

# Annals of the Rheumatic Diseases

---

## Leader

---

### Epidemiology of whiplash: an international dilemma<sup>★</sup>

Whiplash associated disorders have become an international medicolegal and social dilemma. Physicians are not sure what the best therapeutic approach should be, and the courts are finding the topic growing ever more controversial. There are many evident paradoxes in the development and presentation of such disorders. We will focus particularly on the remarkable epidemiological findings covering the “natural history” of this problem, and provide a biopsychosocial model to explain these observations.

#### Epidemiology—international comparisons

A model that considers that chronic symptoms reflect some form of chronic, injury related damage cannot account for wide differences in the prevalence of such behaviours between different countries and even different regions of the same country.

#### SINGAPORE AND AUSTRALIA

The behaviour of reporting chronic symptoms, which once was so commonly observed in whiplash patients in Australia, does not occur in Singapore.<sup>1</sup> This is despite there being at least as many collisions in Singapore. According to J L Balla, the late whiplash syndrome has thus been viewed as a culturally constructed illness behaviour based on indigenous categories and social structural determinants.<sup>1</sup>

Expanding on this observation W B Maguire<sup>2</sup> notes that even in the absence of opportunity for financial gain in Singapore, if there were Singaporeans suffering from severe and long term “whiplash” symptoms, they should still present themselves at the quite sophisticated and free outpatients departments existing in most large Asian cities. Yet he cannot recall having seen one such case.

#### NEW ZEALAND AND AUSTRALIA

There is a very low incidence of whiplash cases in New Zealand, compared with the State of Victoria in Australia, even accounting for the number of vehicles and collisions. Mills and Horne explain that a significant difference between the two systems then was that in Australia the common law system readily offers a route to compensation.<sup>3</sup> This is not to say that financial gain was necessarily the chief factor. As we explain later, it may be that the process of litigation and battling with insurance companies contributes to the behaviour or reporting chronic symptoms. Eliminating the process may be the reason New Zealand patients do so well after acute injury.

#### AUSTRALIA—THEN AND NOW

The effect of legislation changes on the incidence of reporting chronic symptoms from an acute whiplash injury in Australia are impressive.<sup>4,5</sup> In 1987, parliament changed the law: to claim for “whiplash” injury the claimant had to report the accident to the police and bear the first \$317 (indexed for inflation) of medical expenses. In 1988/1989, presumably when there would have been more vehicles on the road than in 1982/1983, there were only 2004 claims for “whiplash”, 47% less than in 1982/1983 and a remarkable 68% less than in 1985/1986.<sup>4</sup>

There are no data to suggest that the patients still exist, but were simply not reporting any problem. Furthermore, the compulsion to bear the first \$317 of medical expenses is unlikely to discourage the innocent injured from making a damages claim as such expenses are invariably spread over weeks or months.<sup>4</sup>

Again, the process of litigation may be the factor to consider, not merely the goal of a monetary reward.

#### UNITED KINGDOM—SOCIAL COPYING

Livingston stated that if chronic whiplash symptoms were a result of physical damage from the acute injury, the existence of a “chronic whiplash syndrome” should have been reported in the United Kingdom not long after it was reported in the United States. Yet, it was not until decades later. Reports of new diseases or forms of trauma spread quickly through the world medical literature, unless they are unique to a particular geographical region or culture. Rear-end collisions have been present in the United Kingdom for as long as in the United States. As Livingston explains<sup>6</sup>: “the *Journal of the American Medical Association* has published papers on whiplash injury since 1953, but 33 years passed before Deans and colleagues described the injury in the *British Medical Journal*, and more time elapsed before the *Lancet* discussed whiplash in British patients. Such a gap suggests that whiplash injury was not a substantial problem in the UK for many years and was partly the result of social copying in the USA, as it was in Victoria, Australia.

#### LITHUANIA

In Lithuania, the acute injury occurs as it does elsewhere in the world. Whiplash injury there, however, does not lead to

---

<sup>★</sup>Adapted from Ferrari R, Russell AS. A minor injury. A mistaken identity. *Hippocrates' Lantern* 1998;5(2):1-7. Aspen Publishers Inc).

a patient reporting chronic symptoms. Lithuania, however, is a country where there is little knowledge or expectation of the potential of a whiplash injury to lead to chronic symptoms, and where involvement of insurance companies, litigation and even the therapeutic community is rare. In this setting, H Schrader *et al* found no increased prevalence of chronic neck pain after a rear-end collision, when compared with the background risk of chronic neck pain in uninjured control subjects.<sup>7,8</sup>

In the 1996 study, the prevalence of any neck pain, for example, in accident victims was 35.1%, and in the general population is approximately 33%. Clearly, being involved in an accident in Lithuania makes one no more likely to report chronic neck pain from that accident injury than not being involved in an accident. This has been confirmed in a prospective, controlled inception cohort study.<sup>8</sup>

#### GREECE

The late whiplash syndrome seems to also be a rare event in Greece. Of 130 accident victims, all suffering acute whiplash injury, 91% recovered in four weeks, the remainder having substantial improvement to the point where their frequency of neck pain was similar to the general population (M Partheni, *et al*. Annual meeting of the North American Spine Society, New York, 1997).

#### GERMANY

The prognosis of acute whiplash injury is also remarkably good in Germany. In a study of physiotherapy treatment, by six weeks the active treatment group and control (healthy) groups were equal in their symptom reporting. Even the group given only a collar for three weeks and no other treatment recovered by 12 weeks. That is, the acute whiplash injury does not seem to confer a greater risk of reporting chronic symptoms than found in the general, uninjured population.<sup>9</sup>

#### INSIDE THE LABORATORY

Experimental collisions with volunteers are less confounded scientifically as they largely avoid the modifying effects of emotional reactions, insurance claims, and compensation or litigation issues. Four decades of such collisions using volunteers have resulted in many individuals with acute neck pain, back pain, and/or headache lasting hours or days. It is important to emphasise, however, that researchers have not found any examples of volunteers with chronic symptoms. This is despite the use of a variety of vehicles, impact directions and speeds, restraint systems, with or without head rests, with varying head inclinations and rotation, with or without tensed neck muscles and more recently with a wide range of young and old, both sexes, non-military volunteers (G P Nielsen, *et al*. Forty First Stapp Car Crash Conference. Society of Automotive Engineers, 1997).<sup>10-13</sup> The collisions experienced in the fairground bumper cars have been shown to be of similar velocity changes to many apparently symptom provoking rear-end collisions with automobiles. Photographs of the bumper car collisions show that the complete absence of neck and upper back support here leads to a much greater cervical hyperextension. Yet, chronic symptoms are not reported.<sup>12</sup>

#### A model

We previously published the view that there is no evidence of chronic damage in the neck or back that results from the acute injury and continues to generate chronic pain.<sup>13,14</sup> Thus, the controversy is what causes the reporting of chronic symptoms that the whiplash victim attributes to the accident and associated "injury". Indeed, the cultural observations compel one to re-evaluate the "injury" (biological) models currently proposed for the late

whiplash syndrome. Two contrasting views exist. One is that whiplash claimants reporting chronic symptoms are malingering. The other is the opposite extreme that some form of chronic physical damage from the initial trauma explains the chronic symptoms. We believe that neither of these models adequately explain the observed behaviours. Although insurance fraud exists, and may be evident to the physician, they commonly judge most claimants to be genuine. Yet, the view that chronic symptoms arise from chronic damage from an acute injury is, as will be seen, insupportable. We offer a third possibility in the form of a biopsychosocial model. This model includes the biological dimension (the possibility of acute injury, and a variety of physical sources for neck pain without any chronic damage from the acute injury), and psychosocial dimensions (the whiplash claimant's belief in the genuineness of their symptoms, and the realisation that accident claimants do not exist in a social and cultural vacuum).

Grade 1 and grade 2 whiplash associated disorders<sup>15</sup> account for most claims of whiplash injury and virtually all of those patients seen by rheumatologists. These are diagnosed purely on the basis of symptom reporting. There are no truly objective findings (that is, findings that are independent of the patient's input). There are many assertions that necropsy studies and anecdotal reports suggest a variety of more serious injuries including significant muscle or ligament tears (that is, more than microscopic bleeding that occurs with minor sprain). These must be discounted. Fatal accidents can hardly reflect the injury in minor collisions, and even non-fatal (but still high velocity) accidents bear little relevance. Moreover, bone scans or magnetic resonance imaging, or both, are routinely capable of detecting significant muscle or ligament tears, disc rupture, spinal joint disruption, or nervous system injury.<sup>14,16</sup> Yet, as we have reviewed, controlled studies of now more than 1000 whiplash patients reporting chronic symptoms have routinely failed to demonstrate such injuries (G Borchgrevink, *et al*. Third Scientific Meeting of the Society of Magnetic Resonance, Nice, France, 1995).<sup>14,16,18,19</sup> In the vast majority of whiplash claimants, radiological findings, whether by radiography or magnetic resonance imaging, are not helpful in demonstrating the injury or source of symptom reporting. Their role is only to exclude a fracture or to locate the source of a clinically evident neurological abnormality. The various, commonly identified 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. Finally, although it has been demonstrated that anaesthetising facet joints (or actually physically interrupting nerve supply to the joints) of the cervical spine may relieve some cases of chronic neck pain<sup>20,21</sup> the inference that facet joints are chronically injured in whiplash claimants remains untenable because of inappropriate patient selection in such studies.<sup>14</sup> Moreover, facet joint injury leading to chronic pain as part of the "whiplash injury" must necessarily be a rare event, given the absence of chronic pain reporting in other cultures following the acute whiplash injury. There is yet no evidence that in most claimants the injury is anything more than a minor sprain, with temporary symptoms.

We suggest that some whiplash claimants behave differently than Singaporeans, Greeks, Lithuanians, or volunteers, because reporting of chronic symptoms represents the intervention of cultural and psychological factors changing the accident victim's behaviour. Many of those factors do not exist in experiment volunteers or evidently in the described accident victims in Lithuania, etc. To say that

psychological or cultural factors are responsible for the behaviour reporting of chronic pain by whiplash claimants, however, is not at all to say that whiplash claimants are malingering. Instead, the symptoms reported are usually genuine, but it is simply not conceivable that they represent an undocumentable progression of what, in most cases, was a minor, acute injury.

#### SYMPTOM EXPECTATION, AMPLIFICATION, AND ATTRIBUTION

It may be more meaningful to consider the mechanisms of symptom expectation, symptom amplification, and symptom attribution in generating the reporting of chronic pain. In certain cultures there is overwhelming information regarding the potential for chronic pain outcomes after whiplash injury, with widespread knowledge of the expected symptoms even among people with no personal experience of having an accident.<sup>22 23</sup> The cultural information may even pervade on a pre-conscious level.<sup>24</sup> This expectation will in turn lead the people 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—symptom amplification.<sup>25</sup>

A number of other factors may lead to symptom amplification. Fear and anxiety about the event, about one's symptoms, or their outcome, are chief among them. The circumstances of the accident immediately create an impression that the minor injury is not benign. The patient's fear may start when paramedics take him out of the car in a special stretcher, apply a hard collar, and warn him not to move. Instructed to ask about neck pain, police suggest a medical visit immediately if there are any symptoms, even if the injured party feels otherwise well. Considered important or necessary responses from the paramedics and police, these nevertheless do create fear. This may amplify symptoms. As indicated by A J Barsky<sup>26</sup>: "Symptoms are intensified when they are attributed to a serious disease than to more benign causes such as ... lack of sleep, lack of exercise, or overwork."

Fear may also be generated later by the responses from physicians after the accident: "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 x ray 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. Considering the multiple sources of information that engender an expectation of chronic disability, it is not surprising such an outcome may evolve. It has been demonstrated that patients with an initial concern or fear of longlasting symptoms and disability have symptoms for a longer duration than those who do not expect or worry less about these possibilities.<sup>27</sup>

Another aspect of symptom amplification occurs when others have the accident 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). Again, as Barsky explains<sup>26</sup>: "Attention to a symptom amplifies it, whereas distractions diminish it. Thus the more frequently ... patients are asked to rate their pain, the more intense they rate it."

In addition, when a form of treatment fails, this may have an important adverse psychological effect on the patient. Patients are likely to assume not that the therapy was inappropriate for their particular problem, but that they have a

resistant or more severe physical injury than realised. This can only serve to increase their fear about future health.

Finally, amidst all the above, there may be symptom attribution. That is, the whiplash patient may be attributing non-accident symptoms to the accident. Neck and back pain are endemic in western civilisation. Thus, had they not been an accident victim, they would still carry some risk of developing such symptoms in the future, and be exposed to this background incidence of neck and back pain. They may have even had such symptoms before, but do not recall that now. It may not be simply that they are hiding this information (although some clearly do this). Instead, an accident victim becomes hypervigilant for symptoms. In the setting of amplification, these previously unintrusive symptoms, largely ignored in daily life, become far more intrusive after the accident. The patient regards them as new, and attributes them to the accident.

Expectation of chronic pain together with the claim process (with or without litigation) may lead whiplash patients to attribute any and all future neck or back pain to the accident. Few tell the whiplash patient to consider that their sudden increase in neck pain a year after the accident is because of some entirely new event. Rather they attribute it to a "flare up of the accident injury." Thus, physicians, lawyers, and therapists may suffer from the same malady of symptom attribution as does the claimant. It is perfectly understandable why a claimant would want to have all of their perceived injuries and suffering documented. There is no doubt, however, that this very same activity leads to symptom amplification and attribution.

Thus, a host of psychological factors, most culturally determined, change the behaviour of some whiplash patients, leading them to expect, amplify, and attribute symptoms in a chronic fashion. We do not conclude that the chronic pain is a result of a psychiatric disease. It may have a variety of physical sources, but not chronic damage following the acute injury. Psychosocial factors lead to amplification and misattribution of pain from such sources in the setting of expectation of chronic pain. When the effect of anxiety, anger, resentment, and money have on symptom reporting are also included—a biopsychosocial model is born.

Although it has been claimed that psychosocial factors before or after the accident are not aetiological in reporting of chronic symptoms,<sup>28</sup> there are a number of reasons to doubt this conclusion, particularly given the cultural observations. Radanov *et al* did not perform a controlled inception cohort study, but rather selectively gathered 117 patients through advertisement. They conclude that psychosocial factors are not relevant in symptom reporting other than as a response to the chronic pain.<sup>28</sup> Interestingly, Radanov *et al* did demonstrate that the prognosis is somewhat better in Switzerland than North America, the absence of litigation in Switzerland being one major difference that may be relevant. Alternatively, the results could be accounted by the selection of study participants, and the other methodological flaws. Others have indicated that the studies of Radanov *et al* are fraught with at least 15 significant methodological flaws or sources of bias.<sup>19 29-31</sup> Kwan and Friel point out that a single group of 117 whiplash claimants, in a single country, with the large number of methodological concerns is incapable of resolving this complex issue.<sup>29</sup> Studies that have improved on this methodology demonstrate that symptom reporting at four weeks after the accident can be predicted by psychological factors,<sup>29</sup> and that symptom reporting at six months after the accident is best predicted by non-accident related stressors.<sup>19</sup> That cognitive therapy alone is effective in removing such symptoms confirms that behaviour patterns may be not only contributory, but aetiological in symptom perception and reporting.<sup>31</sup>

## SUMMARY

In countries with a very low or non-existent prevalence of late whiplash syndrome, accident victims do not routinely hear reports of acute whiplash injury leading to chronic symptoms or disability. They do not witness such behaviour in others, and do not thereby have any expectation of such possibilities. They do not engage in a process that encourages hypervigilance for and attention to symptoms, thus eliminating many factors that promote symptom amplification. They also do not engage in a process that engenders anxiety, frustration, and resentment (that is, battling with insurance companies and proving that your pain is real). They do not change their activity in response to what they, after all, view as a minor injury. They will not amplify pre-accident symptoms, or symptoms or amplify daily life's aches and pains. They will not attribute all these different sources of symptoms to chronic damage they believe the accident caused. There is no cultural information to encourage this chronic pain behaviour being seen in other cultures.

**Treatment of the minor injury**

Diseases desperate grown, by desperate appliance are relieved, or not at all. (William Shakespeare. *Hamlet. Prince of Denmark*. Act IV, Scene III.)

From the point of view of "healing the injury", you would not expect any need for treatment, as the injury is a minor sprain (and no treatment appears to be necessary in experiment volunteers or Lithuanians). Those reporting chronic symptoms, however, have a behaviour that is contrast with these other two groups. Changing the behaviour to prevent chronic symptom reporting is the basis for treatment.

You could begin by reassuring patients that they have received a physical injury that will resolve within days to weeks at most. There is no evidence that whiplash damages neck irreversibly, but rather that other factors lead to ongoing experience of pain. Simply discussing with the patient, and encouraging them to come to terms with, the natural anxieties that occur after an accident may reduce symptoms.<sup>32</sup> Studies demonstrate the existence of increased muscle activity with psychological distress<sup>33</sup> (which may in part explain the findings of Karlsborg *et al*<sup>19</sup>).

It may thus be that the effect of "successful" treatment is an effect on the behaviour of the patient more than an effect on "healing of an injury".<sup>34, 35</sup> Encourage the patient to not change their activity levels after the first day or two of rest. (The Quebec Task Force recommends not prescribing rest beyond four days.<sup>15</sup>) Indeed, whiplash patients who immediately after the accident are told to "act as usual" and not allowed sick leave have a better outcome than those given 14 days sick leave and rest.<sup>35</sup> In keeping with the view of a benign injury, one should thus avoid treatments that give the patient the opposite impression and encourage the sick role (for example, medications, collars, and passive therapy). Many recommend using exercises such as posterior neck muscle stretches and encouraging good posture, both of which have some benefit.<sup>15, 34-37</sup> This is perhaps again because these treatment impress upon the patient that rest and "caution" are not necessary. Furthermore, the development of poor posture itself (from inactivity or a maladaptive response to neck pain) may cause symptoms.<sup>37-42</sup> This merely adds another factor that leads to chronic symptom reporting long after the injury has healed.

In the end, the return of health for whiplash claimants reporting chronic symptoms requires a drastic shift in the medical and social paradigms with which we unravel the true source of their suffering.

ROBERT FERRARI  
ANTHONY S RUSSELL

562 Heritage Medical Research Centre, University of Alberta, Edmonton,  
Alberta, Canada T6G 2S2

Correspondence to: Dr Russell.

- 1 Balla JI. Report to the Motor Accidents Board of Victoria on whiplash injuries, 1984. In: Hopkins A, ed. *Headache, problems in diagnosis and management*. London: W B Saunders, 1988:268-89.
- 2 Maguire WB. Whiplash in Australia: illness or injury? [Letter]. *Med J Aust* 1993;158:138.
- 3 Mills H, Horne G. Whiplash - manmade disease. *N Z Med J* 1986;99:373-4.
- 4 Awerbuch MS. Whiplash in Australia: illness or injury? *Med J Aust* 1992;157:193-6.
- 5 McDermott FT. Reduction in cervical "whiplash" after new motor vehicle accident legislation in Victoria. *Med J Aust* 1993;158:720-1.
- 6 Livingston M. Whiplash injury: misconceptions and remedies. *Aust Fam Physician* 1992;21:1642-4.
- 7 Schrader H, Obelieniene D, Bovim G, Surkiene D, Mickeviciene D, Mickeviciene I, *et al*. Natural evolution of late whiplash syndrome outside the medicolegal context. *Lancet* 1996;347:1207-11.
- 8 Obelieniene D, Schrader H, Bovim G, Miseviciene I, Sand T. Pain after whiplash—a prospective controlled inception cohort study. *J Neurol Neurosurg Psychiatry* (in press).
- 9 Bonk A, Giebel GD, Edelmann M, Huser R. Whiplash in Germany. *J Rheumatol* (in press).
- 10 Gough JP. Human occupant dynamics on low-speed rear end collisions: An engineering perspective. *Journal of Musculoskeletal Pain* 1996;4:11-19.
- 11 DuBois RA, McNally BF, DiGregorio JS, Phillips GJ. Low velocity car-to-bus test impacts. *Accident Reconstruction Journal* 1996;8:44-51.
- 12 Castro WHM, Schilgen M, Meyer S, Weber M, Peuker C, Wörtler K. Do "whiplash injures" occur in low-speed rear impacts? *Eur Spine J* 1997;6:366-75.
- 13 Ferrari R, Russell AS. The whiplash syndrome - common sense revisited. *J Rheumatol* 1997;24:618-23.
- 14 Ferrari R, Russell AS. Authors' reply. [Letter]. *J Rheumatol* 1998;25:1438-9.
- 15 Spitzer WO, Skovron ML, Salmi LR, Cassidy JD, Duranceau J, Suissa S, *et al*. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders. *Spine* 1995;20 (suppl 8):1-73S.
- 16 Borchgrevink GE, Smevik O, Nordby A, Rinck PA, Stiles TC, Lereim I. MR imaging and radiography of patients with cervical hyperextension-flexion injuries after car accidents. *Acta Radiol* 1995;36:425-8.
- 17 Gundry CR, Fritts HM. Magnetic resonance imaging of the musculoskeletal system. Part 8. The spine, section 2. *Clin Orthop* 1997;343:260-71.
- 18 Pettersson K, Hildingsson C, Toolanen G, Fagerlund M, Björnebrink J. Disc pathology after whiplash injury. A prospective magnetic resonance imaging clinical investigation. *Spine* 1997;22:283-8.
- 19 Karlsborg M, Smed A, Jespersen H, Stephensen S, Cortsen M, Jennum P, *et al*. A prospective study of 39 patients with whiplash injury. *Acta Neurol Scand* 1997;95:65-72.
- 20 Lord SM, Barnsley L, Bogduk N. The utility of comparative local anaesthetic blocks versus placebo-controlled blocks for the diagnosis of cervical zygapophysial joint pain. *Clin J Pain* 1995;11:208-13.
- 21 Lord SM, Barnsley L, Wallis BJ, McDonald GJ, Bogduk N. Percutaneous radio frequency neurotomy for chronic cervical zygapophysial joint pain. *N Engl J Med* 1996;335:1721-26.
- 22 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.
- 23 Mittenberg W, DiGiulio DV, Perrin S, Bass AE. Symptoms following mild head injury: expectation as aetiology. *J Neurol Neurosurg Psychiatry* 1992;55:200-4.
- 24 Shorter E. *From paralysis to fatigue*. Toronto: Maxwell Macmillan, 1992:ix.
- 25 Barksy A, Goodson JD, Lane RS, Cleary PD. The amplification of somatic symptoms. *Psychosom Med* 1988;50:510-19.
- 26 Barsky AJ. Amplification, somatization, and the somatoform disorders. *Psychosomatics* 1992;33:28-34.
- 27 Radanov BP, Sturzenegger M, De Stefano G. Long-term outcome after whiplash injury. *Medicine* 1995;74:281-97.
- 28 Radanov BP. Common whiplash-research findings revisited. *J Rheumatol* 1997;24:623-5.
- 29 Kwan O, Friel J. A comment on Radanov BP. Common whiplash - research findings revisited. [Letter]. *J Rheumatol* (in press).
- 30 Drottning M, Staff PH, Levin L, Malt UF. Acute emotional response to common whiplash predicts subsequent pain complaints. A prospective study of 107 subjects sustaining whiplash injury. *Nordic Journal of Psychiatry* 1995;49:293-9.
- 31 Van Akkerveken PF, Vendrig AA. Chronic symptoms after whiplash: a cognitive approach. In: Gunzburg R, Szpalski M, eds. *Whiplash injuries. Current concepts in prevention, diagnosis, and treatment of the cervical whiplash syndrome*. Philadelphia: Lippincott-Raven, 1998:183-91.
- 32 Provinciali L, Baroni M, Illuminati L, Ceravolo MG. Multimodal treatment to prevent the late whiplash syndrome. *Scand J Rehab Med* 1996;28:105-11.
- 33 Kellner R. *Psychosomatic syndromes and somatic symptoms*. Washington: American Psychiatric Press, 1991:195-6.
- 34 McKinney LA. Early mobilisation and outcome in acute sprains of the neck. *BMJ* 1989;299:1006-8.
- 35 Borchgrevink GE, Kaasa A, McDonagh, Stiles TC, Haraldseth O, Lereim I. 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.
- 36 Mealy K, Brennan H, Felton GC. Early mobilization of acute whiplash injuries. *BMJ* 1986;6:27-33.
- 37 McKenzie RA. *The cervical and thoracic spine*. New Zealand: Spinal Publications Ltd, 1990:25-6.
- 38 Cesarani A, Alpini D, Bonvier R, Claussen CF, Gagey PM, Magnusson L, *et al*. *Whiplash injuries. Diagnosis and treatment*. Berlin: Springer, 1996:8.

- 39 Watson DH, Trott PH. Cervical headache: an investigation of natural head posture and upper cervical flexor muscle performance. *Cephalalgia* 1993;13:272-84.
- 40 Harms-Ringdahl K, Ekholm J. Intensity and character of pain and muscular activity levels elicited by maintained extreme flexion position of the lower-cervical-upper thoracic spine. *Scand J Rehab Med* 1986;18:117-26.
- 41 Middaugh S, Kee W, Nicholson J. Muscle overuse and posture as factors in the development and maintenance of chronic musculoskeletal pain. In: Grzesiak R, Ciccone D, eds. *Psychological vulnerability to chronic pain*. New York: Springer, 1994:55-89.
- 42 Hedman TP, Fernie GR. Mechanical response of the lumbar spine to seated postural loads. *Spine* 1997;22:734-43.

### ***Annals of the Rheumatic Diseases* - <http://www.annrheumdis.com>**

Visitors to the world wide web can now access *Annals of the Rheumatic Diseases* either through the BMJ Publishing Group's home page (<http://www.bmjpub.com>) or directly by using its individual URL (<http://www.annrheumdis.com>). There they will find the following:

- Current contents list for the journal
- Contents lists of previous issues
- Members of the editorial board
- Subscribers' information
- Instructions for authors
- Details of reprint services

A hotlink gives access to:

- BMJ Publishing Group home page
- British Medical Association web site
- Online books catalogue
- BMJ Publishing Group books

The web site is at a preliminary stage and there are plans to develop it into a more sophisticated site. Suggestions from visitors about features they would like to see are welcomed. They can be left via the opening page of the BMJ Publishing Group site or, alternatively, via the journal page, through "about this site".