## Dynamic Chiropractic

WHIPLASH / NECK PAIN

## Is the Lumbar Spine Vulnerable in Low-Speed, Rear-Impact Crashes?

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A recent paper by Banks, et al., has suggested that low back injury in low-speed rear-impact crash scenarios is unlikely. According to their abstract:

"The purpose of this study was to define the anatomical arrangement of the lumbar spine in the mid-body sagittal plane of a human volunteer while in three postures: a driving posture; full flexion; and full extension. Radiographic images of the lumbar spine were made of a 33-year-old 50th-percentile male subject seated in a comfortable driving posture. Additional radiographs were made of the lumbar spine while the subject was postured in full voluntary flexion and full voluntary extension. Anterior and posterior midsagittal vertebral end plate positions were plotted on an x-y coordinate system for each posture. Anterior and posterior disk thicknesses, and the positions of the centers of each vertebra were numerically determined using information from the plots. Disk thicknesses were then graphed and comparisons made for each posture. The arrangements of the centers of vertebrae were graphed and compared for the three different postures. The arrangement of the lumbar vertebrae tended toward that of full voluntary flexion while the subject was in a normal driving posture. Anterior disk thickness was a sensitive indicator of posture, while posterior disk thickness was not. While in a driving posture, the lower back approximated a straight-line that was nearly parallel to the seat back axis. The observations support those of an earlier study. Since soft tissue spinal elements can only be damaged by applying tensile forces in excess of their tolerance, the anterior elements of the lumbar spine would not be directly threatened in low-velocity frontal collisions, since anterior elements would be in relative compression. Tension injury to the anterior structures as a result of a rear-end collision would first require reversing the pre-impact conditions imposed by the normal driving posture. Tension injury to the posterior spinal elements resulting from lowvelocity rear-end collisions would be unlikely, since axial compression loading would also diminish tension stress in posterior soft tissue structures. Any compression injury to posterior elements resulting from rear-end collisions would first require reversing the pre-impact conditions imposed by the normal driving posture."

When I saw the name "Howard" and the institution name, "Biodynamics Research Corporation," on the title page of this paper, a smile crossed my face. This is one of the largest - if not the largest - medicolegal firms, or "farms" in the country. They cater primarily to insurance companies and their defense lawyers. This is also the source of some very-often-cited crash-testing literature. Howard, in particular, has historically championed the cause against whiplash-induced temporomandibular disordrs (TMD) over the years. In this TMD literature, he and his colleagues claimed to have proved that the TM joint is exposed to forces (during a typical low speed, 5-6.5-mph delta V crash-testing scenario) that are no more stressful or harmful than everyday flossing. In addition to the faulty study design and rather dubious engineering application (e.g., subjects wearing bite plates only loosely attached to their mouths; the nonphysiological crash conditions with subjects biting down on them; and accelerometers placed far distal to the joint's center of gravity), the conclusions also necessarily contradict a clinical and epidemiological literature pointing to the association of TMJ injury in whiplash trauma exposure, with reports varying from an incidence of four percent to

over 50 percent; in essence, they take a scientific revisionistic approach to reality, skillfully turning a blind eye toward a not significant, albeit relatively poor-quality, contradictory literature. This is something akin to proving a bumblebee can't fly using engineering principles: There may be no errors in the application of the mathematics or physics applied, but the fact that bees can fly demands an explanation.

In the present lumbar study - which was almost certainly designed to be used in future courtroom cases as a foundation for refuting the association between low back injury and rear-impact motor vehicle collisions (MVC) - the authors compared the seated lumbar configuration to that of flexion and extension. This was accomplished using a single male subject seated in a single car seat. Thus, the extent of external validity in this study is unknown. They reported that the flexed position and driving positions of the spine are approximately the same. That statement, of course, is only going to be true for people who cannot bend forward any further while seated. Moreover, their comments about the tolerance and vulnerability to the spine in frontal and rear impact collisions is overly simplistic and seems to assume that only simple bending occurs during this type of trauma. Studies of occupant kinematics<sup>2,3</sup> demonstrate very complex biomechanics, which include a likely flattening of the lumbar spine, followed by compression; ramping; then tension. The rear-impact vector results in a biphasic kinematic, with a pitching forward of the torso in the re-entry and torso overspeed phase and, in both phases, shear effects. This is omitted from discussion in the current paper. A more egregious omission, however, is the clinical and epidemiological literature that shows low back complaints in about 45 percent of cases of whiplash. Reports of the contemporaneous complaints of low back pain go at least as far back in the literature as the mid-1950s<sup>5</sup> and continue through the present, with most authors reporting an incidence of low back pain of 35 to over 50 percent in cases of whiplash injury. 6-12

Despite what we have leaned from human subject crash testing, the exact mechanism of injury remains somewhat opaque. Certainly, we know that the kinematics of the lumbar spine are complex, and that that portion of the spine is subjected to significant acceleration in both x and z vectors. In both cases, the acceleration is less than we see in the neck, but is still often in the range of 6g, even in low-speed crashes (5-6.5 mph delta V). However, in light of what we have leaned about discogenic pain from discography studies (e.g., that discs can be a source of pain as a result of internal disruption, tears or fissures), and what we know about autoimmunology (e.g., that perhaps as a result of an autoimmune reaction as the normally sequestered nuclear antibodies become exposed to the environment external to the disc - and there is animal research validating this physiology), the view that longitudinal ligament tears or disc herniations that produce frank compression on neural structures are likely to be the only valid sources of low back pain, is naÜve at best. In the cervical spine, it has been rather elegantly demonstrated that the facet joint, for example, is one of the chief sources of neck pain in chronic whiplash. 13 It is possible that this joint also plays a prominent role in low back pain. But until we have a better grip on low back pain mechanisms in low speed rear impact crashes, it seems unwise to attempt to dismiss patients' complaints using simple postural studies, coupled with rudimentary and incomplete biomechanical and physiological explanations of pain mechanisms.

## References

- 1. R, Martini J, Smith H, Bowles A, McNish T, Howard R. Alignment of the lumbar vertebrae in a driving posture. *J Crash Prevention and Injury Control* 2(2):123-130, 2000.
- 2. WE, Howard RP, Guzman HM, et al. Analysis of human test subject kinematic responses to low velocity rear end impacts. *SAE Tech Paper Series* 930889 21-30, 1993.
- 3. WE, Howard RP, Poppel JV, et al. Human head and neck kinematic after low velocity rear-

- end impacts: understanding "whiplash." *39th Stapp Car Crash Conference Proceedings* 952724 215-238, 1995.
- 4. RP, Hatsell CP, Guzman HM. Temporomandibular joint injury potential imposed by the low-velocity extension-flexion maneuver. *J Oral Maxillofac Surg* 53:256-262, 1995.
- 5. MM, Rosner S. Symptomatology and treatment of injuries of the neck. *NY State J Med* 55:237-242, 1955.
- 6. AC. Soft tissue injury: long and short-term effect. In: Foreman SM, Croft AC (eds): Whiplash Injuries: The Cervical Acceleration/Deceleration Syndrome. Baltimore, Williams & Wilkins Co., 1988, p 293.
- 7. M. Soft tissue injuries of the neck in automobile accidents: factors influencing prognosis. *J Bone Joint Surg* 56A(8):1675-1682, 1974.
- 8. G, Westman G. Chronic posttraumatic syndrome after whiplash injury: a pilot study of 22 patients. *Scan J Prim Health Care* 9:135-141, 1991.
- 9. T. Extracervical symptoms after whiplash trauma. Cephalalgia 14(3):223-227, 1994.
- 10. MF, Bannister GC. The rate of recovery following whiplash injury. *Eur Spine J* 3:162-164, 1994.
- 11. B, Gargan MF, Bannister GC. Soft-tissue injuries of the cervical spine: 15-year follow-up. *J Bone Joint Surg* 78-B(6):955-957, 1996.
- 12. MD, Croft AC. Late whiplash risk factor analysis of a random sample of patients with chronic spine pain. Submitted.
- 13. L, Lord S, Wallis BJ, Bogduk N. The presence of chronic cervical zygapophysial joint pain after whiplash. *Spine* 20(1):20-26, 1995.

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