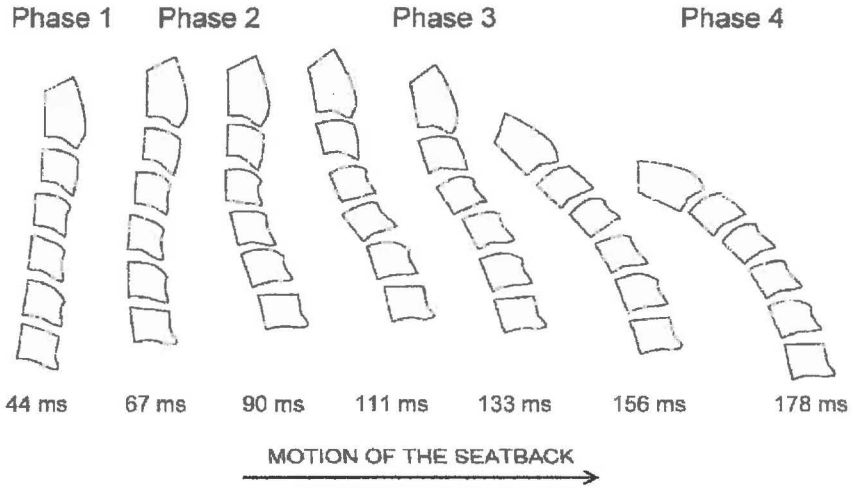

Figure 9 The alignment of the C2 to C7 vertebrae of a volunteer during a rear impact obtained by high-speed radiography for the 4 phases described by Kaneoka and Ono (1998). The alignment at 111 ms also includes the facet capsule and spinous processes to illustrate the possibility of impingement of the facet surfaces.

The exact timing of the events in a volunteer test is quite variable and depends on the acceleration pulse shape and magnitude, the stiffness of the seat back, the angle of the seatback, the posture and anthropometry of the subject, and whether a head restraint was present. The S-shaped response in Phase 2 of the neck in a rear impact has been verified by other studies using cadaver head and necks, whole cadavers and volunteers (Grauer et al.1998; McConnell et al. 1993; Svensson et al. 1993).

If the seat used in the test is fitted with a head restraint, then during Phase 3 the head makes contact and starts to receive additional support. Maximum retraction of the head is most likely to occur before contact with the head restraint (Bostrom et al. 2000). The effectiveness of this extra head support depends on the geometry and stiffness of the head restraint and its mounting on the seat back. A head restraint located at an appropriate proximity to the head, in terms of offset and height, and with ample crush stiffness, has the potential to reduce the neck loads in Phases 3 and 4.

In Phase 4, the motion halts when a restrained subject moves forward into the shoulder portion of the seatbelt. Seatbelts also reduce the upward motion of the torso in Phase 2.Phase 4 may possibly account for the increase in whiplash injury noted with seatbelt use in field accident studies. Based on these phases of motion, there are three distinct periods that have the potential to cause injury to the neck:

- Early in the impact event during the head retraction period and leading to the 'S' shape of the neck (Phase 2);
- Due to the impact with the head restraint, if it is poorly positioned with respect to the head and neck at the time of contact (Phase 3);
- Due to hyperextension for a severe impact with a poorly fitted head restraint or without one (Phase 4); and,
- During the rebound into the seat belt (Phase 4).

7.5 Role of muscles in subject response

As well as Kaneoka et al. (2002), other researchers have investigated the effects of muscular response on the head and neck motion of volunteers in rear-impact tests.

Szabo et. al, (1996) measured the EMG activity of volunteers during low-speed rear impacts. Ten vehicle impacts were conducted using male and female subjects aged 22-54years and with a target vehicle velocity change of 10 km/h (from an impact speed of 16km/h). Accelerometers were affixed to the target vehicle's static centre of gravity and the occupant's head, cervical spine, and lumbar spine. The test protocol was designed to inhibit the subjects from bracing in anticipation of the impacts. The tests were run such that the subjects did not expect the impact. EMG readings were taken from the superficial neck and back muscles of volunteers, including the superior trapezius, sternocleidomastoid, suboccipital cervical extensors, and the para-lumbar muscles.

Typically, initial muscle activity was found to occur 100 to 125 ms after the moment of bumper contact – when the occupant's cervical spine extended during the initial phase of impact. Full muscle tension only developed 60 to 70 ms after the onset of muscle activity – when the cervical spine underwent flexion. The onset of muscle activity commenced while the neck continued to extend and full muscle tension was not achieved until well into the flexion phase. The cervical flexor, cervical extensor and lumbar para-spinal musculature demonstrated similar onset of activity. Consequently, the researchers hypothesized a centrally generated response for the initial onset of muscle activity. The response of the muscles was consistent with a trigger generated by the acceleration of the lumbar spine, and typically occurred 90 to 120 ms following the onset of lumbar spine acceleration.

In a more recent study, Brault et al. (2000) tested 42 male and female subjects (aged 20 to40 years old) in rear impacts at 2 km/h and 4 km/h. The responses of the sternocleidomastoid and the cervical para-spinal muscles (at the C4 to C6 levels) were investigated using EMG. It was found that at 2 km/h the response time for the sternocleidomastoid muscle was 91 (±9) ms while the 4 km/h impact velocity yielded a response of 81 (±8) ms. The females in the group had slightly faster onset times for both muscle groups, but neither the magnitude nor time of the peak muscle-lengthening velocity varied with gender. The researchers made the following conclusions:

The cervical muscles become active in the early phases and are capable of generating forces which modify the head and neck dynamics later in Phases 3 and4 of the motion;

The sternocleidomastoid muscle is activated to contract, while it is lengthening during cervical extension, which is consistent with possible contraction-induced muscle injury;

The arrangement of the neck muscles provides little resistance to the horizontal shear motion between the head and neck pertaining to whiplash; and

The predominately vertical alignment can lead to axial compression loads as a result of muscle contraction.

In seated subject-perturbation tests, Kumar et. al,(1998) showed that the peak head accelerations of subjects who were aware of an impending horizontal perturbation were approximately half as large as those in subjects who were unaware.

7.6 Mechanisms of WAD injury mechanisms in the lower cervical spine

There have been many attempts to relate the phenomenon of soft tissue injury to neck motion following a rear impact. The direct linkage between the mechanical loading from the crash and the injury leading to the observable symptoms is still undefined. The clinical data regarding chronic pain outcomes related to whiplash associated injury has led to a hypothesis that over 50% of whiplash injuries are located within the facet capsules of the cervical spine. The exact timing and mechanism of this injury-causing event to the facet capsule has yet to be determined. Consequently, it is useful to review the main theories about mechanisms of whiplash injury that have been discussed in the literature.

7.6.1 Hyperextension of the neck

Early studies tended to relate whiplash associated injury to hyperextension of the neck. These included primate studies (MacNab 1965), volunteer and cadaver studies (Mertz et. al, 1967) and field accident studies (States et al. 1972). The introduction of head restraints as a result of motor vehicle safety regulation was only partially effective in reducing whiplash associated injury. The increasing levels of whiplash associated injury in the last decade combined with the results of the volunteer testing, which suggests possible injury in the early phase of motion, are indications that simple hyperextension of the neck is not the problem.

7.6.2 Muscle strains

The motion of the head leading to extension of the neck stretches the anterior muscles such as the sternocleidomastoid muscles. One hypothesis is that these muscles are at risk of injury from attempting eccentric contraction during Phase 3 of whiplash motion. Eccentric contraction occurs when a muscle contracts as it is stretched. Studies have shown that muscle failure occurs at forces much larger than maximal isometric force and stretch is necessary to create injury (Garrett et al. 1997). The contraction is due to the stimulation of muscle spindles in the flexor muscles that are being stretched as the neck and head move into extension – Phase 2. At this stage, the large extensor muscles in the back of the neck are moving into compression and are hence unlikely to contract at the time of impact.

A second hypothesis is that the extensor muscles are injured during rebound of the head and neck as they undergo eccentric contraction during the rebound phase of the impact in Phase 4 (Tencer, 1998; Hell et al., 2002). Hell et al. regarded the rebound into the belt system as a possible additional injury source, because the measured head velocities in this phase have been shown to reach higher values than previously expected. This mechanism is consistent with the findings of Garrett et al. (1997) but fails to explain the significant number of belted occupants in severe frontal impacts who do not have neck pain following a crash. Further, the muscle strain mechanism may explain short-term muscle stiffness following the impact, but such injuries typically last only a few days.

7.6.3 Spinal column pressure pulses

Svensson et al. (1993) conducted an animal study to investigate whether whiplash injury was produced by pressure pulses generated in the spinal column. The necks of pigs were exposed to rapid flexion-extension motion in simulated rear impacts. Pressure pulses of up to 150 mmHg were found in the lower cervical spinal canal during neck motion and were greater in magnitude across the vertebral foramen than along the canal. Microscopic analysis of the nerve cells in the spinal dorsal root ganglia (DRG) revealed a leakage of dye from the CFS across the cell membranes, indicating membrane damage.

Eichberger et al. (2000) conducted a total of 21 tests including pressure measurements with 5 cadavers. Sled experiments were performed using a test set-up similar to real rear-end collisions. Impact velocities of approximately 9 km/h and 15 km/h were chosen. The subjects were fitted with 2 triaxial accelerometers on the head and chest, one biaxial accelerometer at

the height of T1, and one angular accelerometer at the head. Pressure measurements in the cerebrospinal fluid (CSF) were performed using 2 catheter-tip pressure transducers, placed subdurally in the spinal canal. The upper transducer was placed at the C1/C2 level and the lower transducer at C6/C7. The researchers found pressure peaks reaching 220 mmHg at approximately 100 ms in the cadavers tested. This confirmed the pressure pulse amplitudes and times obtained in the animal experiments by Svensson et al (1993) were also possible in humans. Injuries to the nerve tissue in the neck resulting from these pressure effects could not be observed due to limitations with the use of cadavers.

7.6.4 Facet impingement

In a series of related studies by Ono et al. (1997), Kaneoka et. al, (1998) and Kaneoka et al. (2002), volunteer subjects were seated on a sled simulating actual car rear-impact acceleration. The motion patterns of cervical vertebrae in the dynamic crash motion and in normal motion were compared using high-speed radiography. The forward and upward motion of the torso combined with the inertia of the head leads to an S-shape formation of the cervical vertebrae. The motion leads to compressive and shear loading of the cervical spine. In this phase of the neck motion, the lower cervical spine becomes extended while the upper spine moves into flexion. Based on the neck radiographs from the volunteer tests, the researchers found that the lower motion segments had the larger the relative rotation angle. The rotation between the fifth and sixth vertebral segments is the largest and earliest (Figure 10).

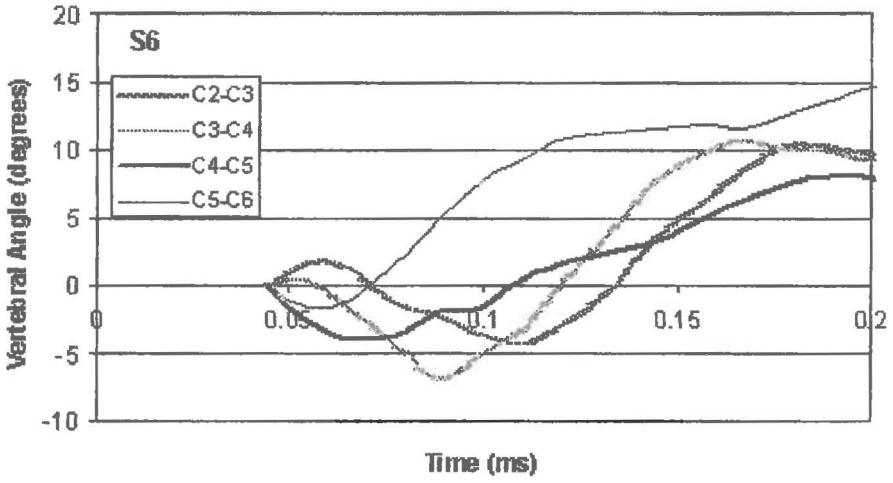

Figure 10. Relative rotation of the cervical vertebra for a volunteer (S6) in a rear impact, from Ono et al. (1997)

To quantify this motion, the position of the instantaneous axis of rotation (IAR) was analyze for the C5/C6 motion segment (Ono et al. 1997). Volunteer neck measurements provided the expected positions of the IAR within the C6 vertebral body, in normal cervical extension (Figure 11).

When the S-shape of the neck occurs in the whiplash motion, the IAR moves upward to a position within the C5 vertebral body (Figure 11). This upward motion of the IAR indicates that the C5 motion at this point is largely one of rotation rather than shear.

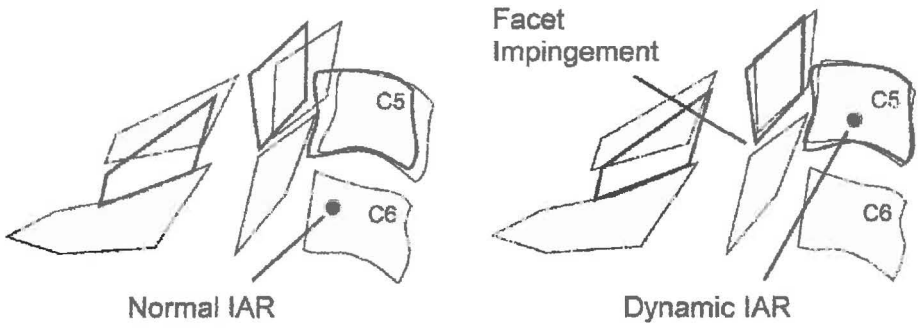

Figure 11. With normal cervical extension motion the IAR is positioned in the C6 vertebral body. When the S shape is reached in the whiplash motion, the IAR moves upward to a position within the C5 vertebral body, after Ono et al. (1997).

This upward shift of the IAR during the crash motion was only observed in the C5/C6 motion segment (Kaneoka et. al, 1998). It was hypothesized that, as a result of the motion, theatrical facet surfaces would collide, resulting in mechanical impingement on the synovial fold or meniscoid in the facet capsule (Kaneoka et al. 2002). Further, it was hypothesized that if this torque is large enough, there was the possibility of tearing the anterior longitudinal ligament or separating of the annulus fibrosus from the end plate of the associated vertebrae (a rim lesion).

Subsequent testing of cadaver head and necks by both Yoganandan et al. (1998) and Pearson et al. (2004) has supported the impingement motion of the facet capsule. Unlike the volunteer measurements by Kaneoka et al. (2002), significant shear displacement was observed in the facet capsule as well as the rotation of the vertebra in both of these studies.

To investigate the facet capsule impingement hypothesis further, Inami et. al,(2000) dissected 20 cervical spines to gain anatomical data of the cervical facet joint meniscoid. The researchers found that five large examples of elliptic-shaped meniscoids projected sufficiently to be impinged by the articular facets of the joint.

7.6.5 Shear

A rear impact causes the seatback to push the torso forward, while the head remains stationary. The effect of the seatback pushing on the cervical spine is to straighten the thoracic spine. The inertia of the head converts this vertical motion of the spine into a compression loading to the cervical spine. This compression has been observed in volunteer and cadaver tests simulating whiplash. As the torso pulls the head forward, a shear force is generated at each level of the cervical spine. Yang et. al, (1996) suggested that this shear force was a candidate to cause soft tissue injury to the intervertebral joints of the cervical spine. Under compression, the cervical vertebrae slide relative to each other and the facet capsules are stretched and possibly torn, resulting in inflammation and pain.

Deng et al. (2000) carried out 26 low-speed rear-end impacts on six human cadavers in a rigid seat. The study showed that the upper cervical vertebrae go into relative flexion with respect to the lower cervical vertebrae during whiplash motion, while the entire neck is in extension (the S-shape). In addition, the upper neck is under flexion when the head contacts the head-rest, while the facets reach peak strain prior to head contact with the head-rest. It was concluded that if stretching of the facet capsular ligaments were the reason for the high incidence of neck pain, the upper cervical spine would sustain a flexion injury while injury to the lower cervical spine would be due to a combination of shear and compression.

Deng et al. (2000) also reported that a 20-degree seatback as compared to a 0-degree seatback resulted in less cervical lordotic curvature, more upward ramping motion of the thoracic spine, and greater relative rotation of each cervical motion segment.

8. Biomedical and biopsychosocial models in relation to WAD

Traditionally, as with most health interventions, the treatment of whiplash associated disorders (WAD) has been based on the biomedical model. However, more recently there has been a move toward the use of a biopsychosocial model (e.g. Nederhand et al., 2003).

As previously mentioned, there is some controversy as to which model is the most appropriate. But with increasing evidence to suggest that other factors, besides crash-related factors, are important in determining outcomes, as well as the use of multidisciplinary treatments, there appears to be increasing evidence in favour of the biopsychosocial model.

8.1 The biomedical model

The biomedical model has been described as a mechanical model of the human body. It is said that it originated with Descartes, and it is the model that dominates medicine in modern times (Walker et. al, 2004). The basis of this model is that there is a direct relationship between the pathology that exists in tissues and the degree and type of symptoms experienced (Daykin et al, 2004; Schultz et al , 2000).

As a consequence of this conceptualization of "biomedical reductionism", the biomedical model is a framework in which the mind and body function separately (Gatchel, 2004). Furthermore, the biomedical model relies on objective scientific truth which is to be found in bodily processes, and puts the physician in the position of being in control of treatment (Schultz et al., 2000). Examples of such a framework can be seen in the literature concerning WAD (i.e. Bogduk et al, 2000; Treleaven et al, 2003; Uhrenholdt et al, 2002).

Schultz et al. (2000), outline a number of consequences arising from the implementation of a biomedical model. They describe that one obvious result is the need to detect underlying pathology relating to the presenting symptoms. Such a need requires the practitioner to gather information from a careful history, as well as a variety of tests including radiographs, laboratory tests and physical examination. The assessment process within the biomedical model has been described as a physician-centred approach (as opposed to a patient-centred approach of the biopsychosocial model). The physician-centred approach is necessary, for example, in acute injury when a person's life may be in danger and a rapid response is required (Larivaara et al, 2001).

In terms of treatment, the biomedical model relies on an approach that aims for a cure, using physical modalities such as medication, surgery and physiotherapy (Schultz et al., 2000).

Conversely, due to this emphasis on "physical" causes and treatment, there is little if any consideration of psychosocial issues within the framework of the biomedical model (Zimmerman et al, 1996).

There is evidence to suggest these issues are important in WAD, as they have been shown to be in relation to other health problems (e.g. Jones et al, 2002; Schultz et al., 2000; Smith et al, 2002; Wickramasekera et al, 1996). In response to findings regarding the importance of psychosocial issues in WAD, an alternative approach which aims to not only deal with the same issues as the biomedical model, but also psychosocial issues, has been proposed. The alternative approach is based on the biopsychosocial model.

8.2 The biopsychosocial model

The biopsychosocial model is a phenomenon that has received a lot of attention recently in medical literature, despite having been around since George Engel described it in 1977 (Engel, 1977). Perhaps it is only due to the recent evidence regarding the relevance of psychosocial issues, that this previously more theoretical model has gained prominence.

While a brief glance at the literature would have one think the biomedical model and the biopsychosocial model are two separate entities, it would come into view the biopsychosocial model is an extension of the biomedical model. It does not ignore biological issues at the expense of the psychosocial. Rather, it pay attention to and expanded on the biomedical model, in an attempt to deal with the health problems that have thus far eluded the reach of this highly effective model.

The biopsychosocial model, as the name implies, is a model of health that considers biological, psychological and social factors, and the interactions between them. These factors are considered in the predisposition, etiology, course, treatment and outcomes related to abnormal states of health (i.e. Alonso, 2004; Caltabiano et al, 2002; Engel, 1977; Gatchel, 2004; Pilgrim, 2002; Suls et. al, 2004; Turk et al, 2002; Walker et al, 2004).

Treatments have arisen out of the biopsychosocial model and generally involve multidisciplinary teams. Examples can be found for chronic back pain (Vendrig, 1999) and chronic pain generally (Burns et al, 2003; Turk, 2001). Similar multimodal treatment programs have also been used in the treatment of chronic whiplash (i.e. Rodriquez et al ,2004; Sterner et al, 2004). With regards to the effectiveness of these interventions, a recent review found conflicting evidence about the effectiveness of these programs with chronic WAD

(Conlin et, al, 2005). However, this study only discussed two studies due to a limited availability of studies of appropriate quality, and so it is difficult to make any definitive judgments about these treatments at this stage. Also, because these studies involve different combinations of biological, psychological and social interventions, it becomes difficult to compare outcomes across studies. As an understanding of these issues evolves, it is expected more research will be able to evaluate these types of treatment regimes. However, in the meantime, in order to assess whether the use of the biopsychosocial model has a theoretical basis, it is necessary to look at the evidence available regarding these issues in relation to WAD.

8.3 Biological, psychological and social factors in WAD

8.3.1 Biological factors

There is general agreement throughout the literature on WAD, that the anatomical structures responsible for the array of symptoms associated with Grade I and II WAD are unknown in the majority of cases (Borchgrevink et al, 1997; McClune et al, 2005; Moog et al, 2002; Pettersson et al, 2004; Radanov et al, 1999; Rodriquez et al, 2004; Silber et al, 2005; Solomon, 2004; Treleaven et al, 2003; Uhrenholt et al, 2002). However, two lines of research give some indication of the possible structures at fault.

Firstly, studies of motor accident fatalities at autopsy have identified the structures damaged in severe impacts. Uhrenholt et al, (2002), recently conducted a review of the literature on the research in this area from 1967-1998. They found that the likely structures damaged in the cervical spine as a result of road accidents include the intervertebral discs, cartilaginous endplates, and the articular surfaces and capsules of the zygapophyseal joints. These lesions were found exclusively in road accident victims at postmortem and not in control groups, and could not be explained by the normal changes associated with aging. In their discussion they highlight the difficulty in identifying such lesions on radiographic examination post injury, which is consistent with reported difficulty in establishing a definitive diagnosis in motor accident victims. They also suggest that while these findings are from studies of road traffic fatalities (i.e. much more severe than typical WAD), they believe it is safe to assume non-fatal road traffic traumas would have similar lesions.

Secondly, the zygapophyseal joints of the cervical spine have been implicated as possible sites of damage through the use of diagnostic blocks (Bogduk et al, 2000). Reviews of the

literature on treatment of WAD have also concluded there is moderate evidence that radiofrequency neurotomy in cases of positive findings from diagnostic blocks, is effective in reducing pain and psychological distress in some cases of WAD (i.e. Conlin et al, 2005). The findings of this research are consistent with the findings of studies on cadavers mentioned above, in that damage to the zygapophyseal joints is in some cases a possible cause for the symptoms of WAD.

From some current research, it appears some biological factors, and in particular zygapophyseal joints, are likely to play a part in the symptomatology of WAD.

8.3.2 Psychological factors

A number of psychological factors have been investigated in relation to WAD. These include but are not limited to, depression, anxiety, coping, pain cognitions (e.g. catastrophising), fear avoidance, somatization, obsessive-compulsive behavior, personality, hostility and distress (Linton, 2000; Mayou et al, 1996; Moog et al, 2002). To measure these constructs a number of different psychometric tools have been used. For the purpose of this section, psychological factors will be separated into three broad areas consistent with the way they have been addressed in the literature on WAD; personality variables, emotional states and cognitions.

Firstly, the construct of personality is difficult to define. There are a number of different theories of personality and some debate about which is the most appropriate. However, a simple definition offered by Coon (1998), is "...a person's unique and relatively stable behavior pattern." The critical word here is stable. Personality is what a person is like most of the time under normal circumstances, and these characteristics are relatively stable. In relation to WAD what is being considered is whether the person's personality, the characteristic way they behave, can predict how they will progress in their recovery.

Findings in relation to personality variables are consistent. For instance, five studies looking at personality variables indicate personality factors do not predict outcomes in WAD (Borchgrevink, 1997; Linder et al., 2000; Pettersson et al., 2004; Radanov et al., 1996; Versteegen et al., 2003). Furthermore, a review of back and neck pain generally, reported similar findings (Linton, 2000). Overall the research suggests personality factors do not predict the course of WAD.

Secondly, there are constructs such as depression, anxiety and stress which will be referred to as emotional states. In contrast to personality factors, these states are generally thought of

as more transient, although they can be persistent in some cases, such as those with major depressive disorders or bipolar disorder. These psychological states have also been studied extensively in the literature on WAD. There is a consistent acknowledgement that WAD is associated with increased prevalence of depression, anxiety and stress (e.g. Ferrari et al., 2005; Solomon, 2004; Sterling et al., 2005; Versteegen et al., 2003; Wallis et al., 1998; Wenzel et al., 2002). However, it is believed by some that these states are a result of the injury and subsequent symptoms, rather than the cause (e.g. Moog et al., 2002; Wenzel et al., 2002). There is also evidence to suggest these emotional states can predict outcomes in WAD (e.g. Richter et al., 2004; Sterling et al., 2005).

Lastly, cognitions have also been studied extensively in research into WAD. Cognitions include a variety of beliefs, attitudes, attributions or expectations. Again, like the states discussed above, these are considered to be more transient than personality variables and arise as a consequence of the injury. There appears to be a general consensus that cognitions such as fear, catastrophising, attention (e.g. hypervigilance) and negative expectations (e.g. Peolsson et al, 2004; Solomon, 2005) have a significant impact on the course of WAD, and in neck and back pain generally (Linton, 2000).

Overall the evidence suggests a variety of psychological factors are influential in WAD. However, some caution is warranted. As is evident, a number of studies have considered a wide range of psychological variables. But few studies have included all of these variable sand it is difficult to say with any certainty, whether all of these variables would remain important if other psychological factors were considered alongside them. Also, many of the measures used in these studies, such as the Symptom Checklist 90 and the Short Form 36 Health Survey (SF-36), are general indicators of psychological wellbeing. However, there are also a number of more specific measures available that have been used and include the Beck Depression Inventory and the Coping Strategies Questionnaire. Due to the array of available measures, caution should be used when assessing the impact of psychological variables, especially if the measures are of doubtful reliability or validity. There are many potential psychological variables of interest and all of them deserve to be assessed through the use of reliable and valid measures, and in the presence of other variables, before statements about their importance can be made.

To summarise, psychological variables of a more *dynamic* nature (i.e. depression, anxiety, fear avoidance, etc.), that can change when an injury occurs, appear to be of more importance than more stable variables (i.e. personality). Also, as such variables are usually the target of cognitive behavioral interventions (e.g. Eccleston, 2001; Frischenschlager et al, 2002), and

cognitive behavioral interventions have some support in the treatment of WAD, it would seem necessary to at least consider these variables in relation to WAD. This is further supported by a recent study on a population in South Australia, which found two components on the SF-36 to be predictors of outcomes after whiplash.

8.3.3 Social factors

Social factors have been of intense interest in the literature concerning WAD. In particular, the influence of compensation systems and cultural idiosyncrasies.

There is wide recognition that compensation systems have an impact on a number of pain conditions, including WAD (e.g. Ferrari et al, 2001; Harris et al., 2005). One study from Canada assessing the prevalence of WAD before and after a change in the compensation system found a reduction in the prevalence of chronic WAD after the change from an at-fault system to a no fault system (Cassidy et al., 2000). In relation to the possible effect of culture on the prevalence of chronic WAD, Ferrari et al, (2001) reported a number of studies have found reduced prevalence of chronic WAD in Lithuania, Greece and Germany. In response to these findings, they discussed further research that was undertaken to distinguish what is different about these countries. These studies found low expectations of chronic symptoms when compared to countries where there is a higher incidence. From this it was hypothesized that because the cultural expectation for the development of chronic WAD is not present in these countries, there are reduced rates of chronic WAD.

It is of relevance to note that evidence suggests that at least these two social factors appear to have an influence on WAD. However, another social factor that has gained attention is that of malingering.

Little research has investigated the prevalence of malingering in WAD populations. One study investigated short-term memory and found a high rate of malingering among patients with WAD (Schmand et al, 1998). A recent study also looked at developing a questionnaire to detect such cases in relation to whiplash (Sartori et al, 2003). However, there is debate about the ability to detect malingering rates amongst many other confounding variables. Such factors include the stress of litigation, pre-existing conditions, unrelated illnesses, influence of third parties, medication or change in psychological functioning (e.g. Ferrari et al., 1999, Ferrari, 2002).

Other studies have reported instances of tertiary gain where health care professionals have benefited from recommending inappropriate treatment (Baer, 1997).Difficulties in detecting

malingering in pain populations generally have also been discussed (Craig et al, 1999). Again, varying results have been found in these populations (Meyers et al, 2000; Mittenberg et al, 2002). A review of exaggeration and malingering in chronic pain found possible rates of 1.25-10.4%, although they describe the evidence as extremely weak, and concluded that at present there was no conclusive way for physicians to detect malingering (Fishbain et al., 1999).

It is evident further research is needed in this area in relation to WAD. As discussed, being able to detect malingering is a difficult task. However, it is clear that malingering does exist (although at low levels) and needs to be taken into consideration when dealing with WAD.

9. Evidence based management of WAD

9.1 Evidence relating to assessment and diagnosis

The most important element of initial assessment and diagnosis of WAD is the identification of patients who are at risk of developing, or who have developed, serious consequences (such as fractures or dislocations) following a motor vehicle accident, so that these issues can be treated appropriately.

Recommendations for history taking and physical examination are primarily based on the Quebec Taskforce (QTF 1995) guidelines. The QTF recommendations were based on the consensus opinion of the QTF. No accepted studies were found by the QTF, which dealt with the value of history taking or physical examination for the positive diagnosis of WAD

Steill (2001) and Bandiera (2003) indicated that the Canadian C-Spine rule is the most appropriate rule to apply in order to correctly diagnose a fracture or dislocation without the necessity of X-raying every person with WAD.

This rule has been validated in emergency department populations and has been shown to be better than unstructured clinical judgment in detecting WAD IV patients. The high reported sensitivity of this test (Steill, 2001) is such that clinicians who follow this rule are extremely unlikely to miss a fracture.

Guez M et al, (2003) in his prospective study on 23 patients with different spinal cord injuries, where six patients had cervical fracture–dislocation, and 17 patients had acute WAD Grade III, concluded that whiplash injury caused increased levels of nervous tissue damage markers in cerebrospinal fluid in three of 17 patients. The increased NFL levels in three of 17 patients with damage in a proportion of patients with WAD with neurological deficit.

Kasch H et al, (2001) compared 141 patients with acute WAD to 40 patients with acute ankle distortion to prospectively determine the sensitivity and specificity of possible predictors for handicap following a whiplash injury. The main outcome was that the Cervical ROM test predicted handicap following an acute whiplash injury with a sensitivity of 73% and specificity of 91%. A combined measure of high pain intensity and seven to 15 no painful symptoms in a semi structured interview demonstrated a sensitivity of 27% and a specificity of 99%. Measurement of CROM may predict handicap following whiplash injury

Ovadia D. et al, (2002) investigated the relative importance of the various available tests in an effort to define the best and most reliable routine tests following whiplash injury. Almost all patients (97%) complained of cervical pain. A minor decrease in ROM was found in 20% of patients. Patients with fractures and dislocations were more restricted. Inability to touch chin to chest was encountered frequently in patients with fracture and instability. 2% of patients had neurological or muscular clinical findings. No significant correlation was found between clinical findings, CT scans and EMG. No correlation was found between scans and MRI findings and complaints of radicular pain. 10% of patients with normal initial X-rays demonstrated an increase in degenerative changes on follow up X-ray. Bone scans identified pathology in six patients. Nerve conduction results were inconsistent. Initial X-rays missed two fractures in subjects with severe degenerative disease. Cervical pain was most common complaint .The chin-to-chest test was a strong indicator of differentiation between less severe and more severe grades of WAD. CT scans and MRI did not add to patient diagnosis. The initial radiograph was the best and most useful imaging modality.

In another study of Kasch H et al, (2002), 19 patients with acute WAD were compared to 20 matched control patients with acute ankle injury, in aim to assess pain and sensorimotor function in the craniofacial region in an unselected group of patients with WAD. The main outcome was that only one episode of jaw pain occurred in each group. No significant difference in temporomandibular joint (TMJ) disorders symptoms between groups was seen at 4 weeks and 6 months post injury TMJ symptoms are rare and whiplash injury is not a major risk factor in the development of TMJ problems.

Steinberg et al, (2005) assessed the value of EMG for patients with Grade I and II WAD and determined whether there is any agreement with clinical and imaging (MRI and CT) findings. 70.6% of EMG studies were normal or showed incidental/unrelated findings. No correlation between CT, MRI and EMG was found. No correlation between patient symptoms, objective findings on clinical examination and EMG. No correlation between EMG, CT and MRI is suggesting these additional studies may not be necessary for the management of patients with WAD.

Sterling M et al, (2004) characterized acute whiplash injury in terms of motor and sensory dysfunction and psychological distress and compared subjects with higher and lesser levels of pain and disability. 80 patients with acute (< 1 month) WAD Grade II–III were compared with 20 healthy controls (patients with WAD were grouped into three groups: mild, moderate or severe, based on NDI scores). All three WAD groups had decreased ROM and increased EMG activity compared with controls. Patients with moderate and severe WAD had greater

joint position error and generalized hypersensitivity. All three WAD groups had psychological distress (greater in the moderate and severe groups) compared with the control group. Patients with higher levels of pain and disability were distinguished by hypersensitivity to a variety of stimuli (potentially central sensitization occurs soon after injury).

9.2 Evidence of treatment of acute whiplash

9.2.1 Active exercise

Range of movement (ROM), mobilizing and muscle re-education / strengthening exercises for the neck and scapular muscles should be implemented immediately, if necessary in combination with intermittent rest when pain is severe. Clinical judgments crucial if symptoms are aggravated by exercise. The aim of muscle re-education /strengthening is to restore appropriate muscle control and support to the cervical region.

There 6 studies – Rosenfeld, 2006; Vassiliou ,2006; Bonk ,2000;Mealy, 1986; Crawford ,2004; McKinney, 1989 that supporting a regime of active exercises over collar immobilization and advice to rest. A common theme of active exercises is strengthening of the neck and scapular muscles, range of movement exercises and mobilizing exercises.

Bunketorp (2006) demonstrated that supervised physical training involving individualized range of motion, stability, proprioception and strengthening exercises was superior to a home exercise program.

9.2.2 Advice to 'act as usual' / reassurance / education

The practitioner should adopt a positive and supportive approach. He should acknowledge that the patient is hurt and has symptoms, and advise that:

- symptoms are a normal reaction to being hurt
- maintaining normal life activities is an important factor in getting better
- staying active is important in the recovery process
- voluntary restriction of activity may cause delayed recovery
- it is important to focus on improvements in function.

Borchgrevink, (1998) is supporting advice (alone) to "act as usual" over advice to rest and immobilization in a soft collar. There were however 3 further studies (Crawford ,2004; Bonk, 2000; Rosenfeld, 2006) that combined advice to 'stay active' or 'act as usual' with active exercise which showed a benefit over collar and / or rest.

Scholten-Peeters (2006) demonstrated similar treatment effectiveness (in terms of pain, headache and work activities outcomes) between general practitioners care given by specially trained practitioners involving education and advice to stay active when compared to physiotherapy education, advice and exercises.

Formal education packages in the form of either videos or pamphlets should be provided. Such education packages should include information to reassure the patient, provide advice regarding return to normal activities, information regarding pain relief and basic exercises aimed at restoring movement to the cervical spine, muscle re-education and strengthening.

Two studies (Brison, 2005 and Oliveira, 2006) demonstrated the benefit of an educational video over usual care in an emergency department setting.

9.2.3 Passive joint mobilization / manipulation

Passive joint mobilization / manipulation may be given in combination with active exercises, in situations where exercise and advice alone are not proving effective, provided there is evidence of continuing measurable improvement. WAD Grade III (decreased or absent tendon reflexes and / or weakness and sensory deficit) is a relative contra-indication for manipulation.

Fernandez de las Penas, (2004a and b), was using the same study population and supported the use of manipulation over a program involving ultrasound, exercises, multimodal therapy and pulsed EMG. Passive joint mobilization was also found to be beneficial in two studies when used in combination with active therapy over rest and collar use (Bonk ,2000; Mealy, 1986). Passive joint mobilization was found to be beneficial when used in combination with posture correction, relaxation exercises and psychological support compared with passive electrotherapy (Provinciali, 1996).

9.2.4 Electrotherapy / Passive modalities

Passive modalities / electrotherapies include heat, ice, massage, TENS, pulsed electromagnetic therapy, electrical stimulation, ultrasound and shortwave diathermy.

These passive modalities / electrotherapies are optional adjuncts to exercise and manual therapy in situations where the patient is not improving with active exercise/advice alone, provided there is emphasis on return to usual activity as soon as possible. The clinician should demonstrate continuing measurable improvement with the use of these modalities.

Three identified studies highlighted a benefit of this form of therapy. Foley et al, (1992) demonstrated a benefit of pulsed EMG plus collar over the use of a collar alone. However subjects in this trial were required to wear a collar for 8 hours per day for 12 weeks - which is strongly contra-indicated. Hendriks (1996) demonstrated a benefit of low frequency interrupted direct current as an adjunct to 'standard' emergency care. Thuile (2002) demonstrated a benefit of magnetic field therapy as an adjunct to medication. In contrast, Provincial (1996) showed benefits of a program involving joint mobilizations, relaxation therapy, posture correction and psychological support when compared to passive electrotherapy involving ultrasound and TENS.

9.2.5 Pharmacotherapy

WAD Grade I - III – Simple (non-opioid) analgesics and NSAIDs can be used to alleviate pain in the short term. Their use should be limited and weighed against known side-effects, which appear to be dose related.

Use of high dose intravenous methylprednisolone infusion for acute management of WAD Grade I - III is not recommended given the potential for side-effects and the method of administration in the one study showing benefit of this form of treatment (intravenous bolus in the first 48 hours post accident).

One study (Gunzberg, 1999) highlighted a benefit of an NSAID (Tenoxicam) over a placebo in terms of pain and function outcomes. Petersson (1998) demonstrated a benefit of an infusion of high dose methylprednisolone administered intravenously in the first 48 hours after an accident compared with a placebo. However this treatment is not recommended given the known side-effects of this drug.

9.2.6 Multimodal therapy

The term 'multimodal therapy' encompasses a range of individual treatment modalities such as joint mobilization, relaxation techniques, electrotherapies and exercises as part of a package to address individual patient deficits such as pain, loss of range of movement and

loss of strength. Treatment packages that are "multimodal" in nature and address a range of patient deficits such as loss of range of motion and strength may be used provided there is continuing evidence of benefit. Ideally, such packages should include an active treatment component.

A number of studies highlighted the benefits of treatment packages involving a number of treatment modalities (Borchgrevink, 1998; Rosenfeld et al, 2006; Vassiliou, 2006; Bonk, 2000; Mealy ,1986; Crawford ,2004; McKinney, 1989, Provinciali, 1996). Only one of these studies (Provinciali ,1996) used the term multimodal therapy. This therapy, which involved passive joint mobilization, relaxation therapy, posture correction and psychological support, was shown to be beneficial over electrotherapy.

9.2.7 Immobilization – collars or collars and rest

Collars should not be prescribed for WAD. If they are prescribed they should not be used for greater than 48 hours. There evidence against the use of cervical collars and advice to rest. Seven studies (Rosenfeld ,2006; Vassiliou, 2006; Bonk ,2000; Mealy, 1986; McKinney, 1989; Crawford ,2004; Borchgrevink ,1998) demonstrated that active treatment was more beneficial than immobilization in a soft collar and rest.

It is not possible to separate the use of a collar and advice to rest, as the studies combined the two features and gave varying advice on the periods of time the collar should be worn. One study (Dehner, 2006) demonstrated no difference in outcome between 2 or 10 day collar immobilization. Gennis (1996) further demonstrated no difference in outcome between collar immobilization with analgesia compared to rest and analgesia.

9.2.8 Surgical treatment

There are no indications for surgical intervention in almost all cases of acute and sub-acute WAD Grades I - III. Surgery should be restricted to the rare Grade III WAD with persistent arm pain consistent with cervical radiculopathy (supported by appropriate investigations) that does not respond to conservative management, or with rapidly progressing neurological deficit. Surgical treatment to reduce dislocation or stabilized cervical spine may be required in WAD IV.

There were no studies identified concerning the benefit of surgical intervention in the treatment of acute WAD Grade I - III.

9.2.9 Cervical pillows

Cervical pillows are not recommended. There were no studies identified concerning the benefit of cervical pillows in the treatment of acute WAD Grade I - III.

9.2.10 Intra-articular and Intrathecal injections

Intra-articular and intrathecal steroid injections are not recommended for acute WAD. There were no studies identified concerning the benefit of intraarticular and intrathecal injections in the treatment of acute WAD Grade I - III.

As noted above, the use of high dose intravenous methylprednisolone infusion for acute management of WAD Grade I - III is not recommended given the potential side-effects. Petersson (1998) demonstrated a benefit of an infusion of high dose methylprednisolone administered in the first 48 hours after an accident compared with a placebo. However this treatment is not recommended given the known side-effects of this drug.

9.2.11 Other interventions

Other interventions include Pilates, Feldenkrais, Alexander Technique, massage and homeopathy are not recommended because no evidence was found to support the use of these treatments in patients with WAD.

9.3 Evidence of treatment of chronic whiplash

9.3.1 Advice to 'act as usual' / reassurance

The practitioner should adopt a positive and supportive approach. He should acknowledge that the patient is hurt and has symptoms, and advise that:

- symptoms are a normal reaction to being hurt
- maintaining normal life activities is an important factor in getting better
- staying active is important in the recovery process
- voluntary restriction of activity may cause secondary complications such as loss of joint range of motion, muscle weakness and loss of cardiovascular fitness all of which will reduce functional capacity and delay recovery.
- it is important to focus on improvements in function.

Stewart (2007) and Jull (2007) demonstrated the benefits of reassurance and advice to "act as usual" as part of a package involving specific muscle control exercises, general exercises and joint mobilization.

9.3.2 Active exercise

Active exercise (in combination with advice) is involving functional exercises, range of motion exercises, strengthening of neck and scapular muscles, and specific strengthening of deep neck flexors.

There is evidence for the use of functional exercises, ie range of movement (ROM), mobilizing and muscle re-education / strengthening exercises for the neck and scapular muscles and in particular exercises for the deep neck flexors in the chronic WAD stage. The aim of these exercises is to restore appropriate muscle control and support to the cervical region and restoration of strength, movement and cardiovascular fitness to allow the performance of everyday tasks. The performance of functional tasks should be reinforced. Emphasis should be placed on the quick return to activities of daily living and work tasks.

Three studies (Stewart, 2007; Jull ,2007, Fitz-Ritson ,1995) highlighted the benefits of an active exercise approach. Stewart (2007) demonstrated the benefits of an individualized, sub-maximal functionally based exercise program, conducted with a cognitive behavioral therapy approach as an adjunct to advice. Jull (2007) demonstrated a benefit of a program involving specific muscle control exercises, kinaesthetic exercises, passive joint mobilization, education and assurance over a self-management program involving advice to stay active, description of an exercise program and postural and ergonomic advice. Fitz-Ritson (1995) demonstrated that phasic exercises (eye-head-neck-trunk and eye-head-neck-arm exercises) plus chiropractic treatment was more beneficial than standard exercises (stretching / isometric / isokinetic) plus chiropractic treatment.

9.3.3 Cognitive behavioral therapy

A cognitive behavioral therapy approach to treatment may be instituted. The basics of this approach include: pacing, shaping, appropriate reinforcement and addressing fear avoidance. There is evidence supporting a cognitive behavioral therapy approach. Two studies (Soderlund ,2001 and Stewart ,2007) demonstrated a benefit of a cognitive behavioral therapy approach as an adjunct to standard physiotherapy treatment and as part of an exercise program respectively. This form of therapy involving gradual resumption of normal activities has been used successfully with other forms of chronic spinal pain.

9.3.4 Passive joint mobilization / manipulation (in combination with active therapy)

Passive joint mobilization / manipulation may be given in combination with exercises in the chronic phase provided there is evidence of continuing measurable improvement.
Reliance on passive therapy alone without an "active" component is not recommended in the chronic phase. WAD Grade III (decreased or absent tendon reflexes and / or weakness and sensory deficit) is a relative contra-indication for manipulation.

One study (Jull ,2007) demonstrated a benefit of passive joint mobilization when used in combination with specific muscle control exercises, kinaesthetic exercises, education and assurance over a self-management program involving advice to stay active, description of an exercise program and postural and ergonomic advice.

9.3.5 Vestibular rehabilitation

A vestibular rehabilitation program may be instituted for patients experiencing dizziness in the chronic phase. There is evidence supporting vestibular rehabilitation. One study (Ekvall Hansson, 2006) demonstrated a small clinical improvement in a group exhibiting dizziness that received a vestibular exercise program compared to a control group receiving no intervention.

9.3.6 Multimodal therapy

Treatment packages that are 'multimodal' in nature and address a range of patient deficits such as loss of range of motion and strength may be used provided there is continuing evidence of benefit. Such packages should include an active treatment component in the chronic phase.

One study (Jull, 2007) demonstrated a benefit of a multimodal program involving specific muscle control exercises, kinaesthetic exercises, passive joint mobilisation, education and assurance over a self-management program involving advice to stay active, description of an exercise program and postural and ergonomic advice.

9.3.7 Radiofrequency neurotomy

There is evidence to support radiofrequency neurotomy for chronic whiplash sufferers whose symptoms have been shown by diagnostic blocks to arise from the lower cervical joints. This surgical technique should only be undertaken after other conservative treatment has been shown to be ineffective and when facet joint pain has been confirmed by a local anesthetic block.

One study (Lord ,1996) demonstrated long term benefit of radio frequency neurotomy to highly selected whiplash patients with confirmed facet joint pain compared with patients receiving a placebo.

9.3.8 Subcutaneous sterile water injections

There is evidence regarding the use of subcutaneous sterile water injections in carefully selected cases. The use of this technique should be limited to practitioners with expertise in such injections.

One study (Byrn ,1993) demonstrated that subcutaneous sterile water injections (2-3mm subcutaneous) over up to 30 trigger points were significantly more effective in providing pain relief and improving ROM immediately and after 8 months than those receiving saline injections.

9.3.9 Collar immobilization

Collar immobilization should not be undertaken with chronic whiplash. There is no evidence to support a regime of collar immobilization with chronic whiplash.

9.3.10 Prescribed rest

A period of prescribed rest is not recommended for chronic whiplash. There is no evidence to support a period of prescribed rest in chronic whiplash.

9.3.11 Surgical treatment (other than radiofrequency neurotomy)

There are no indications for surgical intervention (aside from radiofrequency neurotomy) in almost all cases of chronic WAD Grades I - III. Surgery should be restricted to the rare Grade III WAD with persistent arm pain consistent with cervical radiculopathy (supported by appropriate investigations) that does not respond to conservative management, or with rapidly progressing neurological deficit.

There were no studies identified concerning the benefit of surgical intervention (aside from radiofrequency neurotomy) in the treatment of chronic WAD grade I-III.

9.3.12 Cervical pillows

Cervical pillows are not recommended. There were no studies identified concerning the benefit of cervical pillows in the treatment of chronic WAD grade I-III.

9.3.13 Intra-articular and intrathecal injections

Intra-articular and intrathecal steroid injections are not recommended for acute WAD. There were no studies identified concerning the benefit of intrathecal injections in the treatment of chronic WAD grade I-III. Barnsley (1994), failed to demonstrate any benefit of intra-articular corticosteroid injections over local anesthetic injections in terms of time to return of usual pain.

9.3.14 Botulinum Toxin (Botox) Injections

The use of Botox injections in chronic whiplash is not recommended. There is conflicting evidence with regard to Botox injections. Freund et al (2002) found short-term benefits (4 weeks) in terms of pain and ROM in a group of WAD patients receiving botulinum injections versus those receiving saline. Conversely (Padberg, 2007) found no benefit of botulinum injections in 40 WAD patients compared with those receiving placebo injections.

9.3.15 Electrotherapy

The use of electrotherapy in the treatment of chronic whiplash is not recommended. There were no studies identified concerning the benefit of electrotherapy in the treatment of chronic WAD grade I-III. Evidence favors active treatment (Stewart, 2007; Jull, 2007; Fitz-Ritson, 1995) and reliance on passive therapy should be avoided.

9.3.16 Analgesic injections

Analgesic injections are not recommended for the treatment of chronic WAD. Lemming (2005), demonstrated that three different analgesics produced significant reductions in pain compared with a placebo whilst infusion of drugs was taking place. However no long-term difference in effectiveness of any drug over placebo was demonstrated.

10. Discussion

Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rear-impact or side-impact collisions in a motor vehicle, and can also occur during diving, for example.

In contrast to the hyperextension hypothesis used as an explanation for whiplash injury in the past, Panjabi et al. (1998) observed, in an in vitro experiment on whiplash injury, that the cervical spinal column undergoes a two-phase reaction during whiplash. In the first phase, the spinal column forms as S shape involving flexion of the upper cervical spinal column and hyperextension of the lower cervical spinal column. In the second phase, extension occurs at all levels of the spinal column. On the basis of their observations, the authors concluded that whiplash injury occurs in the first phase, before the neck is fully extended. Thereafter, the lower cervical spinal column is injured during hyperextension. At higher speeds, there is a tendency for injury to occur in the upper part of the cervical spinal column.

The impact may result in injury to bony or soft tissue (i.e., whiplash injury), which in turn may lead to a variety of clinical manifestations. The clinical symptoms, which are known as whiplash-associated disorders, can be classified into five grades of severity. In addition, the time that has passed since the injury can be divided into six phases: less than four days; four days to three weeks; three to six weeks; six weeks to three months; three months to six months, and more than six months.

A primary difficulty in diagnosing whiplash is that the term 'whiplash' essentially describes a mechanism of injury. The mechanism of injury may, in turn, lead to a variety of clinical manifestations, the most common of which is neck pain. As whiplash describes a mechanism of injury rather than a single distinct pathology, studies of diagnosis relating to whiplash and whiplash associated disorders are problematic.

Symptoms following whiplash injury may be diverse and include pain, stiffness, neurological symptoms, dizziness, jaw pain, headache, hearing disorders including tinnitus, memory loss and dysphagia. However, neck pain is the predominant symptom. In 1995, the Quebec Taskforce developed a classification system that was designed to improve management of whiplash by providing a guide to the signs and symptoms of whiplash indicative of the seriousness of the injury sustained. It is important for clinicians to be able to

identify patients who are at risk of developing, or who have developed, serious consequences (such as fractures) following a motor vehicle crash so these issues can be addressed appropriately. Further, it is important that clinicians are able to identify signs and symptoms which indicate various levels of severity so appropriate management can be undertaken.

Whilst being a useful guide for clinicians and researchers alike, most claimants for whiplash injury fall into the WAD grade I and II categories, despite sometimes large variations in their individual presentations. Hartling (2001) and Sterling (2004), among others, have proposed a modification of the WAD II classification to reflect the wide range of signs and symptoms included in this grade. These amended classifications incorporate further physical and psychological signs and symptoms such as change in muscle recruitment patterns, sensory impairment and psychological distress. Correct identification of such signs and symptoms may assist clinicians in diagnosing whiplash patients, assist in determining prognosis and guide future treatment.

Epidemiological data on the incidence of whiplash are mainly derived from insurance claim numbers. Therefore, the reported annual incidence of whiplash varies widely between countries and continents: figures vary from 16 per 100,000 inhabitants each year in New Zealand to 70 per 100,000 inhabitants each year in Quebec, Canada. In the Netherlands, the number of new patients who have experienced whiplash is estimated to be 94–188 per 100,000 inhabitants each year.

There is no consensus in the literature on the prognosis of the consequences of whiplash. The prevalence of long-term complaints (i.e., from six months to two years) varies from 19–60%. A Canadian research group, the Quebec Task Force on whiplash-associated disorders (QTF-WAD), reported that the prognosis is favorable: around 85% of patients return to work within six months after the whiplash injury. Recently, this conclusion has been criticized because the severity and duration of the complaints may have been underestimated.

The results of the evidence review highlight the fact that clinical signs and symptoms following whiplash are diverse. Both acute and chronic WAD are characterized by reduced range of motion of the neck (Armstrong B et al 2005, Sterling M et al 2003, Ohberg F et al 2003). There is an increasing body of evidence that chronic whiplash is characterized by disturbances in motor function, altered joint proprioception, generalized sensory hypersensitivity and psychological distress (Banic B et al 2004, Dall'Alba P et al 2001, Nederhand M et al 2005).

Assessment of these factors may assist clinicians in classifying chronic whiplash patients and determining the type of treatment that is recommended in the future. The picture with

acute whiplash is less clear. Whilst a loss of range of motion (in all planes) is a consistent finding, there is less evidence to support other findings such as altered muscle recruitment or sensory hypersensitivity (Sterling ,2003 and 2004, Nederhand,2000, Kumbhare ,2005, and Kristjannsson ,2002).

Findings of a number of cohort studies identified in the review of prognosis are relevant to both history and physical examination The main findings of the prognosis review indicate that high initial pain levels and high initial disability levels are associated with a poor prognosis (Kasch, 2003 ,Buitenhuis ,2006, Sterling, 2003 ,Richter ,2003). These factors therefore need to be assessed by clinicians. The VAS pain scale and the Neck Disability Index are simple, valid, reliable and responsive tools to measure pain intensity and disability status respectively (Nederhand ,2003).

Despite recent improvements in the diagnostic ability of MRI and CT imaging, until there is a clear correlation between findings using these imaging techniques and the prognosis of individual patients with benefits in terms of treatment, the use of such techniques (or indeed other specialized techniques) cannot be routinely recommended for WAD Grade I and II (Kaale ,2005a, Kaale ,2005b, Kraken's, 2003, Patijn 2001, Eliott 2006). There is a place for such imaging in selected WAD III patients where there is nerve root compression or suspected spinal cord injury.

Measures of psychological distress such as low self efficacy and catastrophising are strongly associated with poorer prognosis, and anxiety may also be associated with poor prognosis. A patient's self-efficacy is their confidence in their ability to perform certain activities. This can be measured using the self-efficacy scale (Kyhlback, 2002). Lower scores were shown by both Kyhlback ,2002 and Soderlund 2,003 to be associated with ongoing pain after whiplash. Catastrophising in this context refers to negative self statements and catastrophising thoughts, as defined on the Coping Strategies Questionnaire catastrophising subscale (Rosenstiel, 1983). Higher scores on the catastrophising subscale were found to be associated with ongoing disability by Kivioja (2005) and Soderlund (2000). Psychological factors such as diverting attention, increased behavioral activity, poor mental health and poor social function are not associated with poor prognosis after whiplash.

There is evidence that poor outcome (in terms of ongoing disability) is associated with educational level (Sterner ,2003, Berglund ,2006). For example, people without a university education were twice more likely to have ongoing disability at 16 months than those with a university education (Sterner, 2003). Similarly, (Berglund ,2006) found that those with a

lower education level (primary/ low secondary) were also nearly twice as likely not to recover at 12 months than those who were more highly educated (college/ university).

There is evidence that poor outcome (in terms of ongoing pain or disability) is not associated with crash- related factors such as speed of impact, direction of impact, presence of a head rest, or awareness of the collision. Crash related factors should therefore not be used to determine prognosis after whiplash (Berglund ,2006; Minton ,2000; Gun, 2005; Crouch 2006; Hendricks, 2005).

Increased sensitivity to cold is associated with ongoing disability after whiplash (Kasch 2005; Sterling 2005). There is evidence that reduced cervical range of motion is associated with ongoing disability after whiplash based on 2 cohorts (Kasch 2001; Sterling 2003).

There is evidence that previous neck pain is not associated with ongoing pain after whiplash (Kivioja 2005).

There is conflicting evidence regarding whether pursuing compensation and/or consulting a lawyer is associated with ongoing pain after whiplash. Three cohorts (Pennie ,1991; Gun 2005; Hendriks, 2005), show no association whereas one cohort (Mayou ,2002) did show an association.

There is conflicting evidence regarding whether pursuing compensation and/or consulting a lawyer is associated with ongoing disability. One cohort found an association (Gun 2005) whereas two cohorts did not (Kasch ,2001; Hendriks ,2005).

10.1 Treatment of acute WAD

Rosenfeld et al (2006), highlighted that a program of early active therapy (within 2 weeks) involving active ROM and McKenzie therapy demonstrated significant short and long term (to three years) benefits in terms of pain and reductions in sick leave over a group receiving advice to rest and soft collar immobilization.

Vassiliou et al (2006) also demonstrated a clinically small but statistically significant benefit of active therapy (strengthening and movement exercises with an elastic resistance band) over 1 week of "day and night" collar immobilization in terms of both short term (6 week) and long term (6 month) pain and disability.

Bonk et al (2000) that active therapy (involving strengthening exercises for neck and scapular muscles, isometric exercises, passive joint mobilization, advice and icing) was superior to 3 weeks of collar in terms of pain at 6 and 12weeks.

Mealy et al (1986), demonstrated significant benefit in terms of pain and ROM in a group receiving early active mobilization (involving home mobility exercises, passive joint mobilization, ice in the first 24 hours and local heat afterwards) compared to a group receiving 2 weeks of collar immobilization and rest.

Crawford et al (2004) demonstrated benefits of a mobilisation program (involving advice to mobilise freely and a self-mobilisation exercise regime) over 3 weeks of collar immobilisation (followed by same mobilisation regime) in terms of speed of return to work. There were no significant differences in terms of pain outcomes.

McKinney et al (1989) demonstrated benefit of active therapy(administered either as mobilisation advice or a package involving active and passive ROM exercises, home exercises and passive electrotherapy) over a group receiving advice to rest (with minimal collar use) and analgesia in terms of short term outcomes (pain and ROM). Advice to mobilise in the early phase reduced the number of patients with symptoms at 2 years.

Borchgrevink et al (1998) demonstrated that acting as usual was superior to 14 days of soft collar immobilisation and sick leave (rest from work) in a number of outcomes including pain during daily activities. It should be noted however, that the clinical effect of treatment would be regarded as small at best.

Gennis et al, (1996) compared subjects who received 2weeks of immobilisation in a soft collar as an adjunct to rest and analgesia to a group that received standard emergency room treatment involving rest and analgesia alone. There was no significant difference between groups in terms of pain and recovery outcomes.

Dehner et al (2006) demonstrated no difference between 2 or 10day collar immobilisation in terms of disability or pain outcomes

Provinciali et al (1996) highlighted the benefits of a multimodal program (passive joint mobilisation, posture correction, relaxation, psychological support) over passive electrotherapy in terms of pain and sick leave.

Brison et al (2005), showed that the use of an educational video (which included reassurance, advice about posture, return to regular activities, exercises and pain relief methods) as an adjunct to usual care in an emergency department was beneficial in reduction of pain and WAD symptoms at 24 weeks.

Oliveira et al (2006) demonstrated highly significant benefits of a 12 minute video (which included education regarding the pathophysiology of whiplash, symptomatology in the first 48hours, treatment in the first 48 hours, treatment after 48 hours including returning to pre-injury activity, recovery period time frame, self-management techniques including muscle

tension reduction and breathing relaxation. There was an overall emphasis on the diagnosis as being muscular in origin) as an adjunct to standard emergency department care in terms of pain, medication use and number of medical consultations.

Ferrari et al (2005) compared an educational pamphlet (involving a 1 page summary with 10 dot points of information from a whiplash book which emphasized reassurance, importance of mobilisation and continuing normal activities) as an adjunct to 'usual care' in an emergency department and demonstrated no benefit of the addition of this pamphlet.

Bunketorp et al (2006) demonstrated in a high quality that supervised physical training involving individualized ROM, stability, proprioception and strengthening exercises was superior to a home exercise program at 3 months. These effects were not maintained at the 9 month follow up.

Scholten-Peeters et al (2006) showed similar effectiveness in terms of pain, headache and work activities between trained doctors care involving education and advice to stay active when compared to physiotherapy education, advice and exercise. Doctors were given training regarding whiplash and appropriate advice for a patient suffering from a WAD.

Foley Nolan, (1992) demonstrated a benefit of pulsed EMG therapy plus collar over a group wearing a collar alone in terms of pain outcome at 2 and 4 weeks and ROM outcome at 12 weeks. However, both groups were required to wear a collar for 8 hours per day for 12 weeks.

Hendriks et al (1996) compared 5 treatments of electrotherapy (low frequency interrupted direct current) as an adjunct to standard emergency department treatment (involving ice, ROM exercises, collar use and advice) and demonstrated a benefit of the electrotherapy in terms of pain immediately post treatment and at 6 weeks.

Thuile and Walzl (2002) demonstrated benefit of magnetic field therapy as an adjunct to standard medication in terms of neck pain and shoulder-arm pain. It is unclear whether these patients were acute, sub-acute or chronic.

Aigner et al, (2006) demonstrated no benefit of laser acupuncture when compared to a placebo control.

Fernandez de las Penas et al, (2004a and b) were found supporting the use of manipulation. In one paper a conventional physiotherapy package was compared to a program involving manipulation and mobilisation of the cervical and thoracic spines. Improvements in terms of pain and ROM were noted in the manipulation group although measurement of outcome was not made at a common time point. The second paper examined dorsal manipulation of the

thoracic spine, again compared to a group receiving a physiotherapy package. Benefits were again demonstrated in the manipulation group.

Passive joint mobilisation has also been shown to be beneficial over rest and collar use when used in combination with active therapy (Bonk et al, 2000; Mealy, 1986). Passive joint mobilisation was also shown to be beneficial when used in combination with multimodal therapy (Provinciali et al, 1996) compared with passive electrotherapy.

Pennie and Agambar, (1990) demonstrated no significant difference in a group receiving traction as part of a physiotherapy package to a group receiving rest and soft collar.

Soderlund et al (2000) demonstrated no benefit of one form of kinaesthetic exercises (involving pressing the head down on points of any imaginary quadrangle whilst lying on their back) as an adjunct to advice and basic exercise in terms of pain or physical measures.

Petersson and Toolanen, (1998) demonstrated the benefits of high dose methylprednisolone administered intravenously in the first 48 hours following admission to a hospital emergency department when compared to a placebo drug. Caution should be taken when considering this treatment given the known side effects of methylprednisolone (Barnsley, 2003).

Gunzberg (1999) was identified which highlighted the benefit of an NSAID (tenoxicam) over a placebo in terms of pain and function (ROM measured with a three dimension measurement device) in a group of 51 acute whiplash patients.

10.2 Treatment of chronic WAD

Jull et al,(2007) demonstrated a benefit in disability and cranio-cervical flexion test scores of a physiotherapy package involving specific muscle control exercises, kinaesthetic exercises, passive joint mobilisation, education and reassurance over a self-management program involving advice to stay active, description of an exercise program and postural and ergonomic advice.

Stewart et al, (2007) demonstrated short term benefit (in terms of pain and disability at 6 weeks) in a group that received an individualized exercise program (conducted with a cognitive behavioral therapy approach) plus advice over a group receiving advice alone. These results were not maintained in the long-term (12 months). Greater benefits were noted for those with higher initial levels of pain and disability.

Fitz-Ritson (1995) found that chiropractic treatment plus 'phasic' exercises (eye-head-neck-trunk and eye-head-neck-arm exercises) produced significantly greater improvements in

disability than chiropractic treatment plus 'standard' exercises (stretching/ isometric/ isokinetic exercises.

Soderlund et al (2001) found no benefit of the addition of a cognitive behavioral approach when added to a standard physiotherapy program for short and mid-term pain outcomes or short and mid term disability outcomes. However significant benefits were demonstrated in ability to perform activities of daily living. The cognitive behavioral approach involved learning basic psychological skills, application to daily activities and maintenance of these skills over time.

Stewart et al (2007) also included a cognitive behavioral component (involving learning of basic psychological skills, goal setting, pacing and education) in the exercise arm of their treatment which was beneficial when compared to advice alone. It is however difficult to separate out the benefit of the cognitive behavioral component from the benefits of active treatment.

Lord et al, (1996) demonstrated a significant benefit in terms of long-term pain relief of radiofrequency neurotomy in WAD patients with confirmed facet joint pain compared with patients receiving a placebo.

Byrn et al (1993) demonstrated) that subcutaneous sterile water injections (2-3mm subcutaneous) over up to 30 trigger points were significantly more effective in providing pain relief and improving ROM immediately and after 8 months than those receiving saline injections.

Barnsley et al (1994) failed to demonstrate any benefit of intra-articular corticosteroid injections over local anesthetic injections in terms of time to return of usual pain.

Freund et al (2002) found short term benefits (4 weeks) in terms of pain and ROM in a group of WAD patients receiving botulinum injections versus those receiving saline. Conversely Padberget al (2007) found no benefit of botulinum injections in 40 WAD patients compared with those receiving placebo injections.

Lemming et al (2005), in a high quality RCT demonstrated 3 different analgesics produced significant reductions in pain compared with a placebo whilst infusion of drugs was taking place. No long term difference in effectiveness of any drug over placebo was demonstrated.

Klobas et al, (2006) demonstrated no benefit of specific jaw exercises on jaw pain as an adjunct to a standard whiplash therapy program involving passive therapy and mobilisation.

Ekvall Hansson et al, (2006) demonstrated a small clinical improvement in terms of postural control and dizziness handicap in a group exhibiting dizziness who received a vestibular rehabilitation exercise program compared to a control group who received no intervention.

11. Summary

In 1995, Spitzer, Skovron et al. found that the epidemiology of whiplash was poorly understood. Since then, two systematic reviews (Côté et al., 2001a; Scholten-Peeters et al., 2003) have given us possibly the clearest picture of the epidemiology of the incidence and prognosis of WAD, despite the conflicting evidence produced. Scholten-Peeters et al. were interested in clinically useful prognostic factors affecting the course of individuals suffering from WAD, whereas Côté et al. were interested in additional factors affecting the course of events following the crash itself. Different criteria were used in each review to assess the level of evidence for several factors affecting the course of WAD, and consequently their conclusions on the importance of some factors differ.

Risk factors for chronicity of symptoms include older age, female sex, initial pain and intensity of headache and initial radicular signs and symptoms. It is apparent that insurance and compensation systems affect the prognosis of whiplash. While initial symptoms are a consistent predictor of chronic whiplash symptoms, the importance of psychosocial factors is more controversial. However a psychosocial view of whiplash injury, in which the transition from acute to chronic injury is significantly determined by non-crash factors, offers an explanation for the variation in the time taken to recover from whiplash symptoms.

There has been much criticism of the biomedical model, but also an acknowledgement of the enormous advances in healthcare that have been made under its rule (i.e. Alonso, 2004; Walker et al., 2004).

The evidence in relation to WAD and a possible rationale for the syndrome is outlined below:

1. The biomedical model of WAD seeks to relate the degree of tissue damage to reported symptoms.
2. The biopsychosocial model of WAD considers biological, psychological and social factors in relation to the course and treatment of WAD.
3. Some biological factors play a role in WAD. A number of structures have been implicated as the cause of symptoms, and particular evidence is available regarding the zygapophyseal joints in the cervical spine, at least in some cases. Apart from this, definitive findings are lacking.

4. Psychological states including depression and anxiety, as well as cognitions such as catastrophising, resulting from whiplash injuries, have a role to play in WAD. The evidence suggests personality or temperament factors do not play a role.
5. Social factors, particularly the compensation system and cultural expectations, have a role to play in WAD. The exact extent of malingering in WAD is unknown.
6. Peripheral and central sensitization, provide a rationale for development and presence of chronic WAD.

What these findings suggest is that using a biopsychosocial approach for the treatment of WAD appears to be appropriate. In fact this is the predominant view at present (Ferrari, 2002; Hendriks et al., 2005; Solomon, 2004). However, what seems to have been lacking in the literature is a reason why such an approach should be adopted, aside from the findings that all these issues seem to have an impact.

Bogduk (2003) raises questions of validity of the diagnosis and effectiveness of treatment in WAD. The fact that it is not possible to make a specific patho-anatomic diagnosis of acute WAD may not be important given that the majority of cases recover within weeks to months. Accuracy of diagnosis becomes more important in chronic cases where symptoms persist for six months or more and there is potential for the downward spiral of chronic pain and disaffection, complicated by ineffective treatments, insurance claims and legal proceedings.

There is very little sound evidence on which to base judgments of the effectiveness of different treatments. The exception is the use of diagnostic blocks of the cervical zygapophyseal joints followed by radiofrequency neurotomy of the affected joint(s). These procedures are very effective, but technically demanding for both the operator and the patient.

There is rather weaker evidence of the effectiveness of other treatments and of the non-effectiveness of yet other treatments. The influence of insurance company policies and their administration has been demonstrated in British Columbia, where changes based on social science principles were successfully introduced to make the claims process more part of the solution and less part of the problem.

There is a clearly evident need for research aimed at identifying effective treatments for chronic WAD, particularly those cases with no identifiable cervical zygapophyseal joint injury. It is only by encouraging the sound evaluation of all aspects of handling WAD cases that effective treatments and procedures will be identified.

The quality of evidence available upon which to judge the effectiveness of treatments for acute and chronic WAD is not high. There is nevertheless enough consistency in the findings of the studies included in the systematic reviews to indicate that some approaches to treatment

are more effective than others. Acute WAD is best treated with early physical activity and active treatments, rather than with passive treatments. For chronic WAD (i.e., cases where symptoms have persisted for more than six months), radiofrequency neurotomy is effective in cases where diagnostic blocks have indicated the presence of injury associated with the cervical zygapophyseal joints. The combination of cognitive behavioral therapy with physical therapy interventions has also been found to be effective.

12. List of figures

13. References

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