

## HEAD INJURY UPDATE

### From an Article Published in the March 1995 Edition of *The Verdict*

by Michael Slater

One of the most difficult problems for lawyers involved in the presentation of traumatic brain injury (TBI) cases is that some doctors are unfamiliar with TBI. Dr. Bernard makes the following observation in the preface to his book *Closed Head Injury: A Clinical Sourcebook* published in 1994:

"After spending many years in an active metropolitan academic neurologic practice, the author noted that many of his patients had characteristic histories and symptoms that appeared in patterns and formed a symptom complex...closed head injury was a single common denominator with these patients, mild to moderate head injury with post-concussion syndrome was a condition the symptom patterns revealed...medical schools do not teach the concept of mild to moderate head injury and many physicians do not understand the problem today."

If the doctor doesn't recognize TBI, what is the lawyer to do? The first step for the lawyer interested in TBI cases is to have an understanding of the mechanism of TBI and a familiarity with the medical and neuropsychological literature.

Publications such as *Brain Injury* and the *Journal of Head Trauma Rehabilitation* will serve to educate the lawyer by providing access to the latest articles and research in TBI. The following publications will also prove useful to the lawyer involved in a TBI case:

- *Neuropsychology for the Attorney*. Sbordone, R.J. 1991, Paul M. Deutsch Press, Inc. A new edition is in press.
- *Mild Head Injury*. Levin, H.S., Eisenberg, H.M. and Benton, A.L. (Eds.) 1989, Oxford University Press.
- *Rehabilitation of Post-Concussive Disorders*. Horn, I.J. and Zasler, N.D. (Eds.) 1992. Hanley & Belfus.
- *Litigating Head Trauma Cases*. Roberts, A.C. 1991. John Wiley & Sons, Colorado Springs. A new edition will be published March, 1995, call 1-800-225-5943.
- *Magnetic Resonance Imaging*. Kucharezyk, J., Moseley, M. and Barkovich, A.J. (eds.) 1994. CRC Press, Inc., 2000 Corporate Boulevard. N.W., Boca Raton, Florida, 33431.
- *Analysis, Understanding and Presentation of Cases Involving Traumatic Brain Injury*. Charles N. Simpkins (Ed.) 1994. National Head Injury Foundation, 1776 Massachusetts Ave., N.W. Suite 100, Washington, DC 20036
- *Closed Head Injury: A Clinical Sourcebook*. Bernard, P.G. 1994, The Michie Company, Charlottesville, Virginia.
- *The Neurolaw Letter*. HDI Publishers, P.O. Box 131401, Houston, Texas 77219

Lawyers representing TBI clients must develop different strategies to deal with challenges not experienced in most personal injury cases. A new field of jurisprudence called Neurolaw has emerged in the last few years to assist lawyers in meeting these new challenges in the identification, evaluation and presentation of a TBI case. Neurolaw was accepted as a practice listing in the 1993 edition of the *Martindale-Hubbell Law Dictionary*. Recognition of Neurolaw as a specific field in personal injury law is a significant step in establishing this emerging discipline within the international legal community.

One of the best ways for the lawyer to stay in tune with recent developments in Neurolaw is to subscribe to The Neurolaw Letter, a monthly publication for legal and medical professionals interested in TBI.

The editorial advisory board of The Neurolaw Letter includes internationally recognized experts in TBI. An example of the type of article appearing is Recognizing Closed Head Injuries by neuropsychologist Dr. Antoinette R. Appel. This article, from the June, 1994 edition is reproduced with the permission of the publisher of The Neurolaw Letter:

### **"Recognizing Closed Head Injuries"**

*Antoinette R. Appel, Ph.D.*

Recognition of an organic basis for closed head injuries is a relatively new phenomenon in the history of both law and medicine. Less than ten years ago, it was impossible to convince people that closed head injuries were not the product of a shared fixed delusional system of the plaintiff, his or her doctor, and counsel. Terms like "litigation psychosis" were remarkably frequent in both the medical literature and the courtroom, causing victims of negligence to become victims again, this time of ignorance. The inability to see the injury caused by closed head trauma led both the legal and medical profession to conclude inappropriately that patients were committing fraud.

Cognizance of the reality of closed head injuries and cognizance of the important role of closed head injuries in producing lasting functional impairment emerged only after technological advancements allowed histological visualization of shearing injuries<sup>1</sup> and imaging of remarkably similar and previously invisible abnormalities in patients who shared both commonality in their complaints and in the events that gave rise to their complaints.<sup>2</sup> After three decades of histological verification, two decades of CT studies, and one decade of MRI studies, the scientific community has begun to realize that even the new imaging technologies are insufficient to allow for the adequate appreciation of the full nature and extent of most closed head injuries.<sup>3</sup> Unfortunately, it will very likely be still another decade before the legal community embraces that fact. Although there is incontrovertible evidence that significant brain injury can exist in the absence of the ability to image it in the living person, many judges, juries and some neuroscientists steadfastly continue to believe that absent visualization, no traumatic brain injury can exist.<sup>4</sup>

The new understanding that visualization of traumatic brain injury may not always be possible, even when the most modern technology is used, and the almost cultist adherence by some to the belief that visualization is a requirement in proof of the existence of a traumatic brain injury, raises the very interesting question of how a closed head injury is recognized and secondarily, once having recognized its existence, how it is that others will recognize its existence and its importance.

Consider a Florida case *State v. Chestnut*.<sup>5</sup> Although this case arises out of criminal law, its lesson cuts across civil and criminal law. Basically, the court found that the proof of the existence of a closed head injury was insufficient to meet Florida's test for admissibility on the issue of *mens rea*. In the civil arena, proof of the existence of a closed head injury without similar demonstration of how both the patient's complaints and the patient's disabilities arise from the injury is insufficient to meet the "feel good" test for compensability. Although large blocks of courtroom time are spent on proof of the existence of an injury, virtually no time is spent

explaining how a particular injury causes the specific complaints the patient/client has. And, even less time is spent relating residual disability to neuropathology. Absent proof of these relationships, there is no particularly good reason for juries to compensate clients appropriately.

Typically, the pathophysiology of traumatic brain injury (TBI) involves diffuse axonal injury caused by shearing forces, ablative injury caused by contusion, metabolic derangements either caused by or causative of edema, hypoxic injury and vascular injury.<sup>6</sup> These mechanisms may or may not produce loss of consciousness, positive imaging studies, or immediate complaints of head-related problems. Nonetheless, residual function impairment arises from these mechanisms of injury and not independent of them. Whereas it is important for the treatment of a patient and the proof of a case to be able to specify the actual mechanism of injury as precisely as possible, the initial recognition that an injury exists usually depends on the stressing of the system caused by making demands on cognitive capacity. This stressing of the system by making demands on cognitive capacity may or may not interact with brain reserve capacity.<sup>7</sup> The bottom line is that failure to document loss of consciousness, failure to obtain positive imaging studies, and failure to find an immediate complaint of head related problems are non-dispositive of the question: "Does a closed head injury exist?" Equally non-dispositive of this question is the unsubstantiated assumption that the amount of damages sustained by vehicles in collisions is directly related to the probability of an occupant sustaining a closed head injury.

It is incorrect to believe that, in cases involving vehicular collisions, increased vehicle damage is an indicator of increased probability of injury. Although the proof is complicated, for collisions that do not cause major deformity of the passenger compartment, at any given collision speed, vehicle crunch is inversely (not directly) related to injury potential. This is because the less crunch there is of the vehicle, the more energy is available to act on the person and thus to cause injury.

The failure to recognize this likely arises in part from conventional injury and accident analyses that stress analysis of linear dynamics and that essentially ignore the physiologically much more important rotational dynamics. It is angular acceleration and angular deceleration, not linear acceleration, that produce the most devastating effect on the brain and that cause axonal injury as well as potential alteration in level of consciousness.<sup>8</sup>

Collision analyses that place importance upon rotation much more closely predict pathophysiological findings and neuropsychological findings observed in patients with closed head injuries than do collision analyses that stress linear acceleration and linear deceleration. Rotation of the areas around the base of the brain typically produces small areas of shear that interfere with transmission of both brainstem to cortex and cortex to brainstem information and it potentially produces impairment in transcortical communication as well. These fundamental injuries interfere with speed of transmission up to and down from cortical areas and also with transcortical passage of information from brain areas with less integrative capacity to brain areas with more integrative capacity. Basically, rotation gives rise to cognitive confusion because information is no longer properly gated or appropriately paced, and so information that should arrive at different cortical areas within tight time constraints does not. This is somewhat akin to throwing parts of an object on a table and hoping that by chance, they will fall in place to form a whole object. This is unlikely to happen and it is equally unlikely that patients suffering from TBI will produce well-integrated thoughts consistently over time.

Neuropsychologists have the responsibility of explaining the relationship between event dynamics and pathophysiology and between locus of injury and examination findings. There are some general rules that are useful in this regard. First, the clinical observation of immediate loss of consciousness implies an injury to the

brainstem caused by rotation of the cortex around the brainstem. This in turn implies an acceleration/deceleration of the brain with the likely production of shear injuries in the brainstem that may only be demonstrable on microscopic examination.<sup>9</sup> The brainstem shear injuries in turn imply interference with transmission of cortically directed information and interference with interpretation of signals from the cortex to the lower brain centres. This invariably produces a period of confusion following upon awakening, with the length of the period of confusion obviously being related to the severity of the brainstem injury. Interestingly, the size of the injury causing this sequence of events can be sufficiently small that routine acute trauma CT scans fail to detect the injury.<sup>10</sup> A corollary of this rule is that injuries that result in delayed loss of consciousness are likely to act by causing edematous compression of the brainstem or ischemia, but are not likely to act by shear. If the edema or ischemia can be rapidly reversed, then it is likely that there will be little or no permanent damage to cell membranes, and thus, no permanent deficits. If, on the other hand, the ischemia or edema persist, then the nerve membrane will be permanently damaged and lasting residual deficits can be expected. It is likely that these observations underlie the noted observation of a direct relationship between length of coma and severity of residual disability in patients sustaining closed head trauma.<sup>11</sup>

The second rule to keep in mind is that injuries to primary sensory or motor nerves, nuclei, tracts, and cortical areas produce focal signs on routine neurological examination. But, injuries to integrative areas do not produce focal signs and are usually missed on routine neurological examinations.

The third rule to keep in mind is that for injuries that are restricted to within integrative areas of a specific system, the size of the injury is directly related to the magnitude of the neuropsychological deficit. This is an example of Lashley's law of mass action.<sup>12</sup>

The fourth rule is that injuries that have coup-contrecoup characteristics are likely to involve linear acceleration and deceleration, which is transitional movement. That is true regardless of whether or not there are also rotationally caused or related injuries. Injuries that involve linear acceleration and deceleration are likely to involve the sliding of the brain over osseous structures and are likely to involve impact of the brain against the inner table of the skull. If these injuries are produced by a force acting from behind the patient, as is typical in the usual rear-end collision, then the injuries are more likely than not to include involvement of the frontal lobe. Because the injuries also likely include some rotation of the brain in the posterior to anterior direction, the inferior frontal lobe surfaces are apt to be involved. Involvement of these surfaces is known to result in behavioral abnormalities. Finally, the fifth rule explains in a theoretical model why mild brain injuries in patients who have previously sustained brain injuries appear to have more devastating consequences than do these identical injuries in patients who have not previously sustained brain injuries. This rule suggests that there is a limited amount and ability for uninjured integrative areas to take over the function of injured integrative areas and that an initial brain injury tends to use up this reserve, making the consequences of a second injury far more devastating than would otherwise be the case. Although Satz relates reserve capacity to brain size, it appears as if the data better fit a function in which brain size is replaced by the percentage of the system specific integrative area remaining unimpaired. Regardless of whether Satz's brain size or a percentage model proves to be correct, one or the other hypothesis will be useful in explaining the disproportionate consequences of a second and subsequent brain injuries.

In addition to the neuropsychologist's responsibility to relate event dynamics to pathophysiology and locus of injury to neuropsychological findings, the neuropsychologist also has the responsibility of demonstrating that the neuropsychological findings cannot be explained by things other than the head injury. To do this the neuropsychologist meticulously reviews the premorbid medical records, the post-event medical records, and all

employment and educational records from both before and after the event that is purported to be causative of the neuropsychological findings. Specific attention has to be directed to those illnesses, whether acute or chronic, which could conceivably result directly or indirectly in similar neuropsychological dysfunctions. The neuropsychologist should be required to explain to both counsel and the jury why such illnesses did not or could not produce the findings being offered as proof of a closed head injury.

Finally, the neuropsychologist should relate the uncompromising truth to counsel and to the client. If there is no head injury, it is wrong to say there is one. The neuropsychologist, by being truthful, can keep the patient from being falsely labeled and, in addition, can keep counsel from expending large sums of money in cases where proof of a head injury will never be possible. Getting into a case late and finding evidence of major proof problems that have never been addressed previously with the patient's attorney can be very uncomfortable for the truthful expert. It is better if that happens early on. There are enough real head injury cases and counsel should not dilute their claims and denigrate these injuries by making claims that cannot be substantiated. On the other hand, counsel must ensure that those who would deny the reality of injuries, merely because they cannot be imaged, are prevented from doing so. It was not so long ago that the conventional wisdom was that there could be no significant brain injury without a loss of consciousness and without visualizable macrodamage. It is now known that is false. I encourage you to remain current, respect each other's knowledge, and work together in the best interest of the patient/client. Clients and patients will then prevail in their efforts to be made whole and no one will ever again be able to suggest that closed head injuries are the product of a shared fixed delusional system of plaintiff, his or her doctor, and counsel.

#### References

<sup>1</sup>Strich, 19 J. Neurol. Neurosurg. Psychiat. 163; Oppenheimer, 33 J. Neurol Neurosurg. Psychiat. 299.

<sup>2</sup>See, e.g. Gentry et al., 9 AJNR 101; Abel-Dayem, et al., 165 Radiology 221.

<sup>3</sup>Wilson, 4 Neuropsychology 261; Widemann et al., 3 Neuropsychology 267.

<sup>4</sup>State v. Zapetis, unpublished, 17th Judicial Circuit, Florida 1993.

<sup>5</sup>538 So.2d 820 (Fla.1989).

<sup>6</sup>Levin, Benton and Grossman, Neurobehavioural Consequences of Closed Head Injury, at 11-33; Miller, 5 Neuropsychology 235.

<sup>7</sup>See, e.g., Satz, 7 Neuropsychology 273; See also "rule 5" infra.

<sup>8</sup>Gennarelli, Head and Neck Injury Criteria, A Consensus Workshop, NHTSA (1981).

<sup>9</sup>Adams et al., 15 Histopathology 49

<sup>10</sup>French and Dublin, 7 Surgical Neurology 171, 172; 44 percent of patients in deep coma with decerebrate posturing had negative CT scans.

<sup>11</sup>Levin, Benton and Grossman, 73-79.

<sup>12</sup>Lashley, Brain Mechanisms and Intelligence (1929); Lashley, 73 Science 245."

The last paragraph of this article will serve as the focal point for a conference sponsored by the Trial Lawyers Association of British Columbia to be held May 19 and 20, 1995 in Vancouver, Litigating Traumatic Brain Injury Cases: Identification, Evaluation and Presentation of the Evidence. This conference is designed to meet the needs of trial lawyers who are involved in litigation of traumatic brain injury cases. Internationally recognized experts in neurology, psychiatry, neuropsychology and neuro-rehabilitation will review the latest developments in recognizing, evaluating and proving traumatic brain injury.

The presenters include members of the editorial advisory boards of such publications as Brain Injury, Journal of Head Trauma Rehabilitation and The Neurolaw Letter and the authors of books such as Neuropsychology for the Attorney, Rehabilitation of Post-Concussive Disorders and Litigating Head Trauma Cases. The American Trial Lawyers who co-authored Litigating Head Trauma Cases and who first identified the term Neurolaw in 1991 will describe the development of this emerging field of personal injury practice.

Topics to be covered at the conference will include:

- Introduction to Neurolaw
- Traumatic brain injury: what the trial lawyer needs to know
- Neurodiagnostic techniques to detect brain damage: EEG, CT scan, MRI, Spect scan, PET scan, brain mapping
- Traumatic head injury or traumatic brain injury: what's the difference?
- Neuropsychological testing: its uses and abuses
- The neuropsychologist and the neurolawyer: how to determine if your client has a brain injury
- Post-concussive disorders: neurorehabilitative assessment and treatment
- Preparing the plaintiff in the mild traumatic brain injury case
- Executive function disorders
- Traumatic brain injury in childhood: special considerations
- Seizure disorders following traumatic brain injury
- Litigating traumatic seizure disorders
- Malingering, hysteria, somatization and factitious disorders: a neuropsychiatric perspective
- Panel discussion of a mild traumatic brain injury case