Prevention of chronic pain after whiplash

R Ferrari

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The acute whiplash injury is a significant health burden for patients and the healthcare system. Traditional approaches to treatment fail to resolve this ever growing medicolegal and social problem. A new biopsychosocial model of whiplash disorder encourages new ways of treating and preventing of the chronic disability. This biopsychosocial model takes into account the mechanism by which acute pain becomes chronic pain, and how this can be prevented. Specific education and treatments encourage a behaviour after whiplash injury that is conducive to more rapid recovery, and provides the whiplash patient with insight into the mediators of chronic pain. The article describes in practical terms how to use education, reassurance, a more judicious use of therapy, and exercise to achieve this goal. Practical guidelines are provided on educating the patient about other symptoms that may cause concern.

Whiplash has been defined as an injury mechanism, an injury, a medicolegal and social dilemma, and a complex chronic pain syndrome. Regardless of how one defines the term whiplash, whiplash associated disorders (WAD) are a frequent contributor to chronic, soft tissue rheumatic disorders. The 1995 Quebec Task Force (QTF) on WAD remarked that this is not only an ever increasing financial burden on insurers, but also an important burden for patients and the healthcare system.¹ There are substantial variances in the epidemiology of whiplash, partly according to the method in which it is studied, but also probably because of real differences in recovery rates in different countries.^{2–3} In Canada, for example, as many as 50% of subjects in Ontario⁴ and Saskatchewan⁵ will have chronic pain at six months after the collision. In another province in Canada (Quebec)¹ and in Switzerland,⁶ this figure is much smaller, in the order of 20% or less. Different study end points and methodologies, as well as different insurance/compensation systems may explain some of this variance. Recovery appears to occur within six weeks in subjects studied in Lithuania,⁷ Germany,^{8,9} and Greece.¹⁰ There are low levels (about 3%–5%) of disability at about two years after the collision in Quebec¹ and Switzerland,⁶ it does take two years for this to be achieved. The same, or better, is achieved within weeks in Lithuania, Greece, and Germany.

The goal for treatment in “whiplash cultures” like Canada, or the United Kingdom, where chronic pain is common is to achieve what other countries seem to be achieving with less therapy, less suffering, and less cost. The QTF on WAD pointed out that there are few studies to support most of the therapies (many of them expensive) used to treat whiplash patients.¹ The QTF define WAD as the various clinical manifestations of a “whiplash injury”. Their grading system is as shown in table 1. By their classification scheme most “whiplash” patients have grade 1 or 2 WAD, these being attributed to “soft tissue injuries”. This commentary will focus on treatment of these

first two grades (that is, patients without cervical spine fractures, dislocations, or clear cut, objective neurological lesions).

COMPONENTS OF THE ASSESSMENT AND TREATMENT PROGRAMME

The art of medicine in whiplash patients

The assessment and management of a patient with WAD requires the application of the art of medicine as well as science. Listening, understanding, empathy, and positive re-assurance are vital roles for the physician. These patients are often anxious and angry and at times resentful. Do not underestimate the patient's perception of the seriousness of their injury. A thorough musculoskeletal examination and neurological examination is reassuring the patient that their injury is being taken seriously and will be properly assessed.

History

Patients will usually offer various symptoms. Specifically ask about pain and stiffness (not just along the spine, but the remainder of the torso and limbs), numbness, visual or balance disturbances, jaw pain and clicking, and try to make an assessment of the emotional impact of the event. For medicolegal purposes, document when and how the collision happened, position in vehicle, use of seat belt, and any suggestion that there was a head strike or loss of consciousness. A past history of musculoskeletal or neurological problems is important to know the level of health that the patient can reasonably be expected to return. From the history alone, one usually has a fairly good estimate that the patient probably had a simple neck sprain and is grade 1 or 2 WAD, but the physical and radiological examination will confirm this further.

Physical examination

A patient seated with drooped shoulders, and head poked forward (the ear lobes should normally align vertically over the shoulder, whereas in poor posture the ear lobes align vertically in front of the shoulders) may be showing maladaptive postures. When seated or standing, a reduced lumbar lordosis will often accompany the head forward, slouched posture. The range of neck and back motion, areas of tenderness, the neurological examination, and a radiograph of the cervical spine allows the patient to be graded according to table 1. The importance of range of motion is questioned by some, as it clearly is influenced as much by pain tolerance and anxiety as it is by a disorder. The poor head forward posture itself limits normal range of motion. (The reader can appreciate this by sitting upright and looking over the shoulder—usually 90 degrees rotation is achieved. If the reader places his/her head in a forward, poked out position, and looks over the shoulder with this posture, lateral rotation may decrease by as much as

Abbreviations: WAD, whiplash associated disorder; QTF, Quebec Task Force (on WAD)

Table 1 The Quebec Task Force on whiplash associated disorders (WAD) classification scheme

Grade	Injury and Symptoms	Signs
1	Probable muscle sprain. Neck stiffness only	No tenderness and normal range of motion Normal reflexes and muscle strength in the limbs
2	Probable muscle and/or ligament sprain. Any combination of neck pain with or without back pain, jaw pain, jaw locking, jaw clicking, limb numbness, dizziness.	Paraspinal tenderness and restricted spine range of motion. Normal reflexes and muscle strength in the limbs.
3	Probable disc protrusion with nerve root impingement. Neck pain, often arm pain or numbness.	Abnormal reflexes and/or muscle weakness, often with sensory changes in a dermatomal pattern suggesting nerve root impingement (typically due to disc protrusion)
4	Cervical fracture and/or dislocation Neck pain, possibly neurological symptoms in limbs, urinary incontinence due to spinal cord involvement.	Possible hyperreflexia, positive Babinski's sign, motor weakness and sensory changes suggesting spinal cord injury. Radiograph reveals fracture and/or dislocation.

50%. This reduction in range with abnormal posture is also true for flexion and extension.) The range of motion decreases with age.³ This is a further caveat regarding excessive reliance on neck range of motion as an important indicator of disorder. Additional aspects of the examination depend in large part on specific symptoms.

Radiological assessment

Many physicians order a cervical spine radiograph to reassure the patient and themselves that the rare possibility of fracture in an apparently otherwise "typical" soft tissue injury has been ruled out. There are recent publications that begin to bring an evidence base to use of radiographs in these patients.^{11, 12} These rules seem to be effective in identifying those patients at risk of fracture. They do not assess the effect on patient re-assurance and recovery. Patients with low impact rear end shunts, no neurology and who can rotate their head 45° in each direction may not need routine radiology. In the patient with neurological signs suggestive of a radiculopathy MRI scan is the most useful investigation. The QTF¹ also recommends that patients with grade 1 WAD should not have a radiograph. Some may disagree and the threshold for radiography will as always depend on the clinical assessment of the patient.

Telling the patient about benign radiological findings can exacerbate concerns. Patients may be told their radiograph shows "straightening of the lordosis" or "disc disease" or "arthritis", all without the explanation that these findings are either normal or age related findings.^{2, 3, 13} Straightening of the cervical lordosis, minor degrees of forward angulation of the cervical spine, or kyphosis are not found any more frequently in those claiming acute whiplash injury compared with asymptomatic controls.¹³ There is little evidence that having an abnormal radiograph (including signs of disc degeneration) at the time the acute injury affects the outcome.¹³

Thus, in the vast majority of whiplash claimants, the various, commonly identified radiological abnormalities do not correlate with symptoms and merely represent the background prevalence of such findings in the general population. Their description, however, seems often to confuse the clinician more than they help, and probably does more to serve the litigious purpose than the patient's health.

Radiological studies (including MRI scans) are reserved for to rule out fracture, dislocation or to investigate neurological signs.

TREATMENT ADVICE BASED ON A MODEL OF CHRONIC PAIN

The advice on treatment in this article concerns patients with grade 1 or 2 WAD. The following advice is based on the author's experience and interpretation of the literature on the prevention of chronic disability after minor spinal injury. Research evaluating this approach is underway.

The biopsychosocial model suggests that we need to change our approach to chronic pain: chronic pain is not "all in the mind", nor is it "all in the body".^{2, 3, 14, 15} 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 1 or 2 disorder as the source for chronic pain. 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 has been explained in detail elsewhere, but is reviewed in brief here to show how it might lead to practical and helpful advice.^{2, 16, 17, 18}

THE BIOPSYCHOSOCIAL MODEL OF CHRONIC PAIN SYNDROME

In North America there is overwhelming information regarding the potential for chronic pain after whiplash injury. There is widespread knowledge in the general population of the outcome expected.^{19, 20} This knowledge may act at a pre-conscious level to produce an expectation of the type and duration of symptoms in WAD.²¹ This expectation may lead the person to become hypervigilant for symptoms, to register normal bodily sensations as abnormal, and to react to bodily sensations with affect and cognitions that intensify them and make them more alarming, ominous, and disturbing. This may result in symptom amplification.²²

The motor vehicle collision is often a frightening event and may immediately create an impression that any injury will be serious. The patient's fear may be increased when paramedics take him out of the car in a special stretcher, apply a hard collar, and warn him not to move. Any symptoms are intensified when they are thought to be attributable to a serious disease rather than benign causes such as lack of sleep, lack of exercise, or overwork.²³

Fear may also be generated later by the responses from physicians after the collision: "You had better see a specialist", "You suffered a little nerve damage", "I am not sure what's wrong with you", "It's just some arthritis of the spine", and "Your radiograph shows degeneration of the spine". Responses of the legal profession like "We had better wait for a few years before settling your claim because you never know how badly off you may become," and "As the representative for the insurance company, we ask that you see one of our specialists," can only serve to increase concern.

Another aspect of symptom amplification occurs when others have the collision victim repeatedly draw attention to the symptoms (that is, every time the patient sees a therapist, or is asked to keep a diary of symptoms, etc). Attention to a symptom amplifies it, whereas distractions diminish it. Thus the more frequent patients are asked to rate their pain, the more intense they rate it.²³

If a type of treatment fails, this may have an important adverse psychological effect on the patient. Patients will probably assume that they have a resistant or more severe physical

Figure 1 The pathway to chronic pain. Reprinted with permission from Ferrari R. *The whiplash encyclopedia. The facts and myths of whiplash*. Gaithersburg, MD: Aspen, 1999:111.

injury than realised. This can only serve to increase their fear about future health.

A collision victim becomes hypervigilant for any symptoms that before the collision might have been disregarded but because of amplification become far more intrusive. The symptom pool may increase even though the acute injury is resolving. These initially minor symptoms may be due to occupation, sports or hobbies, symptoms from medication use, and importantly attributable to maladaptive postures and changes in physical fitness that arise as patients withdraw from normal activities. These various benign, physical sources do not usually cause severe or significant pain but this is where psychological factors begin to become important. This does not mean that the pain is “all in the mind” or that the patient is intentionally exaggerating their symptoms. These pre-conscious influences increase the awareness of otherwise benign physical sources of symptoms to generate a more severe clinical picture and more distress than would be expected.

The biopsychosocial model is not a “psychogenic model”. It postulates that patient expectations, their perception of new symptoms, and how they focus and attribute symptoms will in turn change the character of those symptoms and the patient's behaviour. This model is depicted in figure 1.

THE MANAGEMENT OF WAD GRADE 1 AND 2

The emergency department visit

Once a fracture or neurological injury is excluded the ultimate goal of treatment is an attempt to change the behaviour of injured people so that they come to view their injury and pain as a benign, self limiting problem. Simply telling the patient “this is a minor injury, and do not worry about it” will be in contrast with much of what they have heard or will hear elsewhere. It seems more reasonable to explain to the patient that “while it is true that many people do go on to report chronic pain after an acute whiplash injury, the damage from the acute injury does not cause the chronic pain. Other things do, and you can prevent them from acting to cause chronic pain

for you.” You can reassure the patient that chronic pain will be much less likely to occur if they follow the advice listed in box 1.

The QTF recommends that for grade 1 WAD, that “rest should not be prescribed”, and for grade 2 that “rest > 4 days should not be prescribed”.¹ A recent study confirmed the appropriateness of these recommendations. Borchgrevink *et al* compared whiplash patients being given no sick leave and no collar but told to “act as usual” to a group told to rest for two weeks (given sick leave) and to wear a collar. At six months, the group told to “act as usual” had a much better outcome.²⁶ In Lithuania, Greece, and Germany, whiplash patients routinely return to work early (absence is measured in days not weeks) despite pain, and yet do remarkably well.

You can understand why patients need education (or re-education) early, to deal with anxieties and frustrations in returning to work.²⁷ Such an approach may simply require some straightforward, “no nonsense” advice, as presented in previous publications.^{14 28}

Box 1 Advice for WAD grades 1 and 2

- 1 Maintain normal activities as much as possible even though it may hurt.
- 2 Continue work, or if you must stop work, enter into a very active exercise programme immediately.
- 3 Avoid the development of poor posture because you are inactive or slouch to reduce the symptoms.
- 4 Do exercises (even though some may hurt) that give back normal range of motion.
- 5 Avoid letting the stress of dealing with litigation and insurance people cause more muscle tension.
- 6 Do not pay too much attention and worry over every new ache and pain or symptom.
- 7 Do not wear a collar.
- 8 Do not rely on medications rather than activity to “heal” the injury.

Table 2 Converting the serious to the benign

Symptom	Patient's interpretation	Physician explanation after a thorough history and examination
Cracking or popping sounds	Joint or bone damage	"Many people who have tight muscles will hear noises when they move those muscles. It sounds horrible, but it does not mean anything is wrong with the bones or joints. It is a sound that goes away when the muscle pain is treated, and the muscles are relaxed."
Dizziness, or loss of balance	Brain or inner ear damage	"Dizziness and problems with balance come on for many reasons, none of them serious. The first is what you experience after the collision because your head moved so quickly. After that, the presence of neck pain does not permit smooth movements of your head when you are moving. The mixed signals leads you to experience disorientation and dizziness. The treatment is to get rid of the neck pain. Also, some medications prescribed for pain and sleep unfortunately have dizziness as a side effect. Dizziness is not a sign of anything serious, but a sign that you need to work on getting your neck range of motion back to normal and do the exercises (even though they hurt) that eliminate neck pain. The sooner you do this, the sooner you can stop medications that might actually be causing your dizziness now."
Jaw Pain, jaw clicking, jaw locking	TMJ injury	"When you have neck pain you can get jaw pain because the muscles of the neck often run close to the jaw, and so your jaw region hurts when these muscles hurt. Also, having pain is stressful, and some people will grind and clench their teeth, especially if their sleep is disturbed. This causes jaw pain. The pain is genuine, but the jaw joint is not injured. The treatment is to get rid of the stress of neck pain."
Headaches	Brain damage, TMJ injury	"The neck muscles attach to the skull in many places. Neck pain and its radiation that is generating headaches as well as the stress of having pain. The treatment is to deal with the neck pain."
Chest pain		"The initial chest pain is due to bruising from the seatbelt. After that, people who develop poor posture, with their shoulders forward and head forward, will continue to have chest pain. The cure for this is to correct your posture."
Problems with memory and concentration	Brain damage	"Many things cause your thinking to be affected. Having pain is one, as are some of the medications you are taking, and stress. The cure is to treat your neck pain, reduce medications causing more harm than good, and reduce stress. You do not have brain injury."
Numbness in arms or legs	Nerve damage, pinched nerve	"Numbness in the arms and legs is common with muscle pain, even without nerve damage. I have checked your reflexes and other parts of the nerves carefully, and they are completely normal. The treatment for this numbness is to remove your neck pain."

In addition the patient is given advice on posture and some simple exercises. They should sit with a lumbar roll along the upper part of the lower back for the next few weeks, at work and at home and in the car. The roll goes at around L3. This is not a cushion for comfort, but rather physically blocks movements that allow one to slouch. Detailed advice on neck retraction and back extension exercises are available on the internet (www.emjonline.com).

The efficacy of these specific exercises and the lumbar roll was evaluated by McKinney *et al* and indeed this was found to be beneficial,²⁹ but those data are limited and more studies are needed. Certainly, the head forward posture has been shown to be corrected by neck retractions.³⁰ Lumbar rolls have been tested and found effective for low back pain,³¹ and again were a part of the therapy studied by McKinney *et al*.²⁹ It may be more relevant that the patient comes to understand that any exercise, even when initially painful, is beneficial. Exercise therapy may actually have a profound psychological effect and only a limited physical effect. Nevertheless, if exercises are to be carried out, using the above exercises that have been shown to maintain or improve posture seems most appropriate.

The use of medications and passive therapies has been dealt with in detail elsewhere.^{2,16} The doctor can prescribe a non-steroidal anti-inflammatory drug (NSAID) for the first two weeks, and this can be combined with paracetamol Extra Strength acetaminophen (up to eight tablets per day), which is continued after the NSAIDs are stopped. The patient who remains active and does their exercises will usually improve after the first week, unless something (usually unhealthy advice from others) intervenes. Placing a patient on narcotics, anti-anxiety agents, or antidepressants may convey to them that they have a "serious injury". Of greater concern these very same medications may actually generate symptoms that the patient will not be able to separate from their illness.

The treatment of any other symptoms besides neck or back pain is summarised in table 2. The advice in the table can only follow a sufficient history and physical examination that permits the diagnosis of grade 1 or 2 WAD.

CHRONIC DISABILITY CASES

One matter that remains largely unstudied in whiplash patients is how to deal with the patient who has had passive therapy for a year, say, and has had a marked reduction in activity. For these patient, there is only one study in whiplash patients, and it has its limitations, given there was no control group, and subjects who were not interested in the programme (for various reasons), opted not to participate.³² Vendrig *et al* studied 26 subjects who had all experienced chronic pain for over six months from the time of the collision, and were all not working.³² After a four week programme that included exercise and also an extensive re-education programme to change the way the subject thought about their pain and how they should respond to their pain, 65% of subjects returned to full time work and another 27% to part time (total 92%), these effects lasting at follow up six months later, with 81% of the whole group not using any further treatment after the programme was complete. More studies are needed, but this re-education programme indicates that chronic pain is not intractable and there is a group of patients, if motivated to recover, who will respond to such programmes. Multidisciplinary programmes lasting 4–12 weeks may be prescribed, composed mainly of exercise and cognitive psychotherapy to change the patient's behaviour. It is the experience of many, however that despite these expensive programmes, some patients will withdraw from these programmes early, or continue to report symptoms, and total disability. The risk factors for this behaviour are discussed elsewhere, and if these are not recognised, usual therapeutic efforts will be ineffective.²⁵

CONCLUSION

A biopsychosocial model of whiplash predicts which therapy approach will be most effective. The epidemiology of whiplash in other countries that lack the problem of chronic whiplash teaches us that our own behaviour may influence the patient's behaviour, and that the acute injury itself has little to do with outcome. It is encouraging a behaviour of wellness rather than

serious injury that encourages rapid recovery. Excessive rest, use of collars, withdrawal from activities, over prescribing of medications, and passive therapies all encourage disability. The model of whiplash predicts this. Instead, presenting the acute whiplash patient in a new light emphasises the value of education in changing their behaviour, not their "injury". Although clinical trials are necessary, it is the author's experience and belief that most patients are genuine and want to get better. They will respond gladly to the more helpful approach described above than to the illness affirming approaches so often taken otherwise.

See the journal web site (www.emjonline.com) for additional information regarding this paper.

Author's affiliations

R Ferrari, Room 2G2.06, Walter C MacKenzie Health Sciences Centre, University of Alberta Hospital, Edmonton, Alberta, Canada T6G 2B7
Correspondence to: Dr R Ferrari; rferrari@shaw.ca

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REFERENCES

- 1 **Spitzer WO**, Skovron ML, Salmi LR, *et al*. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders. *Spine* 1995;**20** (suppl 8):1-73S.
- 2 **Ferrari R**, Russell AS. Epidemiology of whiplash—an international dilemma. *Ann Rheum Dis* 1999;**58**:1-5.
- 3 **Ferrari R**. *The whiplash encyclopedia. The facts and myths of whiplash*. Gaithersburg, MD: Aspen Publishers, 1999.
- 4 **Brisson RJ**, Hartling L, Pickett W. A prospective study of acceleration-extension injuries following rear-end motor vehicle collisions. *J Musculoskeletal Pain* 2000;**8**:97-113.
- 5 **Cassidy JD**, Carroll L, Cote P, *et al*. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *N Engl J Med* 2000;**342**:1179-86.
- 6 **Radanov BP**, Sturzenegger M, De Stefano G. Long-term outcome after whiplash injury. *Medicine* 1995;**74**:281-97.
- 7 **Obelieniene D**, Schrader H, Bovim G, *et al*. Pain after whiplash—a prospective controlled inception cohort study. *J Neurol Neurosurg Psychiatry* 1999;**66**:279-83.
- 8 **Bonk A**, Ferrari R, Giebel GD, *et al*. A prospective randomized, controlled outcome study of two trials of therapy for whiplash injury. *J Musculoskeletal Pain* 2000;**8**:123-32.
- 9 **Keidel M**, Rieschke P, Stude P, *et al*. Antinociceptive reflex alteration in acute posttraumatic headache following whiplash injury. *Pain* 2001;**92**:319-26.
- 10 **Partheni M**, Constantoyannis C, Ferrari R, *et al*. A prospective cohort study of the outcome of acute whiplash injury in Greece. *Clin Exp Rheumatol* 2000;**18**:67-70.
- 11 **Stiell GI**, Wells GA, Vandemheen KL, *et al*. The Canadian C-spine rule for radiography in alert and stable trauma patients. *JAMA* 2001;**286**:1841-8.
- 12 **Hoffman JR**, Mower WR, Wolfson AB, *et al*. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. *N Engl J Med* 2000;**343**:94-9.
- 13 **Ferrari R**, Russell AS. Pain in the neck for a rheumatologist. *Scand J Rheumatol* 2000;**29**:1-7.
- 14 **Ferrari R**, Leonard M. Whiplash and temporomandibular disorders—a critical review. *J Am Dent Assoc* 1998;**129**:1739-45.
- 15 **Ferrari R**, Russell AS. Why patients with simple neck strain develop persistent neurological symptoms. *Arthritis Care Res* 1999;**12**:70-6.
- 16 **Ferrari R**, Kwan O, Russell AS, *et al*. The best approach to the problem of whiplash? One ticket to Lithuania, please. *Clin Exp Rheumatol* 1999;**17**:321-6.
- 17 **Ferrari R**. The biopsychosocial model—a tool for rheumatologists. *Baillieres Clin Rheumatol* 2000;**14**:787-95.
- 18 **Ferrari R**, Russell AS. Authors' reply to Drs Barnsley and Bogduk [letter]. *Ann Rheum Dis* 2000;**59**:395-6.
- 19 **Aubrey JB**, Dobbs AR, Rule BG. Laypersons' knowledge about the sequelae of minor head injury and whiplash. *J Neurol Neurosurg Psychiatry* 1989;**52**:842-6.
- 20 **Mittenberg W**, DiGiulio DV, Perrin S, *et al*. Symptoms following mild head injury: expectation as aetiology. *J Neurol Neurosurg Psychiatry* 1992;**55**:200-4.
- 21 **Shorter E**. *From paralysis to fatigue*. Toronto: Maxwell Macmillan, 1992:ix.
- 22 **Barsky A**, Goodson JD, Lane RS, *et al*. The amplification of somatic symptoms. *Psychosom Med* 1988;**50**:510-19.
- 23 **Barsky AJ**. Amplification, somatization, and the somatoform disorders. *Psychosom* 1992;**33**:28-34.
- 24 **Baer N**. Fraud worries insurance companies but should concern physicians too, industry says. *Can Med Assoc J* 1997;**156**:251-6.
- 25 **Ferrari R**, Kwan O, Friel J. Illness behaviour and adoption of the sick role in whiplash claimants. *Forensic Examiner* online journal of the American College of Forensic Examiners [serial online] 1999 Aug. Available from: URL: <http://www.acfe.com>
- 26 **Borchgrevink GE**, Kaasa A, McDonagh, *et al*. Acute treatment of whiplash neck sprain injuries. A randomised trial of treatment during the first 14 days following car accident. *Spine* 1998;**23**:25-31.
- 27 **Provinciali L**, Baroni M, Illuminati L, *et al*. Multimodal treatment to prevent the late whiplash syndrome. *Scand J Rehabil Med* 1996;**28**:105-11.
- 28 **Kent H**. BC tackles whiplash-injury problem. *Can Med Assoc J* 1998;**158**:1003-5.
- 29 **McKinney LA**. Early mobilisation and outcome in acute sprains of the neck. *BMJ* 1989;**299**:1006-8.
- 30 **Pearson ND**, Walmsley RP. Trial into the effects of repeated neck retractions in normal subjects. *Spine* 1995;**20**:1245-50.
- 31 **Williams MM**, Hawley JA, McKenzie RA, *et al*. A comparison of the effects of two sitting postures on back and referred pain. *Spine* 1991;**16**:1185-91.
- 32 **Vendrig AA**, van Akkerveeken PF, McWhorter KR. Results of a multimodal treatment program for patients with chronic symptoms after a whiplash injury of the neck. *Spine* 2000;**25**:238-44.