Centre for
Automotive Safety Research

Whiplash associated disorders: a comprehensive review

Edited by RWG Anderson

CASR REPORT SERIES
CASR016
April 2006

THE UNIVERSITY
OF ADELAIDE
AUSTRALIA

Report documentation

REPORT NO.	DATE	PAGES	ISBN	ISSN
CASR016	April 2006	81	1 920947 15 9	1449-2237

TITLE

Whiplash associated disorders: a comprehensive review

AUTHORS

RWG Anderson, TJ Gibson, M Cox, GA Ryan, RT Gun

PERFORMING ORGANISATION

Centre for Automotive Safety Research
The University of Adelaide
South Australia 5005
AUSTRALIA

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Motor Accident Commission
GPO Box 1045
Adelaide
South Australia 5001
AUSTRALIA

AVAILABLE FROM

Centre for Automotive Safety Research
http://casr.adelaide.edu.au/reports

ABSTRACT

This report is a compendium of papers on aspects of whiplash associated disorders (WAD). The aim of the report is to provide an overview of WAD from different perspectives: epidemiological, engineering, biomechanical, biopsychosocial, and treatment. Two recent studies on WAD in South Australia are also reported. The findings from studies published up until August 2005 are included in this report.

Whiplash associated disorders are a complex phenomenon, triggered by a mechanical event but whose prognosis is affected by many factors including clinical and psychosocial factors. A thorough understanding of these factors provides a basis for dealing with the prevalence of WAD in the community and reducing the incidence of WAD.

KEYWORDS

Whiplash, Injury, Biomechanics, Vehicle safety, Medical treatment

Preface

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.

Much of this literature reveals sharp divisions in opinion over certain aspects of whiplash, specifically related to the importance of psychosocial factors and the effects of compensation on the prognosis of acute pain due to whiplash, in the absence of overt pathological signs. Compounding this are further divisions in the literature along 'professional' lines; for example the engineering literature on vehicle factors and biomechanics does not always acknowledge or incorporate findings from epidemiological and clinical studies of whiplash. The reverse is also true.

In the midst of all of this, it is often difficult to get an overview that encompasses all aspects of whiplash research. In this report our aim has been to present overviews of the whiplash phenomenon from different perspectives: epidemiological, engineering, biomechanical, biopsychosocial, and treatment. Finally we present the results of two recent studies in South Australia that examine factors that affect the outcome of whiplash injury.

Each chapter has been written independently so the report may be read as a compendium of papers on the subject of whiplash injury. As each author has reviewed current knowledge from a particular perspective, it is inevitable that conclusions on some matters have been drawn differently in some instances: it was not an aim to necessarily produce a unified view on the mechanisms of whiplash-associated disorders.

However, a consistent theme throughout this report is the complexity of whiplash-associated disorders. 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 aetiology and prognosis of whiplash by encompassing biomechanical, biomedical, social and psychological factors.

A biopsychosocial approach does not discount the importance of seeking a mechanism of whiplash injury in biomedical terms, and it is likely that biomechanics will explain much about the incidence of whiplash injury, although the exact mechanisms are yet to be fully elucidated. Nevertheless, interventions, such as 'active' headrests, designed to minimise extreme neck motions during rear impact are showing some success in reducing the incidence of the acute injury.

For those who sustain whiplash injury, the prognosis is generally good, with a high rate of recovery. However, a small proportion of cases do go on to have chronic complaints. The characteristics of these chronic complaints and their origins seem to generate the most debate.

About the authors

ROBERT ANDERSON

Dr Robert Anderson is a mechanical engineer with a PhD in engineering for studies on the biomechanics of axonal injury in the brain. He is a Senior Research Fellow and Deputy Director of the Centre for Automotive Safety Research. His research interests span injury biomechanics and epidemiology, vehicle safety and road safety issues.

TOM GIBSON

Tom Gibson is the founder and principal engineer of Human Impact Engineering. He has over 20 years experience in the area of the biomechanics of impact injury causation and mitigation. In 1984 and 1985, with a background in automotive design and development, Tom helped establish a multi-disciplinary crash investigation team investigating head injury mechanisms. He developed a special interest modelling of injury causation and the simulation and design of safety systems. Tom has applied his knowledge of human tolerance to the design, evaluation and development of specialised protective systems for different applications.

MARK COX

Mr Mark Cox graduated with a Bachelor of Applied Science (Physiotherapy) in 1993. After a period of 4 years, treating accident injury and work injury patients in the US and Australia, Mark undertook a Bachelor of Psychology (Honours), completing it at the end of 2004. Mark is currently studying for a Master of Psychology (Clinical) at the University of Adelaide as well working in private practice as a Physiotherapist part time, treating mostly accident injury and work injury patients.

TONY RYAN

Dr Ryan graduated in medicine from the University of Adelaide, South Australia, in 1959, gaining an MD in 1967 for an in-depth study of road traffic crashes and injuries. After being awarded an MPH at Harvard School of Public Health, Dr Ryan spent 18 years at the Department of Social and Preventive Medicine, Monash University, in Melbourne, Victoria, followed by 5 years as Deputy Director of the then NHMRC Road Accident Research Unit at the University of Adelaide. He was Director, Road Accident Prevention Research Unit in the Department of Public Health, University of Western Australia, from 1993 until March 2000. He has published extensively on the epidemiology of road traffic injury and the biomechanics of head, spine and other injuries. He has acted as a consultant for the World Health Organisation in the evaluation of national and international road safety programs and policies.

RICHIE GUN

Dr Richie Gun MB BS, FAFOM, is a medical graduate of the University of Adelaide, specialising in occupational medicine. With Dr Orso Osti he has recently completed two studies on predictive factors of prolonged disability following whiplash injury.

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1 Epidemiology of whiplash associated disorders

Robert Anderson

1.1 Introduction

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 aetiology 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 aetiology is understood and, it seems, even less is agreed upon in the literature.

The aim of this chapter has been to present and describe studies on the epidemiology of WAD, and the intention was to focus on what one might rate as being of the highest level of evidence: meta-analyses, systematic reviews, double-blind randomised controlled trials, case-control studies and well defined and controlled cohort studies. In fact, very few such studies on WAD exist. Nevertheless, there have been several recent systematic reviews that, while subject to some criticism, have served as a guide for this chapter (Spitzer et al., 1995; Côté et al., 2001a; Scholten-Peeters et al., 2003).

1.2 Definitions

1.2.1 Whiplash associated disorders

The term "whiplash-associated disorder" is used to describe the clinical manifestations of whiplash injury. The Québec Task Force on Whiplash-Associated Disorders[1] describes these entities thus:

> *"Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck. It may result from rearend* (sic) *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 (whiplash injury), which may lead to a variety of clinical manifestations (Whiplash-Associated Disorders)."* (Spitzer et al., 1995)

1.2.2 Quebec Classification of Whiplash Associated Disorders

The Québec Classification of Whiplash-Associated Disorders (Table 1.1) was devised by the Québec Task Force in 1995 to assist clinicians in making decisions about the treatment of whiplash injury and symptomatology. It was also proposed to allow research on WAD to be evaluated (Spitzer et al., 1995). The intention was that the classification be refined as more research was performed.

[1] The Québec Task Force on Whiplash-Associated Disorders was formed in 1989 by the Société d'assurance automobile du Québec (SAAQ) to provide an in-depth analysis of the "whiplash problem" (Spitzer et al., 1995).

Table 1.1 The Quebec Classification of Whiplash-Associated Disorders

Grade	Clinical presentation
0	No complaint about the neck No physical signs
I	Neck complaint of pain, stiffness or tenderness only No physical signs
II	Neck complaint, and Musculoskeletal signs*
III	Neck complaint, and Neurological signs†
IV	Neck complaint, and Fracture dislocation

* Musculoskeletal signs include decreased range of motion and point tenderness

† Neurological signs include decreased or absent deep tendon reflexes, weakness and sensory deficits

The implication is that the Classification is an ordered categorical scale with each successive grade severer than the previous one (Spitzer, Skovron 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 alongside an individual with only mild pain.

It is also worth noting that the Classification was devised with two axes: the grade of WAD that corresponds to severity (Table 1.1) on a "clinical-anatomic" axis and a second grade on a time axis that categorises, for each clinical-anatomic grade, the time to recovery: The time categories are: less than 4 days, 4-21 days, 22-45 days, 46-180 days and greater than 180 days (chronic).

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, Brison 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'assurance automobile 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 (see later sections).

1.2.3 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, radicular2 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 (see later sections). 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 (Rø et al., 2000).

1.3 Incidence of WAD

The true incidence of whiplash is difficult to determine, as routine data may not adequately characterise 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 population; 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 population per year (Motor Accidents Authority of NSW, 1999). It should be borne in mind that these differences are just as likely to arise from differences in compensation criteria and coding than from any real underlying phenomenon.

1.4 Systematic reviews of prognostic studies of WAD

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). These reviews, and that of Spitzer et al. (1995) are briefly described and, in following sections, their main findings are expanded upon, supplemented by findings from more recent research reports where appropriate.

Often, systematic reviews can be (purposely) narrow in their consideration, and are designed to answer a specific question. Therefore, the omission of a research report from such a review does not necessarily invalidate the report – it may simply mean that the report did not aim to answer the specific question at hand.

1.4.1 Spitzer et al. (1995)

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 of 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.

[2] Pertaining to the nerve root, radicular signs and symptoms include arm and shoulder pain, loss of muscle strength and sensation abnormalities.

1.4.2 Côté, Cassidy et al. (2001a)

Côté et al. (2001a) (referred to henceforth also as PC) 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). PC propose a conceptual model with which to categorise and systematically review the literature on the prognosis of acute whiplash injury. This model categorises studies on three axes, the first being the "Target Population and Generalisability" of the study. On this axis, a study can be categorised 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 categorised 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 categorised 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. These conclusions are discussed in more detail in Sections 2.5.1 *et seq*.

1.4.3 Scholten-Peeters et al. (2003)

Scholten-Peeters et al. (2003) (henceforth referred to also as GSP) 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 PC, included some studies rejected by PC and, further, they placed a different weight on the evidence produced by studies accepted by both reviews.

GSP 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 PC, GSP 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 PC, GSP applied a systematic ranking protocol to various cohort studies although the criteria were somewhat different in the two studies. Most significantly, GSP 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.)

GSP'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.

1.5 Risk factors in the aetiology and prognosis of whiplash

1.5.1 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, Kullgren 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 towbar at the time of the crash. Crash testing revealed that cars fitted with a tow bar generated higher accelerations than those without.

Later, Krafft, Kullgren and Ydenius (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 minimise whiplash have focussed 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 minimising harmful motions of the neck (Farmer et al., 2003). See Chapter 3 for further discussion on this point.

1.5.2 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, Harder 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 PC and GSP approach consensus on the prognostic value of initial pain. PC found that baseline neck pain, headache and radicular signs and symptoms were predictive of delayed recovery in whiplash patients. GSP also concluded that baseline pain was predictive of delayed recovery. However GSP concluded that there was little prognostic value in initial headache and radicular signs.

HISTORY OF NECK PAIN

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

Bovim, Schrader and Sand (1994) used a randomised 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.

1.5.3 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 - see Chapter 7) 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. PC 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) analysed 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.

GSP 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 GSP 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 GSP 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 GSP 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 PC, but not with GSP.

Satoh, Naito 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 regression showed that females were more likely to still be receiving treatment after 6 months (odds ratio 1.43). Consistent with other findings, lack of immediate symptoms also were significant prognostic factors for recovery within 6 months.

One of the studies reviewed by GSP (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 PC, 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 (c.f. previous discussion) and so the same factors appear to relate to the incidence of WAD. Croft, Lewis 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.

1.5.4 Psychosocial factors

Psychosocial factors affecting the prevalence of chronic WAD are discussed in detail in Chapter 5 in this report. Generalising, 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, Lord 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 criticised for needlessly relying on a "dualistic" and "linear" view of whiplash (Kwan and Friel, 1998) and for restricting the interpretation of the data to preclude a biopsychosocial explanation. (See Kwan and Friel, 1998, for a brief summary of the biopsychosocial approach to the whiplash problem. For a fuller account, see Ferrari and Schrader, 2001.)

Several epidemiological studies have examined the role of psychological factors in the outcome of acute whiplash. Mayou and Bryant (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. As noted previously, this finding appears elsewhere in the literature and is supported by PC and GSP (see Section 1.5.1). Mayou and Bryant conclude that the hypothesis that psychological factors are important aetiologically 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.

GSP 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 GSP 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 preclude the possibility that these factors are a consequence of pain rather than a component of the aetiology of the pain.

1.5.5 Litigation and compensation

Of all factors that have been proposed to explain the prognosis of whiplash-associated disorders, the most contentious is the effect of compensation and litigation 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. GSP, amongst others, have criticised the study of Cassidy et al., (2000) in which time-to-claim-closure was used to indicate recovery, despite the authors 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 and Mior, 1991), the authors analysed 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-end 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 criticised 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 and Croft, 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. Chapter 7 of this report describes 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 tentative conclusion is that the pursuit of compensation through a lawyer adversely affected the prognosis of the injury.

In contrast, GSP 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, GSP 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.

1.5.6 Jurisdictional factors

PC 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 1.5 months (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 aetiology of chronic WAD.

1.6 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.

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2 Vehicle factors in whiplash injury

Tom Gibson

2.1 Introduction

Whiplash injury is examined in this chapter from the perspective of an automotive engineer. For the automotive engineer to be able to design vehicle systems to minimise injuries such as whiplash, the engineer must understand the incidence of the injury, the mechanisms of injury and the human tolerance to the injury.

In this chapter, the results of several field accident studies are reviewed. These studies explored the association between the incidence of whiplash associated neck injury with variables about the crash and the injured occupant. The review is extended to studies that have investigated specific vehicle factors known to be important in whiplash injury causation. These vehicle factors include the structural response of the vehicle, aspects of the design of the seat and the head restraint and the possibility of interaction with other restraint systems. Test methodologies, both regulatory and consumer information based, being developed to minimise the incidence of whiplash injuries in the field are described and the need for an accepted dynamic seat and head restraint test methodology with a matching anthropomorphic test device (ATD) and injury criteria is discussed.

2.2 Accident studies

The analysis of the detailed field accident data can provide useful insights into those human characteristics and vehicle factors that influence the incidence of whiplash injuries in vehicle crashes. The collection of field data on a sufficient number of accidents with the required precision is both difficult and expensive to carry out. The following studies have been important in giving insight into the characteristics of soft-tissue neck injury associated with whiplash.

One of the earlier field accident studies by States et al. (1972) is often referred to and is still relevant. This study included all rear-end crashes reported in Rochester, New York over a three-month period. The cases were followed-up through special police forms, telephone interviews and mail questionnaires. Approximately every 20th case was investigated more thoroughly using vehicle photographs and medical examinations. Data on a total of 691 rear-end crashes were collected, and the following observations were made (none of which were regarded as statistically significant):

- Whiplash was the principal injury to occupants of struck vehicles, totalling 99.3% of all injuries and occurring in 38% of all rear-end crashes. Based on initial police reports, only 17% of all rear-end crashes were determined to have caused a whiplash injury. The proportion rose to 38% after follow-up questionnaires and interviews by the researchers revealed more cases of whiplash injury;
- Head restraints were effective in reducing injury for both the driver and right front passenger. The frequency of crashes causing whiplash injury was 37% where the struck vehicle was head restraint-equipped and 42% in crashes where the struck vehicle was not fitted with head restraints;
- The benefit of head restraints appeared to be more noticeable for female occupants, for whom the frequency of whiplash injury was 38% for those seated in vehicles with head restraints and 51% for those without the benefit of the head restraint;

- Occupants with fixed head restraints appeared better off and had a whiplash injury frequency of 13% (based on the initial police report) compared with a rate of 21% for occupants with adjustable restraints (also based on initial police reports);
- Seat back damage showed no effect on whiplash injury frequency;
- The overall whiplash injury frequency for females was 44%, and 35% for males;
- The whiplash injury frequency for rear and centre front seat occupants was very low at 22%, which was associated with the high usage by younger and smaller occupants;
- The use of lap belts seemed to increase the whiplash injury frequency.

An Australian study by Ryan et al. (1993) followed up 32 individuals with neck strain following a car crash. The subjects were interviewed and given a physical examination soon after the crash and again after six months. Each case vehicle and crash site was inspected and the crash reconstructed. The severity of the crash was assessed by measuring the maximum vehicle residual deformation and estimating the change in velocity. In 22 cases, the impact originated from the rear; the remainder were from the front or side. Neck strain occurred as a result of low severity impacts, with six cases having a velocity change of less than 10 km/h and eight cases resulting in a maximum vehicle residual deformation of less than 50 mm. For rear impacts, maximum residual deformation and velocity change were positively associated with the measures of neck strain severity. Six months after the impact, 19 (66%) of the 29 subjects available for follow-up still showed evidence of injury. There was no statistically significant association between either measure of crash severity and persistence of neck strain at six months. Subjects who were aware of the impending impact were found to have less severe symptoms initially, and were much less likely to experience persisting problems.

A more recent study in the UK by Morris and Thomas (1996) on soft-tissue neck injury was based on the Cooperative Crash Injury Study (CCIS) which commenced in 1983 and ended in 1992. This database contains 11,866 occupants and 6,973 crashed vehicles. They found the following:

- The incidence of soft-tissue neck injury (STNI) for all accident types was found to have been increasing steadily over the data collection period, from 11.2% in 1984 to 22.8% in 1991;
- There was a distinct gender effect for all accident types with the soft-tissue neck injury rate for females increasing at a faster rate than for males. The rate for females had climbed from 14% in 1984 to 31% in 1991, while for males the rise was from 10% in 1984 to 18% in 1991;
- Soft-tissue neck injury occurred in all impact directions with an average injury rate of 16%, but for rear impacts the rate was 38%.
- Seatbelt use increased the overall likelihood of soft-tissue neck injury, with 20% of restrained occupants compared with 8% of unrestrained occupants sustaining neck injury. Seatbelts increased the injury likelihood for males but had no effect on females;
- Rear impacts resulting in neck injuries occur at a lower average speed of 32 km/h compared to 36 km/h for those without injuries. The critical speed for these impacts was between 17.5 and 27.5 km/h;
- Occupant and seating characteristics have some effect. Notably, neck injured females appeared to be using higher head restraints, and were younger and lighter than non-injured females;
- The type of head restraint showed no differences in effectiveness; and,
- There was some tendency for reduced STNI when the seat back was deformed.

Temming (1998) analysed the Volkswagen accident database to investigate the significance of human factors, such as gender, age, height and weight, on the frequency of whiplash

injuries in rear collisions. It was shown that for all combinations of parameters (belted, belted with multiple collisions, and belted with single rear collision) female occupants had a risk of neck injury of between 1.9 and 2.4. times that of male occupants

Temming suggested the following explanations for the gender-specific differences:

- Men have stronger neck muscles, as indicated by the ratio between head volume and neck cross-sectional area;
- Women have longer necks and larger heads relative to their own body weight than men;
- Women sit farther forward in their seats than men; and
- Women may be more inclined to file an insurance claim for whiplash than men.

A Swedish study (Krafft, 1998) found that women with whiplash injuries are more likely to develop long-term symptoms of whiplash than are men with whiplash. In this study 55% of the women and 38% of men who sustained whiplash injuries went on to develop longer term symptoms.

2.3 Engineering for rear impact injury prevention

Engineering interventions aimed at reducing the incidence of crash injury can be designed to influence pre-crash events (reducing the likelihood of the crash), or the crash event itself (minimizing the risk of injury in the crash; see Figure 2.1. Engineering design changes can be applied to the vehicle structure to modify its performance during the crash, or to the individual vehicle components such as seats. The process of designing to minimise an injury requires detailed understanding of the likely injury mechanisms and the availability of appropriate injury tolerance data.

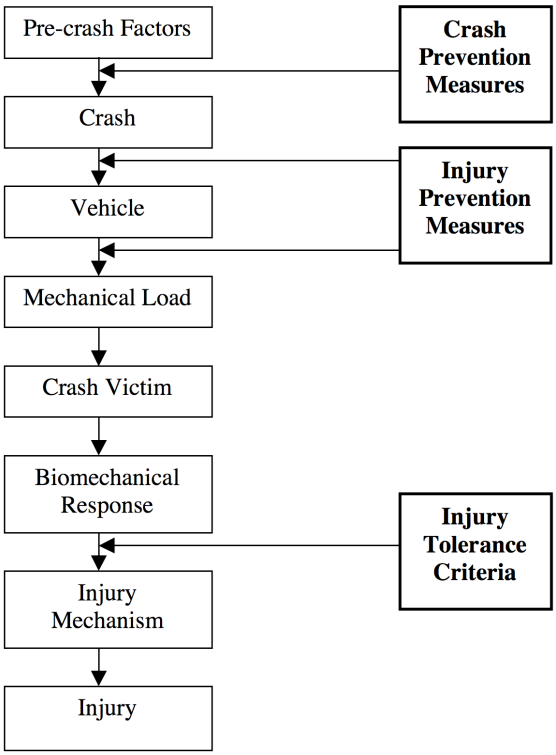

Figure 2.1 The biomechanical injury/load model showing how engineering interventions can reduce crash related injury (Wismans, 1995)

2.3.1 Crash prevention

Certain vehicle features are designed to reduce the likelihood of the crash occurring. Road vehicles have undergone numerous improvements in the performance of braking systems and consistency of operation (such as the user ergonomics, road holding and wet-weather performance). Further improvements in crash prevention are likely to arise mainly from developments in intelligent vehicle systems.

Design changes to vehicles that incorporate modern instrumentation, computing and telecommunications are improving vehicle safety, ergonomics and convenience. Examples of technologies of this type that have already been widely adopted, include anti-lock braking systems (ABS), traction control, and vehicle-attitude control systems, but these have yet to be proven effective in preventing rear-end crashes. Some of the emerging technologies, such as vehicle proximity control, which will allow lane following and obstacle avoidance systems, appear to be likely to have an effect on reducing rear-end crashes.

Road safety engineering can be used to effectively reduce whiplash injury. Whiplash injury has been show to be associated with rear-end impacts, which are common at urban signalised intersections (Navin, Zein & Felipe, 2000). Preventative measures may be undertaken by road engineers to reduce the frequency of rear-end impacts. Several such countermeasures have been designed and deployed in British Columbia. These range from enhancing signal visibility to intersection geometric upgrades and have been proven to be a cost effective method of reducing insurance claims. Some examples of the countermeasures with demonstrated improvements in reducing rear-end crash frequency include:

- Implementation of left and right turn lanes;
- Improvement of intersection lighting;
- Resurfacing of slippery road surfaces;
- Improvement of intersection geometry by realignment;
- Removal of inconsistent signal timing;
- Improvement of signal visibility;
- Improvement of sight distance; and,
- Improvement of pedestrian crossing conspicuity.

2.3.2 Injury prevention

Certain aspects of vehicle structural design can be used to reduce injury once the vehicle is involved in a crash. These interventions are aimed at reducing the mechanical load applied to the victim during the crash. This can be achieved in two ways: the first is by changing the mechanical response of the vehicle in the crash. An example is the incorporation of crushable zones in the rear structure of a vehicle for the purpose of reducing the severity of the crash pulse generated during a rear impact. The other way is to affect the manner in which the load from the vehicle structure during a crash is transmitted to the victim by:

- Promoting ride down;[3]
- Spreading the energy of the impact in a manner appropriate to the body region being loaded;
- Managing the rate of energy transfer by the use of padding materials; and
- Preventing excessive relative motions between the body segments of the crash victim.

[3] Ride down is the term used to describe the coupling of the occupant to the vehicle structure with a safety restraint system to minimize the acceleration levels applied to the occupant, by using as much of the deceleration distance afforded by the deformation of the structure of the vehicle as possible.

Applying these principles to the protection an occupant in a car during a low speed rear impact requires several improvements to typical vehicle designs. The rear structure of the vehicle must be designed to minimize the severity of the deceleration pulse without compromising the structural integrity in a high velocity crash. This must be combined with a seat system designed to promote effective ride down of the occupant of the rear impact crash pulse, while minimising misalignment in the neck and the possibility rebound following the impact.

The injury tolerance level referred to in Figure 2.1 is the threshold of the magnitude of loading at which injury to a specified level of severity is likely to occur to a human[4]. There are large variations in the tolerance to mechanical loading between individuals, dependent on factors such as gender, age, and anthropometric and physiological differences (Yoganandan, Pintar and Cusick, 1997). The tolerance to loading describes the response of living humans to crash loads, and therefore such tolerance values are only of limited use to engineers, who are obviously not going to use human subjects to verify or test new designs. So, the assessment of a specific design during the product development process requires the use of a test methodology based on a surrogate, usually a mechanical crash test dummy, and associated test criteria, the criteria being based on knowledge of human tolerance to mechanical loads.

2.4 Vehicle factors

2.4.1 Effect of head restraints

Kahane (1982) evaluated the effectiveness of head restraints, that comply with the US Federal Motor Vehicle Safety Standard (FMVSS 202), in reducing the overall risk of injury in rear impacts. The study was based on an analysis of the US National Crash Severity Study, the National Accident Sampling System and the Fatal Accident Reporting System. It estimated the reduction in risk of injury to be 17% for integral restraints and 10% for adjustable restraints. The effectiveness of head restraints in reducing whiplash could not be determined, *per se*, because the data did not specifically code this injury type. The in-use median height for adjustable head restraints was found to be less than 660 mm as compared with greaterthan 710 mm for the integral restraints. Considering that the height to the base of the skull of a 50th-percentile U.S. male, seated erectly, is 700 mm, Kahane hypothesised that head restraint heights above 700 mm should give full injury prevention benefits.

Nygren, Gustafsson and Tingvall (1985) found that, consistent with other studies, the use of head restraints decreased the risk of neck injury in a rear-end collision by approximately 20%. Fixed head restraints reduced the risk by 24% and adjustable ones by 14%.

In a study of 33 occupants of Volvo cars, Olsson, Bunketorp and Carlsson (1990) found that neck symptoms lasted longer for increasing horizontal distance between the head and the head restraint. The authors found that a head restraint back-set of more than 10 cm from the back of the head correlated with an increased risk of neck injuries in rear impacts. They also suggested that the risk of whiplash injuries could be decreased by using controlled plastic deformation of the seat back rest integrated with the head restraint, to diminish the relative motion of the head and trunk.

A Dutch study of front-seat occupants found that roughly 40% of male occupants and 50% of female occupants had adjusted their head restraints to the "correct" height – when the top of the head restraint is at least level with the ear or higher (van Kampen 1993). However, it should be noted that a seat that meets the minimum height requirement of the European vehicle regulations enables correct height adjustment only for males shorter than the 25th percentile and for females shorter than the 90th percentile of the Dutch population.

[4] The injury severity level is usually measured by means of the Abbreviated Injury Scale [AIS], which is a six point scale from 0 for no injury to 6 for fatally injured.

2.4.2 Vehicle structural effects

Krafft et al. (2000) studied the influence of crash severity in rear impacts that led to short-term or long-term WAD (i.e. injuries lasting less than or more than one year). The study was completed in three stages.

In the first part of the study, Volvo 240 model vehicles were tested with and without towbars fitted. The fitting of a towbar to a car requires extra structure to be added to the vehicle behind the rear axle and has a stiffening effect. This change to the vehicle structural stiffness at the rear allowed the effect of the vehicle structure on the incidence of neck injury to be investigated. Full-scale crash tests were run at 25 km/h producing a change in velocity in the struck car of 15 km/h. The acceleration produced in the towbar-equipped car by the rear impact was higher (9.6g) than in the car without a towbar (8.0g). The occupant of the towbar-equipped vehicle was also subjected to higher accelerations. The dummy in the towbar-equipped car had a maximum T1 acceleration of 8.9g, compared to 6.7g in the car without a towbar.

In the second part of the study, data was collected from the Folksam insurance company between 1990 and 1993. A random sample of occupants of Volvo 240, Volvo 700 and Saab 900 (1979-1993) model cars, with soft tissue neck injuries, was selected. In 233 crashes, the subject suffered only acute symptoms whereas subjects suffered chronic symptoms in 75 of the crashes. Occupants who suffered chronic symptoms were more likely to have been seated in a car with a tow-bar fitted (odds ratio 1.22).

In the final part of the study an additional 28 rear impacts were collected by Folksam in which a crash pulse recorder was mounted under the driver's seat. The vehicles were inspected and the seat-back deformations were measured. Medical notes and questionnaires were obtained, and possible medical symptoms were followed up at least 6 months after the collision. Fifteen occupants in 11 collisions did not suffer symptoms to the neck. The maximum acceleration levels in these collisions, as measured by the crash pulse recorder, was no more than 6 g. In fifteen collisions, 20 occupants had only acute symptoms and the maximum accelerations did not exceed 10 g. Two occupants had chronic symptoms. In these cases, the accelerations reached 13g and 15g. Krafft et al. (2000) concluded that the stiffness of the vehicle structure influences the severity of WAD outcomes.

2.4.3 Vehicle seat response effects

Foret-Bruno et al. (1991) analysed a French vehicle accident database containing 8,000 crash involved vehicles, and made the following conclusions: deformation of the seat back reduced the incidence of cervical injury in rear impacts; and elastic rebound of the seat back following a rear impact leads to increased neck loading, which the authors demonstrated with a series of sled tests involving dummies and a cadaver.

Parkin et al. (1995) used data from the UK based Cooperative Crash Injury Study. The authors looked specifically at the relationship between seat damage and AIS1 neck injury and reported the following:

- Rear impacts only made up 6.0% of the total of 5,361 crashes studied;
- The seats with no damage became less frequent as the severity of the collision increased;
- An occupant was significantly more likely to suffer AIS1 neck injury if the seat was undamaged; and
- The frequency of AIS1 neck injury was not related to impact severity. This may be related to the greater likelihood of the seat collapsing as the impact severity increases.

Haland et al. (1996) evaluated the difference in the behaviour of the car structure or the seat for two car models: the Opel Corsa and the Peugeot 205. According to the statistics from a Swedish insurance company the Opel had the highest rate of neck injury in crashes, whereas the Peugeot had the lowest rate of neck injury. The vehicle structural responses in rear impacts were compared. The study examined the relative motion between the upper and lower cervical spine in the recently developed rear impact dummy (RID) neck, mounted on a Hybrid III dummy. The researchers were looking for a limited formation of an "S-shape" in the cervical spine, which occurs in the neck during a rear impact. It was found that:

- The standard Opel Corsa seat was worse than the standard Peugeot 205 seat because the neck motion during impact was faster;
- A strengthened standard seat performed worst because the limited deflection (40 mm) increased the velocity of the lower neck; and,
- A modified seat with an increased seat back deflection of 130 mm reduced the velocity of the lower neck.

The authors concluded that seat responses were more important than the vehicle structural responses in mitigating whiplash injuries, and the differences in the performance of the Opel and the Peugeot were to the effects of the seat design alone.

Another Swedish study was based on the same Folksam insurance data of 554 occupants in 195 crashes, as reported in Krafft et al. (2003). It was found that females seated in the rear were at significantly higher risk of injury in a rear impact than when seated in the front. The effect was not as strong for males. This is consistent with the rear seat being likely to be stiffer than the front seats of a vehicle

2.4.4 Seatbelt effects

In a series of sled tests, Viano (1992a) studied occupant retention by the seat during rear impact. Viano found that the retention of an unrestrained (unbelted) dummy was dependent on the degree of deformation of the seat back, which was in turn dependent on the severity of the impact. The limit at which an unrestrained dummy would be retained in its seat was found to be 60° of seat back deformation. Beyond this point, the rearward acceleration of the test dummy was enough to overcome the friction involved, and caused it to ramp up the seat and over the head restraint. For a standard seat designed to meet FMVSS 202 criteria (see Section 2.5.1), this was observed when the peak of the acceleration pulse reached 15.5 g with a velocity change of 9.6 m/s. In another related study, Viano (1992b) demonstrated that lap belt use improved retention of the dummy in the seat in rear impacts and so reduced ramping. This was particularly evident in slightly offset rear impacts (15°), in which the pelvis was kept engaged with the seat.

The effects of shoulder-belt geometry in rear-end collisions were analysed by Krafft et al. (1996), by comparing the outcomes of 2 and 4-door Volvo 240 cars (1975-1994) and 3 and 5-door Saab 900 cars (1979-1993). In these vehicle models, the 2 and 3-door vehicles have the seatbelt shoulder anchorage mounted 27 cm and 23 cm farther back than their respective sedan counterparts. Accident data reported by the police to the Swedish National Bureau of Statistics was used in the study. It was found that the weights of both the struck and striking vehicles, the gender of the occupant, and the seatbelt geometry as indicated by whether the cars were hatchbacks or sedans (with the sedans fairing worse), all influenced the relative risk of soft tissue neck injuries. The authors concluded that the influence of the seatbelt geometry added support to the hypothesis that rebound from the seat is an important part of the injury mechanism for whiplash-associated disorders.

2.4.5 Airbag effects

The effect of airbag deployment on the incidence of whiplash is not clear. Otte (1995) reviewed 41 motor vehicle accidents in Hannover in which airbags were deployed. He found

that half of all airbag inflations caused soft tissue neck injury. Otte concluded that inflation of an airbag induces an extreme motion of the head and cervical vertebrae, giving a higher risk of these whiplash injuries. Conversely another German research group (Langweider, Hummel and Müller, 1996) suggested that the deployment of driver side airbags resulted in fewer soft tissue neck injuries. These researchers proposed that the interception of the head motion with the airbag prevents the hyperflexion of the cervical spine.

In a study for the Australian Transport Safety Bureau (ATSB), Morris et al (2001) evaluated the effectiveness of the Australian Design Rule 69, (Full Frontal Impact Occupant Protection). The study of real crashes compared the injuries and HARM[5] to occupants of vehicles equipped with and without SRS airbags. In terms of whiplash-associated injuries, it was found that 19% of drivers in the airbag cases (n=291) suffered AIS1+ neck injuries, compared with 30% in the non-airbag cases (n=141). The combination of seatbelts with or without airbags gave similar results: 19% of belted drivers with airbags (n=253), and 31% belted drivers without airbags (n=130) suffered AIS1+ neck injuries.

2.4.6 Seat design developments

The first vehicle equipped with a safety system specifically aimed at reducing whiplash associated injury was the SAAB. The aim of the Viano and Olsen (2001) study was to evaluate the field performance of the Saab Active Head Restraint (SAHR) in reducing whiplash in rear crashes. Comparisons were made of single-event rear-end crashes involving Saab 9-5/9-3 equipped with SAHR and Saab 9000/900 fitted with standard head restraints, over a period of 18 months. The design of the SAHR aims to ensure a horizontal trajectory of the head restraint, to lower the loads in an occupant's neck during rear impact. The seat modifications introduced with the SAHR system also addressed lower back injury risk. The seat provides uniform support of the spine by removing the stiff cross-seat structures adjacent to the thoracic and lumbar spine. In the field, the SAHR reduced whiplash injury risks by 75% (±11%): An 18% (±5%) incidence in 85 occupants with standard head restraints to 4% (±3%) in 92 occupants with SAHRs. No SAHR-fitted seats required repair or replacement after the crashes. The SAHR was found to be effective in reducing the incidence of medium to long term whiplash-associated disorders in a sample of rear crashes in Sweden.

Recent research has investigated the effectiveness of these new head restraint and seat designs in reducing neck injury in rear impacts. An Insurance Institute for Highway Safety study was based on the claims data supplied by three of the major US insurance companies, Nationwide, Progressive, and State Farm, (Farmer et al., 2003). Three different seat and head restraint design approaches were studied:

- Improved geometry – to allow the head restraint to be positioned closer to most occupants' heads. Ford adopted this principle in their Ford Taurus and Mercury Sable models between 2000 and 2002;
- Active head restraint – to allow the occupant's torso to sink back into the seat during a rear-end crash, and engage a mechanism in the seat back, which pushes the head restraint up and toward the back of the head. This design was adopted by Saab in 1997 (Viano and Olsen 2001) and in some General Motors and Nissan models; and
- Yielding seat back – to reduce the forward acceleration of the torso in rear-end crashes. The Volvo WHIPS seat design includes a specially designed hinge below the seat back, which allows rearward movement to reduce forward acceleration, without collapse of the seat (Lundell et al. 1998). The Toyota and Lexus whiplash injury lessening (WIL) system allows an occupant to sink farther into the seat back during a rear impact (Sekizuka 1998).

[5] HARM is a measure of the cost of injury

Overall, neck injury claims were reduced, with the benefits greater for women than for men. A 49% reduction was seen in claims for the Volvo S70 compared with similar cars before the WHIPS design was introduced. There was also a 43% reduction in neck injury claim rates for the Saab, General Motors and Nissan models with the active head restraints and an 18% reduction in Ford models with improved geometry. The Toyota WIL system did not show any reduction in neck injuries.

2.5 Minimising whiplash injury

2.5.1 Static head restraint requirements

Regulations that aim to minimise the incidence of neck injury have focussed on the mandatory installation of head restraints and the control of seat back stiffness. These regulations attempt to reduce rearward head motion in crashes and prevent neck injury resulting from hyper-extension. Typical of current regulations is the Federal Motor Vehicle Safety Standard, FMVSS 202, which has, since 1969, required that all passenger cars sold in the U.S. be fitted with head restraints in the front outboard seating positions. FMVSS 202 requires that one of the following conditions is met:

- The head restraint is at least 27.5 inches (700 mm) above the seat reference point when fully extended and the seat back must not deflect more than 4 inches (100 mm) rearward under a 120 lb (54.5 kg) load; or
- The rearward angular displacement of the head reference line is limited to 45°, under a forward acceleration of the seat structure of 8 g.

In the current European regulations, ECE 25.04, the minimum height requirement has been raised to 29.5 inches (750 mm). Effective since 1998, FMVSS 202 also requires head restraints to have a height of at least 29.5 inches in the lowest position and exceed 31.5 inches in the highest position.

The U.S. New Car Assessment Program (NCAP) started with full frontal crash testing of vehicles in 1978, as a means of evaluating the safety of vehicles for consumer information. An increase in the scope of this testing ensued and since 1995 this has included the evaluation of head restraints fitted to vehicles (IIHS 2001). The rating is based on static measurements of the head restraint in its lowest position, with respect to a 50th percentile mannequin (Figure 2.2 and Table 2.1):

- The vertical position of the top of the head restraint with respect to the top of the head (V);
- The horizontal position of the front of the head restraint with respect to the back of the head, or its back-set (H).

Table 2.1 Geometric criteria used to rate head restraint position by the NCAP (Estep & Lund 1995)

Rating	Height, V (mm)	Back-set, H (mm)
Good	< 60	< 70
Acceptable	70 ± 10	80 ± 10
Marginal	90 ± 10	100 ± 10
Poor	> 100	> 110

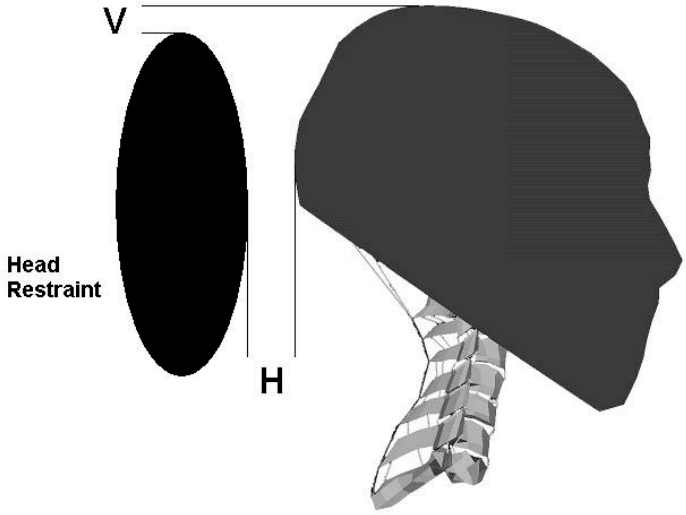

Figure 2.2 The head restraint rating dimensions V and H (c.f. Table 2.1)

The NCAP criteria were used in the evaluation of 164 vehicles from the 1995 model year (Estep & Lund 1995). Under these criteria, only five vehicles were rated as good, eight were acceptable and the remaining 117 were rated as poor. In 1995 only 3% of measured head restraints rated good compared with 45% in 2003, (IIHS 2004). The number of restraints rated as poor has decreased dramatically from 82% in 1995 to 10% in 2003. Nevertheless, these criteria are purely geometric and have not been correlated with injury claims. In a study by Bostrom et al. (1997), the researchers concluded that this rating system for vehicles based on seat-system geometry evaluation did not correlate with available accident data.

2.5.2 Dynamic test requirements

A dynamic test that is suitable for use in the design of seating systems to minimise whiplash associated injury should include both a test protocol which reflects the likely mechanism of injury, and a crash test dummy which responds appropriately under the test conditions.

To assist in the systematic development of a dummy suitable for use in rear impacts, Thunnissen et al. (1996) reviewed the available kinematic response data for the dummy neck and defined performance requirements. These authors found that there was a lack of neck extension response data for humans in comparison to that available for frontal and lateral responses. Thunnissen et al. reported three sets of response data for the neck that were the best available from current data: the moment about the occipital condyles as a function of the head angle (Mertz and Patrick, 1971) and two head rotation time history corridors at different accelerations, based on the relationship between maximum head rotation and average acceleration (Ono and Kanno, 1993). These response requirements were for relaxed car occupants who are unaware of the impending impact.

The requirements reported by Thunnissen et al. (1996) were used in the design of the TNO Rear Impact Dummy (TRID) neck, which was developed from the prototype RID neck developed by Svensson (1993). The response corridors included the effect of the motion of the first thoracic vertebra (T1) during the test, as the neck was designed to be retrofitted onto the relatively stiff Hybrid III dummy to make it suitable for use in rear impact testing. The Hybrid III dummy was developed as a frontal crash test dummy by General Motors in the 1970s and has since formed the basis for most vehicle safety system testing (Mertz, 2002). It has a rigid thoracic spine, which reduces the neck motion caused by the interaction of the hips and thorax with the seat during rear impacts.

A comprehensive set of response requirements for dummy necks in low-speed rear impacts was stipulated by van den Kroonenberg et al. (1998). These were based on 43 volunteer tests conducted at the Allianz Center for Technics, Germany.

Davidsson et al. (2000) reported on the development of the prototype of the BioRID, a dummy designed to be used as a rear-impact dummy for testing seats and head restraints. The performance of the prototype BioRID P3 rear-impact dummy prototype in rear impacts was compared to the ten volunteer tests performed at the Japan Automobile Research Institute by Ono et al. (1999). The BioRID dummy has a flexible spine with lordosis and responds to the pressure from a seat in a rear impact in a biofidelic manner. This dummy is now being manufactured by Denton in the U.S.

The inadequacy of various whiplash countermeasures, which were based on the static measurement of head restraints, has resulted in a proposal for a standard dynamic test for car seats concerning protection in low-speed rear impacts by the International Insurance Whiplash Prevention Group (IIWPG). This proposal consists of a 16 km/h rear-impact sled test of the seat with the BioRID IIe dummy (a fifth generation, commercially available dummy). The proposal is currently available for comment (IIWPG 2003).

The head restraint rating scheme currently in use by the Insurance Institute for Highway Safety is based on a combination of the static assessment of the head restraint position and a dynamic test, with the following characteristics:

- Uses the 50th percentile BioRID dummy with specific positioning instructions;
- Impact sled test with a peak acceleration of 10 g (5 g mean acceleration), and duration of 91 ms;
- Criteria used to assess the seats are:
    - Two seat design parameters measured during the test: time to head restraint contact (must be no more than 70 ms to pass) and torso acceleration (must be less than 9.5 g to pass);
    - Two evaluation criteria measured on BioRID during the test are the maximum neck shear force and maximum neck tension.

2.6 Summary

Field accident studies of vehicle occupants in crashes have shown that there is an increasing prevalence of whiplash associated injury; whiplash associated disorders occur from impacts from all directions but are more likely, and occur at lower speeds, in rear impacts. A distinct gender effect is apparent with female occupants more likely to suffer whiplash associated injury than males and more likely to have longer duration of symptoms. This may be due to differences in stature and strength between females and males.

Static head restraints have only a minimal effect on the incidence of whiplash associated injury whereas seat back deformation appears to reduce whiplash associated injury. The wearing of seatbelts seems to increase the likelihood of whiplash associated injury but being aware of the impending impact may lead to less severe symptoms.

It is possible to mitigate whiplash associated injury by changing the design of vehicles in several areas: developments in intelligent vehicle systems to reduce the numbers of collisions occurring; the use of crushable structures to reduce the crash loads on the vehicle occupant area; and the design of the seat to improved to control the loading to the vehicle occupants in a crash.

The seat design measures which have been effective in reducing whiplash associated injury include improved geometry to minimise head to head restraint standoff, active head restraints that close the standoff in a rear impact; and controlled deformation of the seat back.

For the optimisation of the design of safety systems for vehicles in rear impacts a draft test procedure is being finalised and a specialised biofidelic rear impact dummy, the BioRID, is available. Discussion is still proceeding about appropriate injury criteria.

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3 Biomechanics of whiplash injury

Tom Gibson

3.1 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 3.1.

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 as a pivot about which the head and atlas rotate (see Figure 3.1).

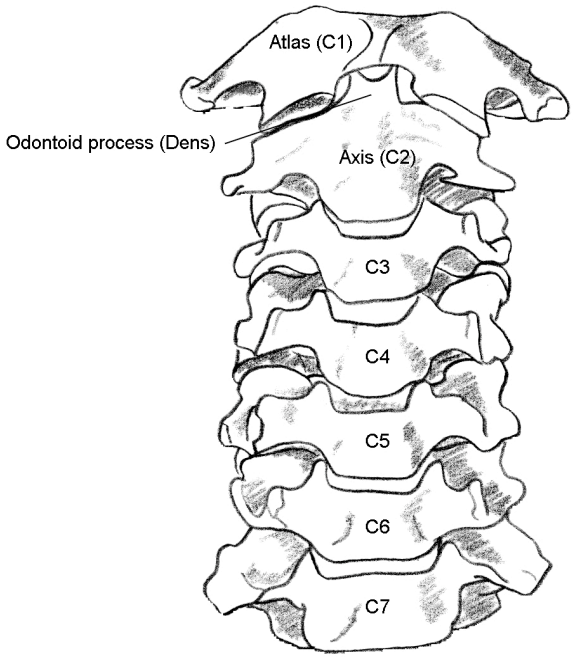

Figure 3.1 Anterior view of the cervical spine showing the odontoid process (dens) of the axis

The vertebrae of the lower cervical spine (C3 to C7) each consist of a cylindrical body and an arch (Figure 3.2). 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.

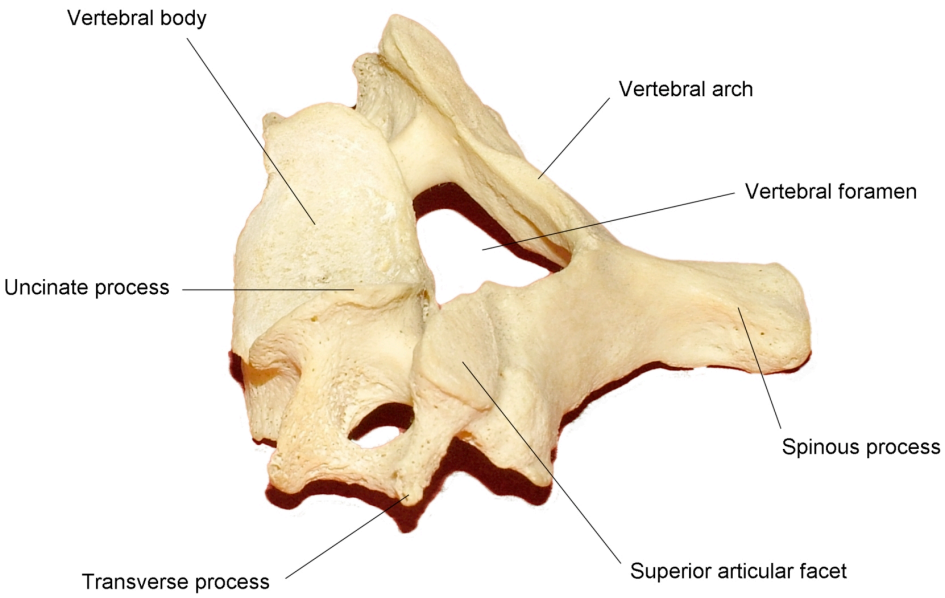

Figure 3.2 View of the C6 vertebra with the main sections indicated

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 3.3).

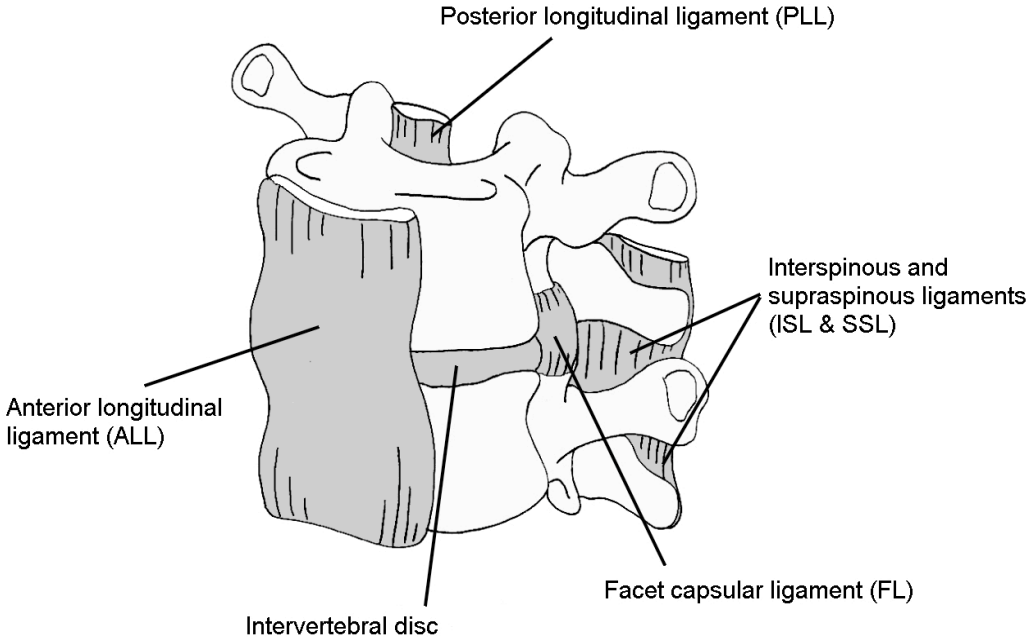

Figure 3.3 Sketch illustrating a cervical spine motion segment and the major ligaments

There are over 40 major muscles involved in controlling neck motion (Stone and Stone, 1990). Any motion of the neck is actuated by a series of these muscles acting in concert.

3.2 Functional anatomy of the cervical spine

3.2.1 The rotation axes of the intervertebral joint

Bogduk and Mercer (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 3.4). 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 3.5).

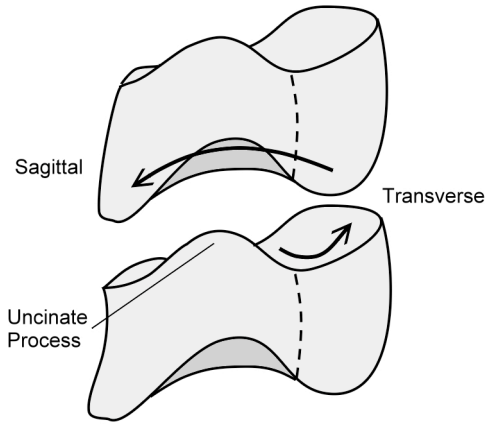

Figure 3.4 The cervical intervertebral joints allow sliding of the upper vertebra in the sagittal plane and rotation in the transverse plane, adapted from Bogduk and Mercer (2000)

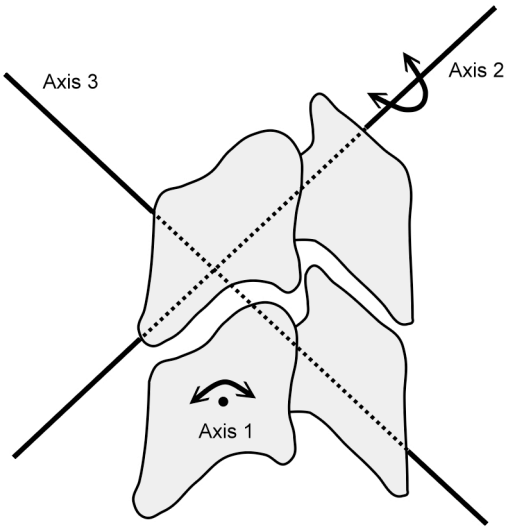

Figure 3.5 A sagittal section of the C5/C6 vertebra showing major axes of rotation: flexion/extension occurs about Axis 1; axial rotation may occur in the plane of the facet capsules around Axis 2; and, no motion is possible about the remaining orthogonal Axis 3, adapted from Bogduk and Mercer (2000)

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.

3.2.2 The structure of the intervertebral disc

Mercer and Bogduk (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 fibres in the anterior AF converge forward and upward towards the line of Axis 2, at approximately 45° to the plane of the intervertebral joint.

3.2.3 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. Mosby's dictionary (1990 edition) 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 sensitised, and the sensation of pain may serve no useful purpose.

Cavanaugh (2000) reviewed the neurophysiology and neuroanatomy of neck pain. The specialised 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 sensitise 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 & Marsland 1988), disc annulus and facet joint ligaments (McLain 1994). Consequently, injury to any of these tissues has the potential to cause neck pain.

3.3 Clinical studies of WAD

In an extensive review of whiplash injury, Barnsley, Lord and Bogduk (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 3.6), were identified, and included the following:

- *Facet capsule injury* - ligament tears, cartilage damage, contusion of the intra-articular meniscus hemarthrosis (joint haemorrhage) 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.

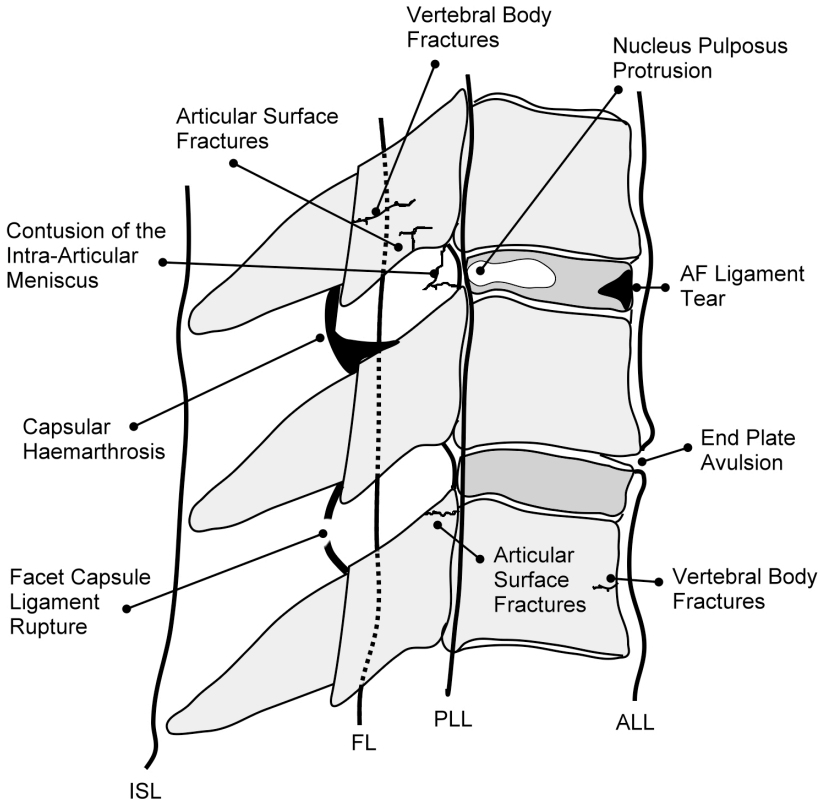

Figure 3.6 A lateral view of a section of the lower cervical spine showing possible whiplash associated injuries, adapted from Barnsley et al. (1995)

3.4 Experimental studies

3.4.1 Introduction

Mertz and Patrick (1967) tested a volunteer and several embalmed cadavers using an impact sled. 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 and Patrick (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 and Kanno (1993), McConnell et al. (1993 and 1995), Geigl et al. (1994), Szabo and Welcher (1996), Ono et al. (1997) and Siegmund, Brault and Wheeler (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).

3.4.2 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 3.7). 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 3.9.

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.

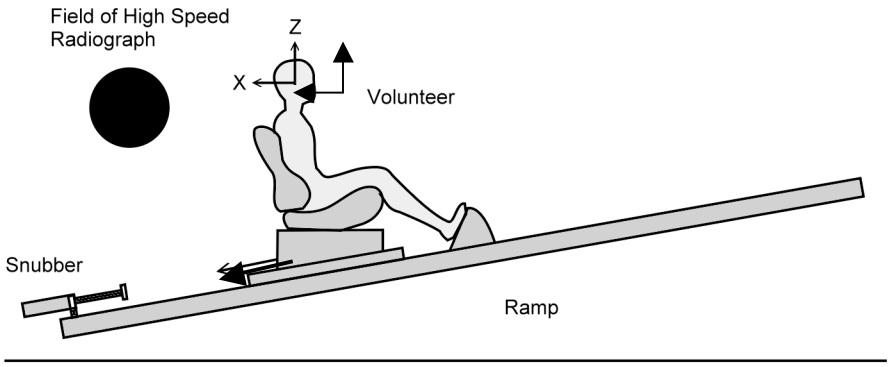

Figure 3.7 Volunteer seated on a sled inclined at 10°,
simulating a car rear impact at 8 km/h (adapted from Kaneoka et al. 2002)

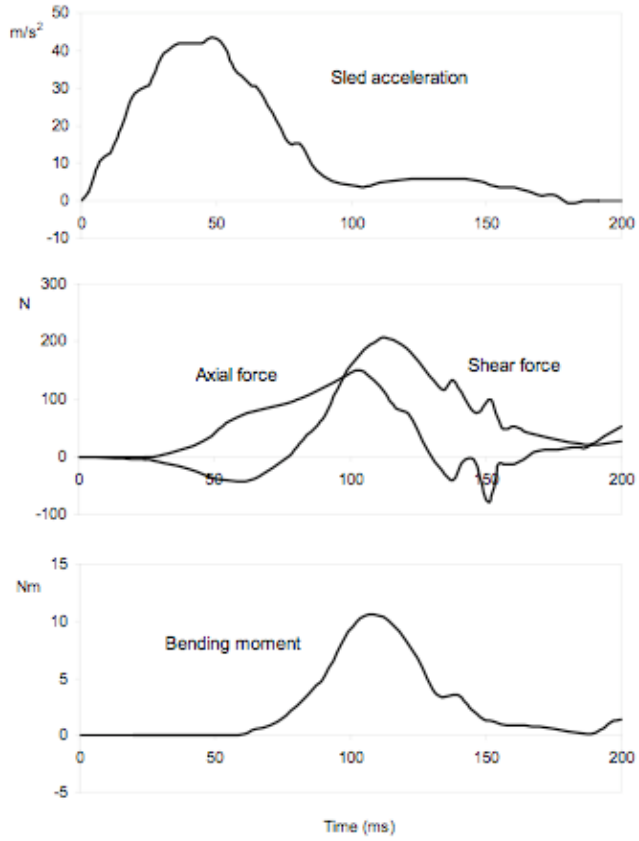

Figure 3.8 The acceleration pulse generated by the sled for the 8 km/h impact speed is shown along with the neck axial and shear forces and flexion/extension bending moment at the occipital condyle for one volunteer (from Kaneoka et al. 2002)

Kaneoka and Ono (1998) divided the motion and head-neck-torso responses of the test subjects into four phases (Figure 3.9):

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.

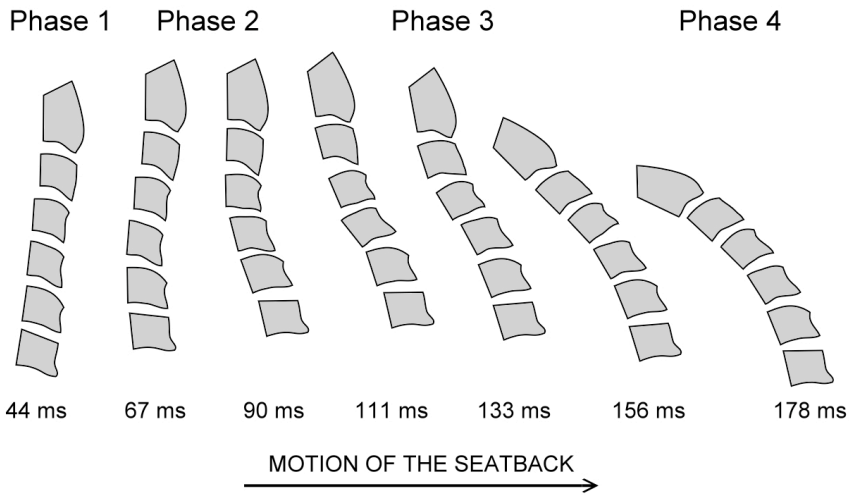

Figure 3.9 The alignment of the C2 to C7 vertebrae of a volunteer during a rear impact obtained by high-speed radiography for the 4 phases described by Kaneoka and Ono (1998). The alignment at 111 ms also includes the facet capsule and spinous processes to illustrate the possibility of impingement of 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 seat back, 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 (see Chapter 3). 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).

3.4.3 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 and Welcher (1996) measured the EMG activity of volunteers during low-speed rear impacts. Ten vehicle impacts were conducted using male and female subjects aged 22-54 years and with a target vehicle velocity change of 10 km/h (from an impact speed of 16 km/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 hypothesised 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 to 40 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 and 4 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, Narayan and Amell (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.

3.5 Hypotheses 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.

3.5.1 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 & Patrick 1967) and field accident studies (States et al. 1972). The introduction of head restraints as a result of motor vehicle safety regulation in the 1980s was only partially effective in reducing whiplash associated injury (as reviewed in Chapter 2). 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.

3.5.2 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.

3.5.3 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.

3.5.4 Facet impingement

In a series of related studies by Ono et al. (1997), Kaneoka and Ono (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. As discussed earlier in this chapter, 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 3.10).

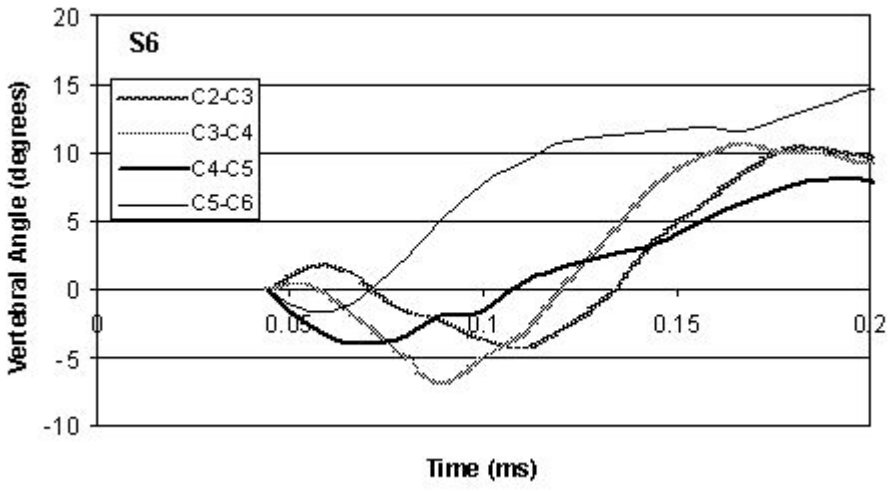

Figure 3.10 Relative rotation of the cervical vertebra for a volunteer (S6) in a rear impact, from Ono et al. (1997)

To quantify this motion, the position of the instantaneous axis of rotation (IAR) was analysed 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 3.11).

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 3.11). This upward motion of the IAR indicates that the C5 motion at this point is largely one of rotation rather than shear.

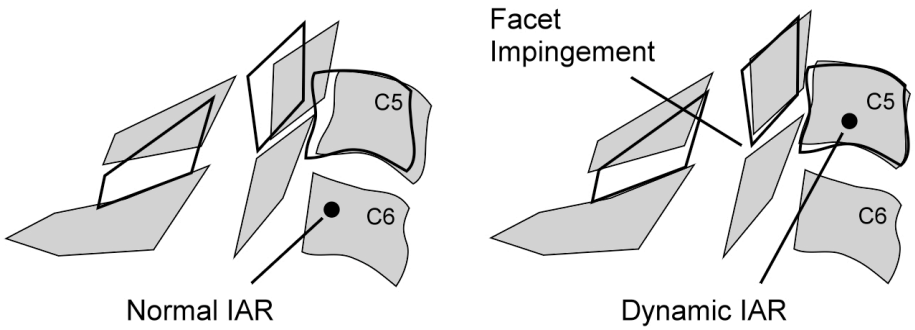

Figure 3.11 With normal cervical extension motion the IAR is positioned in the C6 vertebral body. When the S shape is reached in the whiplash motion, the IAR moves upward to a position within the C5 vertebral body, after Ono et al. (1997).

This upward shift of the IAR during the crash motion was only observed in the C5/C6 motion segment (Kaneoka & Ono 1998). It was hypothesised that, as a result of the motion, the articular 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 hypothesised 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, Kaneoka and Ochiai (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.

3.5.5 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 cadaveric tests simulating whiplash. As the torso pulls the head forward, a shear force is generated at each level of the cervical spine. Yang and Begeman (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.

3.6 Neck injury assessment criteria

3.6.1 The Neck Injury Criterion, NIC

Bostrom et al. (1996) developed the Neck Injury Criterion (NIC) based on a mathematical model of the transient pressure pulses measured by Svensson et al. (1993) in the spinal canal of pigs. These pulses were due to volume changes resulting from forcing the head and torso to translate horizontally relative to each other. Bostrom et al. hypothesised that a neck injury would occur during the initial head/thorax motion, when the spine takes the 'S' shape as the thorax is pushed forward. Anatomically, this is a retraction motion of the neck and it occurs in the first 100 ms of the rear impact, before the head begins to rotate. Injury was thought likely to occur if:

$$NIC = a_{rel} * 0.2 + v_{rel}{}^2 < 15 m^2/s^2$$

where a_{rel} and v_{rel}, are the relative acceleration and velocity between the head (C1) and the upper torso (T1). The criterion for the threshold of human tolerance of 15 m^2/s^2 was estimated to be appropriate.

NIC has been validated with volunteer tests, cadaver tests and dummy tests by Darok et al. (2000). The testing confirmed aspects of the use of NIC. For the volunteers, the peak NIC correlated well with the maximum retraction of the head and no complaints of pain were made below a NIC of 8, while some complaints of pain were made at NIC values of about 10. For the cadavers, a ligament rupture occurred at an NIC of 18.6 and NIC also correlated with the magnitude of the peak pressure readings in the spinal canal.

Kullgren et al. (2003) used a group of 79 rear-impact crashes with known injury outcomes and a crash-pulse recorder fitted to the vehicle to validate the maximum NIC (or NIC_{max}) as a criterion for injury. The crashes were all reconstructed in a mathematical (MADYMO) model of the BioRID II dummy and seat. The model was validated with sled testing. The study found that an NIC_{max} threshold of 15.3 m^2/s^2, where the proportion of occupants with lasting symptoms is 12/13 (sensitivity = 0.92), showed relatively high positive predictive values (33% ± 15%) and very high negative predictive values (99% ± 2%) for neck injury with long lasting symptoms (greater than 1 month)[6].

NIC_{max} has been shown to be sensitive to the major risk factors of a rear impact such as crash pulse, seat deflection characteristics and head-to-head restraint distance (Bostrom et al., 2000).

3.6.2 The N_{km} criterion

The N_{km} criterion is based on the N_{ij} criterion. The N_{ij} criterion is a linear combination of compression load on the neck (F_z) and flexion/extension moment (M_y) across the neck. It was developed to predict serious injury to the neck in frontal impacts (Kleinberger et al., 1998). The N_{km} was developed for rear impacts and uses shear force (F_x) and flexion/extension moment (M_y) (Schmitt et al., 2002).

$$N_{km}(t) = \frac{F_x(t)}{F_{\text{int}}} + \frac{M_y(t)}{M_{\text{int}}} < 1.0$$

The shear force (F_x) and flexion/extension moment (M_y) are both obtained from the upper load cell in a dummy neck and the F_{int} and M_{int} are constants which normalise the load values. Where as NIC_{max} is based on maximum retraction and occurs early in the motion in Phase 2, N_{km} characterises all phases.

Kullgren et al. (2003) used the results of crash reconstructions (described in 3.6.1) to validate N_{km}. The study found that N_{km} could predict neck injury with long lasting symptoms (greater than 1 month). It was found that an N_{km} threshold value of 0.48, predicted long lasting symptoms in 12 of 13 cases (sensitivity = 0.92), and produced equal positive and negative predictive values as those calculated for NIC_{max}.

In correlating NIC_{max} and N_{km} values, Kullgren et al. found that at a given NIC_{max}, there might be a large variation in N_{km}, especially for higher values (>10 m^2/s^2). At a NIC_{max} of 16 m^2/s^2, N_{km} varied between 0.4 and 1.6. The authors suggest that both criteria should be used to predict neck AIS1 injury risk.

3.7 Summary

In this chapter, many of the factors influencing the biomechanics of cervical spinal soft-tissue injury have been summarised. The factors include aspects of the neck anatomy, clinical data, autopsy data, and the results of many experimental studies using animal, human cadaver and human volunteer models to investigate these types of injury. The convergence noted by Barnsley, Lord and Bogduk (1998) of many of these factors with the crash data reviewed in Chapter 2 is becoming more apparent.

Injuries to the facet capsule region of the neck are a major source of post-crash pain. There are several hypotheses of how whiplash associated injury may occur and two of these are related strains within the facet capsule connected with events early in the impact.

There are several possible injury criteria: NIC and N_{km} have been shown to correlate with the duration of symptoms in reconstructions of actual crashes.

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[6] Positive and negative predictive values indicate the probability that an injured and uninjured occupant is correctly classified, respectively.

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4 Biomedical and biopsychosocial models in relation to whiplash associated disorders

Mark Cox

4.1 Introduction

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 in this report, 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 (see Chapter 6), as well as the use of multidisciplinary treatments (see Chapter 5), there appears to be increasing evidence in favour of the biopsychosocial model.

The aim of this chapter is to give the reader an understanding of the two models, particularly as they relate to WAD. This will entail a description of the main tenets of each model, as well as their conceptual basis. This is followed by a discussion of relevant evidence regarding the biological, psychological and social factors associated with WAD. Then, the current understanding of pain processing and persisting pain will be reviewed to illustrate why biological, psychological and social factors are important. The information will then be integrated in order to provide a framework around which WAD may be conceptualised.

4.2 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, Jackson, & Littlejohn, 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 & Richardson, 2004; Schultz, Crook, Fraser, & Joy, 2000). As a consequence of this conceptualisation 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 & Teasell, 2000; Treleaven, Jull, & Sterling, 2003; Uhrenholdt, Grunnet-Nilsson, & Hartvigsen, 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, Kiuttu, & Taanila, 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 & Tansella, 1996). As will be discussed later, 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, Edwards, & Gifford, 2002; Schultz et al., 2000; Smith & Ruiz, 2002; Wickramasekera, Davies, & Davies, 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.

4.3 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 cursory glance at the literature would have one think the biomedical model and the biopsychosocial model are two separate entities, it would appear the biopsychosocial model is an extension of the biomedical model. It does not ignore biological issues at the expense of the psychosocial. Rather, it has taken heed and expanded on the biomedical model, in an endeavour 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, aetiology, course, treatment and outcomes related to abnormal states of health (i.e. Alonso, 2004; Caltabiano & Sarafino, 2002; Engel, 1977; Gatchel, 2004; Pilgrim, 2002; Suls & Rothman, 2004; Turk & Okifuji, 2002; Walker, Jackson & Littlejohn, 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, Kubilus, Bruehl, Harden, & Lofland, 2003; Turk, 2001). Similar mulitmodal treatment programs have also been used in the treatment of chronic whiplash (i.e. Rodriquez, Barr, & Burns, 2004; Sterner & Gerdle, 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, Bhogal, Sequeira, & Teasell, 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 judgements 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.

4.4 Biological, psychological and social factors in WAD

Much research has been done on the aetiology, course, prognosis, treatment and outcomes of WAD. Numerous factors have been investigated as part of this research and they include a number of biological, psychological and social factors. The findings in relation to these factors will be discussed below.

4.4.1 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, Stiles, Borchgrevink, & Lereim, 1997; McClune, Burton, & Waddell, 2005; Moog, Quinter, Hall, & Zusman, 2002; Pettersson, Brandstrom, Toolanen, Hildingsson, & Nylander, 2004; Radanov, Bicik, Dvorak, Antinnes, von Schulthess, & Buck, 1999; Rodriquez, Barr, & Burns, 2004; Silber, Hayes, Liptez, & Vaccaro, 2005; Solomon, 2004; Treleaven, Jull, & Sterling; 2003; Uhrenholt, Grunnet-Nilsson, & Hartvigsen, 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. Some of this research has been discussed in Chapter 4 of this report. Uhrenholt, Grunnet-Nilsson, and Hartvigsen (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 post mortem 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 & Teasell, 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, Bhogal, Sequeria, & Teasell, 2005). Further discussion of this treatment is undertaken in Chapter 5 of this report. 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.

4.4.2 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 behaviour, personality, hostility and distress (Linton, 2000; Mayou & Bryant, 1996; Moog, Quinter, Hall, & Zusman, 2002). To measure these constructs a number of different psychometric tools have been used. A discussion regarding the appropriateness of these constructs and measures is beyond the scope of this report, but when interpreting the findings in the literature it is important to consider psychometric issues. A comment regarding these issues in relation to WAD will be made at the end of this section. 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 behaviour pattern." (p. 519). 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. For this discussion, 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 & Gerdle, 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 variables and 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 behavioural interventions (e.g. Eccleston, 2001; Frischenschlager & Pucher, 2002), and cognitive behavioural interventions have some support in the treatment of WAD (see Chapter 6 of this report), 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 (see Chapter 7 of this report for details).

4.4.3 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 & Schrader, 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 and Schrader (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 discern 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 hypothesised that because the cultural expectation for the

development of chronic WAD is not present in these countries, there are reduced rates of chronic WAD.

While both of these issues are discussed further in Chapter 6, 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, Lindeboom, Schagen, Heijt, Koene, & Hamburger, 1998). A recent study also looked at developing a questionnaire to detect such cases in relation to whiplash (Sartori, Forti, Birbaumer, & Flor, 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, Hill, & McMurray, 1999). Again, varying results have been found in these populations (Meyers & Diep, 2000; Mittenberg, Patton, Canyock, & Condit, 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 (albeit at low levels) and needs to be taken into consideration when dealing with WAD.

4.5 Current knowledge of pain processes

In the previous Section, the current findings in relation to factors that impact on WAD were discussed. These factors include a range of biological, psychological and social factors. From the perspective of the traditional biomedical model it is difficult to make sense of these findings. For instance, it would be expected that a definitive cause would be able to be found for the symptoms of whiplash rather than uncertainty as to which structures have been damaged. Furthermore, it would be expected that the extent of tissue damage would correlate with the intensity and type of symptoms reported. However, the findings in relation to WAD suggests that these issues cannot be wholly explained by biological factors, and it is necessary to also consider psychological and social factors. As these findings do not make sense in a traditional framework, it is useful to try and understand how these findings do make sense. Ironically, the best explanation of why these factors have an influence appears to be based in biology, and in particular the processing of pain. This section will give an overview of the current understanding of how pain is processed and hopefully provide a possible rationale for the findings discussed above in relation to WAD.

Pain has traditionally been thought of as occurring when tissue is damaged and nerve impulses are transmitted from the periphery to the brain, where these impulses are then recognised as pain. This view held until a turning point in pain sciences when Melzack and Wall introduced the Gate Control Theory (GCT) of pain (Melzack & Wall, 1965). The GCT took this view further by introducing the idea of modulation from higher centres within the nervous system (Fields & Basbaum, 1999). This descending modulation acted on the dorsal horn of the spinal cord by either inhibiting or facilitating the transmission of messages from the damaged tissues. The result of this is that pain was no longer only a function of the degree of damage in the tissues, but also a function of the degree to which these messages were being modulated by descending neural pathways. What this meant is that there was potential for pain messages from the periphery to be 'blocked' or intensified.

The most prominent way in which descending modulation has been evident is when little or no pain has been reported in the presence of obvious pathology. There are numerous examples of this, such as cases when soldiers have been wounded during combat but felt little or no pain at the time (Cousins & Power, 1999; Melzack & Wall 1982). Furthermore, the use of distraction has been implemented during treatment as a way of activating the descending modulating systems in order to decrease pain intensity (Nicholas et al., 2000). These examples have demonstrated the inhibitory potential of descending mechanisms but there has been little focus on the opposite scenario of facilitation. In considering this it is useful to know that we are constantly receiving messages from the periphery about actual or potential damage to tissues (Wall & Melzack, 1999). However, because most of the time these messages are not viewed as a threat (i.e. not life threatening), they are not raised to a conscious level where we would be forced to act on them to ensure our safety. It is conceivable then that at times, when a noxious stimulus is of little threat, our descending modulatory system, for a number of reasons, would actually enhance the message thereby bringing it to conscious awareness.

So the GCT demonstrates how descending influences can increase and decrease pain messages from the periphery without changes in the extent of tissue damage. Potential factors that can increase pain in the absence of further damage include stress fatigue, expectation, attention, depression, anxiety, anger, frustration, catastrophising, reinforcement from others, etc (Craig, 1999; Fields & Basbaum, 1999; Rhudy & Meagher, 2000; Weisenberg, 1999). Factors that can reduce pain intensity therefore include the opposite of these constructs such as reassurance regarding safety, remaining calm and distraction. The brain is constantly monitoring a wide variety of information at once to determine what is of importance and noxious stimuli are just one piece of information. Once all of this information has been processed a decision is then made as to whether a stimulus should be acted upon. Only when a stimulus is deemed of sufficient strength and importance, relative to all the information the brain is processing at one time, is it brought to conscious awareness (Wall & Melzack, 1999). In the case of a noxious stimulus, this is most likely to occur when there is a high threat value associated with the stimulus (anxiety/fear) and our safety is deemed to be in jeopardy (Cousins & Power, 1999). Information from the periphery and descending influences then form the basis of pain processing.

Another aspect of pain processing should be noted, and that is the issue of hypersensitivity. Hypersensitivity is a relatively new concept and relates to changes in the nervous system in response to tissue damage. Peripheral and central hypersensitivity can develop, and it has been hypothesised these states are responsible for the phenomenon of chronic pain (Curatolo et al., 2004). Details of these mechanisms can be found in a number of resources (e.g. Wall & Melzack, 1999), but they will only be discussed here briefly.

Peripheral and central hypersensitivity occur in response to pain states. The peripheral and central nervous systems undergo physiological changes that reduce the intensity of the stimulus required to reach a threshold for activation (i.e. they become more sensitive) (Cousins & Power, 1999). Under these conditions two phenomena are possible. Stimuli that previously would have resulted in a mild amount of pain now register a greater intensity of pain (i.e. hyperalgesia), and stimuli that previously would have resulted in no pain now register as painful (allodynia) (Butler & Moseley, 2003). Researchers have suggested we be careful not to suggest that this explains pain in the absence of tissue damage, as there is no evidence to suggest that hypersensitivity persists after tissue healing (Curatolo, 2004), while others suggest that this is possible (Sterner & Gerdle, 2004). Despite this the concept of hypersensitivity is important as it may impact on what stimuli become consciously recognised as being painful, and also the intensity of that pain. It is important to note that factors that may cause sensitivity include both peripheral mechanisms such as the degree and type of tissue damage, as well as psychological factors (Curatolo et al., 2004; Sterner & Gerdle, 2004).

The final aspect of the pain processing system that needs to be discussed is that of neural plasticity. Studies looking at the function of the brain after amputation of a limb, suggest plasticity of the brain contributes to phantom limb sensations (Flor et al., 1995). Similar

changes have been found in chronic back pain patients (Flor et al., 1997). For example, after surgical amputation of upper limbs, reorganisation of the nervous system is thought to be responsible for reports of patients being able to feel the fingers of the amputated limb, on the corresponding side of the face (Doetsch, 1997). Furthermore this process may occur within 24 hours of amputation. The theory is that the face, due to the absence of the limb, is now "taking over" the area of the cerebral cortex usually reserved for the limb (Butler, 2000). Such findings suggest that not only does the nervous system appear to become hypersensitive to pain in some instances, but this process can also occur quite rapidly.

Understanding of pain processing has improved greatly since the formulation of the Gate Control Theory, and particularly in the last decade with findings in relation to sensitisation and plasticity of the nervous system. This new knowledge has led to the ability to explain previously unknown reasons for findings in relation to acute and chronic pain conditions. The final section will attempt to integrate the findings discussed in relation to WAD, with the current understanding of pain processing.

4.6 Summary of biomedical and psychosocial factors in WAD

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 purpose of this section is not to suggest the biomedical model is wrong. The purpose is to provide evidence in relation to WAD, and a possible rationale for the syndrome, also based on evidence. It is up to the reader to determine which sort of model they think is more beneficial.

Firstly, a summary of the key findings 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. The GCT of pain demonstrates how processing of information from biological, psychological and social sources, by the brain, can impact on the intensity of symptoms experienced.
7. Peripheral and central sensitisation, 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. It would appear that the evidence available regarding pain processing mechanisms at least provides a theoretical reason for why these factors have been found to be important.

4.7 How pain processing may be able to explain the characteristics of WAD

Patients, who present with WAD, present with varying degrees of symptoms and varying degrees of identifiable pathology (e.g. Hendriks et al., 2005; McClune et al., 2002). The GCT appears to be able to provide a theoretical explanation for this variation. As discussed, messages from the damaged tissues are sent via the nervous system to the brain. The brain is modulating these messages with other information it is processing at the time. Depending on what this information is and how important it is perceived to be, varying degrees of symptoms will be reported. These may be very closely related to the degree of damage (i.e. if inhibitory and facilitatory influences are balanced), may be less than the degree of damage (e.g. if the person is distracted), or greater than the degree of damage (e.g. if the person is particularly stressed, anxious or depressed). Such mechanisms help to explain why it has been found that some patients with radiological signs report little pain, while others without radiological signs report high levels of pain (Solomon, 2004; Sterner & Gerdle, 2004). The effect the mechanisms involved in the GCT can have on the experience of pain in the acute phase of WAD has recently been discussed (Sterner & Gerdle, 2004).

From the descending inhibitory and facilitatory mechanisms it can also be understood how a variety of psychological and social factors could have an impact on pain intensity. As has been discussed, a number of factors such as attention, mood, emotional state, attitudes and expectations, can alter reported pain levels. These factors, which create nerve impulses in the brain, may result in descending influences on pain perception and thereby variation in reported symptoms. The observed change in the incidence of chronic WAD in a Canadian study looking at the impact of a change in the compensation system, may also be explained by these descending mechanisms. Pain processing by the brain may result in a change in the relative 'importance' of the symptoms. Hence, the relative importance of the symptoms may have been changed under the new compensation system. For example, if a symptom is no longer perceived (even unconsciously) as being as important under a no fault system, this could in turn result in inhibition of messages from the periphery and thereby reduce symptoms over time associated with WAD. The reduced rate of chronic WAD in some countries due to different expectations can be explained by similar mechanisms. A person believes their symptoms will resolve, the brain therefore computes that the pain is not of immediate threat, and reduces the intensity of pain experienced. One result of the way these issues interact is a consequential difficulty in identifying malingering. For instance, there is likely to be a range of malingering from no injury present to injury with reporting of exaggerated symptoms. When you get to the latter it may be difficult to decipher whether the exaggeration is due to malingering or due to impact of descending mechanisms on pain intensity.

More findings can be explained by the GCT. For instance, initial pain score has been found to be a predictor of chronicity (Suissa, Harder, & Veilleux, 2001): if the descending pain facilitatory mechanisms are working (i.e. pain is greater than would be expected in relation to the pathology), it is likely the person is also quite distressed. As has been discussed, psychological distress has been found to be a predictor of chronicity. It would also follow that in such a case, the pain processing mechanism becomes overloaded, and in response, it adapts to having to deal with this by starting to decrease the threshold it takes for a stimulus to be registered as noxious (i.e. peripheral and central sensitivity).

The influence of changes in the nervous system with respect to sensitivity can also explain a number of phenomena related to WAD. The symptom patterns seen in WAD vary greatly and symptoms seem to spread to other areas of the body with time since injury. Central sensitivity may be able to account for the symptoms that are found in areas unlikely to be related to the area in which the injury occurred (Curatolo et al., 2004). In such a case, previously subthreshold stimuli are transformed into suprathreshold stimuli and pain is experienced despite no *noticeable* cause. Such a mechanism may also explain why symptoms persist beyond the time it would normally be expected to take for the tissues to heal. In this case tissues may heal, but because the pain pathways have become sensitised,

stimuli that previously would have resulted in no pain continue and now are registered as 'painful'. A similar mechanism can possibly explain poor responses to traditional 'acute treatments' for chronic WAD. If pain pathways are sensitised, no amount of 'acute-type' treatments are going to help, unless *all* messages can be blocked, such as in the case of radiofrequency neurotomy. On the other hand sensitisation provides some rationale for the effectiveness of exercise and cognitive behavioural therapies (CBT) in these populations. CBT would have an effect on descending mechanisms, as cognitions ultimately are nerve impulses themselves. Also, by managing emotional states such as stress and anxiety, the sensitivity of the pain pathways should theoretically begin to resolve. Exercise may work from the opposite direction. Retraining a sensitised nervous system that normal levels of activity are not dangerous or threatening, may be achieved by gradually increasing levels of activity in the absence of tissue damage. The nervous system then re-learns that the stimuli it is receiving because of the activity are not threatening, but just normal sensations that would be experienced during any activity.

One last issue needs to be clarified. As previously mentioned, findings from research indicate that in a certain group of chronic WAD sufferers, symptoms can be eradicated by radiofrequency neurotomy. If this is the case, the implication is that for pain to be experienced, messages from damaged tissue, or messages from tissues at risk of damage, are required. So, if messages from the periphery are blocked, *no stimuli* can cause pain no matter how sensitive the nervous system is, because no messages are getting through. As has been noted this rules out the possibility of a psychogenic cause for the pain (Curatalo et al., 2004). However, it has been suggested elsewhere that pain sensations can occur without peripheral inputs (Fields & Basbaum, 1999; Sterner & Gerdle, 2004). This assertion may well be supported by the fact that radiofrequency neurotomies do not work for everyone. In these cases, despite no peripheral inputs, pain continues to be experienced. It may be that the "neuromatrix" involved in pain processing in such cases, is continuing to be activated by an unknown mechanism and therefore the symptoms are being perpetuated (Sterner & Gerdle, 2004).

As is evident there are many aspects of WAD that can be explained by current evidence regarding pain processing. One more comment needs to be made with respect to these issues. As all health professionals are aware, every patient that presents to them is different. They all have different thoughts about health, pain, doctors, treatments, medication, exercise, and so on. They also have different temperaments with some being relaxed, some having "Type A" personalities, some being anxious, and some depressed. Furthermore, the circumstances under which each injury occurs differ. Some patients may have been driving at the time of injury, some may have been passengers, and some may have been in stationary vehicles. The brain processes all of this information and the relative importance or 'meaning' of the multitude of stimuli it is receiving will be determined. This in turn will impact on the intensity of pain experienced through the mechanisms of the GCT. There is also the possibility of symptom exaggeration and malingering, which needs to be considered. The main point to make here is that every patient is different, and as a result, trying to consider the information presented here for every patient is a complex task. However, this point perhaps emphasises the need for multidisciplinary interventions as has been suggested in the literature (Moog et al., 2002; Rodriquez et al., 2004; Sterner & Gerdle, 2004), but also a careful assessment of each individual by each discipline. If this is done, then a specifically tailored, and if needed, multi-faceted program of treatment can be developed, aimed at maximising the quality of life for every patient.

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5 Review of current state of management of whiplash-associated disorders

Tony Ryan

5.1 Introduction

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 a diagnosis is made, treatment follows. Those treatments may be ineffective or effective; but sometimes treatment may only seem to be effective, ie, 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.'

This rather stark description sets out the path along which WAD patients may travel in part or in whole. It also highlights the importance of establishing effective methods of diagnosis and treatment for both acute and chronic cases.

The biomechanics of the injury are covered in detail elsewhere in this report. In essence, an upward force on the cervical spine causes abnormal movements of the lower cervical spine with damage to the zygapophyseal and other joints between the vertebrae, at least in some cases.

Bogduk quotes Radanov et al (1995) as showing that whiplash has a good prognosis, with most patients recovering in six months and only about 20% developing chronic symptoms. Bogduk also notes that there is no valid diagnostic technique for acute whiplash. In the vast majority of cases a patho-anatomic diagnosis of acute neck pain cannot be made on the basis of physical examination or medical imaging. In chronic cases neither radiographs nor MRI have been found useful. Only diagnostic block of the cervical zygapophysial joints has been shown to have 'face validity, construct validity and therapeutic utility' (Bogduk 2003).

Given the difficulties in establishing a valid diagnosis, as outlined above, there have been two recent systematic reviews of the treatment of WAD.

5.2 Verhagen et al

Verhagen et al (2004) in a systematic review of conservative treatments for WAD grades I and II, found four new studies since their previous review published in 2001, making a total of 15 studies, all randomised clinical trials, published in English, French, German or Dutch. Conservative intervention was defined as any non-invasive, non-surgical treatment; drug treatments were excluded. A study was included if pain, global perceived effect or

participation in daily activities were one of the outcome measures. Only three studies were judged to be of high quality, the remainder were rated poor. The conservative treatments were divided into active and passive and were compared with each other, with no treatment or with a placebo group. Passive treatment is administered to the patient, eg, soft collar, heat, ultrasound. In active treatment, the patient is an active participant, eg, an exercise program. Clinically relevant improvement was defined as a 15% improvement relative to a control.

5.2.1 Passive intervention v placebo or no treatment

One high quality study (Foley-Nolan 1992) reported positive effects for pulsed electromagnetic therapy applied through a collar compared to placebo at two and four weeks, but no effect at 12 weeks.

Four low quality studies compared a passive treatment with no treatment. Only two of these studies (soft collar v control (Gennis 1996), electro-magnetic field therapy v control (Thuile 2002)) provided data on treatment efficacy, with only short term positive effects and no long term (six months) effects being found. The other two studies examined electrotherapy and iontophoresis v control (Fialka 1989) and ultra-reiz current v control (Hendriks 1996) but provided no data.

5.2.2 Active interventions v no treatment

Only one low quality study was found, comparing traction, massage and exercises (active treatment) with no treatment (Fialka 1989, included three different treatment groups, one of which was the active group). Neck pain was significantly reduced at six weeks.

5.2.3 Active v passive treatments

The authors found two high quality and seven low quality studies. One high quality study compared normal activities with time off work and a soft collar and found little or no difference between the groups at six months follow-up (Borchgrevink 1998). The other compared exercise and psychological education (active) with TENS (trans-epidermal nerve stimulation) and ultrasound (passive) and found small positive differences on pain and global perceived effect and a significant positive reduction in time to return to work with active treatment (Provinciali 1996).

There were seven low quality studies comparing active physiotherapy interventions, which all included some form of exercise, with rest and a soft collar. Five found significant short term differences in pain in favour of the active treatment (Bonk 2000, McKinney 1989a, Mealy 1986), Rosenfeld 2000, Schnabel 2002). One study found no difference between groups (Pennie 1990), but did not report data, and one study reported a significant benefit in the passive group (Fialka 1989).

5.2.4 Active v active treatments

Two low quality studies found conflicting evidence of the additional effectiveness of specific exercises. One found benefit from adding phasic exercises to chiropractic treatment in chronic WAD cases (Fitz-Ritson 1995). The other found no significant effect from kinaesthetic exercises in acute WAD (Söderlund 2000).

After considering the above evidence, the authors found that there was limited evidence that active and passive interventions seemed to be more effective than no treatment. There was a trend suggesting that active interventions were more effective than passive ones, but no clear conclusion could be drawn. They could draw no conclusion about the most effective conservative therapy for chronic WAD, because only one, low quality, trial was found.

5.3 Seferiadis et al

The second systematic review, by Seferiadis et al (2004), had a rather wider scope, covering all modes of treatment in randomised controlled trials published in English from 1962 to May 2003. They found 26 studies, 13 of which were included in the review by Verhagen et al. The criteria for selection were: the intended design was a prospective randomised clinical trial (RCT), the study population included patients with WAD and the publication was in English. The studies were rated for quality of method using three lists of criteria which have been developed for judging methodological quality: the IMLB likelihood of bias in pain research (Jadad et al 1996), the Delphi List (Verhagen et al 1998) and the Maastricht-Amsterdam list of the Back Review Group of the Cochrane Collaboration (Van Tulder et al 1997).

A study was rated as being of high quality if it scored at least 50% of the possible score on all three lists. Seven of the 26 studies were rated high quality. Twelve studies (three were of high quality) were related to acute cases (less than three months duration), the remainder were concerned with chronic cases (three months or more duration), four of which were of high quality.

The authors noted that the large number of papers dealing with physical therapies received consistently lower methodological scores compared with drug and operative surgery RCTs. Physical therapies cannot be administered in a double blind manner, therefore they cannot receive a full score in the criteria lists which have been developed, in the main, for drug therapy.

5.3.1 Treatment of acute WAD

Based on the level of evidence found the following treatments were recommended. In acute WAD, early physical activity was supported by one high quality and several low quality studies.

In the one high quality study, a soft collar and rest was compared with active exercises and physiotherapy either within 96 hours or after 14 days, Active intervention reduced pain at six months and when started within 96 hours Rosenfeld 2003).

Four low quality studies showed improvements for active exercises over rest and a soft collar (Bonk 2000, Borchgrevink 1998, McKinney 1989, Mealy 1986). One study showed no difference between the two groups, but the randomisation process was flawed and blinding of outcome was unknown (Pennie 1990). The addition of coordination exercises to an active treatment program did not change outcome at six months (Söderlund 2000).

High dose prednisolone (Pettersson 1998) and magnetic field therapy via a collar (Foley-Nolan 1992), although supported by one high quality study each, were not recommended for practical reasons. High dose prednisolone, a 24 hour infusion which must be started within 8 hours is costly and requires hospital admission. There is also the risk of side effects. Magnetic field therapy administered by a collar conflicts with the demonstrated effects of soft collar and rest.

5.3.2 Treatment of chronic WAD

In about 50% of cases of chronic WAD, the cervical zygapophyseal joints appear to be the source of the symptoms (Barnsley 1995, Lord 1996). In these cases, radiofrequency neurotomy was shown to be effective in reducing pain in two high quality studies (Lord 1996b, Wallis 1997), while the intra-articular injection of corticosteroids was found not to be effective in another (Barnsley 1994).

There was strong evidence that the addition of cognitive behavioural therapy to physical therapy interventions was effective in reducing pain and increasing activity. This was shown by three low quality studies (Johansson 1998, Provinciali 1996) Söderlund 2001).

One high quality study of melatonin showed an improvement in sleep/wake rhythm but not in other sleep parameters or in quality of life (van Wieringen 2001).

The authors note that high quality RCTs are not common in the field of WAD, and that more research is needed, particularly in the treatment of chronic WAD. They point out that positive brachial plexus tension signs indicate a poor prognosis, and there is a need to evaluate treatment interventions for this condition. This dysfunction may explain the continuing suffering of patients with chronic WAD who do not suffer from zygapophyseal joint pain.

5.4 Other papers reviewed for this report:

One further randomised controlled trial published in English since the above reviews, is an extension of a previously cited study published in German (Schnabel et al 2000), confirmed the finding that active mobilisation resulted in better outcomes than a soft collar (Schnabel et al 2004).

Speldewinde et al, 2001, reported on 97 patients who had undergone diagnostic block of the cervical zygapophyseal joints in private consulting practice. The authors suggest that establishing a diagnosis scientifically prevents these patients' pain being labelled with inaccurate physical or psychosomatic diagnoses and minimises the risk of futile investigations and treatments. These patients can also be offered radiofrequency neurotomy, which is an effective treatment for their symptoms. This paper also reveals one of the disadvantages of the cervical block procedure, in that a substantial proportion of the patients with a successful, positive block, did not agree to undergo a repeat procedure which was required to confirm the diagnosis.

Sterling (2004) and Sterling et al (2004) describe motor, sensory and psychological changes in patients with WAD and suggest a more detailed revision of the Quebec Task Force classification of WAD. The selection process for the patients described in their case series is not described at all. On the basis of their findings they suggest that multi-professional treatment should commence earlier rather than later.

Other methodologically poor papers reported benefits from carpal tunnel release operation for neck and arm pain (Alpar et al 2002), and botulinum toxin (Freund et al 2002). Ryan (2002) in a randomised controlled trial of subjects with chronic symptoms of unspecified duration and source, showed that strength training reduced neck pain more than endurance training.

5.5 Biopsychosocial model

Ferrari (2002) sets out a biopsychosocial model for the development of chronic pain after whiplash, in which he proposes that there are strong social, cultural and psychological influences determining the prevalence of chronic pain after whiplash injury. The model examines the influence of psychological reactions to the injury and the effects this has on the expectation, amplification and attribution of the pain. This model is built on the assumption that most patients are genuine, have a variety of physical sources for pain, but that there is probably no chronic injury from the acute WAD I or II disorder as the source for chronic pain. The author makes no reference to the mechanics of the injury process, nor to the papers demonstrating cervical zygapophyseal joint injury and the effectiveness of radiofrequency neurotomy.

5.6 The role of the insurance company in the treatment of WAD

Harder and Potts (2003) describe the Injury Recovery Program developed by the Insurance Corporation of British Columbia (ICBC), based on social research which explored the effects of unemployment on psychological well-being, as well as the importance of psychosocial factors in resolving the effects of injuries which result in unemployment. The Program's

philosophy was to assist injured individuals in returning to work or usual activities, by focusing on appropriate early intervention and early return to work or activity. Recovery Coordinators (RC) were appointed to each claims centre. The RC acted as an expeditor to ensure that bureaucratic needs and systemic delays did not unnecessarily delay a client's recovery. The RC did not handle the tort aspect of the claim file, (ie, liability, settlement). The program identified six separate but interdependent areas where development was needed. These were the Recovery Coordinator, the Bodily Injury Adjuster, the rehabilitation/treatment network, the treating physicians, management/examiners and the clients. This program was developed over two years. An evaluation in 2000 showed support from physicians and other stakeholders, a reduction in treatment and disability times, and a reduction in lawyer involvement. It was also shown that within the insurance company, the old culture of "defend and deny" was still present and difficult to change, and there was a lack of management resources to allow the RC to reach their program goals. Unfortunately, during 2001, a corporate review resulted in drastic organisational changes with the result that the Injury Recovery Program was dismantled, although its underlaying philosophy was said to have been incorporated in the new company structures.

5.7 Treatment guidelines of the Motor Accidents Authority of NSW

In January 2001 the Motor Accidents Authority of NSW published guidelines for the management of Whiplash Associated Disorders. These guidelines were intended "to assist health professionals delivering primary care to adults with acute or sub-acute simple neck pain after motor vehicle collisions, in the context of third party insurance compensation". They cover the first 12 weeks following the motor vehicle crash. They are based on the recommendations of the Quebec Task Force published in 1995 and updated with new evidence available to 1999. A flow chart is provided which indicates initial management and decision points at 7 days, and at three, six and twelve weeks if the case is not resolving. The document covers diagnosis, prognosis, and treatment. Under diagnosis, guidance is provided for history taking, physical examination, the use of plain radiographs, and specialised imaging techniques in cases of WAD grades I, II and III. Indicators of poor prognosis are noted under "yellow flags" for specific symptoms, radiological findings, psychosocial factors and socio-demographic factors. Treatments are classed as 'recommended', 'recommended under certain circumstances', 'not recommended' and 'not relevant'. In each case these are related to the WAD grades I, II and III. The guidelines are based on a combination of systematic review of the evidence and where the evidence was not available, on consensus of the Working Party. There are accompanying documents for consumers, for the compulsory third party insurance industry, and a technical report which reviews the evidence in detail.

The recommendations within these guidelines are still consistent with the findings of the latest systematic reviews. They are shaped with health practitioners in mind, and provide guidance for the treatment of acute WAD.

5.8 Discussion

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.

A common thread in the reviews examined was the generally poor quality of studies of treatment in WAD. This means that there is very little sound evidence on which to base judgements 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. Collectively, the evidence was considered sufficient for the production of guidelines for the treatment of acute WAD by the Motor Accidents Authority of NSW. One of the virtues of these guidelines is that they provide a series of steps and review points to guide the practitioner through the therapeutic maze for cases of acute WAD.

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 high quality, methodologically sound research aimed at identifying effective treatments for chronic WAD, particularly those cases with no identifiable cervical zygapophyseal joint injury. These studies should take into account the major problems identified in the reviews examined eg, inadequate statistical power, poor case selection and identification, poor randomisation, ignoring the placebo effect, poor follow-up and inappropriate outcome measures and analysis.

It is only by encouraging the sound evaluation of all aspects of handling WAD cases that effective treatments and procedures will be identified.

5.9 Findings

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 (ie, 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 behavioural therapy with physical therapy interventions has also been found to be effective.
- The guidelines for the management of whiplash associated disorders published by the Motor Accidents Authority of NSW could be used as a starting point for influencing the management of WAD in South Australia.
- The experience of the ICBC Injury Recovery Program suggests that changing practice in the insurance company to encourage early intervention in potential chronic cases will have a beneficial effect on outcomes, as well as lowering costs for the insurance company.
- It is evident that there is an urgent need for methodologically sound studies of chronic WAD, to identify effective treatments for the 50% or so of cases which do not have symptoms associated with zygapophyseal joints.

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6 Predictive factors for prolonged recovery and claim settlement for whiplash: findings from two studies in South Australia

Richie Gun

6.1 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 industrialised countries. As the number of cars per head of population has increased, so has the reported rate of whiplash injury. An increase in the rate of such injuries has been noted in a Dutch study of cases reporting to a hospital emergency department over the period 1970 to 1994. Five-yearly rates of whiplash injury (defined in this study as neck sprain due to a car accident) increased nearly 12-fold between the periods 1970-74 to 1990-94 (Versteegen et al., 2000).

However the increase in the number of cars is not enough to explain the rapid increase in the number of whiplash injuries: whereas there was a 12-fold increase in whiplash injury cases, there was only a twofold increase in the number of cars per head of population over the same period. Nor is it likely that the increase can be explained on the basis of increased distance travelled per car. Figures were not available from the early years of the study, but between 1980 and 1994 the estimated increase in the average distance travelled per car was only 7%.

One theory has been the introduction of seat belts. However the Dutch researchers found no sudden increase in these injuries in 1975 when seat belt legislation was introduced, and a number of other studies have failed to show any association between whiplash injury and seat belt use, except for one study which suggested seat belts have a protective effect! The absence of any causal effect is not surprising since the initial head movement following rear-end collision is backwards.

The experience with whiplash has been different in different countries. In contrast to relatively high rates in countries such as Australia, in some countries the condition is unknown. In Lithuania, where there is little awareness of long-term disability following rear-end collisions, a follow-up study has shown that 12 months after rear-end collisions, subjects had no greater likelihood of neck pain and headache than randomly selected controls with no history of a collision. Those who had had symptoms following the collision mostly reported that symptoms abated within a few days, and none persisted for more than 3 weeks. The authors noted that insurance is rarely involved in such cases, and a minority of drivers are insured for personal injury (Obelieniene et al., 1999). This suggests that the existence of universal insurance may be a factor in the occurrence of persisting disability in other countries such as Australia.

Large variations have been found between study findings not only on the frequency of whiplash injury, but also in the resulting duration of the resulting disability.

It might be expected that the severity of the crash might be the main determinant of injury severity: after all injury is the result of energy transfer at a rate beyond the capacity of the target organism to withstand it, and it would be expected that the extent of injury would be proportional to the degree of energy transfer. Yet this has generally been found not to be the case. In a 1996 review of whiplash, Stovner concluded that evidence for a causal link between trauma and chronic symptoms was sparse (Stovner, 1996). More recently. In a review of the literature Côté et al. concluded that there was no consistent evidence that crash-related factors were independently associated with recovery (Côté et al., 2001).

The type of insurance system is also a factor in duration of disability. Cassidy and colleagues have examined the incidence and outcome of whiplash injuries in Saskatchewan following a change in the compensation system from a tort-based system which made payments for pain and suffering to a no-fault system. The change was associated with decreased incidence and improved prognosis of whiplash injury (Cassidy et al., 2000). The findings suggest that an insurance system where financial compensation is determined by the presence of pain may provide barriers to recovery: indeed the authors have suggested that such a system may actually promote persistent illness and disability. Where such systems are absent (e.g. Singapore, New Zealand, Lithuania) prevalence of chronic whiplash syndrome is low or non-existent (Ferrari et al., 1999).

In South Australia we have recently completed 2 studies to identify the likely risk factors for prolonged disability following whiplash.

In the first study, we retrospectively analysed CTP claims records from SGIC, in a search for any association between various factors - age, sex, type and severity of collision, treatment, hiring a solicitor – and prolonged disability, evidenced by delay in settlement beyond 2 years (Osti et al., 2005).

The second study was designed in response to the findings of the first. Unlike the first study, this was a prospective study, in which whiplash subjects were followed for a 12-month period following their vehicle accident (Gun et al., 2005).

Whiplash typically occurs following a rear-end collision, with an initial thrusting backwards of the head, followed by a rebounding forward flexion. Injury from side collisions is included in the definition of whiplash, but not front-end collisions, where the body dynamics are different. However both studies described here included some subjects whose collisions were not typical of those causing whiplash. The findings in relation to these subjects, and their significance, are discussed later.

6.2 Retrospective study using CTP claims data.

The first study was based on data held by State Government Insurance Commission (SGIC) on CTP claims in the period 1993-96.

Records of whiplash claims filed over the period 1993-1996 were obtained with personal identifiers deleted. This data set was divided into two sub-files:

(i) claims settled early ie within 9 months of injury

(ii) claims settled late, ie more than 24 months after the injury.

Subjects who settled between 9 months and 24 months post-injury were excluded from the analysis. Subjects with radiological damage to the cervical spine, neurological deficit and/or significant associated injuries were also excluded.

300 subjects were then randomly selected from each of the subfiles (i) and (ii), giving a study sample of 600 anonymous records.

The following were extracted from each subject file as factors thought to have a possible association with prolonged recovery:

- age
- sex
- occupation
- position in the vehicle (driver, passenger, front or back seat)
- type of collision (front, rear, side, rollover)
- previous or concurrent workers' compensation claim

- previous neck disability
- cost of vehicle repair
- whether the vehicle was driveable after the accident
- whether medical attention was sought on the day of the accident
- whether treatment was obtained from a physiotherapist or chiropractor
- total treatment cost
- whether a solicitor was consulted

Each of these factors was analysed for any association with delayed settlement of a bodily injury claim

To determine whether there was an association between each risk factor and delayed settlement, we computed the number of subjects with the risk factor who settled late (ie more than 24 months after the collision) as a proportion of all subjects with the risk factor.

GENDER DIFFERENCES

Of the 600 cases (300 who settled early and 300 who settled late), 381 of the claimants were female, and 219 were men, that is, the female to male ratio was 1.74. Since the two sub-groups were randomly selected without regard to gender, this suggests a significantly greater rate of whiplash claims in women. (This also represents a proportionate increase risk in women, since a majority of licensed drivers are male.)

Of the 381 women, 53% of the women settled late, compared with 46% of the men. This suggests that women are slightly more likely to settle late, although the excess proportion in women was not statistically significant.

AGE

In all age brackets below 65 years, there were approximately even numbers of subjects who settled early and those who settled late. The highest proportion of claimants who settled late was in the 45-54 age bracket. In contrast, of the 29 claimants aged over 65, only 8 (29%) settled late.

OCCUPATION

For this analysis the subjects were categorised into broad occupational categories – blue-collar worker, white-collar worker, home duties, unemployed, pensioner and student. There was no significant association with late settlement in any occupational category.

TYPE OF COLLISION

For rear-end, side impact and chain collisions there were approximately even numbers of subjects who settled early and those who settled late. However there was a significant difference in relation to claimants who had had a front-end collision – a category not included in the usual classification of whiplash injury. Of the 35 subjects in this category, 28 (80%) settled late.

POSITION IN VEHICLE

None of the positions in the vehicle were predictive of early or late settlement of claim.

WORKERS' COMPENSATION

Only 58 of the 600 claims were subject to workers' compensation, of which 46 (79%) had a late settlement, compared with 52% for non-workers' compensation cases. Thus workers' compensation cases were significantly more likely to have a late settlement (Risk ratio =

1.5, p = 0.001). Thirty-five claimants had had a prior workers' compensation claim, but there was no significant association with a history of a prior workers' compensation claim.

PRIOR NECK DISABILITY

131 subjects had a history of prior neck disability. Fifty-eight percent of these claimants settled late, compared with 48% for those with no prior neck disability. A history of prior neck disability was thus predictive of late settlement (risk ratio 1.2, χ^2=4.3, p=0.04).

DAMAGE TO VEHICLE

As shown in Table 6.1, there was a slight trend towards late settlement with increasing cost of repairs, from 46% of claims where the cost of repairs was below $1000 to 53% where the vehicle was written off, but the trend was not statistically significant. Neither the cost of repairs nor whether the vehicle was driveable after the accident was a significant predictor of late settlement.

Table 6.1 Settlement time by cost of repairs

Cost of repairs	Early claim (9/12mths)	Late claim (>24/12mths)	% with late settlement
<$1000	64	54	46
$1000-2500	85	82	49
> 2,500	86	92	52
Written off	65	72	53

SEEKING MEDICAL ATTENTION ON THE DAY OF ACCIDENT

Claimants who attended a doctor, either in a hospital or elsewhere on the day of the accident were no more likely to settle late than those who did not. However of those within this group who attended a hospital on the day of the accident (155 persons), 58% settled late compared with 47% of the other subjects. Thus attending hospital on the day of the accident is a weak but statistically significant predictor of late settlement.

ATTENDING A PHYSIOTHERAPIST OR CHIROPRACTOR

Four hundred and seventy of the 600 subjects attended a physiotherapist or chiropractor some time between the accident and settlement. Those who attended were more likely to settle late than those who did not seek such treatment.

CONSULTING A SOLICITOR

Three hundred and forty four subjects settled their claim through a solicitor. Of these 75% settled late, compared with only 17% of those who settled direct with the insurer. Thus there was a strong association between consulting a solicitor and likelihood of a late settlement.

MULTIVARIATE ANALYSIS

To examine the impact of these possible risk factors, it was necessary to avoid false conclusions from confounding. For example if it is known that the risk of late settlement is high in those who consulted a solicitor, the high rate of late settlement in those covered by workers' compensation may be due to workers compensation cases being more likely to consult a solicitor rather than from factors related to the workers' compensation system itself. To identify the independent effects of these risk factors, those variables found in the above analyses to be predictive of late settlement were examined simultaneously.

By far the strongest predictor of late settlement was consulting a solicitor: this was associated with a 4-fold increase in risk of late settlement. There were weak associations with previous neck disability, entitlement to workers' compensation and being treated by a physiotherapist or psychiatrist.

TOTAL COST

The median cost was $3907 for the 300 subjects who settled early and $19457 for the 300 who settled late. This difference was statistically significant.

TIME OFF WORK

Time to settlement was not associated with amount of time off work. Of employed subjects, the median time off work was 5 days for those who settled early compared with 4 days for those who settled late.

6.3 Significance of findings

One of the main questions of interest was whether the severity of the collision had any bearing on the duration of disability. All injuries are the result of energy transfer, and it is to be expected that the greater the energy transfer, the greater the severity of injury. Therefore it would be expected that the more severe the collision, the greater the likelihood of prolonged disability. This is an important question to pursue in whiplash cases, since clinical or radiological evidence of physical injury is usually absent, leading to intense debate on whether disability following whiplash is partly or even wholly psychosocial rather than physical in origin.

In the absence of clinical or radiological signs of injury, damage to the vehicle is the only objective marker of the severity of injury to the occupant. If the severity of the injury is a predictor of duration of disability, the degree of vehicle damage (in this case measured by cost of repair) should show some association with recovery. Our failure to detect an association between extent of vehicle damage and settlement time suggests one of two possibilities

(i) that settlement time is not a valid indicator of recovery from whiplash, or

(ii) that prognosis of whiplash injury is related to factors other than severity of collision.

A number of other factors affecting prognosis have been considered in previous studies.

One possibility is a pre-existing neck disorder. Our findings do suggest that this factor is important. According to the insurance data, no fewer than 131 of all claimants (22% of the sample of claimants) had had a prior neck disability, and the multivariate analysis showed a small (15%) excess of delayed settlement which was of marginal significance.

Psychological and social factors have also been considered. Our analysis showed that hiring a solicitor has a powerful, ie four-fold, association with delayed settlement.

The type of insurance or compensation system has been examined in a number of comparative studies. The only opportunity to examine this factor in our study was from the 9.7% of claimants who were covered by workers compensation, as opposed to recourse only to the compulsory third party system insurance. Injury subject to workers' compensation claim was associated with a 50% increased risk of late settlement, but the multivariate analysis to be only weakly predictive. (Correlation analysis showed that this was not due to confounding with consulting a solicitor, i.e. there was no association between having a work-related motor vehicle injury and consulting a solicitor.)

Other psychosocial factors of potential importance could be those relating to the claimants themselves. These attributes of the individual claimants were not included in the insurance database.

To examine these psychosocial factors we designed a follow-up study.

6.4 Aims of the follow-up study

This study was designed to include an assessment of the influence of individual psychosocial factors on the rate of recovery from whiplash. As well as the factors examined in the previous study (eg crash-related factors, prior neck disability, concurrent workers compensation claim), we included a assessment by questionnaire within 6 weeks of injury.

The questionnaire used was the 36-item Short Form Questionnaire (SF-36), a widely-used instrument used to estimate individuals' physical, psychological and social wellbeing. The SF-36 includes eight different sub-scales of functional status: physical functioning, role physical, bodily pain, general health, vitality, social functioning, role emotional and mental health.

The study also addressed another drawback of the insurance-based study, which relied on time to claim settlement as an outcome measure. This assumes that the claim settlement occurs at the time when disability ceases – neither sooner nor later. We retained time to settlement as a measure of recovery, but added some others. The following outcome measures were used:

- Neck Pain Outcome Score (NPOS). This measure is scored according to the extent to which the neck pain interferes with the subject's activities of daily living.
- Visual Analogue Pain Score (VAPS). This is a commonly-used 10-point scale in which the subject assesses his/her pain level.
- Whether the subject had returned to work 12-months following the accident.
- Whether the subject was still receiving treatment 12-months following the accident.
- Whether the bodily injury claim had been settled 12-months following the accident.

147 volunteer subjects were recruited from hospital emergency departments, general practitioners and physiotherapists, and completed a mailed questionnaire within six weeks of the motor accident. The questionnaire included the SF-36 questions, and questions on other possible risk factors as in the insurance-based study. Information on the costs of vehicle repair was not available in this study: the only index of crash severity was from asking the subject whether the vehicle was driveable after the accident.

Subjects were contacted again 12 months after the accident and completed a further questionnaire to assess their recovery according to the outcome measures listed above. 135 of the 147 subjects completed the second questionnaire.

Analyses were then conducted to measure the association between all potential risk factors and the degree of recovery after 12 months, according to the outcome measures.

25 subjects were found to have had front-end collisions, although such collisions are not included in the definition of whiplash injury. We carried out separate analyses including and excluding this group.

6.5 Results of the follow up study

37 subjects were male and 98 female. 87 of the 135 subjects had made at least one claim, (third party, workers' compensation or both) and of these 45 had consulted a lawyer. Of the 87 people having a claim for their whiplash injury, 36 (41%) had at least one of their claims settled within a year.

All but 42 subjects (31%) were still receiving treatment at the one-year follow-up.

Analyses relating to return to work excluded 14 subjects who were not in the workforce at the time of the accident. 108 (89%) out of 121 people had either returned to work one year later, or were not working for reasons unrelated to their whiplash injury.

6.5.1 Crash severity

There was no association between whether the vehicle was driveable after the accident and recovery.

6.5.2 Psychosocial factors

Two of the eight components of the SF-36 Health Questionnaire showed consistent and significantly positive association with all outcome measures. These were the measures of Bodily Pain and Role Emotional. With these measures a higher score means greater subject wellbeing (eg less pain). Bodily Pain and Role Emotional, The Bodily Pain Score is derived from questions on the degree of bodily pain, and the extent to which pain interferes with normal work. Role Emotional Score is derived from questions on reduction of time spent on work or other activities, accomplishing less than one would like, and not performing work or activities as carefully as usual, as a result of emotional problems such as feeling depressed or anxious.

6.5.3 Consulting a solicitor

Consulting a lawyer was associated with a worse outcome for all five outcome measures. Consulting a lawyer was associated with, on average, with an 11-fold greater probability of still receiving treatment, a 5-fold lesser probability of returning to work, a 9-fold lesser probability of claim settlement and a significantly lesser improvement in physical functioning (measured by the Neck Pain Outcome Score) after 12 months. Consulting a solicitor was also associated with a greater pain level after 12 months but the association was not statistically significant.

6.5.4 Other factors

Subjects who had been treated either by a physiotherapist or chiropractor showed statistically lesser improvements in NPOS and VAPS. They were more likely to be still receiving treatment (medical or other) but there was no association with return to work or settlement of claim. Age and sex showed no consistent association with the outcome measures.

6.5.5 Multivariate analyses

As in the insurance-based study, it is possible that some associations could be produced by confounding. For example, the association of consulting a solicitor with adverse outcomes could be due to an association with Bodily Pain Index and Role Emotional (ie whiplash subjects with worse pain and greater effects of anxiety and depression may be more likely to consult a lawyer). To avoid error from confounding, all three variables were entered into multivariate models for each outcome. Even after allowing for Bodily Pain Index and Role Emotional, consulting a lawyer was associated with a 6-point lower NPOS at the end of one year ($p<0.05$). However after a similar adjustment, there was no association with VAPS. Consulting a lawyer was associated with a 7-fold lesser chance of claim settlement ($p<0.01$) and a 7-fold greater chance of still having treatment ($p<0.01$) after one year, but there was no significant association with a return to work.

6.5.6 Excluding front-on collisions

Since the QTF excludes front-on collisions, the data were also analysed without the 25 front-on collision subjects. In this analysis the association between higher score for Role

Emotional and returning to work by the end of one year was no longer statistically significant. The other results were unaffected.

6.6 Applying the SF-36 questionnaire to predict outcome

The findings suggest that the Bodily Pain Index and Role Emotional components of the SF-36 may be useful indicators of duration of disability following whiplash.

The Bodily Pain Index is derived from two questions:

1 *How much bodily pain have you had during the past 4 weeks?*

Possible responses:

- None
- Very mild
- Mild
- Moderate
- Severe
- Very severe

2 *During the past 4 weeks (or since the accident) how much did pain interfere with your normal work (including both work outside the home and housework)?*

Possible responses:

- Not at all
- A little bit
- Moderately
- Quite a bit
- Extremely

The coding and scoring systems are set out in the SF-36 Health Survey Manual (Medical Outcomes Trust, 1994). The Bodily Pain Index is a whole number between 0 and 100, scored so that the greater the pain and disability the lower the score, ie a higher score means greater wellbeing.

The trend of increasing likelihood of having ceased treatment, having returned to work and settled a claim with increasing Bodily Pain Score is shown in Tables 6.2 to 6.4. Tables 6.5 and 6.6 show the association between Bodily pain score soon after the collision with outcome at 12 months, measured as Neck Pain Outcome Score, and as Visual Analogue Pain Score.

A trend towards prolonged disability with lower Bodily Pain Score is apparent, indicating especially that if a sub-group is to be identified as at high risk of prolonged disability and then selected for intensive treatment, those with a score of less than 25 should be selected. The predictive effect is particularly marked for failure to return to work. Although the great majority of subjects had returned by the end of 12 months, 10 of the 13 who had not were in this group of Bodily Pain Score less than 25, ie high reported pain level. A difference of 7 points in the Neck Pain Outcome Score is considered clinically significant, so that a significantly worse outcome is apparent in those with an initial Bodily Pain Score of 25 or less (Table 6.5).

Table 6.2 Trend of increasing Bodily Pain Score at initial interview with still having treatment after 12 months

Bodily pain score	Still being treated	Ceased treatment	Total	% still being treated
0-24	31	7	38	82
25-49	18	6	24	75
50-62	31	14	45	69
63-100	13	15	28	46
Total	93	42	135	

Table 6.3 Trend of increasing Bodily Pain Score at initial interview with not having resumed work after 12 months

Bodily pain score	Not resumed work	Resumed work	Total	% not resumed to work
0-24	10	24	34	29
25-49	1	21	23	5
50-62	1	42	43	2
63-100	1	21	22	5
Total	13	108	121	

Table 6.4 Trend of increasing Bodily Pain Score at initial interview with claim not being settled by 12 months

Bodily pain score	Claim not settled	Claim settled	Total	% claim not settled
0-24	24	6	30	75
25-49	11	9	20	55
50-62	11	15	26	42
63-100	5	6	11	45
Total	51	36	87	

Table 6.5 Trend of increasing Bodily Pain Score at initial interview with Neck Pain Outcome Score after 12 months

Bodily pain score	Mean NPOS at 12 months (max 75)
0-24	44
25-49	50
50-62	58
63-100	64

Table 6.6 Trend of increasing Bodily Pain Score at initial interview with Visual Analogue Pain Score after 12 months

Bodily pain score	Mean VAPS 12 months (range 0-10)
0-24	3.8
25-49	2.7
50-62	2.2
63-100	1.4

The score for Role Emotional is derived from the following question:

"During the past 4 weeks (or since the accident) have you had any of the following problems with your work or other regular daily activities as a result of any emotional problems (such as feeling depressed or anxious)?"

- Cut down on the amount of time you spend on work or other activities
- Accomplished less than you would like
- Didn't do work or other activities as carefully as usual.

The scoring system is based on Yes or No answers to these questions, and the score for Role Emotional is one of 0, 33.3, 66.7 or 100.

Tables 6.7 to 6.11 show an association between a low Role Emotional Score and poor outcome, similar to that with Bodily Pain Score.

Table 6.7 Trend of increasing score for Role Emotional at initial interview with still having treatment after 12 months

Role Emotional Score	Still being treated	Ceased treatment	Total	% still being treated
0	42	7	49	86
33.3	15	5	20	75
66.7	9	9	18	50
100	27	21	48	56
Total	93	42	135	

Table 6.8 Trend of increasing score for Role Emotional at initial interview with not having resumed work after 12 months

Role Emotional Score	Not resumed work	Resumed work	Total	% not resumed to work
0	6	36	42	15
33.3	5	14	19	26
66.7	0	17	17	0
100	2	41	43	4
Total	13	108	121	

Table 6.9 Trend of increasing score for Role Emotional at initial interview with claim not being settled by 12 months

Role Emotional Score	Claim not settled	Claim settled	Total	% claim not settled
0	28	11	39	72
33.3	10	6	16	63
66.7	4	7	11	36
100	9	12	21	42
Total	51	36	87	

Table 6.10 Trend of increasing score for Role Emotional at initial interview with Neck Pain Outcome Score after 12 months

Role emotional score	Mean neck pain outcome score (max 75)
0	47
33.3	50
66.7	61
100	60

Table 6.11 Trend of increasing score for Role Emotional at initial interview with Visual Analogue Pain Score after 12 months

Role emotional score	Mean visual pain score at 12 mths (0-10)
0	3.5
33.3	2.8
66.7	1.6
100	1.9

In summary, the findings suggest that the questions to measure Bodily Pain Score and Role Emotional would be useful prognostic indicators if administered to whiplash subjects soon after the collision. If any subjects are to be selected as at risk of prolonged disability (eg as candidates for intensive management) those with a Bodily Pain Score less than 25 or a Role Emotional Score of 0 should be included.

6.7 General observations on whiplash arising from the study

Both of these studies found no association between crash severity and outcome. Another interesting finding is that inclusion of a significant number of subjects with front-end collisions made little difference to the outcome. Front-end collisions have different dynamics to the typical whiplash, and are excluded in the definition of whiplash injury. The lack of association with the nature or severity of the crash is at odds with the general experience of physical injury, where the severity of injury varies with the quantity of energy transfer. Moreover, in contrast with our studies and studies elsewhere, a review of experimental collisions in volunteers failed to produce cases of chronic symptoms (Ferrari, 1999). The logical conclusion in that either whiplash injury is not an injury at all, or the degree of physical injury is so small that its influence on outcome is completely obscured by other factors. As shown in other studies, one such factor is the existence of an insurance system and the cultural factors arising from it.

Our studies have pointed to the importance of psychosocial factors in affecting the outcome. These factors themselves may well be related to individuals' expectations arising from the insurance system. A review of compensable injuries and health outcomes conducted by the Australasian Faculty of Occupational Medicine has concluded:

Although most people with compensable injuries recover well, a greater percentage of these people have poorer health outcomes than do those with similar but non-compensable injuries

Among the possible causes suggested for this finding, the first was:

"The psychosocial environment of the injured person at the time of the injury" and "This includes societal attitudes towards injury and compensation." (Australasian Faculty of Occupational Medicine Royal Australasian College of Physicians, 2001).

The factor most strongly associated with prolonged disability is retention of a solicitor. This finding is not surprising given the economic dictum that individuals will act in their own financial interest.

It is probable that those subjects with the severest symptoms were, for that reason, more likely to consult a lawyer. Therefore it could be argued that the apparent adverse effect of lawyers on the outcome was no more than a reflection of the fact that they saw the worst affected cases. This is difficult to confirm, as there is no objective marker of injury severity in whiplash injury, particularly in this series where we excluded subjects with neurological or radiological abnormalities. There was a strong correlation between Bodily Pain Index and likelihood of consulting a lawyer, but this does not prove that those who consulted lawyers had more severe injuries. Considering that pain is a subjective experience, the association

simply means that those who feel most pain are also those most likely to consult a lawyer. Nevertheless we found that even after allowing for the initial degree of pain and disability (measured by Bodily Pain Index), consulting a lawyer significantly increased the need for still receiving treatment, reduced the likelihood of returning to work, and of settling the claim, and significantly lowered the degree of improvement in physical functioning (measured by the Neck Pain Outcome Score) after 12 months. Thus even if initial high intensity bodily pain and disability are motivating factors for consulting a lawyer, there is evidence that seeking legal assistance itself adversely affects the outcome of whiplash.

6.8 Acknowledgements

Data for the initial study were provided by the State Government Insurance Commission South Australia.

The follow-up study was supported by the Physical Medicine Research Foundation's Woodbridge Grants and Award Program with the financial support of The Woodbridge Group, Insurance Corporation of British Columbia and State Farm Insurance. A supplementary grant was received from Sofamor-Danek.

Paul Hutchinson of the Centre for Automotive Safety Research analysed the data on which Tables 6.2-6.11 are based.

Thanks are due to Heather McElroy, Dr Diane King, Grant Taylor, Alice Guerin who recruited most of the subjects for the reliability study of the NPO, and to all others who assisted in the recruitment of subjects.

The SF-36 questionnaire is used under licence from QualityMetric Incorporated, Lincoln, RI, USA.

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Acknowledgements

This Report was funded by the South Australian Motor Accident Commission (MAC) through a Project Grant to the Centre for Automotive Safety Research. The MAC Project Manager was Ross McColl.

The Centre for Automotive Safety Research receives core funding from both MAC and the Department for Transport, Energy and Infrastructure.

The views expressed in this report are those of the authors and do not necessarily represent those of the University of Adelaide or the sponsoring organisations.