

[IMAGE]

Dynamic Chiropractic – March 27, 1992, Vol. 10, Issue 07

Whiplash and Brain Injuries: Cognitive Defect or Fertile Imagination?

By Arthur Croft, DC, MS, MPH, FACO

A generally accepted fact is that the most common cause of closed head injury (CHI) is acceleration/deceleration of the brain. Since the brain is a soft structure confined in a non-yielding cranium, the mechanism of injury may be due either to a shearing of axons passing perpendicular to opposing stress planes or due to the impact of the brain against the bony skull. Primarily, the frontal and temporal lobes are injured in this way. A rapid acceleration of the brain is sufficient to produce injury -- head contact is not a prerequisite. Thus, the rapid acceleration and deceleration seen in cervical acceleration/deceleration (CAD) trauma not uncommonly results in CHI.

The fact that even minor head injuries can result in long-term residual complaints such as headache, dizziness, visual disturbance, fatigue, sleep disturbances, difficulty with concentration, and personality changes, etc., was known to even the earliest civilizations. The Edwin Smith Surgical Papyrus, a translation of an Egyptian document dating back some 5,000 years, describes just such a syndrome which today is usually referred to as the postconcussion syndrome (PCS).

The statistics are staggering. In the last 12 years alone, more Americans have died from head trauma than in all of the battles since the founding of this country. Conservative estimates of non-fatal CHI are 2 million per year, with a total cost to society of about \$25 billion annually.¹ Motor vehicle accidents (MVA) are the most common agent in both fatal and non-fatal CHI accounting for more than 40 percent of mild brain injuries. Because CHI runs the gamut from very mild to quite severe, a more precise estimate of the number of patients suffering from PCS is impossible. Many physicians (DCs and MDs alike) have been reluctant to make the diagnosis in disorders of PCS found at the "minor" end of the continuum.

This reluctance has been due largely to be the lack of objective findings, lack of information about this disorder and, in part, due to the suggestion by some that PCS is largely of a neurotic nature, with little or no organic basis. Henry Miller,² a prominent British neurologist, suggested that the majority of these patients

were suffering from "accident neurosis." His writings remain influential in medicolegal settings today in spite of the fact that they have been almost universally debunked for the past 30 years.^{3,4,5}

In the last 2-1/2 decades, we have learned a great deal about these CHIs and the ensuing PCS. We know that the responsible lesion is the diffuse axonal injury (DAI) which consists of axonal swellings (retraction balls), micro-hemorrhages, and chromatolysis of cell bodies. And, while visible on MRI and CAT scans in more severe cases, these lesions are often not detectable by high resolution studies.

We also know that the electroencephalograph (EEG) is no longer a valid diagnostic test for PCS, although some neurologists continue to "rule out" the disorder in this way. Brain stem auditory evoked responses (BAER) and topographic brain mapping are currently the most sensitive electrodiagnostic procedures, although positron emission tomography (PET) scanning and magnetoencephalography (MEG) are promising technologies in this regard. However, these tests may also fail to uncover a significant number of brain injuries.

We know that angular or non-centroidal acceleration can produce this DAI in experimental animals -- a lesion indistinguishable from that seen in man.⁶ We know that the tolerance of the brain to concussion is on an order of magnitude of 1600-1800 rad/sec², an expression of angular acceleration.⁷ We also know that a coronal plane acceleration, such as would occur in a broadside MVA, will produce greater injury than a sagittal plane acceleration.⁶ We know that in primates, non-centroidal accelerations of 4,000-12,000 rad/sec² will produce DAI.

The two current gaps in our understanding of this phenomenon are those of thresholds; in particular -- what is the threshold of DAI in man, in terms of non-centroidal acceleration? The other gap concerns diagnostic thresholds. Even our most sophisticated imaging modalities and electrodiagnostic procedures lack the sensitivity to detect or quantify the more minor of these lesions. It is quite difficult to estimate the number of false-negative results. Psychometric tests such as the Paced Auditory Serial Addition Test (PASAT) have been employed by neuropsychologists as a way of measuring cognitive defects.⁸

Whiplash, with or without head contact, has been shown to be the second most common cause of PCS,⁹ and most recently Radanov, et al.,¹⁰ have described cognitive defects in whiplash victims which they referred to as the "cervicoencephalic syndrome." It is characterized by headache, fatigue, dizziness, poor concentration, disturbed accommodation, and impaired adaptation to light intensity. Cognitive defects included a reduced speed of information processing and difficulty with divided attention. This report supports many others and

provides further and compelling evidence that these cognitive and other defects such as diminished IQ,¹¹ increased sensitivity to sound, disorganized communication skills,¹² slowed reaction time,¹³ and psychosocial problems,¹⁴ are not simply the work of fertile imaginations.

References

1. Goldstein M: Traumatic brain injury: a silent epidemic. *Ann Neurol.*, 27(3):326, 1990.
2. Miller H: Accident neurosis. *Br Med J.*, 919-925, 1961.
3. Mendelson G: Not "cured by verdict." Effect of legal settlement on compensation claimants. *Med J. Austr.*, 2:132-134, 1982.
4. Merskey H: Psychiatry and the cervical sprain syndrome. *Can Med Assoc J.*, 130:1119-1121, 1984.
5. Mendelson G: Persistent work disability following settlement of compensation claims. *Law Instit J.*, 55:342-345, 1981.
6. Genarelli TA, Thibault LE, Tomei G, et al: Directional dependence of axonal brain injury due to centroidal and non-centroidal acceleration. SAE 872197, in *Proceedings of the Thirty-first Stapp Car Crash Conference*, Society of Automotive Engineers, 49-53, 1987.
7. Ommaya AK, Hirsch AE: Tolerances for cerebral concussion from head impact and whiplash in primate. *J Biomechanics*, 4:13-21, 1971.

8. Speed WG III: Closed head injury sequelae: Changing concepts. *Headache*, 643-647, 1989.

9. Barnat MR: Posttraumatic headache patients I: demographics, injuries, headache, and health status. *Headache*, 26:271-277, 1986.

10. Radanov BP, Dvorak J, Valach L: Cognitive deficits in patients after soft tissue injury of the cervical spine. *Spine*, 17(2):127-131, 1992.

11. Gensemer I, Walker JC, McMurry FG, et al: IQ levels following trauma. *J Trauma*, 29(12):1616-1618, 1989.

12. Payne-Johnson JC: Evaluation of communication competence in patients with closed head injury and whiplash. *J Commun Disorder*, 19:237-249, 1986.

13. MacFlynn G, Montgomery EA, Fenton GW, et al: Measurement of reaction time following minor head injury. *J Neurol Neurosurg Psychiatr.*, 47:1326-1331, 1984.

14. Ruys MBM, Keyser A, Gabreeds FIM: Long-term sequelae of brain damage from closed head injury in children and adolescents. *Clin Neurol Neurosurg.*, 92(4):323-328, 1990.

Arthur C. Croft, D.C., M.S., DABCO

Coronado, California

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



Page printed from:

http://www.chiroweb.com/mpacms/dc/article.php?id=43155&no_paginate=true&p_friendly=true&no_b=true