TRAUMATIC BRAIN INJURY: WHAT THE LAWYER NEEDS TO KNOW

Michael J. Slater Q.C.
Slater Vecchio LLP, Vancouver, B.C.

TRAUMATIC BRAIN INJURY: WHAT THE LAWYER NEEDS TO KNOW

Michael J. Slater Q.C.
Slater Vecchio LLP, Vancouver, B.C.

I. Introduction

The number of traumatic brain injury (TBI) cases each year in the United States is reported to be in the range of 1.6 to 3.8 million.[1] This likely translates into 160,000 to 380,000 cases of TBI in Canada each year. This number has been increasing each year partly as a result of the increase in population but also as a result of the increasing awareness of mild traumatic brain injury (MTBI) by the medical community.

MTBI accounts for approximately 75 to 90 percent of all TBI cases.[2] However, the fact that a brain injury is classified as "mild" does not necessarily mean that the effects and consequences for the person who sustains the injury are also mild. In neurology this has been described as "the eloquence of certain portions of the brain".[3] In the case of a stroke, tumor, or trauma, the consequences can be very substantial in the case of damage to an "eloquent area" whereas the consequences can be quite trivial in the case of damage to a "non eloquent area". While the majority of persons recover from a MTBI within 6 months, a significant minority - approximately 10 to 20 percent - never completely recover and are left with one or more of the following problems: physical symptoms (e.g., dizziness, headache, sleep disturbance, quickness to fatigue or lethargy); cognitive deficits (e.g., involving attention, concentration, short-term memory or executive functions); and behavioral changes and/or alterations in degree of emotional responsivity

[1] Granacher, R., *Traumatic Brain Injury: Methods for Clinical and Forensic Neuropsychiatric Assessment* 2nd ed. (Boca Raton, FL: CRC Press, 2008) at xvii; Lezak, M., *Neuropsychological Assessment,* 4th ed. (USA: Oxford University Press, 2004) at 158; McCaffrey, R.J. et al., "Forensic issues in mild head injury" (1993) 8:3 Journal of Head Trauma Rehabilitation 38-47 at 38; Ruff, R., "Two decades of advances in understanding of mild traumatic brain injury" (2005) 20:1 Journal of Head Trauma Rehabilitation 5-18 at 11; Langlois, J.A.; Rutland-Brown, W., & Wald, M., "The epidemiology and impact of traumatic brain injury: a brief overview" (2006) 21:5 Journal of Head Trauma Rehabilitation 375-378.

[2] Alexander, M., "Mild traumatic brain injury: pathophysiology, natural history and clinical management" (1995) 45 Neurology 1253-1260 at 1256; Ruff, *supra* note 1 at 11; Kraus, J. & Chu, L., "Epidemiology" in Silver, M., McAllister, T. & Yudofsky, M., eds., *Textbook of Traumatic Brain Injury* (Washington D.C.: American Psychiatric Publishing Inc., 2005) at 3; Kraus, J.F., & Nourjah, P., "The epidemiology of mild, uncomplicated brain injury" (1988) 28:12 Journal of Trauma 1637-1643; Luerssen, T.G., Klauber, M.R., & Marshall, L.F., "Outcome from head injury related to patient's age: a longitudinal prospective study of adult and pediatric head injury" (1988) 68:3 Journal of Neurosurgery 409-416; Lescohier, I., & DiScala, C., "Blunt trauma in children: causes and outcomes of head versus intracranial injury" (1993) 91:4 Pediatrics 721-725.

[3] This was stated by the neurologist Dr. Donald Calne during the jury trial of *Heringa v. Mah*. A review of the damage award can be seen at [2000] B.C.J. No. 1764 (C.A.).

(e.g., irritability, quickness to anger, disinhibition or emotional lability).[4] These individuals have been referred to as the "miserable minority",[5] the unfortunate minority of MTBI victims who are most likely to find their way into the lawyer's office seeking assistance.

The 1984 decision in *Clark v. Tedesco*[6] is a good example of how far both the medical community and the courts have come in the last 25 years in appreciating the potentially debilitating consequences of mild and moderate brain injuries. The case was an assessment of damages for injuries suffered by a 6-year-old male plaintiff in a motor vehicle accident. The plaintiff hit his head and was unconscious for approximately four minutes. He vomited and was lethargic at the hospital. A CT scan showed dilation in the right lateral ventricle indicating right-sided cerebral atrophy. He experienced balance problems, he was easily frustrated in school and he seemed tired and unable to, or disinterested in, finishing his work. A neurologist concluded that the plaintiff had suffered a blunt head injury and showed temporary, but definite disturbances of behaviour, intellect, and vestibular function. The plaintiff's mother complained about his poor attitude and poor school work following the accident. She was concerned that the brain injury would affect her son's future income earning capacity. The Court did not agree and awarded only $7,000.

Since *Clark*, there has been an explosion of medical literature in the field of traumatic brain injury (TBI). Lawyers can now access this literature to gain a better appreciation of the long term cognitive, behavioural and emotional deficits suffered by victims of TBI. Experts in behavioural neurology, clinical neuropsychology, and neuropsychiatry can assist the trier of fact to understand how a TBI can significantly impair future earning capacity. Experts in rehabilitation science can demonstrate why a victim of a TBI may require substantial rehabilitation and future cost of care. It is no longer unusual to see awards in excess of $1 million in mild traumatic brain injury (MTBI) cases.[7]

Although doctors and neurologists are generally better educated about TBI, there are still many challenges that confront the lawyer in representing TBI victims. I will try to provide a practical overview to the many issues that arise in the prosecution of TBI cases. Much of the information reflects what I have learned in my own litigation experience in TBI cases over the last 25 years. My goal is to arm you with the tools you will need to properly handle a TBI case. I will discuss the common myths that still arise in TBI cases,

---

[4] Kay, T. et al., "Definition of mild traumatic brain injury" (1993) 8:3 Journal of Head Trauma Rehabilitation 86 - 87.

[5] Ruff, *supra* note 1. These victims of MTBI have also been referred to as the "walking wounded": Murray, D.D., "The walking wounded" (1985) 27:8 B.C. Medical Journal 488-489 at 488.

[6] [1984] B.C.J. No. 139 (S.C.).

[7] For examples of MTBI judgments in excess of $1 million see *Roussin v. Bouzenad*, [2005] B.C.J. No. 2682 (S.C.) ($1.3 million); *Lines v. Gordon*, [2006] B.C.J. 3318 (S.C.) varied [2009] B.C.J. No. 471 (C.A.) ($3 million); and *Towson v. Bergman*, [2009] B.C.J. No. 225 (S.C.) ($1.18 million).

the differential diagnosis of a TBI, the issues surrounding recovery from a MTBI, the pathophysiological mechanism of a TBI, why some individuals never fully recover from MTBI, the significance of collateral information, and the issue of malingering and secondary gain.

II. Common Myths in MTBI

In their 1981 text, *Management of Head Injuries*, Jennett and Teasdale concluded that "the damage done by, and the symptoms subsequently suffered after mild head injuries are frequently underestimated [as] …doctors who deal with mildly injured patients are unfamiliar with recent work in this field…".[8] Medical researchers at New York University Medical Center reported in a 1986 article:

> We found gross ignorance and neglect of the long term problems associated with 'mild' head trauma: those injuries where patients spent a brief time (if any) in the hospital, make quick medical recoveries, and were discharged directly home without any perceived need for formal rehabilitation… these patients appeared fine until they attempted to resume their responsibilities at home, work, or school. When they did so, a significant number experienced great difficulty. They complained of inability to remember, concentrate, organize, handle a number of tasks at once, and get as much work done as efficiently as they used to. The relationships with family, peers, and bosses often suffer and they develop psychological problems… In such cases, the unique problem of minor head injury readily became apparent despite swift and complete physical recoveries, and despite no obvious neurological basis for the problems, these persons were experiencing significant cognitive, emotional, and behavioral deficits that seriously interfered with their ability to lead fully functional lives.[9]

This ignorance continued over the next decade as observed by Dr. Bernard, a neurologist and author of the 1994 text, *Closed Head Injury: A Clinical Sourcebook*:

> 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.[10]

---

[8] Jennett, B. & Teasdale, G., "Management of Head Injuries" (1981) 20 *Contemporary Neurology Series*, vol. 20, (USA: F.A. Davis Company, l98l) at 263.

[9] Kay, T., "Minor head injury: An introduction for professionals" (1986) National Head Injury Foundation 1-12 at 1.

[10] Bernard, P.G., *Closed Head Injury: A Clinical Sourcebook* (Charlottesville, Virginia: The Michie Company, 1994).

The same observation was made by noted neurologist, Dr. Michael Alexander, in a 1995 article published in Neurology:

> Postgraduate teaching in neurology does not mirror the high prevalence of this disorder - i.e., most [medical] residents probably do not get proportionate instruction in the diagnosis and management of mild TBI...[11]

This "gross ignorance and neglect" has led to the development of some commonly held myths about MTBI. These myths include:

1. MTBI can not occur without loss of consciousness (LOC);
2. A Glasgow Coma Scale (GCS) score of 15 means no MTBI;
3. You must hit your head to sustain a brain injury;
4. If neurodiagnostic imaging is negative, no MTBI has occurred; and
5. Everyone fully recovers from a MTBI.[12]

If defence experts attempt to rely on these myths, they must be dispelled by experts more familiar with the literature in the field of TBI. This can also be accomplished through the cross-examination of the defence experts on the authoritative literature. This provides the lawyer with the opportunity to educate the trier of fact while at the same time attacking the credibility of the defence experts. The information necessary to dispel these myths is reviewed in this paper.

III Preliminary Information

In order to assess whether a client has sustained a MTBI, the lawyer should obtain the following information before referring the client to experts such as neuropsychiatrists, behavioural neurologists or clinical neuropsychologists, who are skilled in the diagnosis of MTBI:

1. Client's full history pre- and post-accident. The records will eventually surface and it is important to know whether there is anything in the client's history that will affect a differential diagnosis.
2. Witnesses to the accident. Did the client hit their head, lose consciousness, and demonstrate confusion or disorientation? Were there any complaints of headache, dizziness, or nausea at the scene? Did the client appear stunned or confused?

[11] Alexander, *supra* note 2 at 1253.

[12] McLeish, J.A. & Oatley, R.G., *The Oatley-McLeish Guide to Brain Injury Litigation* (Canada: LexisNexis Canada Inc., 2005) at 123-135.

3. Ambulance Crew Report. Check for the Glasgow Coma Scale (GCS) score, any reference to loss of consciousness (LOC), and combative or agitated behaviour. There may be references to trauma to the head such as bruising or lacerations.
4. Post traumatic amnesia (PTA). Does the client recall the events leading up to the accident, the particulars of the accident and the events after the accident? If not, what is the period of PTA.
5. Hospital records. It is important to review not only the initial history, assessment and diagnosis, but the nursing notes which may contain references to cognitive, emotional, and behavioural symptoms consistent with a brain injury.
6. Post accident medical and rehabilitation records from all health care practitioners including physiotherapists, chiropractors, massage therapists, naturopaths, and counsellors. These records may contain references to complaints of headache, dizziness, nausea, as well as problems with memory and concentration.
7. Prior medical records. There may be evidence of a prior brain injury, or other medical conditions that could increase vulnerability to a TBI and explain why the client falls into the 10 to 20 percent of MTBI patients who never recover.
8. School records including any standardized test results.
9. Employment records.
10. Collateral information. Family, friends, teachers, employers, and co-workers can confirm changes in cognitive, emotional, and behavioural functioning following the traumatic event.

The next step is to ensure that the case meets a currently acceptable definition of MTBI.

IV. Differential Diagnosis and Definition of MTBI

Terms like minor head injury, concussion, and post concussive syndrome, posttraumatic syndrome, and traumatic head syndrome have been used interchangeably,[13] which has contributed to difficulties in the diagnosis of MTBI. In 1986, the second issue of the Journal of Head Trauma Rehabilitation was devoted to the topic "Minor Head Injury" although the topic was about MTBI. In 1993, the Journal devoted another issue to the same topic but with the new title, "Mild Traumatic Brain Injury". Dr. Thomas Kay explains that the two conditions are not identical and that symptoms may emanate from either or both conditions:

> Minor head injury refers to an injury to the head, face, and neck area with symptoms caused by damage to the skull, scalp, soft tissues, or peripheral nerves but where there is not necessarily injury to the brain. MTBI refers to a minor head

[13] Bigler, E.D., "Neuropsychology and clinical neuroscience of persistent post-concussive syndrome" (2008) 14 Journal of the International Neuropsychological Society 1-22 at 2.

injury in which there is also damage to the brain, or at least disruption of brain function, as evidenced by alterations of consciousness at the time of injury.[14]

The term "head injury" and even "closed head injury" are no longer employed by TBI experts to refer to brain injury. They are now replaced by traumatic brain injury.

A. American Congress of Rehabilitation Medicine (ACRM)

The first clear definition of MTBI was developed by the American Congress of Rehabilitation Medicine (ACRM).[15] The definition was a consensus reached by a committee of leading experts in the field which established diagnostic criteria for MTBI and was first published in the Journal of Head Trauma Rehabilitation. The definition does not require a loss of consciousness or a blow to the head to diagnose MTBI, a view that was previously held by many neurologists. Another important feature of the definition is that it recognizes that the symptoms of MTBI may not be acknowledged by the patient until the patient attempts to return to normal functioning in which case the evidence for MTBI will have to be reconstructed:

DEFINITION

A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by <u>at least</u> one of the following:

1. any period of loss of consciousness;
2. any loss of memory for events immediately before or after the accident;
3. any alteration in mental state at the time of the accident (e.g., feeling dazed, disoriented, or confused); and
4. focal neurological deficit(s) that may or may not be transient;

but where the severity of the injury does not exceed the following:

- loss of consciousness of approximately 30 minutes or less;
- after 30 minutes, an initial Glasgow Coma Scale (GCS) of 13-15; and
- posttraumatic amnesia (PTA) not greater than 24 hours.

COMMENTS

This definition includes: 1) the head being struck, 2) the head striking an object, and 3) the brain undergoing an acceleration/deceleration movement (i.e., whiplash) without direct external trauma to the head. It excludes stroke, anoxia, tumor, encephalitis, etc. Computed tomography, magnetic resonance imaging, electroencephalogram, or routine neurological evaluations may be normal. Due to the lack of medical emergency, or the realities of certain medical systems, some patients may not have the above factors medically documented in the acute stage.

[14] Kay, T., "Neuropsychological treatment of mild traumatic brain injury" (1993) 8:3 Journal of Head Trauma Rehabilitation 74 - 85.

[15] Kay, *supra* note 4.

In such cases, it is appropriate to consider symptomatology that, when linked to a traumatic head injury, can suggest the existence of a mild traumatic brain injury.

SYMPTOMATOLOGY

The above criteria define the event of a mild traumatic brain injury. Symptoms of brain injury may or may not persist, for varying lengths of time, after such a neurological event. It should be recognized that patients with mild traumatic brain injury can exhibit persistent emotional, cognitive, behaviourial, and physical symptoms, alone or in combination, which may produce a functional disability. These symptoms generally fall into one of the following categories, and are additional evidence that a mild traumatic brain injury has occurred:

1. physical symptoms of brain injury (e.g., nausea, vomiting, dizziness, headache, blurred vision, sleep disturbance, quickness to fatigue, lethargy, or other sensory loss) that cannot be accounted for by peripheral injury or other causes;

2. cognitive deficits (e.g., involving attention, concentration, perception, memory, speech/language, or executive functions) that cannot be completely accounted for by emotional state or other causes; and

3. behavioral change(s) and/or alterations in degree of emotional responsivity (e.g., irritability, quickness to anger, disinhibition, or emotional lability) that cannot be accounted for by a psychological reaction to physical or emotional stress or other causes.

COMMENTS

Some patients may not become aware of, or admit, the extent of their symptoms until they attempt to return to normal functioning. In such cases, the evidence for mild traumatic brain injury must be reconstructed. Mild traumatic brain injury may also be overlooked in the face of more dramatic physical injury (e.g., orthopedic or spinal cord injury). The constellation of symptoms has previously been referred to as minor head injury, postconcussive syndrome, traumatic head syndrome, traumatic cephalgia, post-brain injury syndrome and posttraumatic syndrome.

This ACRM definition has gained widespread acceptance in the field of TBI and is recognized by many neurologists, psychiatrists, physiatrists, and neuropsychologists. In *Reilly v. Lynn,*[16] the British Columbia Court of Appeal affirmed the adoption of this definition by the trial judge. In *S.F.P. v. MacDonald,*[17] the Alberta Queens Bench also adopted this definition. Despite this, some defence neurologists will not accept this definition and will not recognize the Journal of Head Trauma Rehabilitation as an

[16] [1999] B.C.J. No. 2552 (S.C.) aff'd [2002] B.C.J. No. 986 (C.A.) at paras. 273-274 (S.C.). The ACRM definition was also applied in *Lines, supra* note 7 at para. 5 (S.C.).

[17] [1999] A.J. No. 117 at para. 55 (Q.B.).

authoritative source, notwithstanding it is a refereed journal and highly regarded in the field of TBI. It is therefore important to have the definition adopted by one of the plaintiff's experts. The ACRM definition is very similar to Dr. Michael Alexander's definition published in Neurology, a journal that even the most recalcitrant defence expert will have to acknowledge as authoritative:

> Mild TBI is characterized by the following: (1) Head trauma may be due to contact forces or to acceleration/deceleration trauma. (2) The duration of unconsciousness is brief, usually seconds to minutes, and in some cases there is no loss of consciousness (LOC) but simply a brief period of dazed consciousness. (3) When the patient is evaluated in the emergency room or at the scene, the Glasgow Coma Scale (GCS) must be 13 to 15, by common definition. As discussed below, only a score of 13 or 14 is due to confusion or disorientation and will be associated with a longer period of amnesia.[18]

If the expert does not agree with the Alexander definition, it will certainly undermine his or her credibility. At the same time you will be educating the trier of fact.

B. The Center for Disease Control (CDC) definition of MTBI

The Center for Disease Control (CDC) Mild Traumatic Brain Injury Workgroup has published a handbook "Facts for Physicians About Mild Traumatic Brain Injury (MTBI)" which was updated in 2007.[19] This is an excellent summary of the current scientific knowledge in MTBI and should be difficult for defence experts to dismiss. It expands on the ACRM definition and provides a primer for the lawyer to assist in determining whether a case meets the currently accepted diagnostic criteria for MTBI. Their website includes a reference to the Acute Concussion Evaluation (ACE) which is a handy summary of what to look for in determining whether a person has suffered a concussion. Selected references from the handbook are set out below:

> **Definition of Mild Traumatic Brain Injury (MTBI)**
>
> The term mild traumatic brain injury (MTBI) is used interchangeably with the term concussion. A MTBI or concussion is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces secondary to direct or indirect forces to the head. 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 (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 a loss of consciousness (LOC). Duration of symptoms is highly variable and may last from several minutes to days, weeks, months, or even longer in some cases.

[18] Alexander, *supra* note 2 at 1253.

[19] A copy of the handbook can be accessed online at http://www.cdc.gov/ncipc/pub-res/tbi_toolkit/physicians/mtbi/mtbi.pdf.

Neuropathology of MTBI

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.

The ACE provides a convenient checklist on the signs and symptoms to look for in a MTBI:

B. Symptom Check List* Since the injury, has the person experienced any of these symptoms any more than usual today or in the past day?
Indicate presence of each symptom (0=No, 1=Yes). ***Lovell & Collins, 1998 JHTR***

PHYSICAL (10)		COGNITIVE (4)		SLEEP (4)	
Headache	0 1	Feeling mentally foggy	0 1	Drowsiness	0 1
Nausea	0 1	Feeling slowed down	0 1	Sleeping less than usual	0 1 N/A
Vomiting	0 1	Difficulty concentrating	0 1	Sleeping more than usual	0 1 N/A
Balance problems	0 1	Difficulty remembering	0 1	Trouble falling asleep	0 1 N/A
Dizziness	0 1	COGNITIVE Total (0-4) ____		SLEEP Total (0-4) ____	
Visual problems	0 1	EMOTIONAL (4)			
Fatigue	0 1	Irritability	0 1		
Sensitivity to light	0 1	Sadness	0 1		
Sensitivity to noise	0 1	More emotional	0 1		
Numbness/Tingling	0 1	Nervousness	0 1		
PHYSICAL Total (0-10) ____		EMOTIONAL Total (0-4) ____			
(Add Physical, Cognitive, Emotion, Sleep totals) Total Symptom Score (0-22)		____			

Exertion: Do these symptoms worsen with:
Physical Activity __Yes __No __N/A
Cognitive Activity __Yes __No __N/A

Overall Rating: How different is the person acting compared to his/her usual self? (circle)
Normal 0 1 2 3 4 5 6 Very Different

V. The Glasgow Coma Scale (GCS)

Altered consciousness is the most consistent feature of diffuse brain damage resulting from acceleration/deceleration injuries. Neurosurgeons Jennett and Teasdale developed the Glasgow Coma Scale (GCS) as an initial guide to indicate the degree of diffuse brain damage. The GCS generates a score between 3 and 15 based on a person's abilities in eye opening (E), motor response (M), and verbal response (V). It is a quick and easy tool used to assess the severity of TBI in the acute setting or within 48 hours of injury. The GCS (E+V+M) gives a prognosis for survival, not functional outcome.[20]

[20] *Supra* note 8 at 74.

Eye opening (E)

Spontaneous	4
To speech	3
To painful stimulation	2
No response	1

Motor response (M)

Follows commands	6
Makes localized movements to pain	5
Makes withdrawal movements to pain	4
Flexor (decorticate) posturing to pain	3
Extensor (decerebrate) posturing to pain	2
No response	1

Verbal response (V)

Oriented to person, place, and date	5
Converses but is disoriented	4
Says inappropriate words	3
Says incomprehensible sounds	2
No response	1

A GCS score of 13 or higher generally correlates with a mild brain injury, 9 to 12 a moderate brain injury, and 8 or less a severe brain injury. However, a score of 15 does not mean that a brain injury did not occur. The interval between the time of the injury and the time when the GCS is administered is frequently arbitrary, depending on the time it takes for any bystander to call emergency health services, the time for paramedics to arrive at the scene, and the time for paramedics to access and administer treatment to the patient.

The GCS says nothing about focal damage to the brain, only diffuse brain injury. Jennett and Teasdale recognized the limitations at the higher range of the scale. In 1989, Dr. Jennett confirmed that the GCS was never intended to be applied in the assessment of MTBI:

> ... [GCS] was not intended as a means of distinguishing among different types of milder injury. Many of these patients are oriented by the time they are first assessed and therefore score at the top of the Glasgow scale. Yet some of these patients have had a period of altered consciousness, either witnessed or evidenced by their being amnesic for events immediately following injury. Impairment of consciousness is indicative of diffuse brain damage, but there can also be marked local damage without either alteration in consciousness or amnesia...[21] [emphasis added]

[21] Jennett, B., "Some international comparisons" in Levin, H.S., Eisenberg, H.M. & Benton, A.L., eds., *Mild Head Injury* (USA: Oxford University Press, l989) at 24.

VI. Post Traumatic Amnesia (PTA)

Jennett and Teasdale confirmed that it was the duration of post traumatic amnesia (PTA) and not the GCS that was the best "yardstick for assessing severity of head injury."[22] PTA allows the degree of diffuse brain damage to be assessed without any information from witnesses, or from ambulance or hospital records because it depends solely on the recollection of the patient. The authors provided an expanded version of the scale first proposed by Russell in 1946:[23]

Less than 5 minutes	very mild
5 to 60 minutes	mild
1 to 24 hours	moderate
1 to 7 days	severe
1 to 4 weeks	very severe
More than 4 weeks	extremely severe

As Jennett and Teasdale stated in their text:

> Altered consciousness soon after injury is the due to the brain damage already suffered. When first seen in the emergency department it is useful to record whether or not the patient is talking. If he is talking, is he orientated and rational? And if he is, can he remember everything about, and since, the accident? Amnesia for even a few minutes after a blow to the head is evidence of diffuse brain damage.[24] [emphasis added]

VII. The Extended Glasgow Coma Scale (GCS-E)

The definition of MTBI recognizes that an altered state of consciousness can result in MTBI that is not detected by the traditional GCS due to its insensitivity to milder brain damage. The greater sensitivity of PTA as a more reliable measure of MTBI has led to the creation of the Extended Glasgow Coma Scale (GCS-E). The originators of the GCS-E stated:

> A severity index that is more sensitive to the nuances of mild TBI would help resolve the controversy with regard to the sometimes severe consequences to which mild and even very mild brain injuries may give rise. A person with a GCS of 15 on admission or soon thereafter, even if amnesic and hypoaroused [diminished arousal], may be prematurely discharged. Symptoms such as irritability, unreliable memory, and greater fatigue that develop in the days and

[22] *Supra*, note 8 at 90.

[23] *Ibid.*

[24] *Ibid.* at 96.

> weeks after discharge are likely to be attributed to malingering or posttraumatic stress rather than to a concussion syndrome. In developing countries, where there is especially high reliance on the GCS, an admitting score of 14 or 15/15 will often result in a denial of compensation claims, even if the victim is unable to return to employment.[25]

The GCS-E was developed with the support of the World Health Organization Advisory Group on the Prevention and Treatment of Neurotrauma, and has since been adopted as an optional diagnostic variable for the revision of the "Standards for the Surveillance of Neurotrauma". The GCS-E defines 8 levels of PTA and assigns a score that is added to the traditional GCS score. The levels of amnesia are set out in the Amnesia Scale:

Amnesia Scale

Score	
7	No amnesia: client can remember impact, can remember falling and striking a solid surface, etc.
6	Amnesia for 30 minutes or less: client regained consciousness while still in vehicle, in street at scene of incident, in ambulance, or on arrival at hospital.
5	Amnesia of 30 minutes to 3 hours: remembers arriving at emergency room, admission to ward, etc.
4	Amnesia of 3 to 24 hours: determine duration by content of the first memory, which will be for an event in the ward or other hospital procedure.
3	Amnesia of 1 to 7 days.
2	Amnesia of 8 to 30 days.
1	Amnesia of 31 to 90 days.
0	Amnesia greater than 3 months.
X	Cannot be scored, e.g., can speak but responses are inappropriate or unintelligible, cannot speak because unconscious, intubated, facial fractures, etc.

In applying the GCS-E, the GCS is first taken in the usual manner. The Amnesia Scale is then taken and entered after the GCS. For example, if the GCS was 15 and the PTA was 30 minutes then the GCS-E score would be 15:5. The GCS-E recognizes that the duration of amnesia (PTA) is in itself an indicator that a person is not laying down permanent memory and accordingly has suffered an alteration in brain functioning. This information is important in more accurately assessing the degree of brain damage.

The authors of the GCS-E conducted a number of field trials in four hospitals to assess the efficacy of the scale. The GCS-E identified a number of cases that corresponded to

[25] Nell, V., Yates, D.W., & Kruger, J., "An extended Glasgow Coma Scale (GCD-E) with enhanced sensitivity to mild brain injury" (2000) 81 Arch Phys Med Rehab 614-617.

Grade I and II concussions as defined by of the American Academy of Neurology. The authors concluded:

> Flagging these mild cases allows them to be held in the treatment loop until symptoms remit spontaneously, or appropriate early treatment and counseling are given, and reduces the 'cognitive dissonance' between victims of mild TBI and treating professionals. Finally, wide use of the GCS-E would ease the access to compensation for that minority of patients with mild brain injuries who do sustain lasting cognitive-behavioral deficits; this is especially important in developing countries, where the admitting GCS is the gold standard by which the presence or absence of a brain injury is determined.[26] [emphasis added]

VIII. Loss of Consciousness (LOC): Focal and Diffuse TBI

Contrary to the views of some medical experts, the diagnosis of a TBI does not require a loss of consciousness. This was recognized over 15 years ago by the Court in *Chen v. Ruersatt*.[27] The plaintiff, a 59-year-old school custodian, was rear-ended in a motor vehicle accident. He was taken to hospital and released later that night. The diagnosis was a whiplash injury. Prior to the accident, the plaintiff was responsible for the management of the household and was active in physical and recreational activities. After the accident, his lifestyle changed significantly. He no longer had the physical or mental capacity to manage the family finances and household, to maintain employment, to look after rental properties, or to participate in physical and recreational activities. He also became sexually impotent and incontinent. The plaintiff claimed that he suffered from a mild head injury with symptoms including headaches, slowness in movement and cognitive processes, reduced concentration, memory loss, difficulty with balance, vertigo, ringing in his ears, tremor in his right hand and a significant negative personality change (bad tempered and irritable). The Court found that the symptoms were consistent with a head injury notwithstanding an absence of evidence of loss of consciousness:

> There was a great deal of medical evidence given in this case. There was really no dispute that the Plaintiff could have suffered a closed head injury even if there was no loss of consciousness (it is unknown whether the Plaintiff lost consciousness or not - he cannot remember.), and that the symptoms that the Plaintiff is displaying are consistent with such an injury.[28] [emphasis added]

In the case of *Alberta (Workers' Compensation Board) v. Appeals Commission*,[29] Justice Lefsrud of the Alberta Queens's Bench agreed with the reasoning of a Workers'

---

[26] *Ibid.* at 617.

[27] [1993] B.C.J. No. 302 (S.C.), varied [1994] B.C.J. No. 1441 (C.A.).

[28] *Ibid.* at para. 27 (S.C.).

[29] [2003] A.J. No. 1568 at para. 42 (Q.B.).

Compensation Appeals Commission that a TBI can be sustained with no loss of consciousness. These cases are important when facing a medical expert that is out of touch with the brain injury literature.

The most famous and striking example of a severe TBI with no LOC is the classic report of Phineas Gage whose unfortunate injury has been studied by every first-year medical student. Phineas Gage was a foreman of a railroad construction crew in the mid 1800's who sustained a severe frontal lobe injury when an explosive charge propelled an iron bar upward through the lower left side of his face with the point of the bar exiting the top of his skull after passing through the left frontal lobe.[30]

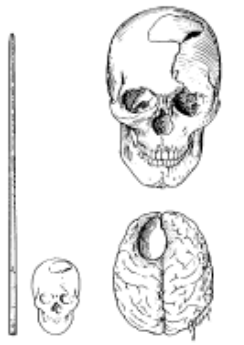

FIG. 8-1. Phineas Gage. On the left is the relationship of the size of the tamping bar to the size and location of the frontal skull lesion. On the right is an enlarged illustration of the skull defect (top) and an artistic rendering of the probable location of brain destruction. (From Stuss and Benson: In: *Neuropsychology of Human Emotion*, edited by Heilman and Satz. Guilford Press, New York, 1983, with permission.)

Phineas Gage never lost consciousness and he was reported to be sitting up and talking shortly after the iron bar went through his left temporal lobe. Physically, Gage made a complete recovery, however, there was a significant change in his personality and emotional behaviour. He went from being a mild-mannered and effective crew supervisor to being an impulsive, aggressive, and unreliable individual who was incapable of working in any capacity. The following description of Phineas Gage after the accident reveals a classic case of orbital frontal lobe injury:

> His physical health is good, and I am inclined to say that he is recovered ... The equilibrium or balance, so to speak, between his intellectual faculty and animal propensities, seems to have been destroyed. He is fitful, irreverent, indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned in turn for others appearing more feasible. A child in his intellectual capacity and manifestations, he has the animal passions of a strong man. Previous to his injury, though untrained in the schools, he possessed a well-balanced mind, and was looked upon by those who knew him as a shrewd, smart businessman, very energetic and persistent in executing all his plans of operation. In this regard his mind was radically changed, so decidedly that his friends and acquaintances said he was 'no longer Gage'.[31]

Focal injuries as occurred in the case of Phineas Gage may be independent of the more diffuse axonal injury (DAI) that leads to a LOC or an alteration in consciousness.

---

[30] Stuss, D.T. & Benson, D.F., *The Frontal Lobes* (New York: Raven Press, 1986) at 121.

[31] Harlow, J.M., "Recovery from the passage of an iron bar through the head" (1868) 2 Publ. Mass. Med. Soc. 327 - 347.

Neurologist, Michael Alexander, wrote in Neurology and Neurosurgery:

> Unlike the patients with primarily DAI [diffuse axonal injury], the severity of a focal injury is not related to LOC and its duration; many patients with severe focal lesions are never unconscious.[32]

Dr. Thomas Kay, the lead contributor to the ACRM definition of MTBI, wrote about the differences between focal and diffuse brain injuries over 20 years ago, well before the clinical use of MRI scans. His comments are still relevant today:

> The alteration of consciousness usually, but not always, involves some brief loss of consciousness. With moderate to severe head injuries, there is a rough correlation between length of coma and severity of injury (as measured by outcome). Within the group of minor head injury, however, when loss of consciousness lasts less than an hour, there is no demonstrable relationship between length of unconsciousness and severity of problems. Significant functional deficits can occur even with transient loss of consciousness.
> …
> It is also possible that significant, long-term deficits can occur in the absence of any documentable loss of consciousness. In such cases the alteration of consciousness may take the form of the patient feeling dazed, confused, or agitated for some period of time, even though consciousness was never lost.
> …
>
> DIFFUSE INJURY
>
> A blow to the head leading to a temporary loss of consciousness is known as a concussion. It used to be thought that concussions were purely transient events, akin to a "short circuiting," with no permanent damage to nerve cells in the brain. It has now been shown that this is not necessarily the case. Using both autopsy studies in humans, and special cell-staining techniques in experiments with animals, it has been demonstrated that even minor blows to the head, leading to only brief loss of consciousness, and apparently complete neurological recovery, can result in stretching and tearing of nerve fibers diffusely (i.e. widely scattered, although not random) throughout the brain. These disruptions of nerve processes can only be seen microscopically.
> In humans, this means that CAT scans and neurological examinations reveal no observable damage to the brain that can be localized to a particular region. Yet there is evidence that the subjective complaints and cognitive problems encountered by some persons after minor head injury may have an organic basis. This is important information because it means that treating such problems as if they were purely psychological in nature will not make the core problems disappear.

[32] Alexander, M., "Neurobehavioural consequences of closed head injury" (1984) 5:20 Neurology and Neurosurgery (Update Series) 1-7 at 5.

The microscopic stretching and tearing occurs because of the mechanical forces transmitted to the brain during trauma. The brain is not a hard, fixed substance. It is soft and custard-like in consistency, composed of millions of fine nerve fibers, and "floats" in cerebral-spinal fluid within the hard, bony skull. When the head is struck suddenly, strikes a stationary object, or is shaken violently, the mechanical force of this motion is transmitted to the brain. Especially when the head has a rotational movement during trauma, the brain mass itself moves, twists, and experiences forces that cause differential movement of brain matter -- much as jello in a shaken bowl will twist and stretch and change its form.

The result of this motion within the brain is that the fine, threadlike nerve cells can become stretched, especially in those areas where rotational forces are likely to produce the most strain. This stretching can temporarily alter the electrochemical functioning of the cells. When the arousal/activating system of the brain is temporarily disrupted in such a manner, consciousness is temporarily lost. The more severe the forces, the longer it will take to regain consciousness.

Most of the nerve cells will eventually return to normal functioning. Many stretched fibers, however, may be permanently damaged, either functioning abnormally, or becoming totally inoperable (if the stretching progresses to tearing). It is the non-functioning of these cells that theoretically provides the organic basis for the deficits experienced after mild diffuse head injury, and where CAT scans and neurological examinations turn up no focal evidence of brain damage. In addition, there is now evidence that the effect of repeated concussions is cumulative. With repeated minor traumas, the severity of the deficits increases, presumably because there is an increase in the number of dysfunctional or non-functional nerve cells.
…

FOCAL INJURY

These focal injuries occur when the soft brain collides with the rough, bony inside surface of the skull during trauma.
…
Especially in acceleration-deceleration injuries such as motor vehicle accidents, where the forward-moving head stops suddenly and strikes a temporary object, the sudden cessation of motion causes the movable brain to continue moving forward and collide with the frontal portions of the hard, bony skull. Because of uneven, rough, ridge-like surfaces in the frontal and basal portions of the inside skull, there is a very high likelihood that contusing (bruising) of the surface of the brain will occur specifically in the frontal and temporal lobes (especially the anterior and basilar regions). Because these particular brain regions are particularly involved in the process of planning, organization, and memory, these cognitive operations are the ones most commonly impaired after focal minor head trauma.

It is important to note that these focal fronto-temporal contusions may be independent of the diffuse injury that leads to unconsciousness (concussion). Patients with concussions may suffer no bruising to the frontal and temporal areas. Conversely, patients may suffer focal contusions without losing consciousness or suffering diffuse injury. Often however, the two types of

> damage occur together, and produce overlapping results; a concussion with temporary loss of consciousness is accompanied by some bruising in fronto-temporal areas.[33]

A clue to a cerebral contusion or focal injury may be found in the report of the behaviour of the patient at the accident scene. Any reference to combative or inappropriate aggressive behaviour even with a GCS of 14 or 15 can indicate a focal injury. Cerebral contusions may not be detected by a standard neurological examination, CT or MRI scans, or standardized intelligence and neuropsychological tests. It is important to retain an expert who is familiar with the pathophysiological mechanism and behavioural correlates of a cerebral contusion as such contusions typically produce significant and often relatively permanent alterations in personality and behaviour.[34]

IX. Whiplash and TBI

Focal injuries and diffuse injuries can result from an acceleration/deceleration movement such as occurs in some whiplash injuries without any direct external trauma to the head. Neuropsychologist Muriel Lezak, in her seminal text *Neuropsychological Assessment* published in 1995 summarizes the literature:

> Bruising can also take place at the moment that rapid deceleration begins or within the first few seconds thereafter as a result of the brain being "slammed" around against the skull's bony protuberances in response to translation forces generated by angular acceleration of the head (J.H. Adams, Graham, and Gennarelli, 1985; D. Pang, 1985, 1989).
> …
> A direct blow to the head is not necessary for this kind of bruising to occur, only rapid deceleration with energy translation to the brain such as occurs when a vehicle comes to a sudden stop (Sweeney, 1992). For example, brain damage can result from a whiplash injury (R.W. Evans, 1992).
> …
> Another neuropsychologically important kind of brain damage that occurs in closed head injury results from the combination of translatory force and rotational acceleration of the brain within the bony structure of the skull (Mendelow and Teasdale, 1984; D. Pang, 1985, 1989). The movement of the brain within the skull puts strains on delicate nerve fibers and blood vessels that can stretch them to the point of shearing (Strich, 1961). Shearing effects, in the form of microscopic lesions that occur throughout the brain (Oppenheimer, 1968) tend to be concentrated in the frontal and temporal lobes (Groswasser, Reider-Groswasser, et al., 1987; Grubb and Coxe, 1978) and the interfaces between gray and white matter around the basal ganglia, periventricular zones, corpus callosum, and brainstem fiber tracts (Mendelow and Teasdale, 1984; D. Pang, 1989).

[33] Kay, *supra* note 9 at 4.

[34] Cummings, J.L., *Clinical Neuropsychiatry* (Florida: Grune & Stratton Inc., 1985) at 57.

> When a moving head comes to a fast stop in an accident, the forward-moving energy (in a motor vehicle) or accelerating energy (in a fall) is translated into rapid acceleration/deceleration expanding and contracting wave-form movements of the brain matter, usually accompanied by the fast rotational propulsion of the brain within the skull. At the neuronal level, this rapid acceleration and deceleration, along with the rotational forces, results in damage to axons in cerebral and brain stem white matter and, in serious injuries, in the cerebellum too (Bostrom and Helander, 1986; R.L. Davis and Robertson, 1985; Gennarelli; Thibault, et al., 1982). This kind of axonal damage, called diffuse axonal injury (DAI), appears as torn axons, shearing of axon clusters, retraction balls consisting of sheared back axonal substance (axoplasm), and reactive swelling of strained and damaged axons. (J.H. Adams, Graham, and Gennarelli, 1985; J.H. Adams, Mitchell et al., 1977; Povlishock and Coburn, 1989).
>
> …
>
> Diffuse axonal injury can occur without any direct impact on the head, as it requires only the condition of rapid acceleration/deceleration such as takes place in whiplash injuries due to acceleration/deceleration forces resulting in rapid flexion-extension movement of the neck (Alves and Jane, 1985; R.W. Evans, 1992; C.M. Fisher, 1982b; Gennarelli, Thibault, et al., 1982; R.S. Parker, 1990; Yarnell and Rossie, 1988).[35] [emphasis added]

In a jury trial in British Columbia Supreme Court 20 years ago, a lawyer was advancing a case of brain injury arising from a whiplash injury. The defence neurologist ran up to the witness box and slammed his fist down on the box and said to the jury, "You can't have a brain injury unless you hit your head!" In the neurologist's file were several articles on concussions including articles by world famous neurosurgeon Thomas Gennarelli. When asked why he had these articles he said he was getting ready for cross-examination. What he didn't have in his file was an article by Gennarelli titled "Mechanisms and Pathophysiology of Cerebral Concussion" published in the Journal of Head Trauma Rehabilitation in which Gennarrelli stated:

> With respect to concussive injuries, injury strains have been proposed to be due to three sources: (1) acceleration of the head, (2) pressure gradients from skull distortion, and (3) stretching of the cervical spine. Of these, only acceleration satisfactorily explains all of the clinical observations.
>
> …
>
> Concussive brain injuries can be viewed as caused by strains induced by head motion. They can occur without impact to the cranium and have little to do with the direct effects of an object that strikes the head, except to the degree that the head impact results in head acceleration or deceleration. Thus, concussions are acceleration-deceleration injuries, and they result from the direction, magnitude, and speed with which the head moves, either from rest or to rest, during the injury sequence. The violent head motions are themselves sufficient to produce strains and distortions within the brain; these result in shearing or stretching of

[35] Lezak, M., *Neuropsychological Assessment*, 3d ed. (USA: Oxford University Press, 1995) at 177-178.

> nerve fibers and the consequent axonal damage that now appears to be the substrate of concussive brain injuries.[36] [emphasis added]

This is an example of how the authoritative literature can be an effective tool in cross-examination to educate the judge or jury while at the same time attacking the credibility of the defence expert.

X. Pathophysiological Mechanism of TBI

Every concussion places unique stress and strain on the brain and no two concussions are identical in terms of how the brain is impacted.[37] Almost 30 years ago, GCS creators, Jennett and Teasdale suggested that the pathophysiological mechanism responsible for an altered state of consciousness is the same for both a concussion (MTBI) or a more severe brain injury:

> Symonds has argued, from a clinical standpoint, that the difference between patients who remain unconscious for days or weeks rather than for minutes or hours could be in the *quantity* of brain damage and not in the *kind* of lesion or its location. He proposed that mild and severe concussion should be recognized; the most obvious pathological counterpart for this would be varying degrees of shearing damage of the white matter. There is some pathological evidence to support this view. Oppenheimer reported microglial stars in patients who had recovered from 'concussion lasting only a few minutes,' but who then died from an unrelated condition.
> …
> Indeed, attention is now shifting away from the brain stem as the site of the lesion responsible for the brief alteration of consciousness implied by the term concussion. An alternative explanation would be shearing lesions of a degree that tear only a few axons, but cause a stretch of many, with subsequent temporary failure of conduction in these nerve fibers. This would provide an explanation for the cumulative effect of repeated mild concussion and would be compatible with the evidence that even mild concussion is associated with structural damage, albeit slight, which leaves its permanent mark in the brain.[38] [emphasis added]

In the cases studied by Oppenheimer, none of the patients suffered anything more than an

---

[36] Gennarelli, T.A., "Mechanisms and Pathophysiology of Cerebral Concussion" (1986) 1:2 Journal of Head Trauma Rehabilitation 23-29 at 25.

[37] Bigler, *supra* note 13 at 13; Viano, D.C., et al., "Concussion in professional football: Brain responses by finite element analysis: Part 9" (2005) 57 Neurosurgery 891–916.

[38] Jennett, *supra* note 20 at 91.

altered state of consciousness or a transient loss of consciousness. Oppenheimer concluded:

> Following a head injury, diffuse microscopic lesions can be seen in a high proportion of human brains. ... They are believed to be mechanical in origin, and can be attributed to:
>
> (1) surface sheering and contusions;
>
> (2) stretching and tearing of small blood vessels;
>
> (3) stretching and tearing of groups of nerve fibres; and
>
> (4) tearing of nerve fibres by a crossing vessel.
>
> They are seen, not only after severe trauma, but also in cases of "concussion". Detailed studies of their sights and distribution could throw light on the mechanics of acceleration injuries of the brain.[39]

Oppenheimer's findings were confirmed in a study published in 1994 in Lancet.[40] The authors were able to examine the brains of five people who suffered a mild concussion (GCS 14 or 15) following motor vehicle accidents. All five then died within 2 to 99 days post injury from other causes. Diffuse axonal injury (DAI), which is microscopic brain damage, was found in all five cases even though none of the patients experienced anything more than an altered state of consciousness.

There is a growing accumulation of data to confirm that enduring pathophysiological effects are associated with MTBI. Recent studies have demonstrated abnormal magnetic resonance spectroscopy (MRS) findings with normal structural imaging. Some loss of brain volume has been demonstrated in MTBI cases with a GCS score of 13 to 15.[41] There are multiple studies where individuals who have suffered a MTBI have had normal structural MRI and CT scan findings but magnetoencephalographic (MEG) abnormalities are significant.[42] Studies using diffusion tensor MRI imaging have shown white matter

---

[39] Oppenheimer, D.R., "Microscopic lesions in the brain following head injury" (1968) 31 Journal of Neurology, Neurosurgery, and Psychiatry 299-306.

[40] Blumbergs, P.C. et al., "Staining of amyloid precursor protein to study axonal damage in mild head injury" (1994) 344 Lancet 1055-56.

[41] Brooks, W.M. et al., "Metabolic and cognitive response to human traumatic brain injury: A quantitative proton magnetic resonance study" (2000) 17 Journal of Neurotrauma 629-640; Garnett, M.R. et al., "Early proton magnetic resonance spectroscopy in normal-appearing brain correlates with outcome in patients following traumatic brain injury" (2000) 123 Brain 2046-2054; Garnett, M.R. et al., "Evidence for cellular damage in normal-appearing white matter correlates with injury severity in patients following traumatic brain injury: A magnetic resonance spectroscopy study" (2000) 123 Brain 1403-1409; Cohen, B.A. et al., "Proton MR spectroscopy and MRI volumetry in mild traumatic brain injury" (2007) 28 American Journal of Neuroradiology 907–913.

[42] Lewine, J.D. et al., "Neuromagnetic assessment of pathophysiologic brain injury induced by minor head trauma" (1999) 20 American Journal of Neuroradiology 857-866.

abnormalities following MTBI.[43] Studies have also confirmed that acute pathological changes in the brain can occur from blows to the head that are below the threshold for producing what would behaviourally be classified as a concussion.[44] These imaging and neuronal injury biomarker studies combined with the post-mortem studies provide indisputable evidence that structural pathology can be present in MTBI. [45]

When structural pathology is not evident, even using the most sophisticated imaging techniques, this does not mean that a brain injury has not occurred. In the words of Dr. Nathan Zasler, co-editor of the recent text *Brain Injury Medicine*:

> Clinicians should remember that gross absence of proof is not necessarily proof of absence. In unsophisticated hands there may be no evidence whatsoever that someone has had a significant injury, whereas in different hands and to other eyes the patient may indeed have objective examination findings clinically as well as neurodiagnostically.[46] [emphasis added]

CT and standard MRI scans depict brain structure and lack the resolution to visualize the microscopic damage which occurs in MTBI cases. However, MTBI produces not only structural injury, but metabolic injury as well.[47] Positron emission tomography (PET) is routinely used clinically in oncology. It is a computerized scanning technique that produces a picture showing the distribution of radioactivity in the brain after the injection of a radioactive isotope. Whereas CT and MRI show a static picture of brain structure, PET reflects brain function by showing metabolic activity in different areas of the brain. It provides an illustration of brain dysfunction by monitoring alterations in the amount of glucose that specific areas of the brain consume.[48] PET has been used to explain why symptoms of MTBI can be present in the absence of any structural damage.

Even if CT and MRI scans do not demonstrate objective findings of a brain injury, and the PET evidence is not admissible, it is important to remember the real issue: whether "but for" the accident, your client would not be experiencing the problematic symptoms.

---

[43] Arfanakis, K.et al., "Diffusion tensor MR imaging in diffuse axonal injury" (2002) 23 American Journal of Neuroradiology 794-802.

[44] Zetterberg, H. et al., "Neurochemical aftermath of amateur boxing" (2006) 63 Archives in Neurology 1277–1280.

[45] Bigler, *supra* note 13 at 7.

[46] Zasler, N.D., "Mild Traumatic Brain Injury: Medical Assessment and Intervention" (1993) 8:3 Journal of Head Trauma Rehabilitation 13-29 at 29.

[47] Collins, M., "In the Midst of a Paradigm Shift: Data-Based Management of Sports-Related Concussion" (2007) 4:4 Brain Injury Professional 8-13 at 9; Giza, C.C. & Hovda, D.A., "The neurometabolic cascade of concussion" (2001) 36 Journal of Athletic Training 228-235.

[48] In *Wolfin v. Shaw* (1998), 43 B.C.L.R. (3d) 190 (S.C.) the court held that PET did not meet the test for novel scientific evidence. A decade later may produce a different result. See Slater, M.J., "Admissibility of PET Scan Evidence" (February, 1999) 79 The Verdict.

Justice MacLeod sums this concept up nicely with respect to MTBI in the recent Alberta case of *Labrecque v. Heimbeckner*:

> The Plaintiff's position is that Sarah suffered a moderate traumatic brain injury with resulting symptoms which persist today. The Defendant's position is that if she suffered a TBI at all, it was of the mild variety and was not something that contributed to her problems on-going six months after the accident.
> …
> [I]t is not surprising that there are differing opinions on the subject because, in the absence of unequivocal objective findings of brain damage, there are many possible explanations for the Plaintiff's behaviour. On the other hand, simply because there are no unequivocal objective signs of brain damages it does not mean that there is not any because it can occur microscopically such that it is not discernable in a C.T. scan or even an M.R.I. Furthermore, while experts in this area are used to dealing with degrees of TBI, including mild, moderate, and severe, these labels only reflect statistical probabilities of recovery and probabilities of long term sequelae. However, not all mild or moderate TBIs are the same. More importantly they do not affect all people the same way. For example, those with existing personality disorders may be more vulnerable. Similarly, those with a drug dependancy may also be more vulnerable because a TBI may make it more difficult for them to deal with their problem. Accordingly, to assess damages in this case I do not find it necessary to decide whether Sarah suffered a mild TBI or a moderate TBI. The important issue is the extent to which, if any, her existing and future disability is contributed to by the accident as opposed to pre-existing factors.[49] [emphasis added]

XI. Recovery from MTBI

In her 1995 text, Muriel Lezak suggested that MTBI was likely to leave some residual deficits:

> I no longer use the term "recovery" when discussing brain damage. Brain damage that is severe enough to alter the level of consciousness even momentarily, or to result in even transient impairment of sensory, motor, or cognitive functions, is likely to leave some residual deficits.[50]

In a review of the literature,[51] Erin Bigler points to several studies[52] demonstrating that persons with a history of MTBI from which they had supposedly clinically "recovered"

[49] [2007] A.J. No. 1462 at paras. 102, 113 (Q.B.).

[50] Lezak, *supra* note 35 at 175.

[51] Bigler, E.D., "Neuropsychological results and neuropathological findings at autopsy in case of mild traumatic brain injury" (2004) 10 Journal of International Neuropsychological Society 794-806.

developed dementia years later. According to Bigler, "these studies support the presence of a permanent neuropathologic basis to mild TBI, even though clinical "recovery" has occurred."[53]

Just last month the Boston University School of Medicine found that evidence of chronic traumatic encephalopathy (CTE), a degenerative brain disease caused by brain injury, was found in the brain of a deceased 18 year old male who had previously suffered multiple concussions. This finding is consistent with findings of CTE in six former National Football League (NFL) players who also experienced concussions,[54] and findings that some athletes who sustained a MTBI demonstrated adverse effects over 30 years later even when they appeared asymptomatic to friends and family.[55]

The groups at the highest risk for MTBI are infants and children (ages 0-4), children and young adults (ages 5 to 24), and older adults (ages 75 or older).[56] Recovery time from MTBI may be longer for children and adolescents.[57] With respect to gender differences in MTBI, women have an increased likelihood of developing postconcussional syndrome, have more severe symptoms, and take longer to recover.[58]

---

[52] Guo, Z. et al., "Head injury and the risk of AD in the MIRAGE study" (2000) 54 Neurology 1316-1323; O'Meara, E.S. et al., "Head injury and risk of Alzheimer's disease by Apolipoprotein E genotype" (1997) 146 American Journal of Epidemiology 373-384; Plassman, B.L. et al., "Documented head injury in early adulthood and risk of Alzheimer's disease and other dementias" (2000) 55 Neurology 1158-1166; Schofield, P.W. et al., "Alzheimer's disease after remote head injury: An incidence study" (1997) 62 Journal of Neurology, Neurosurgery, and Psychiatry 119-124.

[53] Bigler, *supra* note 51 at 795.

[54] A summary of the findings can be found at http://www.bumc.bu.edu/busm-news/2009/01/28/new-evidence-links-head-trauma-brain-disease-in-football-players/.

[55] De Beamount, L. et al., "Brain function decline in healthy retired athletes who sustained their last sports concussion in early adulthood." Online: (2009) Brain <http://brain.oxfordjournals.org/cgi/reprint/awn347v1>.

[56] Bazarian J. et al., "Mild traumatic brain injury in the United States, 1998-2000" (2005) 19:2 Brain Injury 85-91.

[57] Field, M. et al., "Does age play a role in recovery from sports-related concussion? A comparison of high school and collegiate athletes" (2003) 142:5 The Journal of Pediatrics 546-553; Pellman, E.J. et al., "Concussion in professional football: recovery of NFL and high school athletes assessed by computerized neuropsychological testing - Part 12" (2006) 58:2 Neurosurgery 263-74.

[58] Hall, R.C. & Chapman, M.J., "Definition, diagnosis, and forensic implications of postconcussional syndrome" (2005) 46 Psychosomatics 195–202. Bazarian J.J. et al., "Epidemiology and predictors of post-concussive syndrome after minor head injury in an emergency population" (1999) 13 Brain Injury 173–189; Corrigan J.D. et al., "Early identification of mild traumatic brain injury in female victims of domestic violence" (2003) 188 (May suppl) American Journal of Obstetrics & Gynecology S71–S76. Farace E. & Alves W.M., "Do women fare worse: a meta-analysis of gender differences in traumatic brain injury outcome" (2000) 93 Journal of Neurosurgery 539–545.

A. The "Miserable Minority"

Defence experts will invariably point to articles stating that the majority of MTBI patients will fully recover within 6-12 months of the injury.[59] However, as previously stated, there are a growing number of studies indicating that not only can damage from MTBI be permanent, but it can lead to progressive degenerative changes.[60] For example, TBI has been described as the most robust environmental Alzheimer's disease risk factor in the general population.[61]

While there is a direct relationship between the severity of concussion and the likelihood of symptoms lasting more than three months,[62] concussion severity alone is a poor predictor of who will experience long term symptoms.[63] The 10 to 20 percent of persons, the "miserable minority",[64] who never completely recover[65] are left with one or more physical symptoms, cognitive deficits, behavioral changes, or alterations in degree of emotional responsivity.[66] In an article in the Journal of Head Trauma Rehabilitation, Professors Dikmen and Levin state:

> Not all patients with mild head injury complain of posttraumatic symptoms, and most improve without further intervention. In a fraction of the cases, however, the postconcussion symptoms do persist and may evolve into the so-called postconcussional syndrome. Owing to the high incidence of mild head injuries

[59] Carroll, L.J. et al., "Prognosis for Mild Traumatic Brain Injury: Results of the WHO collaborating centre task force on mild traumatic brain injury" (2004) 43 Journal of Rehabilitation Medicine 84-105.

[60] MacKenzie, J.D. et al., "Brain atrophy in mild or moderate traumatic brain injury: A longitudinal quantitative analysis" (2002) 23 American Journal of Neuroradiology 1509-1515; Guo, *supra* note 52; O'Meara, *supra* note 52; Plassman, *supra* note 52; Schofield, *supra* note 52.

[61] Guo, *ibid.*; Plassman, *ibid.*

[62] Hessen, E., Nestvold, K., & Sundet K., "Neuropsychological function in a group of patients 25 years after sustaining minor head injuries as children and adolescents" (2006) 47 Scandinavian Journal of Psychology 245–251.

[63] Guskiewicz, K.M. et al., "National Athletic Trainers' Association Position Statement: Management of Sport-Related Concussion" (2004) 39 Journal of Athletic Training 280–297.

[64] Ruff, R., Camenzuli, L., & Mueller, J., "Miserable minority: emotional risk factors that influence the outcome of a mild traumatic brain injury" (1996) 10:8 Brain Injury 551-565; Ruff, *supra* note 1.

[65] Ruff, *supra* note 1; Alexander, *supra* note 2 at 1256. For a discussion of the long term affects of MTBI see McAllister, T., "Mild brain injury and the postconcussion syndrome" in Silver, M., McAllister, T. & Yudofsky, M., eds., *Textbook of Traumatic Brain Injury* (Washington D.C.: American Psychiatric Publishing Inc., 2005) at 281.

[66] Kay, *supra* note 4.

> this fraction of cases translates into a sizable group of patients, who may be significantly disabled in resuming their preinjury lifestyle.[67] [emphasis added]

This "sizable group of patients" has not changed dramatically over the last 25 years. What has changed is the recognition by the medical profession, and the courts, of the potentially debilitating effects of MTBI.

B. The "thin skull" or "eggshell personality"

Members of the "miserable minority" may fall into the category of the "thin skull" or "eggshell personality" case. These individuals will have suffered long-term effects of a MTBI not because they are all malingerers or are looking to capitalize on secondary gains, but because they have a greater susceptibility (physical and/or psychological) to this type of injury. This susceptibility can arise as a result of prior concussive injuries from which the individual made what appeared to be an uneventful recovery, or as a result of a particular personality type rendering that individual more vulnerable. Pre-morbid factors predispose those with a history of neuropsychiatric disorder to be more likely to experience long term effects of MTBI.[68] In terms of compensation, the appropriate question should be whether the physical and/or emotional consequences suffered by the individual are genuine and whether they arise as a result of the accident.

The lawyer should be aware of some risk factors that can complicate the recovery process. Empirical research has identified that the following factors have be associated with a longer period of recovery from a MTBI:

- History of MTBI[69]
- Headache History[70]
- Developmental History[71]
- Psychiatric History (such as history of depression/mood disorder, anxiety, and/or sleep disorder)[72]

---

[67] Dikmen, S.S. & Levin, H.S., "Methodological issues in the study of mild head injury" (1993) 8:3 Journal of Head Trauma Rehabilitation 30-37 at 31.

[68] Ponsford, J., "Rehabilitation interventions after mild head injury" (2005) 18 Current Opinions in Neurology 692– 697.

[69] Guskiewicz, K. et al., "Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study" (2003) 290:19 JAMA 2549-55; Collins, M.W. et al., "Cumulative effects of concussion in high school athletes" (2002) 51 Neurosurgery 1175-181; Iverson, G. et al., "Cumulative effects of concussion in amateur athletes" (2004) 18:5 Brain Injury 433-43.

[70] Mihalik, J. et al., "Posttraumatic migraine characteristics in athletes following sports-related concussion" (2005) 102:5 *Journal of Neurosurgery* 850-55; Collins, M.W. et al., "Headache following sports-related concussion: To play or not to play" (2003) 31:2 American Journal of Sports Medicine 168-173; deKruijk, J. et al., "Prediction of post-traumatic complaints after mild traumatic brain injury: Early symptoms and biochemical markers" (2002) 73:6 Journal of Neurology, Neurosurgery, and Psychiatry 727-32.

[71] Collins, M.W. et al., "Relationship between concussion and neuropsychological performance in college football players" (1999) 282:10 JAMA 964-970.

A tortfeasor who injures another must accept the risk that his victim suffers from a frail skull or an unusual susceptibility to injury. The thin skull situation arises where greater harm was caused because of the susceptibility to trauma. Accordingly, a victim of MTBI who does not recover as quickly as one might expect or who suffers a more significant disability due to a prior concussive injury, is entitled to recover damages to the full extent of the injuries.

The thin skull rule was first enunciated by Lord Justice Kennedy in *Dulieu v. White & Sons*,[73] in the following statement:

> If a man is negligently run over or otherwise negligently injured in his body, it is no answer to the sufferer's claim for damage that he would have suffered less injury, or no injury at all, if he had not had an unusually thin skull or an unusually weak heart. [74]

The authorities are clear that the thin skull rule applies to emotional as well as physical susceptibility, although there appears to be a need to differentiate between pre-accident susceptibility and post-injury mental attitude.[75] Physical injury which triggers personality change is compensable.[76] In his text, *Canadian Tort Law*, Linden canvasses a number of cases where the courts have awarded full compensation for the "vulnerable personality".[77] Linden quoted Justice Lane in the case *Malcom v. Broadhurst*:

> ...there is no difference in principle between an eggshell skull and an eggshell personality... Exacerbation of her nervous depression was a readily foreseeable consequence of injuring her... Once damage of a particular kind, in this case psychological, can be foreseen, ... the fact that it arises or is continued by reason of an unusual complex of events does not avail the defendant. [78]

---

[72] Moore, E.L., Terryberry-Spohr, L. & Hope D.A., "Mild traumatic brain injury and anxiety sequelae: a review of the literature" (2006) 20:2 Brain Injury 117-32; Mooney, G., Speed, J. & Sheppard, S., "Factors related to recovery after mild traumatic brain injury" (2005) 19:12 Brain Injury 975-87; Mather, F.J., Tate R.L., & Hannan, T.J., "Post-traumatic stress disorder in children following road traffic accidents: A comparison of those with and without mild traumatic brain injury" (2003) 17:12 Brain Injury 1077-87.

[73] [1901] 2 K.B. 669.

[74] *Ibid.* at 679.

[75] Cooper-Stephenson, K., *Personal Injury Damages in Canada* (Toronto: Carswell, 1996) at 856; *Gray v. Gill*, [1993] B.C.J. No. 2389 (S.C.).

[76] See *Kovach v. Smith*, [1972] 4 W.W.R. 677 at 685 (B.C.S.C.) where the plaintiff prior to the accident was happy, healthy and employed. He recovered from his physical injuries but developed a serious paranoid illness resulting in a change in his personality. He was unable to work and became difficult to live with and at times became violent towards his family. Justice McIntyre held that the plaintiff had a predisposition to emotional reaction and applied the thin skull rule in awarding full compensation.

[77] Linden, A.M. & Feldthusen, B., *Canadian Tort Law*, 8th ed. (Canada: LexisNexis, 2006) at 380-390.

[78] *Ibid.* at 388 citing *Malcom v. Broadhurst*, [1970] 3 All E.R. 508 at 511.

C. Individual vulnerability

Individuals may be differentially susceptible to identical trauma to the brain for a variety of reasons. Every brain and every personality is unique. The concept of "individual vulnerability" was introduced by Dr. Kay to explain the persistence of symptoms in a significant minority of MTBI cases:

> The concept of "individual vulnerability" suggests that a large number of variables will influence how the injury will affect the person, and that each person has a given level of "vulnerability" on each of these dimensions. We know least about neurologic vulnerability. Individual differences in brain structure, hormonal and neurotransmitter balances, and other biologic systems may make one brain more susceptible to, say, an excitotoxic cascade than another brain. Other factors such as age, drug or alcohol abuse, or prior central nervous system (CNS) damage may also increase neurologic vulnerability, magnifying the functional effect of loss of a relatively small number of nerve cells. In addition, a wide variety of psychosocial and personality variables, including family dynamics, type of work, and many more, help determine how each individual person will uniquely react to the trauma of an accident, the presence of symptoms, and the persistence of subtle but real changes in cognitive capacity. (2)(3)(22). The interaction of these neurologic and psychological variables determines an individual vulnerability for each person who suffers a concussion and helps account for the inconsistency in outcomes after apparently similar neurologic events.
>
> …
>
> Failure to medically diagnose mild TBI and anticipate the cognitive and behavioral sequelae exacerbates the psychological deterioration of the person. When a person with a genuine mild TBI suddenly finds him - or herself forgetting things, making errors, and taking longer and requiring more effort to do things that used to be automatic; when the person starts becoming disorganized, irritable, and getting into conflicts with friends, co-workers, and family; and when he or she is told by professionals that there is nothing wrong, that he or she should get on with life, then nothing exists to validate the experience that something is wrong, and the sense of self begins to erode. If subsequent medical follow-up fails to provide quick and useful diagnostic feedback on the post-concussive state, the person is in danger of spiraling downward into failure, frustration, fear, avoidance, and loss of confidence and self esteem, and ultimately the person feels like he or she is "going crazy". If this psychological deterioration continues unabated, it can become more debilitating than the primary, neurologic deficits that fuel it.
>
> …
>
> No variable is more complex and important in understanding functional disability after mild TBI than personality. The situation is most clear in the extremes. A history of well-adjusted personality functioning in a flexible individual who has shown the ability to deal well with stress makes more credible the conclusion that true organic damage has occurred when there is a sudden and dramatic drop in ability to function after mild TBI.
>
> …
>
> While there is no established taxonomy of personality styles at risk for a dysfunctional response to mild TBI, five examples can be cited from our clinical

experience. First, we have noted that persons who are **highly driven - often obsessive-compulsive - overachievers** whose sense of self is tightly bound up with intellectual pursuit and achievement, are greatly at risk for a catastrophic breakdown of the self after a mild TBI in which real cognitive problems persist. Often these persons have histories of success and achievement, but lack an inner self esteem. The "cognitive slippage" from a mild TBI is sufficient to "knock out" the high-performance mental engine that propelled them with sufficient speed to keep them emotionally afloat; the effects of a neurologically real mild TBI are psychologically devastating because they steal away the only source of self-esteem. The subtle neurologic event unmasks the underlying lack of sense of self. It is a mistake to write such persons off as having purely psychological problems; their psychological devastation must be seen as a secondary reaction to a primary neurologic event. They are not imagining or creating their problems, although their personalities do magnify the impact of the problems.

Second, we have noted that **persons who suffered emotional deprivation as children,** when they are injured in ways that retribution cannot be extracted (as is often the case in mild TBI) may become extremely hostile and dysfunctional in the presence of permanent symptoms.

…

Third, **persons with strong tendencies toward dependency** are often immobilized by the symptoms of mild TBI, especially in the acute state, and respond by decreasing activity and increasing their anxiety about their inability to function. Even when symptoms actually resolve over time, the self-paralysis dynamic, set in motion early on, can take on a life of its own and continue to render the dependent person dysfunctional long after the primary sources of deficit have receded. Besides arguing strongly for an early interventional model (to counteract the self-isolating tendencies of the dependent person), such a possibility renders it imperative for the evaluator to weigh the contribution of a dependent personality style when determining the causes of functional disability.

Fourth, the term **"borderline" personality** has been widened beyond its strict diagnostic criteria to include a group of high functioning, often very intelligent persons, who appear to have high levels of emotional rigidity and impaired capacity for deep human relationships, and often manifest some "borderline" characteristics in a mild form, such as affective instability, intense anger, feelings of emptiness, and fear of rejection. Such persons, especially when they have poor social support systems, also do quite poorly after mild TBI when real neuropsychological deficits persist. Their coping threshold seems to have been exceeded by the increase in symptoms, and they become overwhelmed by the difficulties they face. Again, the identification of such a personality dynamic should *not* be the cause of negating the impact of the neurologic event; it simply means that in understanding the level of dysfunction and planning a program of rehabilitation, these personality factors need to be taken into account.

Fifth, a different set of problems is presented by the **person with tendencies toward grandiosity,** inflated self-belief, and other elements of a narcissistic personality style. Such persons often minimize, deny, or hide the difficulties they are having, to the extent that their life must crumble around them before they will acknowledge to others the difficulty they are having. Unfortunately,

> when the reality finally hits, the narcissistic wound can be enormous and devastating, presenting a unique therapeutic challenge.
>
> Again, these personality factors may not be in evidence during cursory history-taking, nor do they mean that real, organically based deficits do not exist. They do, however, complicate the neuropsychological diagnosis and present additional challenges to the process of rehabilitation.[79]

D. Prior traumatic brain injury

The cumulative deleterious effects of concussion have been recognized since the research by Gronwall and Wrightson published in 1975.[80] Dr. James Kelly, in a review of the literature, confirmed the effects of repeated cerebral concussions:

> ... repeated concussions that are spaced near in time to each other can lead to catastrophic neurologic injury. This has been reported in the literature as the "second impact syndrome," which is the development of brain swelling after a second concussion while an individual is still symptomatic from an earlier concussion.
>
> ... even repeated concussions spaced distant in time from each other can impart cumulative neurological damage reflected in documented neuropsychological decline in mental performance, atrophy on repeated neuroimaging studies, and the development of dementia (global intellectual decline) with Parkinsonian features first noted in boxers and termed *dementia pugilistica*.[81]

In MTBI, the fine thread-like nerve cells become stretched and either cease to function or function abnormally. It is the malfunction of these cells that provides the organic basis for the deficits experienced after MTBI. Repeated trauma increases the severity of the deficits. This was recognized by Dr. Kay over 20 years ago: [82]

> …there is now evidence that the effect of repeated concussions is cumulative. With repeated minor traumas, the severity of the deficits increases, presumably because there is an increase in the number of dysfunctional or non-functional nerve cells... even after "complete recovery," the occurrence of additional minor head traumas (especially common in sports) may eventually produce noticeable deficits, even though none is any worse than the first - implying that the initial "complete recovery" was really a decrement in nervous system integrity too

[79] Kay, T., "Neuropsychological Diagnosis: Disentangling the multiple determinants of functional disability after mild traumatic brain injury" in Horn, I.J. & Zasler, eds., *Rehabilitation of Post-Concussive Disorders* (Philadelphia: Hanley & Belfus, 1992) at 113.

[80] Gronwall, D. & Wrightson, P., "Cumulative effect of concussion" (1975) 2 Lancet 995-997.

[81] Kelly, J.P., "Traumatic brain injury in sports: are the risks appreciated?" (1995) Current Therapy in Sports Medicine 21-24.

[82] Kay, *supra* note 9 at 3.

> small to notice behaviorally... many persons who later in life develop significant emotional, interpersonal, or behavioral problems, are found to have, upon careful interviewing, a history of minor head trauma at some time in their lives.[83] [emphasis added]

Dr. Lezak is of the view that each brain injury has an exponential effect:

> Repeated head injuries tend to have a cumulative effect on cognition as a second, even mild concussion, leaves the victim somewhat more compromised than if this had been the sole injury (Gronwall, 1989b, 1991; with Wrightson, 1975). Moreover, a single traumatic injury to the brain doubles the risk for a future head injury, and two such injuries raises the risk eightfold (Gaultieri and Cox, 1991).[84] [emphasis added]

This vulnerability to subsequent brain injury is the subject of more recent comment by noted neurosurgeon Thomas Gennarelli in the chapter "Neuropathology" published in the 2005 *Textbook of Traumatic Brain Injury*:

> Given that some structural damage is likely in all forms of TBI, an important determinant of outcome is the preinjury condition of the brain. In other words, a good recovery is more likely in a healthy individual with no pre-existing brain disorders who experiences TBI than in an individual with a similar level of injury who, either because of pre-existing developmental or acquired disorders, had abnormal brain function before injury. The outcome, even after relatively mild brain injury, in an individual who has already experienced cerebrovascular disease or brain injury is likely to be worse than if such premorbid conditions were not present.[85] [emphasis added]

It is important to know whether a client has fully recovered from a prior concussion or MTBI. If not fully recovered from a prior brain injury, a person is more susceptible to future brain injuries. This is because it takes less force to injure a brain that has not fully recovered.[86] Similar to when a person sprains their ankle, if enough time is not given for the ankle to fully heal, it will be more susceptible to re-injury. A fall that would normally result in no injury (or a very minor strain) in a healthy ankle can result in significant injury in an ankle that has not fully recovered from a prior sprain. Brain injuries should be viewed the same way.

If your client has a history in sports where concussions may have resulted, this should be brought to the attention of the trier of fact. Guskiewicz found that football players with a

---

[83] *Ibid*.

[84] Lezak, *supra* note 1 at 188.

[85] Gennarelli, T. & Graham, D., "Neuropathology" in Silver, M., McAllister, T. & Yudofsky, M., eds., *Textbook of Traumatic Brain Injury* (Washington D.C.: American Psychiatric Publishing Inc., 2005) at 27.

[86] Collins, *supra* note 47.

history of concussion were more likely to have future concussions, and prior concussions may be associated with a prolonged recovery of neurological function.[87]

As previously mentioned, repeated trauma to the head can also cause CTE, a neurodegenerative disease characterized by the buildup of a toxic protein throughout the brain. The protein impairs normal functioning of the brain and eventually kills brain cells.[88] The presence of CTE can only be detected in subjects upon death, but the research clearly shows its presence in individuals who experienced multiple concussions.

The Acute Concussion Evaluation (ACE) for physicians lists prior concussions as a risk factor for protracted recovery and must be canvassed by both the lawyer and the medical professional. If in doubt, previous medical records should be reviewed as there may be evidence that would justify the diagnosis of a prior MTBI under the criteria established by the ACRM or CDC definitions. The prior concussion may explain to the judge or jury why someone falls into the "miserable minority" group of MTBI victims who go on to have long lasting problems.

XII. Significance of Collateral Information

In many MTBI cases, the GCS is 15 and the medical records may not show any loss of consciousness or any indication of PTA. In *Neurological Assessment*, Muriel Lezak states:

> ...it is not uncommon to find no notes reporting altered mental status in the emergency room record or hospital chart, even when the patient is later observed to suffer from fairly debilitating mental dysfunction.[89]

For this reason, it is important to use sources beyond medical records to demonstrate the condition of your client. One of these methods is neuropsychological assessment, however this type of assessment has its limitations and should not solely be relied on. If the client was functioning at a high level before the accident, the neuropsychological assessment may not demonstrate any deficits. It is for this reason that Varney and Menafee suggest that the best information of changes in cognitive, emotional, and behavioural functioning will come from collateral witnesses who knew the patient before and after the traumatic event:

> Patients with TBI, particularly when mild, may perform normally on a wide variety of neuropsychological measures and may appear relatively normal within the structure of standard psychological interviews. At the same time, they are often substantially impaired in independent, self-determined "adult" behaviours and activities of daily living. Thus, there has been increasing recognition of the

[87] Guskiewicz, *supra* note 59.

[88] *Supra* note 54.

[89] Lezak, *supra* note 1 at 172.

> importance of obtaining information from collateral informants (e.g., parents, spouses, siblings, coworkers) who are familiar with the patient. Collaterals are capable of elaborating on traditional cognitive deficits (e.g., reliability of memory) and are also qualified to assess the social, behavioral, and interpersonal changes experienced by an individual as a result of a head injury because they interact with the patient on a day-to-day basis. Information obtained from collaterals may often differ markedly from information given by the patient. Patients with TBI may provide inaccurate histories, overreport or underreport symptomatology, and lack insight concerning their behaviour and its effect on others in their environment. Because these individuals are likely to fall within normal ranges on traditional batteries of neuropsychological tests and may appear normal during a psychological interview, psychosocial symptoms (which often render the individual ineffective in daily functioning) may be overlooked by the most astute observer without collateral information.[90] [emphasis added]

More recent statements of the value of collateral information from reliable sources can be found in the 2005 *Textbook of Traumatic Brain Injury*.[91] One of the major reasons for using collateral information is due to the inadequacy of the neuropsychological assessment. These assessments are almost never conducted in real world settings, therefore the ecological validity (how the assessment reflects abilities of the patient in real world scenarios) of neuropsychological assessments should be a concern. These limitations are well documented in the literature. Bigler states:

> … cognitive skills, in particular working memory and executive function, can place much higher demands on neural integrity in the real world than what can be assessed by any current clinical neuropsychological technique in the laboratory.[92]

In many cases, unless a significant cognitive demand is placed on the subject that requires more than typical cognitive effort, no difference between pre- and post-accident ability will be determined.[93] Therefore, it is particularly important to obtain collateral information for patients who appear to be functioning normally at home, but are experiencing fatigue or frustration from tasks assigned in the workplace.

Standardized neurological tests are unable to detect neurobehavioural problems such as irritability, rapid mood swings, poor safety judgment, problems with emotional and behavioural regulation, loss of curiosity, impulsivity, disinhibited speech or behaviour,

---

[90] Varney, N.R & Menefee, M.S., "Psychosocial and executive deficits following closed head injury: Implications for orbital frontal cortex" (1993) 8:1 Journal of Head Trauma Rehabilitation 32-44 at 33.

[91] Arlinghaus, K, Shoaib, A, & Price, T., "Neuropsychiatric Assessment" in Silver, M., McAllister, T. & Yudofsky, M., eds., *Textbook of Traumatic Brain Injury* (Washington D.C.: American Psychiatric Publishing Inc., 2005) at 60; McAllister, *supra* note 63 at 300.

[92] Bigler, *supra* note 13 at 12.

[93] Bigler, *ibid.* at 8; Chen, S.H. et al., "A study of persistent post-concussion symptoms in mild head trauma using positron emission tomography" (2003) 74 Journal of Neurology, Neurosurgery, and Psychiatry 326–332.

use of crude of coarse language, circumstantial and tangential thinking, poor frustration tolerance, inability to feel compassionate or show affection toward others, loss of libido, disinhibited social behaviour, rapid fatigue, viscosity, emotional lability, apathy, inappropriate social behaviour, egocentricity, poor judgment, anhedonia (inability to experience pleasure), inability to plan for future events, reduced responsiveness to environmental cues, hyperverbosity, or hypersomnia.[94]

If collateral witnesses tell you that your client has, following a traumatic event, undergone a dramatic change in their personality, is unable to control their behaviour or regulate their emotion, has less social tact, poor impulse control, an inability to empathize with others, marked egocentricity, frequent use of crude and coarse language, exhibits inappropriate social behaviour, has poor frustration tolerance, rapid mood swings, poor judgment, and has little or no awareness of how their neurobehavioural problems affect others, loss of motivation, loss of energy and drive, loss of pleasure from the environment, pseudodepression, psychomotor retardation, changes in eating habits, loss of sex drive, loss of curiosity, and obsessive-compulsive behaviour, then these are red flags for frontal lobe damage.[95] Another problem with neuropsychological assessments is that even with frontal lobe damage, the individuals will often perform in the normal range in standardized neuropsychological tests.[96]

The neuropsychological assessment is performed in a highly structured setting and it has often been said that the neuropsychologist replaces the frontal lobes during the testing. Given the deficiencies and insensitivity of the neuropsychological assessment, the courts recognize and place significant weight on the evidence of collateral witnesses who confirm changes in the functioning of the plaintiff following a traumatic event.[97] In *Warder v. Insurance Corp. of British Columbia*,[98] Justice Bouck found lay evidence very compelling in deciding whether the plaintiff suffered a MTBI, particularly when such evidence was contrasted with evidence from the expert witnesses who only examined the

---

[94] Sbordone, R., "Critical issues that arise when neuropsychologists assess individuals who have sustained traumatic brain damage" (Paper presented to the Trial Lawyers Association of British Columbia, 28-29 March 2008) at 19; Sbordone, R., "Ecological validity issues that arise in medi-legal cases when neuropsychologists are asked to assess patients with traumatic brain injuries" in Handbook of Forensic Neuropsychology (New York, New York: Springer Press, 2009 in press).

[95] Sbordone (2008), *ibid.* at 23.

[96] Bigler, E.D., "Frontal lobe damage and neuropsychological assessment" (1988) 3 Archives of Clinical Neuropsychology 279-297; Damasio, A.R., "The Frontal Lobes" in Heilman, K.M. & Valenstein, E., eds., *Clinical Neuropsychology*, 2d ed. (New York: Oxford Press, 1985) 409-460; Mesulam, M.M., "Frontal Cortex and Behaviour: Editorial" (1986) 19 Annals of Neurology 320-325; Zangwill, O.L., "Psychological deficits associated with frontal lobe lesions" (1986) 5 International Journal of Neurology 395-402.

[97] *Warder v. Insurance Corp. of British Columbia*, [1993] B.C.J. No. 644 (S.C.); *Hosseini-Nejad v. Roy*, [1998] B.C.J. No. 3038 (S.C), affirmed [2000] B.C.J. No. 1291 (C.A.); *Datta v. Rowan*, [1993] B.C.J. No. 1683 (S.C.); and *Brown v. Lalani*, [2005] B.C.J. No. 1225 (S.C.).

[98] *Ibid.*

plaintiff after the accident and therefore had little information of what the plaintiff was like before. Justice Bouck stated:

> 15 Except for item 7, almost all of these complaints cleared up within two years of the accident. But, as counsel for the plaintiff put it in her excellent written briefs, he has still not regained his "spark." He cannot function anywhere near the same level as he did before 1 March 1989. His memory is bad, he has problems expressing himself on paper, he cannot initiate projects in the same way he did before the accident, he is not as good a teacher as he used to be, he is a less effective politician, he is not as good a husband and father, and he has lost much of his self-esteem. All of this has seriously affected his enjoyment of life.
>
> 16 Medical experts differ on a diagnosis. Since Mr. Warder had no significant period of unconsciousness, defence experts say that is an indication he did not suffer any organic brain damage. On the other hand, neuro-psychological testing tends to support the allegation of organic brain damage. Summarizing all of the evidence on this issue, I am satisfied he suffered a minor degree of brain damage that is interacting with his depressed psychological state. He continues to be disabled.
>
> 17 Almost all the specialists agree that psychological therapy over a period of six to eight months should help him improve from his present state. Few are predicting an immediate recovery. What struck me about the severity of his condition was the evidence coming from people who knew him well before the accident and then saw the significant change in his performance after the accident. This kind of evidence is very compelling when compared to evidence from others who only examined him after the accident and had little personal knowledge of him before that time. [emphasis added]

It is important to obtain a list of all the family members, friends, co-workers, and supervisors who can attest to physical, cognitive, emotional, and behavioural changes. These witnesses must be interviewed so you can select which witnesses will present the most compelling story of what your client was like before and after the traumatic event. Your neuropsychologist should interview one or more of the collateral witnesses so that this information can be incorporated into the report. Alternatively, this information can be provided to the expert with instructions to assume that the collateral information is true. Even in cases where there is a GCS of 15, no LOC and minimal PTA, if there is sufficient credible collateral evidence on which the trier of fact can base their decision, a finding of a MTBI with lasting effects on your client may be found.

In *Lines v. Gordon,*[99] the plaintiff suffered a blow to the head and experienced PTA as a result of a MVA. The defence neurologist stated in her report that the symptoms the plaintiff was experiencing were entirely due to the medication he was taking, and in no way could be caused by a brain injury. Even though the defence expert's report was not relied on, and it was clear that the judge believed the experts of the plaintiff who said that

[99] *Supra* note 7.

his symptoms were consistent with a TBI, a large portion of the judgment was spent considering the testimony of collateral witnesses. Justice Lander of the British Columbia Supreme Court stated:

> These "lay" witnesses painted a broad picture of Mr. Lines' pre-accident and post-accident functioning which reveals a very different person after December 9, 2001. The evidence reveals that it has been what might be considered almost a 180 degree alteration in this man's behaviour patterns and, most importantly, as to his ability to function sufficiently well day to day in this society and to be competitively employed.[100]

In relying heavily on the collateral information of lay witnesses, Justice Lander awarded the plaintiff over $3.5 million in damages. The British Columbia Court of Appeal upheld the majority of this decision, but reduced the final award by $480,000.[101] This is an excellent example of how important and effective collateral witnesses can be to your client's case.

The importance of collateral witness information was also confirmed in *Labrecque v. Heimbeckner*, where Justice Macleod stated:

> The best evidence as to whether the accident had any affect on her brain function is the evidence of those who knew her best prior to the collision and to compare their assessment of her prior to the collision with their observations and the observations of those caring for her or associating with her post collision.[102]

The judge or jury will not know what your client was like before the accident. The best way to tell the story is through the evidence of collateral witnesses. Pick the witnesses who can testify to your client's abilities and accomplishments before the accident. This must be done before you introduce any medical evidence and before you call your client. It is not recommended that your client be called at or near the beginning of the trial. Wait until the collateral witnesses have told the story of how your client has changed following the accident and the experts have shown the judge or jury how this is consistent with a TBI. It is not recommended that your client be in the courtroom before testifying. Witnesses may not feel comfortable testifying about significant changes in your client if he or she is present in the courtroom.[103]

---

[100] *Ibid.* at para. 87 (S.C).

[101] *Ibid.* (C.A.).

[102] *Supra* note 49 at para. 125.

[103] I only have my client in the courtroom at the beginning of the trial to introduce the client to the court. I bring the client back to court only for the purpose of testifying. I do not see any advantage to having the client present during the testimony of other witnesses.

XIII. Malingering, Secondary Gain and Accident Neurosis

While it would be naive to believe that malingering and secondary gain are not factors in some cases of MTBI, the literature indicates that such cases of outright malingering are not as common as once believed.[104] In an article entitled "Malingering Aspects of Mild Head Injury", the authors noted:

> Miller contributed greatly to a controversy by claiming that accident neurosis occurs subsequent to head injury. With his views, he fueled a long-standing controversy between opposing attorneys, and his work has been quoted frequently. He reported that nearly all his patients (48 of 50) demonstrated substantial, if not complete, recovery 2 years after their claims were settled. Many others in subsequent studies have disputed this assertion, however. In a study of 500 patients with post-traumatic psychoneurosis, Thompson reported that financial settlement did not significantly alter the course of the illness. In an earlier study, Thompson found that of 190 individuals with posttraumatic psychoneurosis only 15% reported that their symptoms were better after litigation was finalized. More pertinent to the field of brain trauma, Kelly and Smith reported that few of their concussed patients who had not returned to work by the time of the settlement returned to work subsequent to their settlements. Mendelson suggests that the term *compensation neurosis* is invalid because it is not supported by criteria that typically are utilized to validate a disease entity. His study of 1992 demonstrated that 75% of those injured in compensation accidents failed to return to gainful employment, even 2 years after the settlements were finalized.[105]

Dr. Muriel Lezak, in her text *Neuropsychological Assessment*, reviewed the literature with regard to the effect of compensation claims:

> Insufficient or inappropriate behavioural examinations of head trauma can lead to unjust social and legal decisions concerning employability and competency, can invalidate rehabilitation planning efforts, and can confuse patient and family, not infrequently adding financial distress to their already considerable stress and despair (Nemeth, 1991; Varney and Shepherd, 1991).
>
> In this vein, it should be noted that patients seeking compensation for their injuries do not present more symptoms or deficits on testing than similar patients who do not have compensation claims (Rimel, Giordani, Barth, et al., 1981; Stuss, Ely et al., 1985), but the claimants may tend to complain more than other patients (McKinlay, Brooks, and Bond, 1983). A negative kind of support for the conclusion that litigation or compensation has little effect on patient behaviour was the finding that at three months post trauma, half of a group of mildly injured

[104] Berrol, S., "Terminology of post-concussion syndrome" in Horn, I.J. & Zasler, N.D. eds., *Rehabilitation of Post-Concussive Disorders* (Philadelphia: Hanley & Belfus, 1992) 1-8 at 4; Lezak, *supra* note 32 at 191.

[105] Ruff, R.M., Wylie, T. & Tennant, R., "Malingering and malingering-like aspects of mild closed head injury" (1993) 8:3 The Journal of Head Trauma Rehabilitation 60-73 at 61.

> patients had not returned to work, yet none had compensation claims (R. Diamond et al., 1988). In fact, Shinedling et al (1990) reported not only no test differences between suing and nonsuing patients, but that both groups were deeply involved in denying their trauma-related deficits. Bornstein and his colleagues (1988) failed to find any differences in emotional status between patients involved in compensation issues and those who were not. However, Rutherford (1989) suggests that the stress of being in litigation could affect the duration of symptoms, noting that this effect would not be apparent at six weeks, but would become evident some time late. Yet L.M. Binder (1986) notes that "the effect of compensation claims and preinjury pathology is often secondary to organic factors," pointing out that patients with enduring symptoms are the ones most likely to sue.[106] [emphasis added]

Dr. Berrol also commented on the prevalence of malingering in a chapter published in the text *Rehabilitation of Post-Concussive Disorders*:

> Some terminology has evolved to imply that persistent symptoms have no clinical basis, in spite of the literature described above. Persistence of symptoms has been suggested as having a direct relationship to the desire for compensation. …Others who have attempted to establish a relationship between symptoms and desire for compensation have failed to do so. …On the other hand, large multicenter studies in which unselected patient populations are evaluated have identified statistically significant numbers of mild head injured with longstanding symptoms that affect daily function and who have no potential for litigation. Others have argued that such pejorative terminology, which implies a preconceived concept of causation, not infrequently leads to inappropriate clinical management. Many investigators have actively sought to identify relationships between postinjury personality complaints and the issue of personal gain and compensation – or litigation and premorbid personality disorders – without success.[107]

Most neuropsychologists will incorporate "motivation" tests designed to determine if the patient is trying to deceive the examiner. While failure does not mean that your client is necessarily malingering, passing these tests is a positive sign that your client is presenting his or her best effort.

XIV. Conclusion

TBI litigation is very costly. The lawyer will want to ensure that the case meets the applicable diagnostic criteria. Severe and even moderate TBI cases will not pose much difficulty at the diagnostic stage. It is the MTBI case that presents the difficulty. Is the client a member of the "miserable minority", the 10 to 20 percent of MTBI victims that do not recover? If the answer is yes, then the challenge remains to marshal the evidence necessary to educate the trier of fact so that the award of damages will properly reflect

[106] Lezak, *supra* note 35 at 191.

[107] Berrol, *supra* note 104.

the debilitating deficits that can be associated with MTBI. Remember the previously referred to observation of Muriel Lezak with regard to "recovery" from MTBI:

> I do not use the term "recovery" when discussing brain injuries. Damage that is severe enough to alter the level of consciousness even momentarily, or to result in even transient impairment of sensory, motor, or cognitive functions, is likely to leave some residual deficits.[108] [emphasis added]

If defence counsel or the defence experts make conclusions based in whole or in part on the MTBI myths discussed, it is imperative to show the trier of fact both why and how these myths are untrue. In addition, the limitations on current diagnostic criteria should be made clear. In the absence of positive neuroimaging, the most powerful evidence in a MTBI case will be the collateral evidence attesting to the significant cognitive, emotional, and behavioural changes following the traumatic event. If a client has sustained a MTBI, then the lawyer should be able to find several compelling witnesses that can testify to the "residual deficits" referred to by Lezak. It is the cumulative effect of these witnesses that will convince the judge or jury that your client is a member of the 10 to 20 percent of persons who never recover from a MTBI.

[108] Lezak, *supra* note 1 at 162.