by Robert Luce, Esq.

Proving a "Mild" Traumatic Brain Injury: A Complex but No Longer Impossible Task

Traumatic Brain Injury, especially brain injury categorized as "mild" (MTBI), has historically presented one of the biggest challenges for trial lawyers because there often are no visible markers or clear signs of brain injury. Three important areas of development—research on brain injuries and veterans conducted by the Department of Defense, research on brain injuries and athletes, conducted by Center for the Study of Traumatic Encephalopathy and other groups, and comprehensive, ongoing study conducted by the Centers for Disease Control and Prevention (CDC)—have provided critical advances in both understanding the signs and the long-term effects of brain injury and in increasing public awareness and acceptance that invisible injuries to the brain can have devastating consequences. While proving MTBI is still a highly sophisticated, complex area of law, the new research and the public attention it has brought, combined with advances in brain imaging technology, mean that it is no longer an impossible task.

Minor Complaints or Symptoms of MTBI?

Accident victims with obvious physical trauma sometimes mention other more subtle problems such as insomnia, fatigue, dizziness, nausea, imbalance, irritability, difficulty concentrating or remembering new information, sensitivity to light, visual disturbances, or loss of ability to smell. Sometimes the complaint is as vague as "I just don't seem like myself." These feelings are often first reported hours, days, or even weeks after the accident. Frequently, they become a focus of attention only when the physical trauma is less acute or when the accident victim tries to return to work or school.

Sometimes these subtle complaints improve over time; occasionally they do not. Some people report that these symptoms got worse instead of better after they first became evident. Some of these accident victims have no observable evidence of head trauma, no reported loss of consciousness and, where diagnostic images are taken, negative CT scans and MRIs. Friends, family members, and employers sometimes complain that although the physical trauma has begun to heal, the victim does not seem to be getting his or her act together, is disorganized, stays up half the night, and is difficult to get along with. Sometimes the victim gets more and more depressed, occasionally to the point of being suicidal. Many lose their jobs and/or their marriages fall apart.

In the past, many lawyers have overlooked these complaints, or, worse yet, have concluded that even the physical injuries are "worth" less in the context of a negligence claim because the client is unlikeable and comes across as "malingering." This sentiment is sometimes "confirmed" when the accident victim is finally referred for neuropsychological testing and shows deficits on all measures, including measures designed to assess "effort"—which unfortunately are sometimes inaccurately labeled as "malingering tests." The negative judgment can be further confirmed when the treating physician or neuropsychologist suggests (ignoring the prevailing understanding in this area) that the accident victim could not have a brain injury of any significance because there is no evidence of a loss of consciousness.

Information has surfaced in the past ten years that should cause a completely different light to be cast on the patient presentation I have described—information that substantially increases the likelihood of being able to prove a brain injury under these circumstances and recover substantial damages. Three recent developments are particularly significant.

Brain Injuries and Veterans

One key development is that the symptoms I have described have been exhibited in a remarkably high percentage of returning Iraq and Afghanistan War veterans over the past decade. Many of them were passengers in armored vehicles that struck IEDs and who appeared to have been "protected," at least from obvious, serious physical injuries. It has become apparent to the military, over time, that a large number of veterans who were previously thought to be suffering from Post-Traumatic Stress Disorder or malingering, have, in fact, suffered brain injuries. Autopsies of veterans who may have died of other causes reveal widespread microscopic damage to brain cells, damage that no existing imaging technology is capable of visualizing.

Traumatic Brain Injury has become known as the "signature" wound of these recent wars, and this has led to a significant increase in research funding. On January 12, 2012, First Lady Michelle Obama and Dr. Jill Biden announced the "Joining Forces" initiative involving 120 medical schools (including Dartmouth) that are committing to conduct new research and clinical trials on TBI to improve the understanding of and effective treatment for this condition. The U.S. Defense Department has created "Centers for Psychological Health and Traumatic Brain Injury" to focus on early detection and monitoring of MTBI or concussions, which account for most TBI diagnoses. They have developed clinical guidelines and support tools and have promoted the use of several advanced scanning techniques, including diffusion tensor imaging (DTI), single photon emission computed tomography, and functional MRIs, which Department of Defense experts describe as "especially promising" in early detection of MTBI.[1] The Defense Department guidelines help to explain why MTBI can produce the symptoms I have described and how, in some of these cases, the injuries can be "objectively" confirmed through advanced imaging techniques.

Brain Injuries and Athletes

A second development that brings more awareness and understanding of MTBI is the growing recognition in the context of sports that concussions, even without loss of consciousness, can have severe permanent consequences. This recognition has led to the adoption of concussion guidelines in both amateur and professional sports, sometimes at the insistence of players' unions. Many former professional athletes and their families have gone public with their common experience of depression, sleep disorders, and mental fatigue, and several suicides have been linked to histories of concussions.[2]

The Center for the Study of Traumatic Encephalopathy (CSTE) has reported stunning findings from the examination of tissue posthumously culled from retired NFL athletes.[3] These studies showed that the concussion injuries these athletes experienced—injuries not evident on conventional MRIs or CT scans—caused significant brain damage that was only visible microscopically, a condition labeled "chronic traumatic encephalopathy" (CTE). Most of the former NFL athletes whose brain tissue has been examined reveal this damage throughout the brain tissue, on both the superficial aspects of the brain and deep inside. A 2009 article published by the Amer-

ican Association of Neuropathologists describes this condition as a progressive disorder.[4] Widespread news reports of this finding have helped to raise public awareness of the potential consequences of concussions or MTBIs.

Centers for Disease Control: Comprehensive Study of TBI

The third development has emerged from the work of the U.S. Department of Health and Human Services' Centers for Disease Control and Prevention (CDC). When Congress passed the *Children's Health Act of 2000*, the legislation included instructions requiring the CDC to focus attention on MTBI. As a result, the CDC formed the Mild Traumatic Brain Injury Working Group, composed of leading experts in the field. The Working Group has published several guidelines (which have been periodically updated) for physicians, patients, and coaches, and in 2003, it provided an extensive report to Congress. (The most recent guideline, published in 2012, is titled "Returning to School After a Concussion, A Fact Sheet for School Professionals.") This information is publically available on CDC's website.[5]

Both the CDC and Defense Department findings have a high degree of credibility, since they are endorsed by respected federal agencies. The CDC's conclusions and recommendations are particularly valuable for both TBI victims and their representatives. The following contents from the CDC publications are particularly relevant to the many TBI victims with symptoms like those I described above:

- "In recent years, public health and health care communities have become increasingly aware that the consequences of mild traumatic brain injury (MTBI) may not, in fact, be mild. Epidemiologic research has identified MTBI as a public heath problem of large magnitude, while clinical research has provided evidence that these injuries can cause serious, lasting problems."[6]
- "A concussion is a brain injury. All concussions are serious. Concussions can occur without loss of consciousness or other obvious signs."[7]
- "Some symptoms may appear right away, while others may not be noticed for days or months after the injury, or until the person starts resuming their everyday life and more demands are placed upon them."[8]
- "MTBI is caused by a blow or jolt to the head that disrupts the function of the brain. This disturbance of brain function is typically associated with normal structural neuroimaging findings, i.e., CT Scan, MRI. MTBI results in a constellation of physical, cognitive, emotional and/or sleep-related symptoms and may or may not involve loss of consciousness."[9]
- "Unlike more severe TBIs, the disturbance of brain function from MTBI is related more to dysfunction of brain metabolism rather than to structural injury or damage. The current understanding of the underlying pathology of MTBI involves a paradigm shift away from a focus on anatomic damage to an emphasis on neuronal dysfunction involving a complex cascade of ionic, metabolic and physiologic events. Clinical signs and symptoms of MTBI such as poor memory, speed of processing, fatigue, and dizziness result from this underlying neurometabolic cascade."[10]
- "Diagnosing MTBIs can be challenging as symptoms of MTBI are common to those of other medical conditions (such as post-traumatic stress disorder [PTSD], depression, and headache syndromes), and the onset may occur days or weeks after the initial injury."[11]
- "Research indicates that up to 90% of concussions do not involve Loss of Consciousness"[12]
- "Physicians should be aware that symptoms will typically worsen or re-emerge with exertion."[13]
- "Sometimes people do not recognize or admit that they are having problems. Others may not understand why they are having problems and what their problems really are, which can make them nervous and upset."[14]
- "In general, recovery may be slower among older adults, young children and teens. Those who have had a concussion in the past are also at risk of having another one and may find that it takes longer to recover if they have another concussion."[15]

Mild Traumatic Brain Injury Defined

CDC's Report to Congress recommended a conceptual definition of MTBI, drawn in large part from the American College of Rehabilitation Medicine (ACRM). It is similar to the definition adopted by the World Heath Organization (WHO). This definition plays a key role in most MTBI cases. Although it is reasonably clear on its face, as I will discuss, it can be challenging to apply in many cases because of imperfect information. The definition is as follows:

> [A]n injury to the head as a result of blunt trauma or acceleration or deceleration forces that result in one or more of the following conditions:
>
> Any period of observed or self-report-

ed:

- Transient confusion, disorientation, or impaired consciousness;
- Dysfunction of memory around the time of injury [antegrade or retrograde amnesia];
- Loss of consciousness lasting less than thirty minutes.

Observed signs of neurological or neuropsychological dysfunction, such as:

- Seizures acutely following injury to the head;
- Among infants and very young children: irritability, lethargy, or vomiting following head injury;
- Symptoms among older children and adults such as headache, dizziness, irritability, fatigue or poor concentration, when identified soon after injury, can be used to support the diagnosis of mild TBI, but cannot be used to make the diagnosis in the absence of loss of consciousness or altered consciousness.[16]

One of the differences between this definition and the ACRM definition is that ACRM includes "dazed" as well as "confused or disoriented" in explaining what constitutes "altered consciousness." There is professional dispute on this issue, as discussed in a 2009 National Academy of Neuropsychological Education Paper published in the Archives of Clinical Neuropsychology[17] (the "Position Paper"). The Position Paper identifies other challenges in diagnosing MTBI that are more practically significant than this subtle difference in wording. For example, "even when patients are reviewed by emergency medical personnel at the scene of the injury, various acute symptoms, including a brief LOC, might have been present prior to their arrival at the scene."[18]

Furthermore, "[b]ecause post-traumatic confusion or amnesia usually persists for a period beyond LOC, patients are typically unable to accurately self-report if—and for how long—they were unconscious. Some patients assume they were unconscious during the period for which they have no recall." This can lead to a situation where the plaintiff reports being unconscious until "waking up" in the ambulance, yet is reported to have been talking at the scene. This discrepancy can create a credibility issue, if not clearly understood. Even assessing loss of memory for events before or after the accident can be challenging since many patients report what they were *told* about the accident instead of what they actually *remember*, without drawing this fine, but important distinction.

Focal Neurologic Signs of MTBI

The definitions do not spell out various focal neurologic signs that occasionally appear and, as the Position Paper indicates, should be used to diagnose MTBI. These signs may be

> associated with injury to one or more of the systems affecting vision, hearing, language, sensory-perceptual, or motor functions. The *most common* focal signs of brain injury,[19] including those in the WHO definition, are: post-traumatic seizures, intracranial lesions (e.g. contusion, hematoma, hemorrhage, or edema) anosmia/hyposmia; other cranial nerve deficits; visual field cuts, diplopia, or other visual symptoms caused by CNS injury; acute non-fluent (expressive) aphasia; and gait/balance problems.[20]

Needless to say, when any of these conditions are confirmed, the challenge of proving a brain injury is much less.

One of the conditions on this list that is often overlooked is anosmia, or loss of sense of smell. There is a substantial body of work (and several peer-reviewed articles) from Nils Varney that provides powerful evidence indicating that post-traumatic anosmia is a clinical sign of significant orbito-

frontal damage (the olfactory nerve passes between the bony cribiform plate and the orbitofrontal cortex) and, where present, is usually associated with very poor long-term social and vocational outcomes.[21]

It is generally accepted that, although the prognosis is favorable for the majority of MTBI patients, a significant minority of patients develops a chronic, often-debilitating constellation of signs and symptoms known as "chronic post-concussive syndrome."[22] Most of the patients exhibiting these long term disabilities (including many athletes as discussed above) do not exhibit any focal signs of brain injury. Autopsy studies tell us that in most cases, the injury to the brain is microscopic, at the cellular level.

Proving MTBI

Since even the most powerful diagnostic imaging is not capable of examining the brain cells of living patients, we are left trying to infer the injury from its footprints, either chemical (as in the case of some of the newer advanced imaging techniques) or cognitive (for example, the pattern of cognitive deficits associated with brain injuries as understood by the field of neuropsychology). These inferential methods are fraught with potential problems. Neuropsychologists have developed refined tests for determining if a patient is demonstrating the full "effort" necessary for testing to produce valid results, but unfortunately there are many reasons why patients may be unable to give full effort other than malingering, as discussed in Murial Lezak's leading text on neuropsychological evaluation.[23] When this occurs, however, the inferential tool of neuropsychology becomes essentially useless as a tool for proving the injury.

The CDC findings reflect the prevailing understanding that brain trauma initiates a metabolic process that can have destructive consequences over a course of time—in other words, that the delay in certain symptoms is normal and not a justified basis, standing alone, to question credibility. This has been well understood for years. For example, a respected learned treatise, *Greenfield's Neuropathology*, describes this process as follows:

> The delayed consequences of the primary injury have only recently begun to be understood. These are various events that have been triggered by the primary injury and include neurobiological processes involving cellular dysfunction such as free radical formation, receptor mediated mechanisms, calcium and inflammation mediated damage. In various combinations, and in various severities, the resultant cellular dysfunction defines the nature and extent of the primary injury, the outcome of which may not become apparent for several days or even weeks after the injury.[24]

In many cases, the most persuasive proof must necessarily come from "before" and "after" fact witnesses. Many cases have been won based not on the science, but on the testimony of exceptionally credible before and after witnesses—either independent witnesses like a respected employer, clergy, or leader of a non-profit the victim was actively involved with, or less independent witnesses like a young child who innocently talks about how mommy or daddy are different.

Another potentially useful method to "authenticate" a brain injury is through a qualified biomechanical analysis of both the acceleration/deceleration and rotational forces involved in the accident and a comparison of those forces to tolerance data available from public agencies. If persuasive evidence can be offered that the forces exceeded the tolerance of the typical human brain, the "differential diagnosis" of brain injury becomes more persuasive.

Brain injury is different in one critical way from other serious injuries like orthopedic injuries. For reasons that are still not fully understood, there is tremendous variability in the way different brains respond to trauma and recover from trauma. This makes it much harder to predict what will happen in any individual case. Some expectations have changed radically over time, based on evolving research. For example, it used to be thought that the brains of young children are especially malleable and would therefore recover from trauma easier than adults. Recent research demonstrates that it is exactly the opposite: young children may be particularly susceptible to permanent consequences, especially in the area of social dysfunction.[25] Current researchers, like Dr. Thomas McAllister at Dartmouth, are looking at factors such as genetics to try to understand this variability.

The attention now being given to traumatic brain injury, including MTBI, makes it less challenging to bring brain injury claims. It is now much more generally understood that someone who looks fine and can walk and talk can in fact have a disabling condition caused by injury to the brain. The attention being given to TBI has also accelerated research on better methods of diagnosis and treatment. Imaging technology is increasingly effective at detecting the "footprints" of brain injury. For example, on March 2, 2012, the *Journal of Neurosurgery* published very exciting research regarding a new, powerful tool developed at the University of Pittsburgh Medical Center for visualizing the footprints of microscopic brain damage called "high definition fiber tracking." This technology promises to be even

more sensitive than the current Diffuse Tensor Imaging (DTI) technology.[26]

Other footprints are being discovered, including footprints in the blood. For example, patients with TBI have been shown to have significantly higher levels of glial fibrilary acidic protein (GFAP) than patients without such injuries, a finding that has led to work on diagnostic blood tests. Not surprisingly, some of this work has been funded by the Department of Defense and, to some extent, the National Institutes of Health.[27] We still know, however, that many injuries exist without obvious footprints and cannot be fully confirmed until autopsy.

The proof of brain injury can be one of the most complex and challenging tasks of a trial lawyer, a task requiring up-to-the-moment knowledge of the rapidly evolving research. Fortunately, the future holds great promise for making this task less daunting.

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[1] *See* Molly A. Burgess, *New Developments Lead to Early TBI Detection* (Apr. 2, 2010), http://www.defense.gov/news/newsarticle.aspx?id=58587.

[2] The University of North Carolina's "Center for the Study of Retired Athletes" has focused on this issue. Several research papers can be accessed on its website at www.csra.unc.edu.

[3] The research of the "Center for the Study of Traumatic Encephalopathy" at Boston University, as well as good background information on sports concussions, can be found at www.bu.edu/cste.

[4] Ann C. McKee et al., *Chronic Traumatic Encephalopathy in Athletes: Progressive Tauopathy After Repetitive Head Injury*, 68 J. Neuropathology & Experimental Neurology 709, 709 (2009).

[5] Centers for Disease Control, *Injury Prevention & Control: Traumatic Brain Injury*, http://www.cdc.gov/TraumaticBrainInjury (last visited Mar. 19, 2012).

[6] Centers for Disease Control, *Report to Congress on Mild Traumatic Brain Injury in the United States: Steps to Prevent a Serious Public Health Problem*, http://www.cdc.gov/traumaticbraininjury/pdf/mtbireport-a.pdf.

[7] Centers for Disease Control, *Heads Up: Concussion in Youth Sports, A Fact Sheet for Coaches*, http://www.cdc .gov/concussion/pdf/coaches_Engl.pdf.

[8] Centers for Disease Control, *Injury Prevention & Control: Traumatic Brain Injury—Concussion*, http://www.cdc.gov/concussion/signs_symptoms.html.

[9] Centers for Disease Control, *Facts for Physicians*, at p. 2, http://www.cdc.gov/concussion/headsup/pdf /Facts_for_Physicians_booklet-a.pdf.

[10] *Id.* at p. 3.

[11] *Id.* at p. 4.

[12] *Id.* at p. 6.

[13] *Id.* at p. 7.

[14] CDC, *Facts About Concussion and Brain Injury*, supra note 8.

[15] *Id.*

[16] CDC, *Report to Congress*, supra note 6.

[17] Ronald M. Ruff et al., *Recommendations for Diagnosing a Mild Traumatic Brain Injury: A National Academy of Neuropsychology Education Paper*, 24 Archives of Clinical Neuropsychology 3, 5 (2009).

[18] *Id.*

[19] This is not a complete list of focal signs of injury, only the most common ones.

[20] Ruff et al., *supra* note 17, at p. 7.

[21] *See, e.g.*, Nils R. Varney et al., *Quantitative PET Finding in Patients with Posttraumatic Anosmia*, 16 J. Head & Trauma Rehabilitation 259 (2001).

[22] This is addressed in Thomas W. McAllister, *Mild Brain Injury and the Postconcussion Syndrome, in* Textbook of Traumatic Brain Injury 279, 300-01 (Jonathan M. Silver et al. eds., 2005). Dr. McAllister is a neuropsychiatrist practicing at Dartmouth Medical Center.

[23] Muriel D. Lezak, Neuropsychological Assessment 771-72 (4th ed. 2004).

[24] David I. Graham & Peter L. Lantos, Greenfield's Neuropathology 197 (6th ed. 1997).

[25] Stefanie Rosema et al., *Social Function in Children and Adolescents After Traumatic Brain Injury: A Systematic Review 1989-2011*, 29 J. Neurotrauma (forthcoming 2012), *available at* http://online.liebertpub.com/doi/abs/10.1089/neu.2011.2144.

[26] Samuel S. Shin et al., *High-Definition Fiber Tracking for Assessment of Neurological Deficit in a Case of Traumatic Brain Injury: Finding, Visualizing and Interpreting Small Sites of Change*, 116 J. Neurosurgery (forthcoming 2012), *available at* http://thejns.org/doi/pdf/10.3171/2012.1.JNS111282.

[27] Drissa Zongo et. al., *S100-B Protein as a Screening Tool for the Early Assessment of Minor Head Injury*, 59 Annals Emer. Med. 209 (2012).