10 Whiplash-associated disorders

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Previous accounts used different terms – whiplash injury, hyperextension injury, acceleration injury, soft tissue neck injury, cervical strain, cervical sprain – to describe the lesion that may occur after and the consequences of a typical car accident: a rear-end motor vehicle collision.

When a vehicle is struck from the rear, the occupants rarely have any warning and do not brace the muscles to prevent head movement. As a result, the body is propelled forwards and the neck hyperextends backwards well beyond the normal range of allowable movement (Fig. 10.1). This violent motion is followed by a less rapid forwards recoil into flexion that can often result in a head injury if the head impinges on the windscreen.

Although rear-end impacts are most common and result in hyperextension–hyperflexion injuries, other types of car accident may also cause whiplash-type injuries and, in any trauma, complex head and neck movements may occur, leading to different lesions that resemble whiplash.

Definition

Medical literature, in an attempt to find a proper definition, has so far described 'whiplash injury' in terms of the mechanism of the accident, the type of lesion that is caused or the clinical appearance after the injury. In 1995[1] the Quebec Task Force (QTF) proposed the following definition:

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

The term 'whiplash-associated disorders' (WAD) indicates the clinical features that result from an accident in which two elements – acceleration, followed by deceleration – are responsible for the traumatic forces that act on the cervical spine and related structures.

Incidence

As the result of increases in availability and use of cars worldwide, motor vehicle accidents have become very frequent with, as a result, an enormous increase in whiplash-type trauma. It is one of the most common mechanisms of injury to the cervical spine.

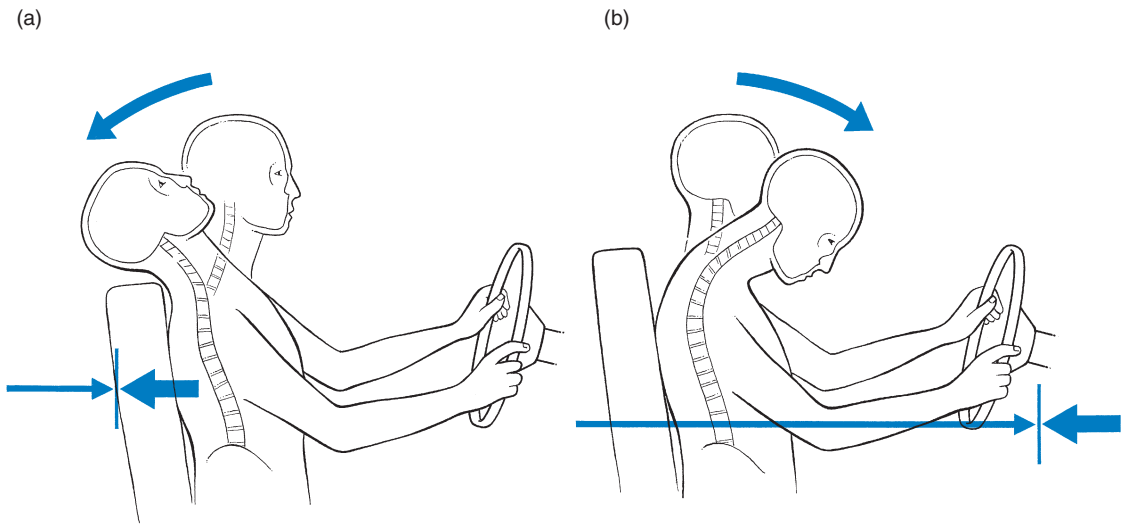


Fig 10.1 • Whiplash injury: (a) hyperextension on impact, followed by (b) recoil into flexion.

The incidence is not precisely known. A figure of 1 per 1000 people per year has been suggested.[2]

The QTF mentions figures on whiplash injuries in Canada. In 1987 in the province of Quebec there were approximately 131 whiplash injuries per 100 000 vehicles per year – 70 injuries per 100 000 inhabitants. This cost the Canadian government CAN $19 000 000, of which 70% was income compensation. The female : male ratio is about 1.5 : 1 and the main age group 20–24 years.

Other studies in Canada mention 5000 whiplash cases a year in the province of Quebec, accounting for 20% of all insurance claims after motor vehicle accidents.[3,4]

In the United States 11 300 000 car accidents were reported for the year 1991, of which 2 690 000 were rear-end collisions and caused 85% of all whiplash injuries.[5]

Classification

The QTF, persuaded that proper diagnosis is difficult to achieve, has proposed two classifications: one according to the severity of the symptoms and signs (grades) (Box 10.1) and one according to the time elapsed since the accident (stages) (Box 10.2).

Neither classification suggests what lesion is present nor stipulates the type of tissue damage. They reflect only the clinical appearances that occur after acceleration–deceleration injury.

Pathology

Depending on the movement of the head during the accident, several lesions may occur, ranking from severe to moderate to slight. Hyperextension is the most common mechanism, followed by hyperflexion and lateral flexion.[6]

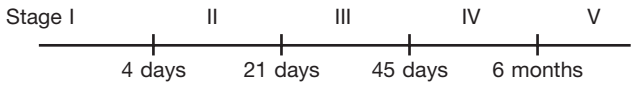
Box 10.1

Clinical (Quebec) classification of whiplash-associated disorders

Grade	Clinical presentation
0	No neck complaint No physical sign(s)
I	Neck complaint involving pain, stiffness or tenderness only No physical sign(s)
II	Neck complaint AND Musculoskeletal sign(s)
III	Neck complaint AND Neurological sign(s)
IV	Neck complaint AND Fracture or dislocation

Box 10.2

Classification according to the time elapsed since the accident



Severe lesions

Hyperextension and distraction of the neck may rupture the anterior longitudinal ligament as well as some discs. A ruptured disc can lead to backward displacement of the vertebra lying

above it – the upper facets then slide downwards on the lower – with damage to the spinal cord as a result.[7] Spinal cord injuries after motor vehicle accidents occur most often in young car users in the 15–24 year age group.[8,9]

Pure hyperextension may also cause compression of the spinal cord in those cases in which retrolisthesis or spinal stenosis already existed. In other instances, compression fractures of the posterior elements may occur.

Hyperflexion injury may lead to fractures of the vertebral body – most fractures of the atlas[10] and of the axis[11] are the result of motor vehicle accidents – and/or to disruption of posterior ligaments and occasionally facet joint luxation.

Less frequently, lesions of arteries, veins, neural structures, oesophagus and retropharyngeal tissues may occur.

Other lesions

Less severe lesions are much more frequent and may involve the intervertebral discs, the zygapophyseal joints, and the cervical ligaments and muscles. These lesions may occur in isolation but are more often combined and therefore are sometimes difficult to recognize. The common complaint is neck pain.

Discodural and discoradicular interactions

Recent retrospective studies have shown that the occurrence of disc lesions after whiplash injury is quite high[12,13] and one prospective study indicates the value of clinical diagnosis.[14] Most disc lesions are endplate avulsions and ruptures of the anterior annulus fibrosus.

As the result of the hyperextension element during the trauma the disc may have fissured. The subsequent flexion or hyperflexion element causes displacement of disc material in a posterior direction. Davis *et al* describe a number of posterolateral disc lesions with radicular symptoms as the result of a hyperextension whiplash trauma.[12] These herniations seemed to develop only after the acute phase and it took a few weeks for the radicular symptoms to appear. In postmortem studies Taylor *et al* describe the intervertebral disc as the most frequently damaged structure.[15–17] Jónsson *et al*[18] Also confirmed the large number of disc lesions after whiplash, and during surgery were able to confirm the findings from magnetic resonance imaging (MRI).

Posterocentral protrusions lead to central, bilateral or unilateral pain in a multisegmental distribution: pain in the neck, trapezius and upper scapular area. On examination a symmetrical (mimicking a full articular pattern) or asymmetrical pattern of limitation is found. In acute cases the picture may be torticollis-like. For a detailed description of disc pathology, see page 145.

Facet joint problems

Whiplash may also lead to problems at the level of the zygapophyseal joint capsules.[19] Lord *et al* undertook a placebo-controlled prevalence study after whiplash and found that chronic cervical facet joint pain was common.[20]

Pain is felt unilaterally and is usually quite localized. A convergent or divergent motion pattern may occur, although any asymmetrical pattern is compatible. (Facet joint pathology is discussed on p. 163).

Ligamentous lesions

Ligaments can become overstretched, leading to minor lesions,[12] or may become adherent as the result of post-traumatic immobilization. They present with vague stretching pain felt at the end of range of those movements that stretch the ligament (see p. 168).

Muscular lesions

Muscular lesions, mostly anteriorly, are described in clinical studies,[21,22] on echography,[23] in experiments in animals[24,25] and in postmortem studies.[26] Muscles, particularly their occipital insertions, can be strained during injury. The subsequent pain will be quite localized and can be elicited during either contraction or stretching – the contractile tissue pattern (see p. 169).

Medicolegal consequences

As WAD automatically involve compensation claims, they have considerable consequences. If a significant number of patients remain with some disability – and this number still seems to be increasing – the costs of diagnosis, treatment and indemnity become progressively higher.

The different parties involved in the approach to WAD are:

- The patient who is seeking help and a refund of money. For most patients the two elements do not influence each other but for a number of people the compensation claim is essential. This may result in absence from work, illness behaviour, social disability, malingering and fraud.
- Doctors who are looking for a diagnosis. Physicians treating the patient wish to reach a diagnosis and to confirm it with technical investigations. They also – because of the often-complex nature of the syndrome – use an extensive pattern of treatment techniques. Doctors who work for the insurance company are sometimes biased and tend to over-diagnose the condition as 'psychogenic' or 'simulation'.
- Insurance companies who are trying to minimize their payments. As the result of the lack of consensus about diagnosis and treatment of WAD, insurance companies see their compensation payments and indemnities rise considerably. They exert pressure on governments in order to keep these expenses under control.
- Lawyers who are protecting either the insurance company or the patient. Discussions about confirmation of the lesions and the consequences for patients' professional activities lead to an increase in litigation.

It is clear that eligibility for compensation for pain and suffering furthers the perseverance of symptoms and a tendency to

chronicity. Where this compensation can be eliminated, a decrease in incidence and an improved prognosis are seen.[27]

Psychological problems

Most WAD start as an ordinary trauma followed by purely physical disturbance. When subsequent treatment is unable to rehabilitate the patient after a short period of time, secondary emotional and psychological changes may supervene. They raise the patient's awareness of neck pain and subsequently aggravate and perpetuate the pain, or even turn a simple neckache into chronic pain and disability.[28]

Diagnosis

Clinical picture

Making a diagnosis in patients who have undergone a whiplash-type injury is no different from other patient groups. It requires proper history taking, inspection and careful functional examination, including a neurological evaluation.

Severe lesions should be recognized and the patient immediately treated as necessary. Most of these conditions are classified as grade IV and fall outside the scope of this book. They will probably be recognized clinically when warning signs are present but certainly be detected by performing the necessary technical investigations: radiography, computed tomography (CT), scintigraphy and/or MRI.

Technical investigations in post-traumatic neck patients are mandatory but the decision as to which imaging technique to use should be based on clinical grounds. Radiographs in patients with soft tissue injuries are often negative: no fractures or luxations are found. The finding most commonly obtained is loss of the normal cervical curvature on a lateral view.[29] CT and MRI are not very helpful in recent cases but may become important when the condition persists, although their use is still controversial. Scintigraphy may be useful to screen for occult fractures.[30]

The reader is referred to the section on non-discogenic disorders (see Ch. 9) for further information about the diagnosis of non-mechanical conditions.

In most cases the condition is *not severe* and clinical examination initially suggests some of the above-mentioned conditions, although the picture may sometimes be vague and difficult to interpret.

Diagnostic difficulties

Most patients present a genuine clinical pattern. The symptoms and signs are clear and not too difficult to interpret. Some patients give a more diffuse picture, probably because there is a combination of lesions. The examiner should then concentrate on the features that are understood and compatible with a known syndrome.

Most difficult are those patients who exaggerate or simulate their symptoms. They pretend to have problems in the hope of persuading the examiner or of making people believe their story. The examiner should look for inherent unlikely details and inconsistencies in the history and functional examination, which will enable a positive diagnosis of 'psychogenic pain' or 'malingering' to be made.

Symptoms

Immediately after the accident the patient is stunned and confused, and complains mainly of any head problem that is present. A feeling of discomfort in the neck, often associated with some degree of nausea, may develop. Although not usually reported, up to 60% of patients have evidence of concussion with momentary loss of consciousness. Examination in a hospital emergency department may not reveal any positive signs and discharge follows. In the following hours and days, a cluster of symptoms may then develop: soreness, tenderness and swelling in the anterior neck region, stiffness and restriction of movement at the neck, headache, visual and auditory disturbances, dizziness, concentration and memory disturbances, pain in the upper thorax, scapular area, shoulder and arms, and numbness or paraesthesia in the upper limbs, accompanied by a feeling of heaviness and weakness. Most symptoms gradually disappear but the majority of patients are left with pain in the neck, radiating to the scapular area or to the shoulder region.

The examiner should enquire about the mechanism and velocity of the trauma so that a judgement can be made about the severity of the injury and so that a prognosis can be reached.[31] Other important information relates to the time interval between the accident and the onset of symptoms, which often is 2–3 days.[32] Many patients have associated low back pain[33] and this is noted and assessed later.

Important information can be obtained by asking the patient to describe exactly the localization of the symptoms: central or bilateral symptoms suggest a condition lying at the midline, whereas unilateral symptoms may stem from either a central or a unilateral condition.

The examiner also concentrates on the inherent likelihood of a certain type of lesion: for example, multisegmental pain in disc lesions, segmental pain in facet lesions, and local pain in muscular lesions. When a patient continuously presents inconsistencies or an improbable combination of symptoms and this is later confirmed during the functional examination, examiners should be on their guard.

Signs

Inspection of the position of the patient's neck may indicate an acute condition: for example, when a torticollis-like picture is found or when muscle spasm is present.

When the head is fixed in flexion with central or bilateral pain, a posterocentral disc displacement is clearly present.

Articular signs – pain on movement with or without limitation – suggest involvement of the intervertebral joint or the facet joints, certainly when movement is shown to be restricted. Discodural or discoradicular interactions are considered when articular signs are accompanied by dural or radicular signs (see p. 145). When there is no dural or nerve root involvement, the possibility of a condition involving the facet joint exists; the pain is purely unilateral, i.e. when either a convergent or a divergent pattern is found. Limitation of movement indicates that the joints are responsible.

End-range pain is typical of ligamentous or muscular conditions, the latter also giving rise to positive resisted movements (see p. 169).

Because combined lesions are not at all uncommon, the clinical picture may become difficult and therefore hard to interpret. It should be looked at in the light of the anatomical findings and compared to the clinical pictures known to occur in the cervical spine (see Ch. 8).

Natural history

The natural history of WAD is difficult to predict. An extensive study in Switzerland found that 30% of patients still complained of symptoms, even 4–7 years after the accident.[34] Another study showed that increasing age and the severity of the initial neck pain were predictors of lasting symptoms after 6 months.[35] Objective neurological signs and degenerative changes on radiographs or MRI (spondylosis, diminution of the diameter of the spinal canal) may be associated with a poor prognosis.

In 30–40% the condition needs up to 1 year to recover, ache diminishing quite considerably in the first 2 months. The pain usually disappears but, when no treatment is given, movements may remain restricted.

Chronicity

Not all patients will develop chronic symptoms after whiplash injury. In most instances it is a benign, self-limiting condition. All patients destined to recover will do so in the first 2–3 months after trauma. Those who do not may claim symptoms for several more years. Recent studies have indicated that between 14 and 42% of patients develop chronic symptoms and that about 10% have permanent difficulties,[36–42] although patients may improve even after many years.[43] A predisposing factor for chronicity could be pre-existing spondylosis.[44]

The QTF study clearly demonstrates that the most common whiplash injury – without bony or cord lesions – is essentially a benign and self-limiting condition. A small number of patients are refractory and responsible for the enormous costs incurred by the injury: about 50% of all costs are spent on an eighth of the total number of victims.

Of the 1 000 000 whiplash injuries per year in the USA, the majority of patients become asymptomatic after a limited number of weeks or months. According to certain statistics, 20–40% continue to have invalidaty symptoms for several years.

In those countries where the entity of 'chronic pain' resulting from rear-end collisions is not known and consequently there is no fear of long-term disability leading to indemnity and litigation, symptoms after whiplash injury are self-limiting, lasting for only a short period of time, and there is no evolution towards the chronic stage.[45]

Therapeutic approach

There is still some discussion about how to approach patients with lasting symptoms from WAD. It is difficult to find objective signs and physicians are therefore divided into two groups: those who believe the patients and those who think in terms of psychogenic pain, personality and chronicity because of concerns about financial compensation.

There is some consensus about the possibility of encouraging the patient to develop an active and positive attitude towards the problem. In those patients who do not present with intensive neck pain immediately following the accident, who do not show clinical signs of cord or root compression and in whom routine radiographs show no bony abnormalities, all further extensive and expensive examinations should be avoided.

Patients should be encouraged to remain active and functional. In the acute phase analgesics and anti-inflammatories should be administered but only temporarily. A collar should not be used for more than a few days and should be replaced by early mobilization of the cervical spine. The patient should be taught how to perform active movements, and prolonged physiotherapy must be avoided in order not to push the patient into a passive disabled attitude.

Such an approach is also recommended by the QTF in order to avoid prolonged disability.[46] An early return to work is advocated as one of the best measures to avoid chronicity. Immobilizing measures such as bed rest and collars are best avoided.

Specific treatment

In those cases where the diagnosis is clear, the condition must be treated properly along with the lesion.

When a discal pattern is found and thus a discodural or discoradicular interaction is present, manipulation is performed immediately because it is unwise to leave the displacement untreated; posterocentral protrusions draw out osteophytes fairly quickly, and this may lead to a situation in which extension or one rotation becomes permanently blocked. Because a posterocentral protrusion is present, great care must be taken in the choice of techniques and operation; rotation movements are avoided and the therapist must be experienced. In acute cases with gross deviation, manipulation is performed every day. Traction in the direction of the deviation, followed by pure traction manipulations without articular movement, quickly leads to full recovery. The more moderate and long-standing cases require more treatments, performed once or twice a week over a few weeks. The techniques for posterocentral

protrusions are used: straight pull, lateral flexion, anteroposterior gliding and traction with leverage (see p. 201).

Facet joint lesions can be treated either with steroid infiltration or with deep transverse massage; in more chronic cases slow stretching is used (see p. 201).

Ligamentous lesions are best treated with deep transverse massage, unless there are adhesions which can be manipulatively broken. Muscular lesions respond to deep transverse friction or infiltration with local anaesthetic. If a combination of ligament and muscle damage exists, the muscle should be treated first.

Proper treatment and re-examination on a regular basis are the guarantees of a maximal therapeutic result.

 Access the complete reference list online at **www.orthopaedicmedicineonline.com**

References

1. Scientific Monograph of the Quebec Task Force on Whiplash-Associated Disorders. *Spine* 1995;**20(8S)**:22S.
2. Barnsley L, Lord S, Bogduk N. Whiplash injuries. *Pain* 1994;**58**:283–307.
3. Girard N. Statistiques descriptives sur la nature des blessures. Québec: Régie de l'assurance automobile du Québec, Direction des services médicaux et de la réadaptation, April. Internal Document, 1989.
4. Giroux M. Les Blessures à la colonne cervicale: importance du problème. *Le Médecin du Québec* 1991;**Sept**:22–6.
5. Van Goethem JWM, Biltjes IGGM, van den Hauwe L, et al. Whiplash injuries: is there a role for imaging? *Eur J Radiol* 1995;**22**:8–14.
6. Hohl M. Soft-tissue neck injuries. In: The Cervical Spine Research Society, Sherk HH, editor. *The Cervical Spine*. 2nd ed. Philadelphia: Lippincott; 1989. p. 436–41.
7. Kinoshita H. Pathology of hyperextension injuries of the cervical spine. *Paraplegia* 1994;**32**:367–74.
8. Northrup BE. Evaluation and early treatment of acute injuries to the spine and spinal cord. In: The Cervical Spine Research Society, Clark CR, editor. *The Cervical Spine*. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 541–9.
9. Myers BS, McElhaney JH. *Cervical Spine Injury Mechanisms. Biomechanics and Prevention*. New York: Springer; 1993. p. 311–61.
10. Kurz LT. Fractures of the first cervical vertebra. In: The Cervical Spine Research Society, Clark CR, editor. *The Cervical Spine*. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 409–13.
11. Clark CR, White AA III. Fractures of the dens: a multicenter study. *J Bone Joint Surg* 1985;**67A**:1340–8.
12. Davis S, Teresi L, Bradley W, et al. Cervical spine hyperextension injuries: MR findings. *Radiology* 1991;**180**:245–51.
13. Hamer AJ, Gargan MF, Bannister GC, Nelson RJ. Whiplash injury and surgically treated cervical disc disease. *Injury* 1993;**24**:549–50.
14. Pettersson K, Hildingsson C, Toolanen G, et al. Disc pathology after whiplash injury. A prospective magnetic resonance imaging and clinical investigation. *Spine* 1997;**22(3)**: 283–7.
15. Taylor JR, Finch PM. Neck sprain. *J Aust Fam Physician* 1993;**22**:1623–9.
16. Taylor JR, Twomey LT. Acute injuries to cervical joints: an autopsy study of neck sprain. *Spine* 1993;**18(9)**:1115–22.
17. Taylor JR, Twoney LT. Disc injuries in cervical trauma. *Lancet* 1991;**338**:1340–3.
18. Jónsson H Jr, Cesarini K, Sahlstedt B, Rauschning W. Findings and outcome in whiplash-type neck distorsions. *Spine* 1994;**19**:2733–43.
19. Ketroser DB. Whiplash, chronic neck pain, and zygapophyseal joint disorders. A selective review. *Minn Med J* 2000;**83(2)**: 51–4.
20. Lord SM, Barnsley L, Wallis BJ, Bogduk N. Chronic cervical zygapophysial joint pain after whiplash. A placebo-controlled prevalence study. *Spine* 1996;**21(15)**: 1737–45.
21. Frankel VH. Pathomechanics of whiplash injuries to the neck. In: Morley TP, editor. *Current Controversies in Neurosurgery*. Philadelphia: Saunders; 1996. p. 39–50.
22. Jeffreys E. Soft tissue injuries of the cervical spine. In: *Disorders of the Cervical Spine*. London: Butterworth; 1980. p. 81–9.
23. Martino F, Ettore GC, Cafaro E, et al. L'Ecographia musculo-tendinea nei traumi distorvi acuti del collo. *Radiol Med Torino* 1992;**83**:211–5.
24. Macnab I. Whiplash injuries of the neck. *Manitoba Med Rev* 1966;**46**:172–4.
25. La Rocca H. Acceleration injuries of the neck. *Clin Neurosurg* 1978;**25**:209–17.
26. Jónsson H Jr, Bring G, Rauschning W, Sahlstedt B. Hidden cervical spine injuries in traffic accident victims with skull fractures. *J Spinal Discord* 1991;**4**:251–63.
27. Cassidy JD, Carroll LJ, Coté P, et al. Effect of eliminating compensation for pain and suffering on the outcome of insurance claims for whiplash injury. *NEJM* 2000;**342(16)**:1179–86.
28. Waddell G. *The Back Pain Revolution*. Edinburgh: Churchill Livingstone; 1998. p. 173–86.
29. Daffner RH. Evaluation of cervical injuries. *Sem Roentgenol* 1992;**27**:239–53.
30. Kitchel SH. Soft-tissue neck injuries. In: The Cervical Spine Research Society, Clark CR, editor. *The Cervical Spine*. 3rd ed. Philadelphia: Lippincott-Raven; 1998. p. 351–5.
31. Gebhard JS, Donaldson DH, Brown CW. Soft tissue injuries of the cervical spine. *Orthop Rev* 1994;**(Suppl 1)**:9–17.
32. Jolliffe VM. Soft tissue injury of the cervical spine: consider the nature of the accident. *BMJ* 1993;**307**:439–40.
33. Taylor JR, Finch PM. Neck sprain. *Aust Fam Physician* 1993;**22**:1623–5.
34. Dvorak J, Valach L, Schmidt S. Cervical spine injuries in Switzerland. *J Manual Med* 1989;**4**:7–16.
35. Radanov BP, di Stefano G, Schnidrig A, Ballinari P. Role of psychosocial stress in recovery from common whiplash. *Lancet* 1991;**338**:712–5.
36. Hodgson SP, Grundy M. Whiplash injuries: their long-term prognosis and its relationship to compensation. *Neuro-Orthop* 1989;**7**:88–91.
37. Hildingsson C, Toolanen G. Outcome after soft-tissue injury of the cervical spine. A prospective study of 93 car-accident victims. *Acta Orthop Scand* 1990;**61**:357–9.
38. Pettersson K, Kärrholm J, Toolanen G, Hildingsson C. Decreased width of the spinal canal in patients with chronic symptoms after whiplash injury. *Spine* 1995;**20(15)**:1664–7.
39. Barnsley L, Lord S, Bogduk N. Clinical review. Whiplash injury. *Pain* 1994;**58**: 283–307.
40. Maimaris C, Barnes MR, Allen MJ. 'Whiplash injuries' of the neck: a retrospective study. *Injury* 1988;**19**:393–6.
41. Gargan MF, Bannister GC. Long-term prognosis of soft-tissue injuries of the neck. *J Bone Joint Surg* 1990;**72B**:901–3.
42. Olsson I, Bunketorp O, Carlsson G, et al. An in-depth study of neck injuries in rear end collisions. In: *Proceedings of the International IRCOBI Conference on the Biomechanics of Impacts*. France: Bron-Lyon; Sept 12–14. 1990. p. 269–80.
43. Olivegren H, Jerkvall N, Hagstrom Y, Carlsson J. The long-term prognosis of whiplash-associated disorders (WAD). *Eur Spine J* 1999;**8(5)**:366–70.
44. Bonuccelli U, Paverse N, Lucetti C, et al. Late whiplash syndrome: a clinical and magnetic resonance imaging study. *Functional Neurol* 1999;**14(4)**:219–25.
45. Obelieniene D, Schrader H, Bovim G, et al. Pain after whiplash: a prospective controlled inception cohort study. *J Neurol Neurosurg Psychiatry* 1999;**66(3)**:279–83.
46. Scientific Monograph of the Quebec Task Force on Whiplash-Associated Disorders. *Spine* 1995;**20(8S)**:36S.