Dynamic Chiropractic

WHIPLASH / NECK PAIN

Neck Pain and Whiplash Associated Disorder (WAD), Part 2

Mark A. King, DC

Editor's note: Part 1 of this article appeared in the Feb. 12, 2007 issue. To access a printable version online, visit www.chiroweb.com/archives/25/04/09.html.

Clinicians and researchers have known for many years that accidents involving rear-end collisions cause more neck pain than side-impact or frontal-impact collisions. This was noted as far back as 1882 with railway accidents. The culprits are the head and neck structures that lag as the body goes forward, creating a laxity in the ligaments and subsequent injury upon hyperextension.

In 1997, Yang, et al., noted that the cervical spine is subjected to a compressive force (from weight of the head) during a rear-impact collision. The problem occurs when the body starts to move forward and the head remains still. At approximately 120 milliseconds into the impact, the head begins its movement backward. The weight of the head compressing down upon the cervical spine causes subsequent laxity in the cervical ligaments, leading to a decrease in stability at the time when there is a shear force acting upon the lower cervical spine.[1] This combination of axial compression and shear force may be responsible for the higher incidence of neck injuries in rear-impact accidents. Shear forces occur when two bodies - in this case, vertebral bodies - slide in opposite directions from each other.

While cervical myofascial injury is the most common problem with whiplash-associated disorders (WAD), the cervical zygapophyseal joints are routinely injured in these accidents.[2] Researchers have helped document this by injecting the facet joint or electrically stimulating the nerves that supply the facet joints and evaluating the referred pain pattern. The referred pain patterns are similar to the pain distribution experienced by WAD patients.[3] In 1996, Lord, et al., in the study I mentioned earlier, injected the medial branches of the dorsal rami in patients with WAD pain. The authors found that 60 percent of the patients had evidence of facet joint injury.[2] In another study, Lord, et al., performed radio-frequency neurotomy on the same nerves of another group of WAD patients. There was a poor long-term outcome, however. Fifty-eight percent reported relief of symptoms for a few months.[4]

From these studies and many others, it is clear there is a relationship between the WAD injury and the injury to the cervical facet joints, specifically. The disc also can be injured in these traumatic WAD injuries. I am appreciative of the work of Dr. Oliphant showing the safety of treating lumbar disc herniations with spinal manipulation. With caution, I suspect these principles also can be applied to the cervical spine, as far as safety of manipulation with cervical disc injuries. In 2004, Dr. Oliphant wrote an article showing the high level of safety with regard to manipulations with lumbar disc herniations.[5] Several things can happen to the disc in WAD injury, including a horizontal tear of the vertebral endplate, separation of the disc from the vertebral endplate, acute disc herniation, or swelling. The real problem starts with the diagnosis of these disc lesions. Conventional CT, MRI, X-ray, CT myelography, or even EMG are common, but are more relevant

when there is neurologic compression and inflammation. While MRI is routinely used now, it may not tell you the extent of the cervical disc lesion, so disc lesions related to these accidents are difficult to fully quantify.

WAD injuries accelerate the degenerative changes in the cervical spine, and there have been several interesting studies regarding this. Evans stated that there was acceleration in the development in the degenerative changes following whiplash injuries.[6] Hamer, et al., reported that WAD predisposes to premature degenerative changes.[7]

One of my favorite articles was found in *JMPT* in 2004, although it is not specifically related to whiplash-associated disorders. Cramer, et al., performed a study in which an external fixation device was placed on the spine of small animals for varying lengths of time. After the devices were removed and the animals were analyzed, degenerative changes were noted in the area of fixation.[8] This research could imply that restoring normal motion following injury would help slow down these degenerative changes. Chiropractic adjustments, stretching of shortened muscles, work on soft tissues and low-tech rehab could be key manual interventions. You also may need to add some nutritional recommendations to these injured patients for optimal healing. The progression is injury causing inflammation, and then causing collagen crosslinks/adhesions/scarring, leading to degenerative joint changes.

As clinicians, a big part of our job is preventing the development of chronic conditions. If DCs do what is clinically indicated and provide the best care for these WAD injuries, we should be the health care providers of choice for these patients. Learn more about how to manage these cases, do what is best for your patient, and build the practice of your dreams.

References

1. Yang KY, et al. "On the Role of Cervical Facet Joints in Rear End Impact Neck Injury Mechanisms." Warrendale, Penn: Society of Automotive Engineers, 1997; sae970497.
2. Lord SM, et al. Chronic cervical zygapophysial joint pain after whiplash: a placebo-controlled prevalence study. *Spine,* 1996;21(15):1737-45.
3. Fukui, et al. Referred pain distribution of the cervical zygapophyseal joints and cervical dorsal rami. *Pain,* 1996;68(1):79-83.
4. Lord SM, et al. Percutaneous radio-frequency neurotomy for chronic cervical z-joint pain. *NEJM,* 1996;335(23):1721-6.
5. Oliphant D. Safety of spinal manipulation and treatment of lumbar disc herniations. *JMPT,* 2004;27(3):197-210.
6. Evans RW. Some observation on whiplash injuries. *Neurology Clin,* 1992;10(4):975-97.
7. Hamer, et al. Whiplash injury and surgically treated cervical disc disease. *Injury,* 1993;24(8):549-50.
8. Cramer, et al. Degenerative changes following spinal fixation in a small animal model. *JMPT,* 2004;27(3):141-54.

MAY 2007