BCMJ, Vol. 44, No. 5, June 2002, page(s) 252-256—Article

James R. Taylor, MD, PhD

The pathology of whiplash: Neck sprain

Clinical studies alone cannot adequately describe the pathology of severe whiplash, as most imaging is unable to demonstrate it.

ABSTRACT:

Common spinal lesions consistently observed in autopsy spines, cadaver spines from crash studies, and experimental neck extension injuries in primates include disc tears, avulsions, and facet hemarthroses. Radiographs are unable to demonstrate many of these cervical spinal lesions that can be observed at autopsy after fatal injury. Similar injuries may be observed in patients with severe whiplash, particularly when the physician is alerted to the sites most vulnerable to injury.

Introduction

The terms *whiplash* and *acceleration injury* to the neck describe an injury mechanism without specifying a pathology.[1,2] *Neck sprain* assumes a ligamentous injury.[3] *Whiplash* assumes no head impact, though in modern vehicles, the back of the head impacts on the head restraint, unless there is hyperextension over the top of a low-positioned head restraint. However, hyperextension is not essential for a neck sprain to occur. Neck sprain may result from a rear-end impact or a blow to the forehead. It is usually a multilevel extension sprain in a well-aligned spine. By contrast, a severe flexion injury is often a one-level fracture dislocation with risk of spinal cord injury. Fracture dislocations are outside the scope of this review.

The Quebec Task Force on WAD report provided a useful classification of whiplash-associated disorders (WAD) according to their severity[4] (see the Table in the article "Incidence of whiplash-associated disorder.). This was based on the clinical characteristics of the disorder, as the authors could not describe the pathology. They excluded from consideration the strong inferences that can be drawn from cadaver and animal studies that described such pathology.

Most soft-tissue lesions observed in autopsy studies were not visible on postmortem radiographs.[5-8] Even magnetic resonance imaging (MRI) grossly underestimates the extent of injuries found at surgery.[8] Radiologists emphasize the difficulty of demonstrating the pathology of neck extension injuries.[9-12] This deficiency in objective imaging methods may explain some of the inadequacy in diagnosing organic disorders in severe whiplash. Recent developments have found objective evidence of nociceptive pain in specific injured joints in patients who may otherwise have been dismissed with psychological disorders.[13] Most of these significant physical injuries with organic pathology appear to have occurred in collisions with a high force of impact.[14]

This paper does not consider WAD associated with low-impact collisions. It describes lesions seen in fatal injuries and experimental injuries in cadavers and living primates, the same

lesions that can also be demonstrated in living patients. The cadaver injuries serve as a guide to those that may occur in severe WAD patients where alert radiologists and physicians may identify the problem.

Pathology of injuries in well-aligned spines

Macnab used a sled-seat, accelerated backwards to impact, to produce neck hyper-extension injuries in monkeys.[2] Anterior distraction caused tears of longus colli with retro-pharyngeal hematomas, tears of the anterior longitudinal ligament (ALL), and avulsion of the disc from the vertebral body. Posterior compression caused facet joint hemarthroses. The disc and ligament injuries were among the most reproducible lesions. He described similar unhealed disc injuries in eight patients operated on within 2 years of injury. Seven patients obtained pain relief following anterior interbody fusion. The disc lesions were not seen on X-rays. Similar experimental observations showed injuries to muscles, discs, and facet joints in primates.[15]

Autopsy and cadaver studies

Disc avulsions were described in 12 juveniles as epiphyseal separation.[16] In 50 fatal cranio-spinal injuries, Davis and colleagues observed a higher proportion of upper cervical injuries than in survivors of injury.[17] In subaxial injuries, disc lesions predominated. Muscle ruptures were rare, though muscle hemorrhages were common. In crash tests using cadavers, similar disc and facet-joint injuries were found.[13,18] The consistent nature of these soft-tissue injuries clearly points to the most likely sites of injury.

Sixteen cervical spines from fatal motor-vehicle collisions showed disc and facet lesions, though 16 controls did not show these lesions.[6] A second study of 235 formalin fixed, deep frozen cervical spines used a band saw to cut regular thickness 2.5 mm sagittal slices. This blunt trauma fatality study included 95 from motor-vehicle trauma and 51 from fatal falls or blows. Injuries were observed in 138 spines, covering a wide age range, but mainly young people, frequency peaking at 20 to 25 years. The most common cause of death was head injury, followed by chest and abdominal injuries. Spinal injuries were the least common cause of death; most spinal injuries were incidental to the fatal injuries. Disc and facet injuries were equally common as previously described.[7,19,20] Almost half of the specimens showed fracture dislocations; these are not relevant to this article, which describes soft-tissue extension-type injuries in well-aligned spines. Examples of common injuries are shown in the Figure.

Upper cervical soft-tissue injuries

The most common injury was bruising of the intra-articular synovial folds of the lateral atlanto-axial joints with or without hemarthrosis. The lateral atlanto-axial joint surfaces are both convex in the sagittal plane, permitting rocking movements in flexion and extension. Anterior and posterior gaps are filled by large vascular synovial folds. In normal movements these move out of the way of the closing articular surfaces, but in crashes lasting a few milliseconds, the synovial folds are exposed to being nipped, bruised, or ruptured. Posterior synovial fold bruises were often associated with a hematoma located behind the joint and around the dorsal root ganglion of C2. This ganglion is surrounded by thin-walled veins in a small compartment deep to the obliquus capitis inferior. The veins may be damaged and the hematoma may track along the greater occipital nerve as it arches below the inferior oblique

muscle. Injuries to the C1-2 synovial folds were seen in 88 (60%) of the cervical spines, usually independent of any fracture or dislocation.

Alar ligament rupture was uncommon, except in fracture dislocations; unilateral rupture results in contralateral rotary hypermobility.[21] Damage to the vertebral artery at the level of the atlas, with intimal dissection and antemortem thrombosis, was seen in one well-aligned spine.

Disc, facet, and dorsal root ganglion injuries

Cervical subaxial extension injuries contrast with thoraco-lumbar flexion-compression fractures, which target vertebral bodies. The cervical spine is more vulnerable to extension due to the paucity of anterior muscles. Even in combined flexion and extension violence, we still mostly observe extension-type injuries. Cervical disc, ligamentous, and zygapophyseal joint injuries were four times more frequent than vertebral fractures. Both extension and side flexion injuries may injure cervical dorsal root ganglia.

Age-related fissures

Cervical discs differ from thoracic and lumbar discs.[22] The translation that is part of normal cervical movement causes a shear effect across the discs, forming lateral uncovertebral clefts at puberty and complete transverse fissures through the posterior half of each disc by the mid-30s. We must distinguish these age-related fissures from injuries.

Disc injuries

Continuing with our pathoanatomic study, acute injuries observed at autopsy were classified as minor and major.[6,7] Minor injuries to the anterior annulus included transverse tears at the vertebral rim (rim lesions) and annular hemorrhage without a rim lesion. Hemorrhage was seen in children, as young discs contain small superficial annular vessels that disappear in adults. In the minor injuries, the ALL remained intact. Major injuries included linear avulsions of the disc from the vertebral body and disc herniations. Avulsions were accompanied by partial or complete tears to the anterior longitudinal ligament. The major injuries were confined to one or two levels, most often at C5-6 and C6-7. The minor injuries were often multilevel and were most frequent at C3-4 and C4-5. Both rim lesions and avulsions were seen at the upper disc margin more often than the lower disc margin.

Rim lesions were the most common disc injuries, followed by avulsions, and then by herniations. The disc usually tears in linear fashion along the disc vertebral junction in subjects under 55 years. In subjects over 55, irregular disc disruption was more common. The longus cervicis, on the front of the spine, was seldom torn, even when the ALL was ruptured, but bleeding into the muscle was common. Herniations were usually contained by an intact posterior longitudinal ligament. Large herniations impinge on the dura or spinal cord. They may include annular fragments, central disc material, or part of a cartilage plate torn off the vertebral end plate. None of these lesions were seen in controls who died of nontraumatic causes.

Anterior distraction injury

In an extension mechanism sprain, it would appear that the anterior annulus tears first, then with disc avulsion there is partial or complete rupture of the ALL. The anterior muscles tear last. The ALL is more compliant than the annulus and the muscle is the most compliant of all, allowing stretch without rupture. Posterior herniations were less frequent than avulsions. Imaging demonstrated herniations, but it may miss avulsions, which occur along an anatomical line where the vertebra meets the disc, and alignment is maintained by contraction of intact anterior muscles, despite significant injury.

Facet injuries

Facet-joint injuries were signaled by hemarthrosis or bruising of the intra-articular synovial folds. Hemarthrosis most often resulted from capsular or synovial damage, but articular cartilage tears or fractures were also common. Small fractures of the articular surface and the subchondral bone were often seen, with hemorrhage into the joint. Undisplaced fractures included facet tip fractures, fractures across the facet base, or vertical pillar fractures. The facet fractures were seldom visible on the postmortem radiographs.

Secondary pathology in discs and facets

Late pathologies may be secondary to the initial injury rather than the direct result of it. Osti and colleagues made surgical rim lesions in the anterior annulus of sheep discs and found that, after an interval, they resulted in disc and facet-joint degeneration.[23,24]

Dorsal root ganglion injuries

Hemorrhage into the vascular dorsal root ganglion, often with associated neural tissue disruption, was seen in 14% of the fatal injuries, usually in spines with mild ligamentous injuries. Dorsal root ganglion injury was identified in one-third of the subgroup who survived injury for more than 1 hour, as the dorsal root ganglion hemorrhage became obvious when circulation was maintained for more than 1 hour after injury.[25]

Comparability with whiplash

Two main difficulties in comparing these injuries with whiplash are that the majority of neck injuries described above were secondary to head injuries, and that the average severity of injury was greater than in cases of WAD alone. A comparison of lesions in those with head injuries and those with no evidence of head impact showed that the spinal injuries were similar, despite a slight excess of upper cervical lesions in those with head injuries. There is a range of overlap in severity between the probable impact velocities in fatal injuries and in severe WAD.

Studies in survivors of injury showed that similar injuries were found in severe WAD patients. We reported two autopsies where the disc injuries found, at 14 months and 3 years after the injury, corresponded with clinically recorded symptoms and signs.[26] Two subsequent autopsies were done on survivors of injury who committed suicide 3 years after injury: a man aged 28 years, injured in a motorcycle crash, and a woman car driver aged 40 years, with WAD. Both had normally aligned spines on postinjury radiographs. Sectioning revealed disc and facet injuries that correlated with their antemortem symptoms. Most of the pathology in these four cases was not evident in vivo.

Finch and Taylor[27] found vacuum clefts (rim lesions) in the extension films of functional views in WAD patients, occasionally with annulus calcification. Davis described annular tears, disc herniations, and fluid in facet joints in MRI scans of severe whiplash injuries.[28] Facet "hot spots," as demonstrated by bone scans with SPECT imaging in young patients with chronic pain and focal facet tenderness, probably indicate traumatic pathology.

Summary

The consistency of the disc and facet pathology observed in postmortem studies of fatal injuries, experimental injuries in cadavers, and in living primates points to the reality of the type of lesions that can occur in severe neck sprain. Although these lesions can be visualized in some patients, they may also exist with severe WAD where imaging studies are normal. Here, an alert physician may or may not make the diagnosis of clinical pathology with further focused imaging or other investigative studies. Clearly, normal imaging does not exclude organic pathology.

Competing interests

Dr Taylor has spoken at conferences on the topic of whiplash for many years, often as an invited speaker.

References

- 1. Gay JR, Abbott KH. Common whiplash injuries of the neck. JAMA 1953;29:1698-1704.
- 2. Macnab I. Acceleration injuries of the cervical spine. J Bone Joint Surg 1964;46A:1797-1798.
- 3. Porter KM. Neck sprains after car accidents: A common cause of long term disability. BMJ 1989;298:973-974. PubMed Citation
- 4. 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(8S):1S-73S. PubMed Citation
- 5. Jonsson H, Bring G, Rauschning W, et al. Hidden cervical spine injuries in traffic accident victims with skull fractures. J Spinal Disorders 1991;4:251-263. PubMed Abstract
- 6. Taylor JR, Twomey LT. Acute injuries to cervical joints. Spine 1993;18:1115-1122. PubMed Abstract
- 7. Taylor JR, Taylor MM. Cervical spinal injuries: An autopsy study of 109 blunt injuries. J Musculoskeletal Pain 1996;4:61-79.
- 8. Jonsson H, Cesarini K, Sahlstedt B, et al. Findings and outcome in whiplash-type neck distortions. Spine 1994;19:2733-2743. PubMed Abstract
- 9. Weir, DC. Roentgenographic signs of cervical injury. Clin Orthop 1975;109:9-17. PubMed Abstract
- 10. Edeiken-Monroe B, Wagner LK, Harris JH. Hyperextension dislocation of the cervical spine. AJR 1986;146:803-808. PubMed Abstract
- 11. Gerrelts BD, Petersen EU, Marby J, et al. Delayed diagnosis of cervical spine injuries. J Trauma 1991;31:1622-1626. PubMed Abstract
- 12. Woodring JH, Lee C. Limitations of cervical radiography in the evaluation of acute cervical trauma. J Trauma 1993;34:32-39. PubMed Abstract

- 13. Barnsley L, Lord S, Bogduk N. Whiplash injury. Pain 1994;58:283-307. PubMed Citation
- 14. Radanov B, Dvorak J. Spine update: Impaired cognitive functioning after whiplash injury of the cervical spine. Spine 1996;21:392-397. PubMed Abstract
- 15. Wickstrom J, Martinez J, Rodriguez R. The cervical sprain syndrome: Experimental acceleration injuries to the head and neck. In: Selzer ML, Gikas P, Huelke D (eds). Prevention of Highway Injury. Ann Arbor, MI: Highway Safety Research Institute, 1967:182-187.
- 16. Aufdermaur M. Spinal injuries in juveniles: Necropsy findings in twelve cases. J Bone Joint Surg 1974;56B:513-519. PubMed Citation
- 17. Davis D, Bohlman H, Walker A, et al. The pathological findings in fatal cranio-spinal injuries. J Neurosurg 1971;34:603-613. PubMed Citation
- 18. Shea M, Wittenberg RH, Edwards WT, et al. In vitro hyperextension injuries in the human cadaveric cervical spine. J Orthop Res 1992;10:911-916. PubMed Abstract
- 19. Taylor JR, Finch PM. Acute injury of the neck: Anatomical and pathological basis of pain. Ann Acad Med Singapore 1993;22:187-192. PubMed Abstract
- 20. Schönström N, Twomey L, Taylor, JR. The lateral atlanto-axial joints and their synovial folds: An in vitro study of soft tissue injuries and fractures. J Trauma 1993;35:886-892. PubMed Abstract
- 21. Dvorak J, Panjabi M, Gerber M, et al. CT-functional diagnostics of the rotary instability of the upper cervical spine: An experimental study on cadavers. Spine 1987;12:197-205. PubMed Abstract
- 22. Taylor JR, Twomey L, Levander B. Contrasts between cervical and lumbar motion segments. Crit Rev Phys Rehab Med 2000:12:345-371.
- 23. Osti OL, Vernon-Roberts B, Fraser RD. 1990 Volvo Award in experimental studies. Anulus tears and intervertebral disc degeneration. An experimental study using an animal model. Spine 1990;15:762-767.PubMed Abstract
- 24. Moore R, Crotti T, Osti O, et al. Osteoarthrosis of the facet joints resulting from anular rim lesions in sheep lumbar discs. Spine 1999;24:519-525. PubMed Abstract
- 25. Taylor JR, Twomey LT, Kakulas BA. Dorsal root ganglion injuries in 109 blunt trauma fatalities. Injury 1998;29:335-339. PubMed Abstract
- 26. Taylor JR, Kakulas BA. Neck injuries. Lancet 1991;338:1343. PubMed Citation
- 27. Finch P, Taylor JR. Functional anatomy of the spine. In: Waldeman S, Winnie A (eds). Interventional Pain Management. Philadelphia: Saunders, 1995:39-64.
- 28. Davis SJ, Khangure, MS. A review of magnetic resonance imaging in spinal trauma. Aust Radiol 1994;38:241-253. PubMed Abstract

James R. Taylor, MD, PhD

Dr Taylor is a visiting professor at the Australian Neuromuscular Research Institute, QE 2 Medical Centre, Nedlands, Western Australia.