SPINE Volume 31, Number 24, pp 2820–2826
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# Magnetic Resonance Imaging Assessment of Craniovertebral Ligaments and Membranes After Whiplash Trauma

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**Study Design.** Review article.

**Objectives.** To review the literature on soft tissue lesions of the upper cervical spine in whiplash trauma with focus on imaging.

**Summary of Background Data.** Whiplash injury is associated with chronic impairment in a substantial number of patients. There are different opinions as to the nature and prognosis of this condition, and the role of diagnostic imaging is debated.

**Methods.** Review the literature on the anatomic source of the chronic whiplash syndrome. Review the literature on imaging of the upper cervical spine, emphasizing on the author's own research.

**Results.** MRI shows structural changes in ligaments and membranes after whiplash injury, and such lesions can be assessed with reasonable reliability. Lesions to specific structures can be linked with specific trauma mechanisms. There is a correlation between clinical impairment and morphologic findings.

**Conclusion.** Whiplash trauma can damage soft tissue structures of the upper cervical spine, particularly the alar ligaments. Structural lesions in this area contribute to the understanding of the chronic whiplash syndrome.

**Key words:** alar ligament, transverse ligament, tectorial membrane, posterior atlanto-occipital membrane, whiplash injury, cervical spine, magnetic resonance imaging. **Spine 2006;31:2820–2826**

Whiplash is an acceleration-deceleration mechanism of energy transfer to the neck, which in turn may lead to a variety of clinical manifestations. It may result from rear-end, frontal, or side impact motor vehicle collisions, but also from fall traumas or other mishaps (whiplash-associated disorders).[1,2]

Most investigators who have studied the natural history of whiplash patients have found long-term symptoms in 24% to 70%, among whom 12% to 16% are severely impaired many years after the accident interfering with their job and everyday activities.[3–6]

Trying to understand the nature of chronic whiplash syndrome, several cervical structures have been in focus. The facet joints as an anatomic source of chronic pain after whiplash trauma have been investigated in several reports.[7,8] These authors conclude that the prevalence of cervical facet joint pain (C2–C3 or below) is 60%.[9] The morphologic correlate to such pain is not known. Post mortem studies of traffic victims have shown that facet joints as well as other structures are vulnerable to cervical trauma,[10] and one may extrapolate that such injures also may occur in whiplash trauma.

A few studies suggest that whiplash trauma causes structural changes predisposing to premature degenerative disc disease.[11,12] Ovadia *et al* found that 10% of patients with normal radiographic findings in the acute stage of whiplash injury developed new degenerative changes at 2- to 3-year follow-up.[13] Micro-avulsions and endplate clefts could possibly cause such degeneration.[10,14] Surgical fusion alleviated pain in some whiplash patients with presumed segmental instability combined with disc protrusion.[15]

The value of flexion and extension views in chronic whiplash syndrome has been evaluated in several reports. Dvorak *et al* found slight hypermobility in the upper and middle cervical levels in the late stage of whiplash injury compared with a healthy population.[16] Such increased angulation can be confined to certain segments only.[17,18] However, other studies showed significantly decreased range of motion in chronic whiplash syndrome compared with asymptomatic individuals.[19–21]

Whiplash injury is not a lethal entity, and its pathologic anatomy cannot be studied directly. Current hypotheses on etiology are based on clinical studies, postmortem studies, and imaging studies. In this presentation, we review the literature on soft tissue lesions of the upper cervical spine focusing on imaging.

## *Craniovertebral Soft Tissue Structures and Whiplash Trauma*

**CT Evaluation of Alar Ligament Insufficiency.** The alar ligaments are the main restraints to axial rotation and lateral bending in the upper cervical spine.[22,23] In hyperextension-hyperflexion injuries, CT has been used to estimate range of rotation in the craniovertebral joints. Increased rotation toward one side was taken as a sign of insufficient alar ligament on the opposite side. In a study of 43 patients with clinically suspected rotatory instability after traffic accidents, 60% showed increased passive rotation at C0–C1 and at C1–C2, strongly suggesting alar ligament insufficiency.[24] The same authors obtained functional CT of 137 whiplash-injured individuals with therapy-resistant neck pain and 35 healthy adults. A ro-

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Acknowledgment date: February 8, 2006. First revision date: April 25, 2006. Acceptance date: April 29, 2006.
The manuscript submitted does not contain information about medical device(s)/drug(s).
Institutional and Foundation funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

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tation at C0–C1 of more than 7° and at C1–C2 more than 54° was regarded as pathologic. As much as 33% of the whiplash group showed signs of increased rotation between these segments.[25] Antinnes *et al*[25a] found asymmetric rotation reaching pathologic values at the level of C0–C1 in 36% of 423 whiplash patients and postulated that alar ligament lesions are responsible for this asymmetry. Surgical fixation of the upper cervical joints has been reported beneficial in selected patients, offering support to the hypothesis of instability of these joints.[26] However, others have not been able to reproduce such instability.[27] The reason could be that functional CT is difficult to standardize. The method depends on the patient's ability to relax, which is difficult because rotation of the head beyond its normal range causes pain and dizziness.[24]

**Postmortem Study of Craniovertebral Ligaments.** Postmortem studies of fatal head/neck injuries document that craniovertebral ligaments are vulnerable to trauma even though there is no dislocation. Saternus and Thrun reported 11 alar ligament lesions of a total of 30 cases.[28] Another study of 21 head-neck injuries also without dislocation showed either laceration or sprain of the alar ligaments in 20 cases. Thirteen of these had injury of the dura mater and the tectorial membrane as well, either lacerations, loosening from the clivus or from the C2 body. One had total rupture and one a partial laceration of the transverse ligament.[29]

**MRI of the Alar Ligaments.** Several investigators have examined the alar ligaments with MRI. Willauschus *et al* studied 8 healthy volunteers and 7 patients with severe upper neck pain after trauma and found no signal changes indicating a lesion, and no difference between the groups.[30] Wilmink and Patijn examined the alar ligaments in 12 patients with chronic whiplash syndrome and 6 controls. The ligaments could be identified in all cases, but alar ligament damage as a causative factor of the impairment could not be proven.[31]

Looking for structural alterations in normal alar ligaments, Pfirrmann *et al* studied 50 asymptomatic individuals.[32] They found asymmetric high signal intensity in the majority of cases and concluded that since such alterations frequently were found in a noninjured population and the clinical relevance of such structural changes is limited. However, only 80% of the alar ligaments could be visualized. Reasons for this low detection rate could be inadequate imaging protocol: low spatial resolution (slice thickness, 3–4 mm) and insufficient contrast resolution (T1- and T2-weighted sequences).[32] Studying the alar ligaments in 15 young volunteers, Roy *et al* found high signal intensity in one third of the ligaments. The reliability in assessing signal abnormality was poor. However, they used a 0.5T MR scanner with a vertical open bore, which has low image quality compared with conventional 1.5T scanners.[33]

**Imaging Protocol for the Craniovertebral Junction.** Searching for a possible morphologic correlate to craniovertebral instability,[24,34] the authors of the present paper developed an imaging protocol specially designed for ligamentous structures in the upper cervical spine. High spatial resolution and good contrast between tissues were obtained by using proton-density-weighted sequence with 2-mm-thick sections. Examinations were performed in three orthogonal planes with the head fixed in neutral position using a standard head coil. Axial sections covered from the foramen magnum to the base of the dens, coronal sections from the anterior arch of the atlas and halfway through the spinal canal, and sagittal sections covering from one occipital condyle to the other. Imaging parameters are given elsewhere.[35]

**Study Population.** We examined 92 whiplash injured individuals (33 males and 59 females with a mean age of 40 years; range, 14–61 years) sustaining a frontal or rear-end collision 2 to 9 years (mean, 6 years) previously. They were prospectively registered, but only those who met the criteria for whiplash-associated disorder 2 (neck pain, decreased range of motion, and palpation tenderness) after 3 months were included. Neck radiographs in the acute phase were normal. Those with other neck injuries of any kind were excluded. As a control group, we included 30 individuals without any known neck injury (11 men and 19 women; mean age, 46 years; range, 28–66 years).[36]

**Image Interpretation and Grading Criteria.** The whiplash group and the control group were mixed in random order and signal intensity within the ligaments was graded twice at a 3-month interval by three radiologists blinded to clinical information and which group they belonged to. For the alar ligaments, we based our grading on the ratio between any high-signal part and the total cross-sectional area in sagittal sections (Figure 1). A lesion should be seen in coronal images as well.[36] The same grading principle was used for the transverse ligament.[37] A transverse ligament lesion should be seen in at least two imaging planes. Depending on the sharpness of its curvature around the dens, the high signal part of the total cross-sectional area was assessed either in coronal or sagittal section (Figure 2). Grading criteria are given in Table 1.

The tectorial- and the posterior atlanto-occipital membranes were evaluated on sagittal sections only. The tectorial membrane is adherent to, and indistinguishable from, the dura (Figure 3A). A normal membrane/dura complex showed a mean thickness of 1.4 mm and a transverse width of 15 mm.[35] A membrane/dura complex with reduced thickness, similar to that of the dura alone, was regarded abnormal (Figure 3B). Grading criteria were based on the fraction of the total transverse width showing such reduced thickness.[38]

The posterior atlanto-occipital membrane appeared as a dark band fused with the dura (Figure 3A). We based our grading of the membrane/dura complex on struc-

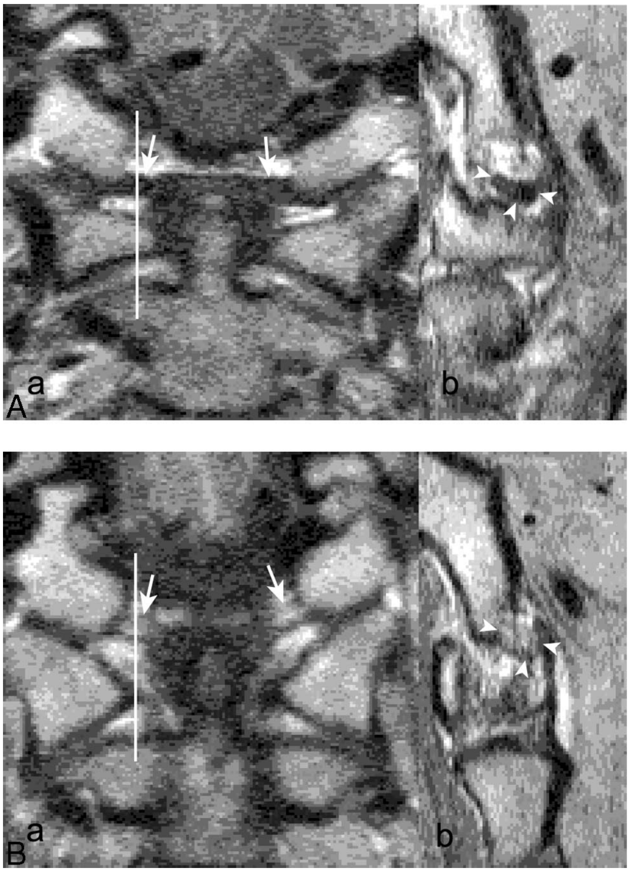

Figure 1. A, Coronal, 2-mm-thick proton density-weighted (PDW) section (a) of normal alar ligaments extending from the upper tip of the dens to the occipital condyles (arrows). Sagittal view (b) of the right alar ligament (at the line in a) shows low signal intensity in the cross-sectional area (arrowheads). B, An 18 year-old woman sustaining rear-end collision 2 years previously. Both alar ligaments (arrows) show high signal intensity in coronal view (a). Sagittal view (b) of the right alar ligament (at the line in a) shows high signal intensity in the entire cross-sectional area (arrowheads), classified as Grade 3.

tural changes of the adjacent dura: dural hump, dural ridge, or dural flap. Dural hump was interpreted as an elongation of the dura and regarded as a sign of minor sprain, whereas dural ridge or flap was regarded as major sprain or rupture (Figure 3C).[38] Grading criteria for both membranes are given in Table 2.

### *Structural Changes in Injured and Noninjured Individuals*

**Observer Variation.** The consistency in grading varied both between observers and for the different structures evaluated. Generally, we found better intraobserver than interobserver agreement. Using dichotomized groups, we obtained good agreement between observers (kappa values) for the posterior atlanto-occipital membrane (0.64–0.82), moderate to good for the alar ligaments (0.46–0.65), and moderate for the transverse ligament (0.43–0.46) and for the tectorial membrane (0.41–0.56).[36–38]

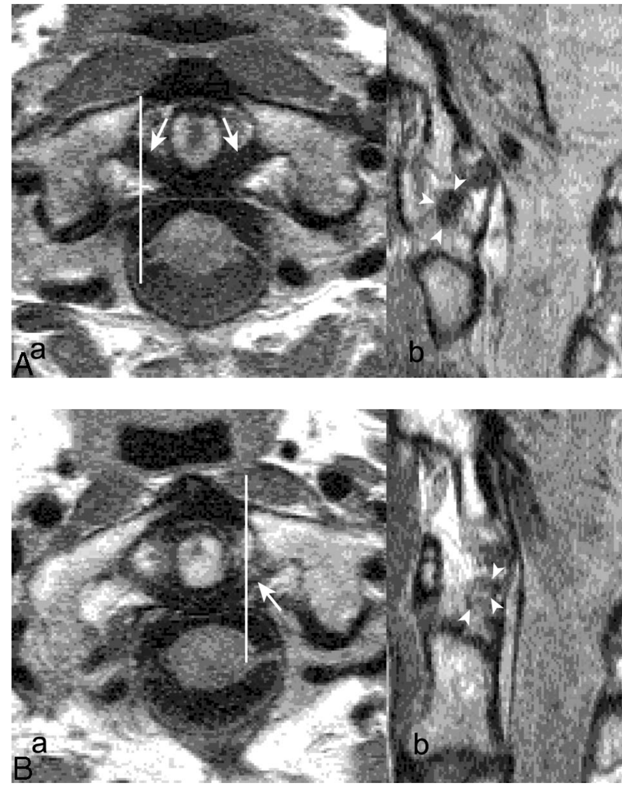

Figure 2. A, Axial (2-mm-thick PDW) section of a normal transverse ligament (arrows) arching around the dens axis (a). Sagittal view (b) near the right insertion (at the line in a) shows low signal intensity in the cross-sectional area (arrowheads). B, A 58-year-old woman involved in a frontal collision 8 years previously showing high signal intensity at the left insertion (arrow). Sagittal view (b) near the left insertion (at the line in a) shows high signal intensity in the entire cross-sectional area (arrowheads), classified as Grade 3.

**MRI Findings: Whiplash Group *Versus* Control Group.** We found significantly more high-grade (2 or 3) changes in the whiplash group compared with the control group for all structures evaluated. The frequency of high-grade changes was highest for the alar ligaments (66.3%) and lowest for the tectorial membrane (17.4%).

There were no Grade 3 changes in the control group; however, two alar ligaments were assigned as Grade 2. These two ligaments showed intermediate signal intensity, which did not apply to our grading criteria. Such changes could either be due to loose fiber structure or sprain. There were six Grade 2 transverse ligaments in the control group. The majority of these cases showed

**Table 1. Grading Criteria for the Alar and Transverse Ligaments**

| Grade | Criteria |
|---|---|
| 0 | Low signal intensity throughout the entire cross section |
| 1 | High signal intensity in one third or less of cross section |
| 2 | High signal intensity in one third to two thirds of cross section |
| 3 | High signal intensity in two thirds or more of cross section |

Criteria are based on the ratio between any high-signal part and the total cross-sectional area of the ligament.

Figure 3. Midsagittal (2-mm-thick, PDW) sections of the craniovertebral junction. A, Normal anatomy. The tectorial membrane (arrows) is fused with the dura mater and extends from the C2 body to the clivus. The posterior atlanto-occipital membrane (arrowheads), also fused with the dura mater, extends from the posterior arch of the atlas to the occipital bone. B, A 40-year-old woman sustaining frontal collision 4 years previously. Upper part of the tectorial membrane (arrows) is absent; only the dura is shown. C, A 46-year-old woman sustaining rear-end collision 11 years previously. The flap combined with thinning of the atlanto-occipital membrane/dura complex was classified as Grade 3 (arrowheads).

**Table 2. Grading Criteria for the Tectorial and the Posterior Atlanto-Occipital Membranes**

| Grade | Criteria |
|---|---|
| Tectorial membrane | |
| 0 | A membrane/dura complex thicker than the dura alone in all sagittal sections |
| 1 | Only the dura seen in one third or less of transverse width |
| 2 | Only the dura seen in one third to two thirds of transverse width |
| 3 | Only the dura seen in two thirds or more of transverse width |
| Posterior atlanto-occipital membrane | |
| 0 | Smooth and well-defined membrane/dura complex |
| 1 | A dural hump traversing the membrane/dura complex |
| 2 | A tent-shaped dural ridge traversing the membrane/dura complex |
| 3 | A dural flap traversing the membrane/dura complex |

decreased signal intensity in the anterior atlantal space obscuring the ligament. Such changes could either be due to degenerative or reparative changes. Details are given in Table 3.[39]

**Association Between MRI Findings and Head Position.** A total of 45 of the 92 whiplash patients had neutral and 47 had rotated head position at the moment of impact.[42] We found significantly more high-grade (2 and 3) alar ligament lesions among those with rotated head position (85.1%) than among those with neutral head position (46.7%, $P < 0.001$). A total of 61.7% of those with rotated head position had alar ligament Grade 3, whereas only 4.4% had Grade 3 in the group with neutral head position. The association between rotated head position and high-grade lesions was more pronounced after rear-end than after frontal collision (93.8% *vs.* 31.8%, $P < 0.001$). There was no such association for the other structures (Table 4).[39]

**Table 3. MRI Grading in the Control Group and in the Whiplash Group**

| MRI Grade | Control Group [no. (%)] | WAD Group [no. (%)] | $\chi^2$ Test (*P*) |
|---|---|---|---|
| Alar ligaments* | | | <0.001 |
| 0–1 | 28 (93.3) | 31 (33.7) | |
| 2–3 | 2 (6.7) | 61 (66.3) | |
| Transverse ligament | | | 0.044 |
| 0–1 | 24 (80.0) | 55 (59.8) | |
| 2–3 | 6 (20.0) | 37 (40.2) | |
| Tectorial membrane† | | | 0.016 |
| 0–1 | 29 (100.0) | 76 (82.6) | |
| 2–3 | 0 (0.0) | 16 (17.4) | |
| Posterior atlanto-occipital membrane† | | | 0.014 |
| 0–1 | 28 (96.6) | 70 (76.0) | |
| 2–3 | 1 (3.4) | 22 (24.0) | |

*Highest assigned grade if different between right and left.
†One missing value in the control group (not examined).

**Table 4. MRI Classification of Ligaments and Membranes in 92 Whiplash Patients by Head Position and Impact Direction at the Moment of Impact ($P$ values for $\chi^2$ test between groups)**

| | Head Position | | | Impact Direction | | |
|---|---|---|---|---|---|---|
| MRI Grade | Neutral [no. (%)] | Rotated [no. (%)] | $P$ | Frontal [no. (%)] | Rear-end [no. (%)] | $P$ |
| Alar ligaments* | | | <0.001 | | | 0.15 |
| 0–1 | 24 (53.3) | 7 (14.9) | | 15 (27.8) | 16 (42.1) | |
| 2–3 | 21 (46.7) | 40 (85.1) | | 39 (72.2) | 22 (57.9) | |
| Transverse ligament | | | 0.64 | | | <0.001 |
| 0–1 | 28 (62.2) | 27 (57.5) | | 21 (38.9) | 34 (89.5) | |
| 2–3 | 17 (27.8) | 20 (42.5) | | 33 (61.1) | 4 (10.5) | |
| Tectorial membrane | | | 0.92 | | | 0.15 |
| 0–1 | 37 (82.2) | 39 (83.0) | | 42 (77.8) | 34 (89.5) | |
| 2–3 | 8 (17.8) | 8 (17.0) | | 12 (22.2) | 4 (10.5) | |
| Posterior atlanto-occipital membrane | | | 0.71 | | | <0.001 |
| 0–1 | 35 (77.8) | 35 (74.5) | | 34 (63.0) | 36 (94.7) | |
| 2–3 | 10 (22.2) | 12 (25.5) | | 20 (37.0) | 2 (5.3) | |

*Highest assigned grade if difference between right and left.

**Association Between MRI Findings and Impact Direction.** A total of 54 of the 92 whiplash patients sustained a frontal and 38 a rear-end collision. High-grade changes in the transverse ligament were significantly more common in frontal than in rear-end collision (61.1% *vs.* 10.5%, $P < 0.001$). That was true for the atlanto-occipital membrane as well (37.1% *vs.* 5.3%, $P < 0.001$). There was no such difference for the alar ligaments or for the tectorial membrane (Table 4).[39]

**Association Between MRI Findings and Disability.** The 92 whiplash patients and 30 control persons completed the Neck Disability Index (NDI) score, a modification of the Oswestry Low Back Pain Index, comprising 10 single items related to activity of daily living. This score reflected either degree of neck pain or difficulties performing certain activities.[40]

We found significantly higher NDI score in the whiplash group than in the control group for all items, particularly for neck pain, head ache, reading abilities, concentration, car driving, and activity level in general ($P < 0.001$). In the whiplash group NDI score increased significantly with increasing MRI grading for the alar ligaments ($P = 0.002$). The same tendency was found for the transverse ligament ($P = 0.059$), but not for the membranes.

Lesions only to the alar ligaments (no other findings) were associated with higher score than lesions only to the transverse ligament. Lesions only to the atlanto-occipital membrane showed the same score as those with abnormal alar ligaments. Analysis with mutual adjustment for all four structures showed that the strongest symptom marker is high-grade lesion (Grade 2 or 3) in one or both alar ligaments. Finally, we found a highly significant increase in NDI score with increasing number of high-grade lesions ($P = 0.003$).[41]

## ■ Discussion

The soft tissues of the craniovertebral joints have until recently been unexposed to diagnostic imaging with the exception of a few case reports of major disruption.[42] By use of high-resolution MR imaging, subtle structures of this area are now available for morphologic assessment with reasonable reliability.[36–38] In a case-control study of whiplash patients, a substantial number of high-grade ligamentous lesions were discovered. Only minor changes were found in the control group.[39]

This study confirms former hypothesis that the alar ligaments are particularly vulnerable to neck trauma when the head is rotated at the moment of impact.[24] When the head rotates, the alar ligaments twist around the dens. Reaching 90° rotation, these ligaments are maximally tightened and obtain an anteroposterior orientation. Not unexpected, such tightened anteroposteriorly oriented alar ligaments are more vulnerable to hyperextension-hyperflexion trauma than relaxed, transversely oriented ligaments.[41] Cadaver dissection has shown that transection of the alar ligaments caused increased rotation and lateral flexion in the upper cervical joints.[43] Thus, there is reason to believe that MRI verified alar ligament lesions could cause such instability.

The transverse ligament and the atlanto-occipital membrane showed significantly more high-grade lesions in frontal than in rear-end collision. In frontal collision, an anterior translation of the head takes place,[44] and this trauma mechanism strains the transverse ligament. Despite the fact that extensive signal changes were found in this ligament, we never saw anterior translation of the atlas. However, flexion and extension views are necessary to disclose such translation.

The atlanto-occipital and the tectorial membranes restrict hyperflexion in the craniovertebral joint.[45] Both of these membranes showed more lesions in frontal than in rear-end collision, indicating that hyperflexion is the predominant trauma mechanism. The atlanto-occipital membrane is elastic. When this membrane is stretched beyond its elastic properties, the hyperflexion forces are transmitted to the adjoining dura, which can sprain or even rupture.

NDI score increased significantly with increasing alar ligament grading. The alar ligaments appeared to play a major role also in combination with lesions in other structures, which support former hypothesis that these ligaments are important as a causative factor of pain and disability in chronic whiplash syndrome.[46]

## ■ Conclusion

The number of high-grade changes in whiplash patients compared with noninjured individuals indicates that these lesions are indeed caused by a whiplash trauma. The different spectrum of lesions in frontal *versus* rear-end impacts and in neutral *versus* rotated head position support this view. There is association between NDI score and MRI findings, particularly for the alar ligaments. Our findings add support to the hypothesis that injured soft tissue structures in the upper cervical spine, particularly the alar ligaments, play an important role in the understanding of the chronic whiplash syndrome.

### ■ Key Points

- By use of high-resolution MRI, it is possible to assess ligaments and membranes in the craniovertebral junction with reasonable reliability.
- Significantly more high-grade lesions in a whiplash injured than in a noninjured population.
- There is association between high-grade changes in the alar ligaments and clinical impairment.
- There is association between specific lesions and specific trauma mechanisms.

## References


1. Spitzer WO, Skovron ML, Salmi LR, et al. Scientific monograph of the Quebec Task Force on Whiplash-Associated Disorders: redefining 'whiplash' and its management. *Spine* 1995;20(suppl):1–73.
2. Jakobsson L, Norin H, Bunketorp O. Whiplash-associated disorders in frontal impacts: influencing factors and consequences. *Traffic Inj Prev* 2003;4:153–61.
3. Borchgrevink GE, Lereim I, Royneland L, et al. National health insurance consumption and chronic symptoms following mild neck sprain injuries in car collisions. *Scand J Soc Med* 1996;24:264–71.
4. Bunketorp L, Nordholm L, Carlsson J. A descriptive analysis of disorders in patients 17 years following motor vehicle accidents. *Eur Spine J* 2002;11:227–34.
5. Radanov BP, Sturzenegger M, De Stefano G, et al. Relationship between early somatic, radiological, cognitive and psychosocial findings and outcome during a one-year follow-up in 117 patients suffering from common whiplash. *Br J Rheumatol* 1994;33:442–8.
6. Squires B, Gargan MF, Bannister GC. Soft-tissue injuries of the cervical spine: 15-year follow-up. *J Bone Joint Surg Br* 1996;78:955–7.
7. Barnsley L, Lord SM, Wallis BJ, et al. The prevalence of chronic cervical zygapophysial joint pain after whiplash. *Spine* 1995;20:20–5.
8. Lord SM, Barnsley L, Bogduk N. The utility of comparative local anesthetic blocks versus placebo-controlled blocks for the diagnosis of cervical zygapophysial joint pain. *Clin J Pain* 1995;11:208–13.
9. Lord SM, Barnsley L, Wallis BJ, et al. Chronic cervical zygapophysial joint pain after whiplash: a placebo-controlled prevalence study. *Spine* 1996;21:1737–44.
10. Jonsson H Jr, Bring G, Rauschning W, et al. Hidden cervical spine injuries in traffic accident victims with skull fractures. *J Spinal Disord* 1991;4:251–63.
11. Watkinson A, Gargan MF, Bannister GC. Prognostic factors in soft tissue injuries of the cervical spine. *Injury* 1991;22:307–9.
12. Hamer AJ, Gargan MF, Bannister GC, et al. Whiplash injury and surgically treated cervical disc disease. *Injury* 1993;24:549–50.
13. Ovadia D, Steinberg EL, Nissan MN, et al. Whiplash injury: a retrospective study on patients seeking compensation. *Injury* 2002;33:569–73.
14. Taylor JR, Twomey LT. Acute injuries to cervical joints: an autopsy study of neck sprain. *Spine* 1993;18:1115–22.
15. Jonsson H Jr, Cesarini K, Sahlstedt B, et al. Findings and outcome in whiplash-type neck distortions. *Spine* 1994;19:2733–43.
16. Dvorak J, Panjabi MM, Grob D, et al. Clinical validation of functional flexion/extension radiographs of the cervical spine. *Spine* 1993;18:120–7.
17. Griffiths HJ, Olson PN, Everson LI, et al. Hyperextension strain or 'whiplash' injuries to the cervical spine. *Skeletal Radiol* 1995;24:263–6.
18. Kristjansson E, Leivseth G, Brinckmann P, et al. Increased sagittal plane segmental motion in the lower cervical spine in women with chronic whiplash-associated disorders, grades I-II: a case-control study using a new measurement protocol. *Spine* 2003;28:2215–21.
19. Dall'Alba PT, Sterling MM, Treleaven JM, et al. Cervical range of motion discriminates between asymptomatic persons and those with whiplash. *Spine* 2001;26:2090–4.
20. Puglisi F, Ridi R, Cecchi F, et al. Segmental vertebral motion in the assessment of neck range of motion in whiplash patients. *Int J Legal Med* 2004;118:235–9.
21. Prushansky T, Pevzner E, Gordon C, et al. Performance of cervical motion in chronic whiplash patients and healthy subjects: the case of atypical patients. *Spine* 2006;31:37–43.
22. Panjabi M, Dvorak J, Crisco J III, et al. Flexion, extension, and lateral bending of the upper cervical spine in response to alar ligament transections. *J Spinal Disord* 1991;4:157–67.
23. Dvorak J, Panjabi M, Gerber M, et al. CT-functional diagnostics of the rotatory instability of upper cervical spine: 1. An experimental study on cadavers. *Spine* 1987;12:197–205.
24. Dvorak J, Hayek J, Zehnder R. CT-functional diagnostics of the rotatory instability of the upper cervical spine: 2. An evaluation on healthy adults and patients with suspected instability. *Spine* 1987;12:726–31.
25. Dvorak J, Penning L, Hayek J, et al. Functional diagnostics of the cervical spine using computer tomography. *Neuroradiology* 1988;30:132–7.

25a. Antinnes JA, Dvorak J, Hayek J, et al. The value of functional computed tomography in the evaluation of soft-tissue injury in the upper cervical spine. *Eur Spine J* 1994;3:98–101.

26. Volle E, Montazem A. MRI video diagnosis and surgical therapy of soft tissue trauma to the craniocervical junction. *Ear Nose Throat J* 2001;80:41–8.
27. Patijn J, Wilmink J, ter Linden FH, et al. CT study of craniovertebral rotation in whiplash injury. *Eur Spine J* 2001;10:38–43.
28. Saternus KS, Thrun C. Traumatology of the alar ligaments. *Aktuelle Traumatol* 1987;17:214–8.
29. Adams VI. Neck injuries: III. Ligamentous injuries of the craniocervical articulation without occipito-atlantal or atlanto-axial facet dislocation: a pathologic study of 21 traffic fatalities. *J Forensic Sci* 1993;38:1097–104.
30. Willauschus WG, Kladny B, Beyer, et al. Lesions of the alar ligaments. In vivo and in vitro studies with magnetic resonance imaging. *Spine* 1995;20:2493–8.
31. Wilmink JT, Patijn J. MR imaging of alar ligament in whiplash-associated disorders: an observer study. *Neuroradiology* 2001;43:859–63.
32. Pfirrmann CW, Binkert CA, Zanetti M, et al. MR morphology of alar ligaments and occipitoatlantoaxial joints: study in 50 asymptomatic subjects. *Radiology* 2001;218:133–7.
33. Roy S, Hol PK, Laerum LT, et al. Pitfalls of magnetic resonance imaging of alar ligament. *Neuroradiology* 2004;46:392–8.
34. Dvorak J, Panjabi MM. Functional anatomy of the alar ligaments. *Spine* 1987;12:183–9.
35. Krakenes J, Kaale BR, Rorvik J, et al. MRI assessment of normal ligamentous structures in the craniovertebral junction. *Neuroradiology* 2001;43:1089–97.
36. Krakenes J, Kaale BR, Moen G, et al. MRI assessment of the alar ligaments in the late stage of whiplash injury: a study of structural abnormalities and observer agreement. *Neuroradiology* 2002;44:617–24.
37. Krakenes J, Kaale BR, Nordli H, et al. MR analysis of the transverse ligament in the late stage of whiplash injury. *Acta Radiol* 2003;44:637–44.
38. Krakenes J, Kaale BR, Moen G, et al. MRI of the tectorial and posterior atlanto-occipital membranes in the late stage of whiplash injury. *Neuroradiology* 2003;45:585–91.
39. Kaale BR, Krakenes J, Albrektsen G, et al. Head position and impact direc-

tion in whiplash injuries: associations with MRI-verified lesions of ligaments and membranes in the upper cervical spine. *J Neurotrauma* 2005;22: 1294–302.
40. Vernon H, Mior S. The Neck Disability Index: a study of reliability and validity. *J Manipulative Physiol Ther* 1991;14:409–15.
41. Kaale BR, Krakenes J, Albrektsen G, et al. Whiplash-associated disorders impairment rating: neck disability index score according to severity of MRI findings of ligaments and membranes in the upper cervical spine. *J Neurotrauma* 2005;22:466–75.
42. Labler L, Eid K, Platz A, et al. Atlanto-occipital dislocation: four case reports of survival in adults and review of the literature. *Eur Spine J* 2004;13:172–80.
43. Dvorak J, Panjabi M, Gerber M, et al. CT-functional diagnostics of the rotatory instability of upper cervical spine. 1. An experimental study on cadavers. *Spine* 1987;12:197–205.
44. Penning L. Backward hypertranslation of the head: participation in the whiplash injury mechanism of the cervical spine? *Orthopade* 1994;23:268–74.
45. Harris JH, Yeakley JW. Hyperextension-dislocation of the cervical spine: ligament injuries demonstrated by magnetic resonance imaging. *J Bone Joint Surg Br* 1992;74:567–70.
46. Dvorak J, Schneider E, Saldinger P, et al. Biomechanics of the craniocervical region: the alar and transverse ligaments. *J Orthop Res* 1988;6:452–61.