

§400 Controversies

§405 Mild Traumatic Brain Injury and Post-Concussion Syndrome

The fragility of the brain has been recognized for centuries. Gama, in 1835, wrote, “Fibers as delicate as those of which the organ of mind is composed are liable to break as a result of violence to the head.” (Evans RW. The postconcussion syndrome and the sequelae of mild head injury. *Neurology of Trauma* 1992;10:815-847.)

Mild traumatic brain injury is a major problem in our society. Ruff et al (Ruff RM, Camenzuli L, Mueller J. Miserable minority: emotional risk factors that influence the outcome of a mild traumatic brain injury. *Brain Injury* 1996;10(8):551-565.) wrote this about the extent of the problem:

“Epidemiological evidence suggests that each year in the United States alone, over 1,300,000 individuals suffer MTBI. Ongoing studies at New York University further suggest that, after 1 year, about 10% of their MTBI sample continues to complain of post-concussional symptoms, and about 11% of those who were previously employed have not returned to work. Thus, if even a small proportion (10%) present with persistent problems, over 130,000 individuals are affected each year in the United States. Thus, the Miserable Minority represents a sizable challenge to the health-care system.”

There is a tremendous amount of confusion, controversy, and misinformation on the issue of MTBI, post-concussion syndrome (PCS), and whiplash injuries.

On one side of the equation are physicians who believe that the Postconcussion Syndrome (PCS) is faked. In fact, in a recent survey (Evans RW, Evans RI, Sharp MJ. The physician survey on the post concussion and whiplash syndromes. *Headache* 1994;34:268-274.) of physicians’ attitudes about PCS and whiplash, nearly 25% of family practice doctors and 23% of orthopedists reported that they questioned the authenticity of PCS. They were even more likely to question the validity of whiplash.

On the other side is another group of physicians that strongly believe that the psychiatric and attentional difficulties after whiplash are a result of neurological damage to the brain.

Complicating the whole issue is the role of chronic pain. Pain as a result of tissue damage can also overlap with the symptoms found in patients with MTBI. (Radanov BP, Hirlinger I, Di Stefano G, Valach L. Attentional processing in cervical spine syndromes. *Acta Neurologica Scandinavica* 1992;85:358-362; Radanov BP, Begres, Sturzenegger M, Augustiny KF. Course of psychological variables in whiplash injury—a 2-year follow-up with age, gender and education pair-matched patients. *Pain* 1996;64:429-434; Ruff RM, Camenzuli L, Mueller J. Miserable minority: emotional risk factors that influence the outcome of a mild traumatic brain injury. *Brain Injury* 1996;10(8):551-565; Schmand B, Lindeboom J, Schagen S, et al. Cognitive complaints in patients after whiplash injury: the impact of malingering. *Journal of Neurology, Neurosurgery and Psychiatry* 1998;64:339-343; Taylor AE, Cox CA, Mailis A. Persistent neuropsychological deficits following whiplash: evidence for chronic mild traumatic brain injury? *Archives of Physical Medicine and Rehabilitation* 1996;77(6):529-535; Wallis BJ, Lord SM, Barnsley L, Bogduk N. Pain and psychologic symptoms of Australian patients with whiplash. *Spine* 1996;21(7):804-810.)

Kessels et al (Kessels RPC, Keyser A, Verhagen WIM, et al. The whiplash syndrome: a psychophysiological and neuropsychological study towards attention. *Acta Neurologica Scandinavica* 1998;97:188-193.) studied a group of 24 whiplash patients in regard to this issue, with no definitive answer. They surmised that the patients' distress could, "...be related to both emotional distress caused by the accident itself and to additional experienced stressing life events, either prior to the accident or during recovery. However, these high scores could also be the consequence of organical lesions, causing physical complaints (e.g. neck pain or headache) that lead to psychological stress."

Packard and Ham wrote that, "...many patients sustaining MHI suffer from significant difficulties with concentration, memory, and/or thinking. The ability to concentrate and remember important details is often a major factor in determining an individual's ability to function in society. Sometimes these symptoms may be overlooked or disbelieved because of a 'minor' injury, no car damage, low velocity impact, and so forth. There also may be a prejudice against these patients because of legal proceedings or physician and attorney bias. Cognitive deficits may only be evident when an individual is under a great deal of stress, overtired, or trying to do more than one thing at a time. We have found that many patients are only aware of cognitive difficulty under these circumstances or when attempting to return to work or increase their activity level. These difficulties may be compounded when patients are evaluated with head imaging studies (CT or MRI) that are 'normal.' Unfortunately, a 'normal' imaging study is often considered to mean normal function." (Packard RC, Ham LP. Evaluation of cognitive evoked potentials in post-traumatic headache cases with cognitive dysfunction. *Headache Quarterly, Current Treatment and Research* 1996;7(3):218-224.)

§405.1 Etiology

As we saw in the section on Biomechanics, tremendous forces can be applied to the body during a “mild” whiplash accident.

Parker and Rosenblum (Parker RS, Rosenblum A. IQ loss and emotional dysfunctions after mild head injury incurred in a motor vehicle accident. *Journal of Clinical Psychology* 1996;52(1):32-43.) provide a thorough summary of the causes of MTBI:

“TBI needs only sufficient angular rotation (without impact) to occur, although unreported head impact in MVA is common. Rear-end impact causes the head on the relaxed neck to be left behind momentarily. Front-end collision causes the head to be propelled forward. The head moves in a combination of planes (lateral and sagittal) and torsion around the tethering neck, which changes the brain’s position and movement relative to the enclosing skull and often causes impact within the confining space. It is unpredictable which structures will be over-stressed and damaged. TBI is determined by: the point of impact and its direction, velocity, and accelerating or decelerating effect; whether the neck is braced; whether the rotation is accelerated or steady; the relative strength of the head-neck junction; ratio of brain mass to head mass; duration of the impact impulse; and characteristics of the scalp. Intensive head movement causes contusions of the frontal and temporal poles from *translational* motion, most severe at the crests of gyri, but which can extend subcortically. Lacerations occur of the base of the brain from *lateral* or angular motion. *Rotation* in the coronal plane causes shearing of internal structures and penetrating blood vessels and tensile and compression strains. Head impact causes skull and brain deformation; energy directed inwardly coup, contre-coup and/or diffuse brain injury, and pressure gradients.”

Friedmann et al (Friedmann LW, Marin EL, Padula PA. Biomechanics of cervical trauma, in *Painful Cervical Trauma: Diagnosis and Rehabilitative Treatment of Neuromuscular Injuries*, Ed. Tollison CD, Satterthwaite JR 1992;10-19.) wrote, “Patients receiving whiplash injury of the neck can also suffer from a cerebral concussion. If the head is thrown forward and then strikes the steering wheel or windshield, a head injury can occur. Also, mechanical deformation of the brain occurs during the acceleration/deceleration phase of the injury and a concussion can occur without the head actually striking anything.”

Teasell and McCain (Teasell RW, McCain GA. Clinical spectrum and management of whiplash injuries, in *Painful Cervical Trauma: Diagnosis and Rehabilitative Treatment of Neuromuscular Injuries*, Ed. Tollison CD, Satterthwaite JR 1992;292-318.) elaborate: “It has been proposed that rotational brain shear injuries occur as a result of sudden movement of the skull, especially sudden angular acceleration, resulting in surface trauma to the cerebral cortex and cerebellum. This theory posits that the brain lies relatively free within the skull. As the head moves, the brain, because of its inertia, tends to maintain its position within the vault. The moving skull may therefore concuss the brain either as it rotates backward or as it accelerates forward.”

Research by Otte et al (Otte A, Ettlin TM, Nitzsche EU, et al. PET and SPECT in whiplash syndrome: a new approach to a forgotten brain? *Journal of Neurology, Neurosurgery, and Psychiatry* 1997;63:368-372; Otte A, Goetze M, Mueller-Brand J. Statistical parametric mapping in whiplash brain: is it only a contusion mechanism? *European Journal of Nuclear Medicine [Letter]* 1998;25:306-312; Otte A, Mueller-Brand J, Fierz L. Brain SPECT findings in late whiplash syndrome. *The Lancet*, June 10, 1995;345:1513-1514.) have used the much more sensitive PET and SPECT scans on whiplash patients, and have found evidence of brain dysfunction. In one study, they wrote:

“[Our] hypothesis is that parieto-occipital hypometabolism may be caused by activation of nociceptive afferences from the upper cervical spine. By contrast, the areas of hypometabolism seen in areas other than parieto-occipital may mainly be explained by brain contusion and not by the effects of activated nociceptive afferences on brain metabolism. In addition, hypometabolism in parieto-occipital regions cannot be excluded in some cases as part or entirely a consequence of diffuse axonal lesions due to acceleration forces.” (Otte A, Ettlin TM, Nitzsche EU, et al. PET and SPECT in whiplash syndrome: a new approach to a forgotten brain? *Journal of Neurology, Neurosurgery, and Psychiatry* 1997;63:368-372.)

In a later study (Otte A, Goetze M, Mueller-Brand J. Statistical parametric mapping in whiplash brain: is it only a contusion mechanism? *European Journal of Nuclear Medicine [Letter]* 1998;25:306-312.) they elaborate on this issue. The authors hypothesized that if head trauma was responsible for the cerebral lesions, they should only exist in one of two areas—the left frontal and the right parieto-occipital regions. They did indeed find lesions in these areas. They also, however, found areas of hypoperfusion in the left parieto-occipital region.

“As the patients were looking to the right side at the time of the accident, a contusion mechanism could be possible for the left frontal and the right parieto-occipital region, regardless of whether this was produced directly by hitting the head onto the steering wheel or by the acceleration forces producing indirect head impact. If whiplash injury were only a form of mild head injury with a contusion mechanism, the additional left parieto-occipital hypoperfusion in the above patients could not be explained. Therefore, we think that parieto-occipital hypoperfusion in whiplash patients may still be hypothesized to be elicited from lesions of neuroceptive afferents from the cervical spine. Of course, this does not mean that brain contusions need not be carefully evaluated, as additional contusion is well known to have an effect on clinical after injury.” (Otte A, Goetze M, Mueller-Brand J. Statistical parametric mapping in whiplash brain: is it only a contusion mechanism? *European Journal of Nuclear Medicine [Letter]* 1998;25:306-312.)

Torres and Shapiro (Torres F, Shapiro SK. Electroencephalograms in whiplash injury. *Archives of Neurology* 1961;5:28-35.) tested this theory by giving whiplash patients electroencephalogram (EEG) tests. They found that 46% of these patients showed abnormal EEG readings. Abnormal EEGs after whiplash have also been found by Jacome et al (Jacome DE. EEG in whiplash: a reappraisal. *Clinical Electroencephalography* 1987;18:41-45.) and Ettlin et al. (Ettlin TM, Kischka U, Reichmann S, Radii EW et al. Cerebral symp-