



Dynamic Chiropractic – January 26, 1999, Vol. 17, Issue 03

The New Model of Whiplash, Part II

Cervical Spine Afferents Cause More Than Pain

By Arthur Croft, DC, MS, MPH, FACO

In a recent study, a group of late whiplash patients was compared to a control group via positron emission tomography (PET) and single photon emission computed tomography (SPECT) examination.¹⁵ PET measures the metabolism of glucose in the brain, while SPECT measures the brain's perfusion (or regional cerebral blood flow). Although the sample size was small, the authors reported a significant hypoperfusion and hypometabolism in the brains of the CAD patients. These abnormalities were focused in the so-called watershed area of the brain, the parieto-occipital region. While these abnormalities might result from direct mild traumatic brain injury (MTBI), the authors speculated that activation of nociceptive afferents from injured upper cervical regions might activate vasoactive peptides which might produce the findings.

The patients also had measurably abnormal neuropsychological tests. The questions that arise from this study are: 1) were the metabolic and perfusion abnormalities that were found the result of direct acceleration injury to the brain? 2) were they the result of the cognitive difficulties commonly measured in chronic CAD patients, which in turn were the result of chronic pain? 3) were the cognitive abnormalities directly attributable to the 'lesions' seen, or were they related only to chronic pain? or 4) were the 'lesions' the result of afferent activity in the trigeminal region, as the authors speculated, and if so, could the cognitive difficulties be attributed to them or simply to chronic pain?

Adding to the mystery, the results of this study meshed rather poorly with those of another paper published this year.¹⁶ Here again, PET and SPECT studies were performed on late whiplash patients and correlated with neuropsychological test scores. These authors found no lesions in the parietotemporo-occipital area, but did find abnormalities in the frontopolar region which correlated with the results of the Beck Depression Inventory. The study suffers from the same small sample size as the previous one and potentially also from selection bias: some of the patients had only neck pain.

Since the authors concluded that neither PET nor SPECT were useful for "routine evaluation" of late whiplash patients; and since most clinicians do not turn to brain imaging or scanning studies in the evaluation of other than potentially brain-injured patients; and since one of the most common symptoms of this (brain-injured) subgroup of patients is headaches, it is possible that a larger, more carefully selected group might have yielded more meaningful results. Another question left unanswered by the study was whether the abnormalities found on PET and SPECT were the result of the neurophysiological consequences of chronic pain -- as it is commonly seen in CAD and is the subject of several other papers by some of these authors. Thus we are left with the quintessential chicken/egg question of cause and effect.

Making matters more difficult still has been the recent finding that nontrauma depression patients also have been shown to have similar PET and SPECT abnormalities. I suspect the answers will be forthcoming in future work. I spoke with one of the authors (Ettlin) of the previous paper recently at a conference in Switzerland and was informed that a much larger study is underway.

Another recent study showed that vestibular hyper-reactivity and abnormalities of the vestibulo-ocular reflex (VOR) may be the result of chronic pain as well.¹⁷ In their group of CAD patients, peripheral labyrinthine function was found to be intact and there was no evidence of cervico-ocular reflex (COR) (i.e., cervical nystagmus). The authors theorized that VOR enhancement might be a plastic adaptation to diminished cervical range of motion (are you chiropractic physicians listening?). On the other hand, some of these abnormalities might also be the result of brain stem injury, as has been suggested by others in the past.

Another fascinating study comes from Heikkila and Wenngren.¹⁸ The cervicocephalic kinesthetic sensibility of a consecutive group of CAD patients (not late whiplash patients in this case) were tested for their ability to reposition the head and neck while blindfolded. A control group performed the same tests. Subjects wore a light bicycle helmet with an attached laser light. This was shone on a wall target and zeroed in the neutral cervical position by the researchers. Patients and control subjects were then instructed to turn to the side and then return to the neutral starting position.

CAD patients had significantly more repositioning error, suggesting a deficit in proprioceptive input from the cervical spine. These abnormalities also correlated with oculomotor dysfunction, leading the authors to conclude that abnormal oculomotor function (seen in 62% of the patient group) might be related to cervical proprioceptive abnormalities. Most interesting was the fact that most of the 26% of CAD patients who were asymptomatic at the time testing (at average, 1-2 years postinjury) showed high levels of repositioning error.

The authors also noted correlations between repositioning errors, reduced range of motion and oculomotor dysfunction, but no details were provided.

In a related study, Tjell and Rosenhall¹⁹ measured smooth pursuit using the smooth pursuit neck torsion (SPNT) test. Smooth pursuit is a measurement of the ability to smoothly track a moving object. Overshoot and undershoot are the ocular neurological equivalents of pastpointing. Again, a consecutive series of CAD patients was compared to matched controls. Fifty of the CAD patients reported dizziness; 25 did not. Smooth pursuit gain was measured after turning the head 45 degrees to the right and left. In both CAD groups, neck torsion reduced smooth pursuit gain significantly, but the sensitivity and specificity in the dizzy group was 90/91%, compared to only 56% sensitivity in the non-dizzy group.

The question generated in this study was: were the SPNT test results the result of proprioceptive abnormalities or nociceptive activity? Based on the differences between dizzy and non-dizzy patients, the authors surmised the former. Chiropractic physicians have long known that spinal manipulation can -- at least on a temporary basis -- relieve dizziness in such patients, thus lending further empirical support to this conclusion. It is in the upper cervical spine where most proprioceptive activity is generated.

Before I leave the subject, some of you might be yearning to remind me that muscles have proprioceptive afferents too. Yes, I haven't dismissed muscles entirely as important contributors of pain. Even as mere end organs for this symphony of physical hardship, they play a mighty important role. Others of you might have missed my mentioning of the lowly intervertebral disc. Clearly it is important and has been known to herniate or rupture after CAD injury. In fact, my thinking here has changed too. Whereas we once thought of disc herniation (rupture, prolapse, etc.) as an acute event, I now see it as a process: initiated perhaps early in life as a result of biomechanical aberrations and microtraumatic events; catalyzed more acutely by a motor vehicle crash injury; developing rapidly in the ensuing weeks or months into a fully developed lesion.

But the role of the lowly disc extends well beyond that of non-innervated spacer/shock absorber once attributed to it. In 1992, a group of researchers actually found -- in addition to innervation by pain fibers -- Pacinian-like corpuscles and Golgi tendon organ-like structures suggesting a more complex biomechanical servo type of role.²⁰ Might these mechanoreceptor afferents also contribute to the clinical conditions discussed here? I think it is quite likely. This also suggests the importance of reducing disc loading through maintenance or restoration of normal curves.

What Hope for Improved Seats and Head Restraints?

From the papers that I have reviewed here (and from more that I've omitted) it has become clear that the risk of injury in LOSRIC certainly will never be eliminated outright. Risk reduction, however, is an achievable goal. In fact, in my first book, written 12 years ago, I recommended a "seat back designed to absorb acceleration forces by incorporating a system that would allow a limited slow collapse of the seat back with fluid-damping characteristics."²¹ This just about perfectly describes Volvo's new seat which is found in the new S80 model. Saab also has a revolutionary new seat back/head restraint design which it is incorporating into some models. We will be crash-testing some of these seats next year at this Institute.

In the meantime, as hopeful as we are about these new seats, they depend on optimal occupant positioning, ideal occupant stature (they are designed for 50th percentile male body types), and the notion that the injury mechanisms can be interrupted before an injury occurs. It appears to me that the last variable in the equation may give us the most trouble. Virtually all of the recent tests suggest that the injury-producing event (i.e., hyperextension of the lower cervical segments and significant shear and compression) occurs within the first 200 msec or so. This sometimes occurs before the head makes contact with even a well-positioned head restraint with minimal backset.

My Canadian colleagues recently reported the results of their human volunteer testing.^{22,23} In these crash tests the backset was minimal and occupant positioning ideal. Still, the head contact with the restraint occurred between 70 and 100 msec or so. Even with such optimal laboratory conditions, fully 29% and 38% of volunteers sustained minor injury at crash velocities (ΔV) as low as 2.5 and 5 mph, respectively.

Conclusions

We are still far from understanding all there is to know about CAD injuries and their sequelae, but our fundamental understanding had advanced greatly, even from the time I first became interested in this phenomenon nearly 20 years ago. As is often the case in science, the closer we look, the more complicated the thing becomes, and the more we come to appreciate the depth of our ignorance. At the same time, we are that much closer to finding ways to prevent or reduce the significance of these injuries and to more effectively diagnose and treat them. And we are in a better position to make headway in the struggle to bring this huge public health problem to the attention of insurers, legislators and automobile manufacturers.

References (#'s 1-14 refer to Part I of "The New Model of Whiplash")

1. Macnab I. Acceleration injuries of the cervical spine. *J Bone Joint Surg* 1964;46A(8):1797-1799.
2. Bogduk N, Marsland A. The cervical zygapophyseal joints as a source of neck pain. *Spine* 1988;13(6):610-617.
3. Dwyer A, Aprill C, Bogduk N. Cervical zygapophyseal joint pain patterns I: a study in normal volunteers. *Spine* 1990;15(6):453-457.
4. Svensson M, Lovsund P. A dummy for rear end collisions: development and validation of a new dummy neck. International IRCOBI Conference on Biomechanics of Impacts. Verona, Italy, September 9-11, 1992;299-309.
5. Croft AC. Module 1 of *Whiplash: the Masters' Program*, Coronado, Spine Research Institute of San Diego, 1996, p. 17.
6. Grauer JN, Panjabi MM, Cholewicki J, Nibu K, Dvorak J. Whiplash produces an s-shaped curvature of the neck with hyperextension at lower levels. *Spine* 1997;22:2489-2494.
7. McConnell 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, 1995, p. 215-238.
8. McConnell 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, 1993, p. 21-31.
9. Ono K, Kaneoka K, Wittek A, Kajzer J. Cervical injury mechanism based on the analysis of human cervical vertebral motion and head-neck-torso kinematics during low speed rear impacts. 41st Stapp Car Crash Conference Proceedings. *SAE paper* 973340, 1997, p. 339-356.
10. Yang KH, Begman PC, Muser M, Niederer P, Waltz F. On the role of cervical facet joints in rear end impact neck injury mechanisms. SAE SP-1226, *Motor Vehicle Safety Design Innovations, SAE Paper* 970497, 1997, p. 127-129.
11. Croft AC. Biomechanics. In: Foreman SM, Croft AC (eds). *Whiplash Injuries: the Cervical Acceleration/Deceleration Syndrome*, second edition. Baltimore: Williams & Wilkins, 1995, p. 66-71.
12. Barnsley L, Lord S, Wallis BJ, Bogduk N. The presence of chronic cervical zygapophysial joint pain after whiplash. *Spine* 1995;20(1):20-26.
13. Taylor JR, Twomey LT. Acute injuries to cervical joints. *Spine* 1993;18(9):1115-1122.
14. Woodward MN, Cook JCH, Gargan MF, Bannister GC. Chiropractic treatment of chronic "whiplash"

- injuries. *Injury* 1996;27(9).
15. Otte A, Ettlin TM, Nitsche EU, Wachter K, Hoegerle S, Simon GH, Fierz E, Moser E, Mueller-Brand J. PET and SPECT in whiplash syndrome: a new approach to a forgotten brain? *J Neuro Neurosurg Psychiat* 1997;63:368-372.
 16. Bicik I, Radanov BP, Schafer N, et al. PET with (18)fluorodeoxyglucose and hexamethylpropylene amine oxime SPECT in late whiplash syndrome. *Neurology* 1998;51:345-350.
 17. Fischer AJEM, Verhagen WIM, Huygen PLM. Whiplash injury. A clinical review with emphasis on neuro-otological aspects. *Clin Otolaryngol* 1997;22:192-201.
 18. Heikkila HV, Wenngren BI. Cervicocephalic kinesthetic sensibility, active range of cervical motion, and oculomotor function in patients with whiplash injury. *Archives of Physical Medicine & Rehabilitation* 1998;79:1089-94.
 19. Tjell C, Rosenhall U. Smooth pursuit neck torsion test -- a specific test for cervical dizziness. *American Journal of Otology* 1988;19:76-81.
 20. Mendell T, Wink CS, Zimny ML. Neural elements in human cervical intervertebral discs. *Spine* 1992;17(2):132-135.
 21. Croft AC. Biomechanics. In: Foreman SM, Croft AC (eds). *Whiplash Injuries: the Cervical Acceleration/Deceleration Syndrome*. Baltimore: Williams & Wilkins, 1988, p. 68-69.
 22. Siegmund GP, King DJ, Lawrence JM, Wheeler JB, Brault JR, Smith TA. Head/neck kinematic response of human subjects in low-speed rear-end collisions. *SAE Technical Paper 973341*, 1988, p. 357-385.
 23. Brault JR, Wheeler JB, Siegmund GP, Brault EJ. Clinical response of human subjects to rear-end automobile collisions. *Archives of Physical Medicine & Rehabilitation* 1998;79:72-80.
-

Click [here](#) for more information about Arthur Croft, DC, MS, MPH, FACO.



Page printed from:

http://www.chiroweb.com/mpacms/dc/article.php?id=35076&no_paginate=true&p_friendly=true&no_b=true