TRYING A MILD TRAUMATIC BRAIN INJURY CASE: WHAT YOU NEED TO KNOW

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No head injury is too severe to despair of,
nor too trivial to ignore
Hippocrates 400 BCE

I. Introduction

"Occasionally a seemingly innocuous event can have tragic consequences."[2] These are the opening comments of Mr. Justice Weatherill in *Wallman v John Doe*.[3] The innocuous event? A $673 rear-end collision. The tragic consequences? A mild traumatic brain injury (mTBI).

The facts. On December 4, 2006, the plaintiff, Dr. Wallman, was driving to the Whistler Health Care Centre where he worked as an emergency room physician. It was a cold and snowy morning. He stopped at a red light. When the light turned green he started forward, but had to stop for a snowplow that entered the intersection. He heard a roaring engine behind him. There was a bang and a flash of white light. He had a vague recollection of speaking to the bus driver that rear-ended him, but no recollection of preparing a statement for the bus driver to sign. He did not recall driving to the Whistler Health Care Centre, but recalled attending an intubation course later that day. He was confused and could not follow what was going on.

In the days, weeks, and months following the collision, Dr. Wallman was nauseated, confused, and disoriented. He struggled with double and blurry vision, sensitivity to light and sound, headaches, irritability, and difficulty with his memory. The diagnosis? An mTBI that Dr. Wallman claimed prevented him from returning to his work as a physician. He also claimed losses from his real estate business.

Plaintiff's counsel presented a case of a man whose "life changed instantly and dramatically."[4] He called experts experienced in the field of mTBI, as well as friends, work colleagues, and family members to tell the judge the story of how Dr. Wallman became a member of the "miserable minority," the 10-20% of victims of mTBI who continue to experience persistent disabling problems.

The defence argued that the damage to the vehicles was so minor that the plaintiff could not have suffered the injuries he alleged. They said the plaintiff was a malingerer and must have fabricated his evidence. He was burnt out, exhausted by his home life, and angry with ICBC for denying an earlier claim. The collision was an opportunity to change his life and get retribution against ICBC. They attacked the diagnosis of mTBI and causation.

The trial lasted 35 days and the court heard evidence from over 40 witnesses. Weatherill J. was impressed with the lay evidence, which trumped the competing expert evidence. He found that Dr. Wallman had suffered an mTBI caused by the collision that developed into a post concussion syndrome. Dr. Wallman's life was forever altered by the seemingly innocuous event and he was awarded over $5.9 million in damages.

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[1] This paper is an update of "Litigating a Mild Traumatic Brain Injury Case" published in the Spring 2011 edition of the Verdict. I would like to thank Nicole Kelly for her assistance with this paper.

[2] *Wallman v Doe* 2014 BCSC 79 at para 1. (under appeal)

[3] *Wallman*, *supra*.

[4] *Wallman*, *supra*, at para 5.

The risk to Dr. Wallman and his counsel in proceeding to trial was significant. There is an inverse relationship between the severity of the traumatic brain injury (TBI) and the cost of litigation. Unlike moderate and severe TBI cases, mTBIs share symptoms with other medical conditions leading to issues with diagnosis and causation. More experts get retained, the cost of litigation goes up, and the likelihood of settlement goes down. A ten-day trial morphs into 20 or 30 days or more. Disbursements can easily exceed $200,000 and the lawyer's investment in time and overhead can be even higher. The client is exposed to paying defence costs if unsuccessful.

In *Wallman v John Doe*, the upside in damages justified the risk in proceeding to trial. The decision is an excellent example of the many considerations that go into an mTBI case. This paper reviews what the trial lawyer needs to know to successfully try an mTBI case.

II. Diagnosis of a Mild Traumatic Brain Injury

A TBI is defined as "an alteration in brain function, or other evidence of brain pathology, caused by an external force."[5] A TBI can be mild, moderate, or severe. Historically, the terms minor head injury, concussion, post concussive syndrome, posttraumatic syndrome, and traumatic head syndrome were used interchangeably.[6] This created difficulty diagnosing mTBI. Neuropsychologist Dr. Thomas Kay explained that minor head injury and mTBI 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 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.[7]

The term "head injury" and even "closed head injury" are no longer employed by experts when referring to TBI.

A. American Congress of Rehabilitation Medicine (ACRM)

The first clear definition of mTBI was developed by the ACRM. The definition does not require a loss of consciousness or a blow to the head and recognizes that the symptoms of mTBI may not be acknowledged by the patient until they attempt to return to normal functioning.

> **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 (eg, 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

[5] Menon, D.K., Schwab, K., Wright, D.W., & Maas, A.I., "Position Statement: Definition of Traumatic Brain Injury" (2010) 91 Archives of Physical Medicine and Rehabilitation 1637-1640, at 1637.

[6] Bigler, E.D., "Neuropsychology and clinical neuroscience of persistent post-concussive syndrome" (2008) 14 Journal of the International Neuropsychological Society 1-22 at 2; McCrea, M.A., *Mild Traumatic Brain Injury and Postconcussion Syndrome*, (New York: Oxford University Press, 2008), at 9.

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

- 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. 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, behavioural, 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 (eg, 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 (eg, 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 (eg, 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 (eg, 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.[8]

This definition gained widespread acceptance and is recognized by many neurologists, psychiatrists, physiatrists, and neuropsychologists. Our court has also accepted the definition.[9] However, some defence neurologists refuse to accept this definition and will not acknowledge the *Journal of Head Trauma*

[8] Kay, T., et al. "Definition of mild traumatic brain injury" (1993) 8:3 Journal of Head Trauma Rehabilitation 86-87. Available online at https://www.acrm.org/pdf/TBIDef_English_Oct2010.pdf.

[9] *Reilly v Lynn* [1999] B.C.J. No. 2552 (S.C.) aff'd [2002] B.C.J. No. 986 (C.A.) at paras. 273-274 (S.C.); *Lines v. Gordon et al. and ICBC*, [2006] B.C.J. No. 3318 at para. 5 (S.C.), varied 2009 BCCA 106, and *Adamson v. Charity*, 2007 BCSC 671 at para 200.

Rehabilitation as an authoritative source, notwithstanding it is a refereed journal and highly regarded in the field of TBI. It is therefore important that one of your experts adopts this definition or another recognized definition of mTBI. The definition should be included in the written report of the expert and served pursuant to the Supreme Court Civil Rules.

The ACRM definition is very similar to neurologist Michael Alexander's definition published in *Neurology*, a journal that even the most truculent 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.[10]

By using the authoritative literature to cross-examine defence experts, plaintiff counsel educates the trier of fact and undermines the credibility of defence experts who disagree with recognized definitions of mTBI.[11]

B. Center for Disease Control (CDC) definition of mTBI

The CDC published a handbook "Heads Up: Facts for Physicians about Mild Traumatic Brain Injury."[12] It expands on the ACRM definition and is a convenient resource in determining whether a case meets the accepted diagnostic criteria for mTBI. The following excerpt from the CDC highlights the shift in emphasis from structural injury or damage to dysfunction of brain metabolism:

> **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.
>
> **Neuropathophysiology 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.

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[10] Alexander, M., "Mild traumatic brain injury: pathophysiology, natural history and clinical management" (1995) 45 Neurology 1253-1260, at 1253.

[11] In *Young v. Anderson*, 2008 BCSC 1306 the Court preferred the evidence of the experts who adopted the ACRM definition to the experts who did not adopt the definition.

[12] http://www.cdc.gov/concussion/headsup/pdf/Facts_for_Physicians_booklet-a.pdf

Signs and symptoms

Signs and symptoms of MTBI generally fall into four categories: physical, cognitive, emotional, and sleep, and may include:

Thinking/ Remembering	Physical	Emotional/ Mood	Sleep
Difficulty thinking clearly	Headache Fuzzy or blurry vision	Irritability	Sleeping more than usual
Feeling slowed down	Nausea or vomiting (early on) Dizziness	Sadness	Sleep less than usual
Difficulty concentrating	Sensitivity to noise or light Balance problems	More emotional	Trouble falling asleep
Difficulty remembering new information	Feeling tired, having no energy	Nervousness or anxiety	

Some of these symptoms may appear right away, while others may not be noticed for days or months after the injury, or until the person starts resuming their everyday life and more demands are placed upon them.[13]

C. World Health Organization (WHO) definition of mTBI

The WHO Collaborative Center Task Force also published an authoritative definition of mTBI. The Task Force definition is similar to that of the ACRM:

> MTBI is an acute brain injury resulting from mechanical energy to the head from external forces. Operational criteria for clinical identification include: (i) 1 or more of the following: confusion or disorientation, loss of consciousness for 30 minutes or less, post-traumatic amnesia for less than 24 hours, and/or other transient neurological abnormalities such as focal signs, seizure, and intracranial lesion not requiring surgery; (ii) Glasgow Coma Scale score of 13-15 after 30 minutes post-injury or later upon presentation for healthcare. These manifestations of MTBI must not be due to drugs, alcohol, medications, caused by other injuries or treatment for other injuries (e.g. systemic injuries, facial injuries, or intubation), caused by other problems (e.g. psychological trauma, language barrier or coexisting medical conditions) or caused by penetrating craniocerebral injury.[14]

III. Essential Information

The following information is critical. It should be obtained early on and definitely before referring the client to mTBI experts:

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[13] http://www.cdc.gov/concussion/signs_symptoms.html and http://www.cdc.gov/concussion/headsup/pdf/Facts_for_Physicians_booklet-a.pdf. See the Ontario Neurotrauma Foundation, "Guidelines for Concussion/Mild Traumatic Brain Injury and Persistent Symptoms" Second Edition September 2013 http://onf.org/documents/guidelines-for-concussion-mtbi-persistent-symptoms-second-edition for the Canadian equivalent.

[14] Carroll L.J., Cassidy, J.D., Holm L. et al. "Methodological issues and research recommendations for mild traumatic brain injury: The WHO Collaborative Center Task Force on Mild Traumatic Brain Injury. (2004) 43 (suppl) Journal of Rehabilitative Medicine 113-24, at 115.

1. Client's full history pre- and post-traumatic event. It is important to know whether there is anything in the client's history that will affect a diagnosis of mTBI.
2. Witnesses to the traumatic event. Did the client hit his or her head, lose consciousness, or demonstrate confusion or disorientation? Were there any complaints of headache, dizziness, or nausea at the scene? Did the client appear stunned or confused?
3. Ambulance crew report. Look for the Glasgow Coma Scale score, any reference to loss of consciousness, and combative or agitated behaviour. There may be references to trauma to the head such as bruising or lacerations.
4. Pre and post traumatic amnesia. Does the client recall the moments leading up to the traumatic event, the particulars of it, and the moments after? If not, what is the period of amnesia?
5. Hospital records. It is important to review not only the initial history, assessment, and diagnosis, but also the nursing notes which may contain references to cognitive, emotional, and behavioural symptoms consistent with an mTBI.
6. Post-traumatic event 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 an mTBI.
8. School records including any standardized test results.
9. Employment records. Contrast pre and post traumatic event performance.
10. Collateral information. Family, friends, teachers, employers, and co-workers can confirm changes in cognitive, emotional, and behavioural functioning following the traumatic event.

IV. Common Defence Arguments

There are many common myths surrounding mTBI that have been perpetuated by individuals who are, in the words of Thomas Kay, "guilty of gross ignorance and neglect of the long-term problems" associated with mTBI.[15] These myths include:

1. An mTBI cannot occur without loss of consciousness (LOC);
2. A Glasgow Coma Scale (GCS) score of 15 means no mTBI;
3. Whiplash cannot cause a mTBI;
4. If neurodiagnostic imaging is negative, no mTBI has occurred; and
5. Everyone fully recovers from an mTBI.[16]

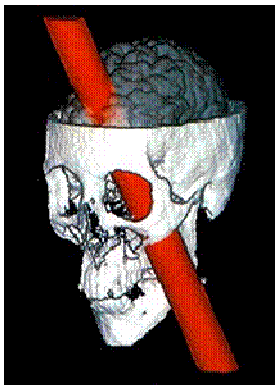
When defence counsel and their experts try to perpetuate these myths you can counter with the medical literature. Have your experts refer to authoritative literature in their reports. This will educate the judge or jury. Then use the literature to cross-examine the defence experts. If they agree the opinions expressed in the literature, then it will become evidence in the case. If they disagree, then their credibility is diminished.

A. There was no loss of consciousness

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

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

The definition of mTBI does not require an LOC. Phineas Gage is the most famous and striking example of a severe TBI with no LOC. He sustained a severe frontal lobe injury while working as a foreman with a railroad construction crew in 1848. 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.[17]



Phineas Gage never lost consciousness. He was reported to be sitting up and talking with the iron bar protruding from his left temporal and frontal lobes. Although he made a complete physical recovery, his personality and emotional behaviour changed significantly. Previously a mild-mannered and effective crew supervisor, Phineas Gage became impulsive, aggressive, and unreliable. He became incapable of working in any capacity. The following description of Phineas Gage 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'.[18]

Focal injuries, such as that of Phineas Gage, may be independent of diffuse axonal injury (DAI) that leads to LOC or alteration in consciousness. Unlike patients with DAI, the severity of focal injury is not related to LOC and its duration.[19]

In diagnosing a TBI, the behaviour of an injured person at the traumatic event scene can be indicative of a cerebral contusion or focal injury. Any reference to combative or aggressive behaviour can indicate a focal injury despite no LOC. Cerebral contusions may not be detected by a standard neurological examination, CT or MRI scans, or standardized neuropsychological tests. Experts familiar with the pathophysiological mechanism and behavioural correlates of a cerebral contusion can explain how cerebral contusions produce significant and often permanent alterations in personality and behaviour.[20]

Over twenty years ago the British Columbia Supreme Court, in *Chen v. Ruersatt,* recognized that LOC was not a prerequisite for an mTBI.[21] More recently in *Cikojevic v. Timm*, Brown J. unequivocally stated that "a loss of consciousness is unnecessary for the diagnosis of mTBI."[22]

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

[18] Harlow, J.M., "Recovery after severe injury to the head" (1968) 2 Publ. Mass. Med. Soc. 327 - 346.

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

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

[21] [1993] B.C.J. No. 302 (S.C.), varied [1994] B.C.J. No. 1441 (C.A.).

[22] 2010 BCSC 800, at para. 250.

B. Glasgow Coma Scale score was not less than 15

Neurosurgeons Jennett and Teasdale developed the GCS as a 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).

	1	2	3	4	5	6
Eye Opening	No response	To painful stimulation	To speech	Spontaneous	N/A	N/A
Motor Response	No response	Extensor (decerebrate) posturing to pain	Flexor (decorticate) posturing to pain	Makes withdrawal movements to pain	Makes localized movements to pain	Follows commands
Verbal Response	No response	Says incomprehensible sounds	Says inappropriate words	Converses but is disoriented	Oriented to person, place, and date	N/A

It is a quick and easy tool used to assess the severity of TBI in the acute setting or within 48 hours of injury. However, the GCS (E+V+M) gives a prognosis for survival, not functional outcome.[23] A GCS score of 13 or higher generally correlates with mTBI, 9 to 12 a moderate TBI, and 8 or less a severe TBI. However, a score of 15 does not mean that a TBI 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 may be administered several times. When that occurs, it is prudent to have all the GCS scores interpreted according to a timeline.[24]

The GCS is not sensitive to the defining characteristics of mTBI.[25] The GCS score says nothing about focal damage to the brain, only diffuse brain injury.

i. Post Traumatic Amnesia

The duration of post traumatic amnesia (PTA) - not the GCS score - is the best "yardstick" for assessing severity of TBI.[26] PTA can be used to assess the degree of diffuse brain damage without any information from witnesses, or from ambulance or hospital records. It depends solely on the recollection of the patient. Jennett and Teasdale note that "amnesia for even a few minutes after a blow to the head is evidence of diffuse brain damage."[27] Jennett and Teasdale expanded the original PTA scale for measuring TBI severity:[28]

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

[23] Jennett, B. & Teasdale, G., "Management of Head Injuries" (1981) 20 *Contemporary Neurology Series*, vol. 20, (USA: F.A. Davis Company, 1981) at 74.

[24] Ruff, R.M., et al., "Recommendations for diagnosing a mild traumatic brain injury: A national academy of neuropsychology education paper" (2009) 24 Archives of Clinical Neuropsychology 3-10, at 8.

[25] McCrea, *supra* note 6, at 17.

[26] Jennett, *supra* note 23, at 90.

[27] *Ibid.* at 96.

[28] *Ibid.*

PTA continues to be "the primary and most specific diagnostic indicator of injury."[29] After the first 24 hours, PTA assessments at weekly intervals may predict important outcomes including the likelihood of the patient becoming employed or being able to live independently.[30]

ii. Extended Glasgow Coma Scale

Due to the insensitivity of the traditional GCS and the greater sensitivity of PTA, the Extended Glasgow Coma Scale (GCS-E) was created. The GCS-E was developed with support from the WHO Advisory Group on the Prevention and Treatment of Neurotrauma, and was adopted as an optional diagnostic variable for the revision of the "Standards for the Surveillance of Neurotrauma." The GCS-E defines eight levels of PTA and assigns a score that is recorded along with the traditional GCS score. The levels of amnesia are set out in the "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.

If the GCS was 15 and the PTA was 30 minutes, the GCS-E score would be 15:5. The GCS-E recognizes that the duration of PTA is an indicator that a person is not laying down permanent memory and accordingly has suffered an alteration in brain functioning.

C. No positive diagnostic imaging

CT and standard MRI scans depict brain structure and lack the resolution to visualize the microscopic damage which occurs in mTBI cases.[31] Every TBI places unique stress and strain on the brain and no two TBIs are identical in terms of how the brain is impacted.[32] However, the pathophysiological mechanism responsible for an altered state of consciousness is the same for both an mTBI and a more severe TBI.[33] Acute pathophysiological changes in the brain can also occur from blows to the head that are below the

[29] Ruff, R.M. et al., *supra* note 24, at 6.

[30] Brown, A.W. et al., "Predictive utility of weekly post-traumatic amnesia assessments after brain injury: a multicentre analysis" (2010) 24:3 Brain Injury 472-478.

[31] Bigler, E.D. "Neuroimaging in mild traumatic brain injury" (2010) 3 Psychological Injury and Law 36-49, at 41.

[32] Bigler, *supra*, note 6, at 13; Viano, D.C., et al., "Concussion in professional football: Brain responses by finite element analysis: Part 9" (2005) 57 Neurosurgery 891–916.

[33] Zink, B.J., Szmydynger-Chodobska, J. & Chodobski, A. "Emerging concepts in the pathophysiology of traumatic brain injury" (2010) 33 Psychiatric Clinics of North America 741-756, at 752

threshold for producing what would behaviorally be classified as an mTBI.[34] Jennett and Teasdale recognized this, stating:

> 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.[35] [emphasis added]

When structural pathology is not evident, even using the most sophisticated imaging techniques; this does not mean that a brain injury has not occurred.[36] In the words of Dr. Zasler, co-editor of the 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.[37] [emphasis added]

A 1994 study examined the brains of five people who suffered an mTBI (GCS 14 or 15), all of whom died 2 to 99 days post injury from other causes.[38] There was no positive evidence on neuroimaging. However, microscopic examination of the brains showed diffuse axonal injury in all five cases.

In 2007, Macleod J. in *Labrecque v. Heimbeckner*[39] recognized that brain injury can still be present notwithstanding negative CT or MRI scans. The plaintiff experienced PTA, and had lacerations and swelling of her face, but it could not be determined whether or not she experienced a LOC. Macleod J. stated:

> 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.

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

[35] Jennett, *supra* note 23, at 91.

[36] CT remains the acute standard for neuroimaging of MTBI but it is only sensitive to gross abnormalities and is typically used to rule out more serious and life-threatening injury. Bigler, *supra* note 31, at 42.

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

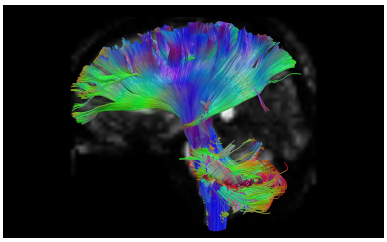
[38] Blumbergs, P.C. et al., "Staining of amyloid precursor protein to study axonal damage in mild head injury" (1994) 344 Lancet 1055-1056.

[39] [2007] A.J. No. 1462 (Q.B.).

…

> [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 damage 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 … 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 dependency 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.[40]

Enduring pathophysiological effects are associated with mTBI; however, little is known about the pathophysiology that follows traumatic axonal injury.[41] Abnormal magnetic resonance spectroscopy was found with normal structural imaging, with some loss of brain volume demonstrated in mTBI cases with a GCS score of 13 to 15.[42] Individuals who have suffered an mTBI can have normal structural MRI and CT scan findings, but magnetoencephalographic abnormalities that are significant.[43]



Diffusion tensor imaging (DTI) is an MRI application using the diffusion of water to image the brain. Unlike MRI, DTI provides a more direct measure of the integrity of white matter fibers[44] and thus may be more sensitive to milder forms of damage.[45] Recently studies using DTI have shown white matter abnormalities following mTBI[46] and in those with a history of mTBI.[47] Research also suggests that DTI may predict recovery in TBI patients, particularly with mTBI that causes axonal injury not identified in CT or MRI scans.[48]

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[40] *Ibid.* at paras 102 and 113 (Q.B.).

[41] Pasternak O et al., "Hockey Concussion Education Project, Part 2. Microstructural white matter alterations in acutely concussed ice hockey players: a longitudinal free-water MRI study" (2014) Journal of Neurosurgery 1-9 published online February 4, 2014 DOI: 10.3171/2013.12.JNS132090

[42] 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.

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

[44] Alger, J.R., "The Diffusion Tensor Imaging Toolbox" (2012) 32 The Journal of Neuroscience 7418-7428.

[45] Huang et al., "Integrated imaging approach with MEG and DTI to detect mild traumatic brain injury in military and civilian patients" (2009) 26 Journal of Neurotrauma 1213-1226.

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

[47] Sasaki, T et al, "Hockey Concussion Education Project, Part 3. White matter microstructure in ice hockey players with a history of concussion: a diffusion tensor imaging study" (2014) Journal of Neurosurgery 1-9 published online February 4, 2014; DOI 10.3171/2013.12.JNS132092.

[48] Huang, *supra*, note 45 at 1213; Belanger, H.G., Vanderploeg, R.D., Curtiss, G. and Warden, D.L., "Recent neuroimaging techniques in mild traumatic brain injury" (2007) 19 Journal of Neuropsychiatry and Clinical Neurosciences, 5-20.

Free-water MRI has also demonstrated acute micro-structural changes in the brain following mTBI.[49] Susceptibility-weighted imaging is another emerging area of imaging following mTBI.[50] Further development of these newer imaging modalities may provide objective confirmation of mTBI and ease the task of lawyers in educating judges and jurors.[51]

In addition to structural injury, mTBI produces metabolic injury.[52] Positron emission tomography (PET) 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 conventional MRI show a static picture of brain structure, PET reflects brain function by showing metabolic activity in different areas of the brain. It illustrates brain dysfunction by monitoring alterations in the amount of glucose that specific areas of the brain consume.[53] PET has been used to explain why symptoms of mTBI can be present in the absence of any structural damage.

The lack of objective evidence of mTBI has also spurred researchers to focus on possible biomarkers of mTBI. Studies show promise for the identification and use of proteins and molecules to diagnose mTBI, assess severity of injury, and predict prognosis.[54] These imaging and neuronal injury biomarker studies combined with the post-mortem studies provide indisputable evidence that structural pathology can be present in mTBI.[55]

D. Everyone recovers from mTBI

Defence experts often point to articles stating that the majority of mTBI patients will fully recover within 6-12 months of the injury.[56] Twenty years ago, neuropsychologist Dr. Muriel Lezak recognized 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.[57]

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[49] Pasternak, O. et al., "Hockey Concussion Education Project, Part 2. Microstructural white matter alterations in acutely concussed ice hockey players: a longitudinal free-water MRI study" (2014) Journal of Neurosurgery, published online February 4, 2014; DOI 10.3171/2013/12/JNS132090.

[50] Helmer, K. et al., "Hockey Concussion Education Project, Part 1. Susceptibility-weighted imaging study in male and female ice hockey players over a single season" (2014) Journal of Neurosurgery, published online February 4, 2014; DOI 10.3171/2013.12.JNS132093.

[51] De Caro, S. & Kaplen, M.V., "Current issues in neuroloaw" (2010) 33 Psychiatric Clinics of North America 915-930, at 920.

[52] 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.

[53] 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. See Slater, M.J., "Admissibility of PET Scan Evidence" (February, 1999) 79 The Verdict. Since the *Wolfin* decision PET scans have passed the *Daubert* admissibility test in the United States and it may be that PET scans will now be admissible in Canada.

[54] Shahim, P. et al., "Blood Biomarkers for Brain Injury in Concussed Professional Ice Hockey Players" (2014) JAMA Neurology published online March 14, 2014 DOI:10.1001/jamaneurol.2013.367; Zink, B.J., Szmydynger-Chodobska, J. & Chodobski, A. "Emerging concepts in the pathophysiology of traumatic brain injury" (2010) 33 Psychiatric Clinics of North America, 741-756, at 752; Jagoda, A.S. "Mild traumatic brain injury: Key decisions in acute management" (2010) 33 Psychiatric Clinics of North America, 797-806, at 799; and Pasinetti, G.M., Fivecoat, H. & Ho, L. "Personalized medicine in traumatic brain injury" (2010) 33 Psychiatric Clinics of North America, 905-913, at 911.

[55] Bigler, *supra* note 6, at 7.

[56] 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.

[57] Lezak, M., *Neuropsychological Assessment*, 3d ed. (USA: Oxford University Press, 1995) at 177-178 at 175.

The term "mild" is a misnomer for many persons with MTBI, with a recent study showing that 20 to 30% of patients continue to experience clinically significant sequelae and over 80% remain symptomatic at a 1-year follow up.[58] A study of former athletes found altered neuropsychological and motor indices more than 30 years after they sustained an mTBI.[59]

Long term studies demonstrate that persons with a history of MTBI from which they had supposedly clinically "recovered" developed dementia years later.[60] These studies also "support the presence of a permanent neuropathologic basis to mTBI, even though clinical 'recovery' has occurred".[61] There are also a growing number of studies indicating that damage from mTBI can lead to progressive degenerative changes.[62] An mTBI has been described as the most robust environmental Alzheimer's disease risk factor in the general population.[63]

i. "Miserable Minority"

While there is a direct relationship between the severity of concussion and the likelihood of symptoms lasting more than three months,[64] concussion severity alone is a poor predictor of who will experience long term symptoms.[65] Approximately 10 to 20 percent of persons never completely recover,[66] and are left with one or more physical symptoms, cognitive deficits, behavioral changes, or alteration in degree of emotional responsivity.[67] These individuals have been referred to by neuropsychologist Ronald Ruff as the "miserable minority."[68]

ii. "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 suffer long-term effects of mTBI, not because they are malingerers or are looking to capitalize on secondary gains, but because they have a greater susceptibility (physical and/or

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[58] McMahon, P, et al., "Symptomatology and functional outcome in mild traumatic brain injury: result from the prospective TRACK-TBI study" (2014) 31 Journal of Neurotrama 26-33, at 32.

[59] 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.

[60] 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.

[61] *Ibid*. at 795.

[62] 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, 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.

[63] Guo, *ibid.*; Plassman, *ibid*, and DeKosky, S.T., Ikonomovic, M.D., & Gandy, S. "Traumatic brain injury – football, warfare, and long-term effects" (2010) 363:14 New England Journal of Medicine, 1293-1296, at 1295. 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/.

[64] 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.

[65] Guskiewicz, K.M. et al., "National Athletic Trainers' Association Position Statement: Management of Sport-Related Concussion" (2004) 39 Journal of Athletic Training 280–297; and Riggio, S. "Traumatic brain injury and its neurobehavioral sequelae" (2010) 33 Psychiatric Clinics of North America, 807-819, at 816.

[66] 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; Alexander, M., "Mild traumatic brain injury: pathophysiology, natural history and clinical management" (1995) 45 Neurology 1253-1260 at 1256, and *Burdett v. Eidse*, [2010] B.C.J. No. 289, at para. 198. For a discussion of the long term effects 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.

[67] Kay, *supra* note 7; and Riggio, *supra* note 65, at 808.

[68] 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 66.

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. Those with a history of neuropsychiatric disorder are more likely to experience long term effects of mTBI.[69] Research shows the following factors are associated with a longer period of recovery from an mTBI: history of mTBI[70]; headache history[71]; developmental history[72]; psychiatric history (such as history of depression/mood disorder, anxiety, and/or sleep disorder).[73] The outcome for TBI is also worse for females than for males.[74]

A victim of mTBI who does not recover as quickly as might be expected or who suffers a more significant disability due to a prior concussive injury is entitled to compensation for the full extent of the injuries. This thin skull rule applies to emotional and physical susceptibility, although there appears to be a need to differentiate between pre-accident susceptibility and post-injury mental attitude.[75] Physical injury that triggers personality change is compensable.[76] In *Canadian Tort Law*, Linden canvasses a number of cases where the courts have awarded full compensation for the "vulnerable personality."[77] Linden refers to the following comments of Lane J. 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]

iii. Individual Vulnerability

The term "individual vulnerability" was introduced by Dr. Thomas 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

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

[70] Guskiewicz, K. et al., "Cumulative effects associated with recurrent concussion in collegiate football players: the NCAA Concussion Study" (2003) 290:19 JAMA 2549-2555; Collins, M.W. et al., "Cumulative effects of concussion in high school athletes" (2002) 51 Neurosurgery 1175-1181; Iverson, G. et al., "Cumulative effects of concussion in amateur athletes" (2004) 18:5 Brain Injury 433-443.

[71] Mihalik, J. et al., "Posttraumatic migraine characteristics in athletes following sports-related concussion" (2005) 102:5 *Journal of Neurosurgery* 850-855; 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 727-732.

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

[73] McCrea, *supra* note 6, at 112; 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 117-132; Mooney, G., Speed, J. & Sheppard, S., "Factors related to recovery after mild traumatic brain injury" (2005) 19:12 Brain Injury 975-987; 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 1077-1087.

[74] Farace, E. & Alves, W.M., "Do women fare worse: a metaanalysis of gender differences in traumatic brain injury outcome" (2000) 93 Journal of Neurosurgery 539-545.

[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,* 9th ed. (Canada: LexisNexis, 2011) at 384-391.

[78] *Ibid.* at 390 citing *Malcom v. Broadhurst*, [1970] 3 All E.R. 508 at 511.

> 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. 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 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.[79]

Kay referred to the following personality styles that were susceptible to long term problems following an mTBI:

1. Persons who are highly driven;
2. Persons who suffered emotional deprivation as children;
3. Persons with strong tendencies toward dependency;
4. Persons with high levels of emotional rigidity and impaired capacity for deep human relationships, and who manifest "borderline" characteristics in a mild form;
5. Persons with tendencies toward grandiosity, inflated self-belief, and other elements of a narcissistic personality style.[80]

iv. Prior TBI

The cumulative deleterious effects of concussion have been recognized since 1975.[81] An mTBI causes the fine thread-like nerve cells to become stretched and either cease to function or function abnormally. It is

[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] *Ibid*.

the malfunction of these cells that provides the organic basis for the deficits experienced after mTBI. Repeated trauma increases the severity of the deficits. 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).[82]

Neurosurgeon, Dr. Thomas Gennarelli, comments on this vulnerability to subsequent 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.[83] [emphasis added]

A study of the effects of concussion on football players found that a prior concussion increased the likelihood of prolonged recovery of neurological function. Players with a history of a concussion were more likely to have future concussions.[84]

Repeated trauma to the head can also cause chronic traumatic encephalopathy (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.[85] 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.[86] Evidence of CTE has been discovered in numerous deceased former athletes.[87] However, a 2014 study suggests that there may still be some controversy regarding CTE.[88]

The lawyer must inquire whether the client has (1) experienced a prior mTBI whether diagnosed or not, and (2) if so, have they fully recovered from the mTBI. The previous medical records may reveal evidence justifying a diagnosis of mTBI under the criteria established by the ACRM, CDC or WHO definitions. The prior mTBI may explain to the judge or jury why the plaintiff falls into the "miserable minority".

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

[82] Lezak, M., *Neuropsychological Assessment,* 4th ed. (USA: Oxford University Press, 2004) at 188.

[83] 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.

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

[85] Bigler, *supra* note 61, at 795.

[86] DeKosky, *supra* note 58, at 1295.

[87] Leahy, S. "Doctors: Late NFL player Chris Henry had brain damage at time of death at age 26" USA Today, June 28, 2010, online: USA Today <http://content.usatoday.com/communities/thehuddle/post/2010/06/doctors-late-nfl-player-chris-henry-had-brain-damage-at-time-of-death-at-age-26/1>. Keating, P., "Researchers find brain trauma in Henry" ESPN, June 28, 2010. online: ESPN <http://sports.espn.go.com/nfl/news/story?id=5333971>. 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/.

[88] Randolph, C., "Is Chronic Traumatic Encephalopathy a Real Disease?" (2014) 13 Current Sports Medicine Reports 33-37.

v. Malingering and Secondary Gain

Malingering and secondary gain may be factors in some mTBI cases, but the literature indicates that cases of outright malingering are not as common as once believed.[89] Malingering is defined as the "intentional reporting of symptoms for personal gain."[90] The authors of the article "Malingering Aspects of Mild Head Injury" 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.[91]

Lezak has expressed a contrary view after reviewing the literature regarding the effect of compensation. She concluded that persons seeking compensation are the ones that have enduring symptoms. That is why they sue. In her opinion, misguided allegations of secondary gain leads to unjust social and legal decisions:

> 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 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)

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[89] 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 57, at 191.

[90] American Psychological Association. DSM-V, diagnostic and statistical manual on mental disorders.54th edition. Arlington (VA): American Psychiatric Association; 2013, at 326.

[91] 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.

> 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.[92] [emphasis added]

Most neuropsychologists incorporate "motivation" tests to determine if the patient is trying to deceive the examiner. There is no gold standard in determining whether someone is malingering and there are numerous criticisms leveled at the validity of the various tests.[93] While passing these tests confirms the subject is exhibiting his or her best effort, failure does not necessarily mean the subject is malingering. Decisions regarding credibility are outside the scope of the expert opinion and should be left for the trier of fact.

V. Collateral Witnesses

It is not uncommon in mTBI cases for there to be "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."[94] In addition, the neuropsychological assessment may not demonstrate any deficits. It is for this reason that Varney and Menafee suggest that the best information regarding 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 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.[95]

More recent statements on the value of collateral information from reliable sources can be found in the 2011 *Textbook of Traumatic Brain Injury*.[96] One of the major reasons for using collateral information is due to the inadequacy of the neuropsychological assessment. These assessments are rarely conducted in real world settings, therefore the ecological validity (how the assessment reflects abilities of the patient in real world scenarios) of the assessments should be a concern. These limitations are well documented in the literature and the defence expert should recognize this. Unless a significant cognitive demand is placed on the subject that requires more than typical cognitive effort, there may be no difference between pre- and post-accident ability.[97] Neuropsychologist, Erin 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.[98]

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[92] Lezak, *supra* note 82, at 191.

[93] De Caro, supra note 51, at 923.

[94] Lezak, *supra* note 82, at 172.

[95] 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.

[96] Arlinghaus, K., Pastorek, N., & Graham, D., "Neuropsychiatric Assessment" in Silver, J., McAllister, T. & Yudofsky, M., eds., *Textbook of Traumatic Brain Injury* 2nd (Washington D.C.: American Psychiatric Publishing Inc., 2011), at 57.

[97] Bigler, *supra* note 6, at 8; and 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.

[98] Bigler, *ibid*., at 12.

Neuropsychological test scores of brain injured patients are often unrelated or poorly related to measures of everyday functioning and their behavior in real-world settings.[99] Neuropsychological assessment often occurs in a highly structured setting and the neuropsychologist effectively replaces the frontal lobes during the testing. Accordingly, standardized neurological tests are often unable to detect neurobehavioural problems.[100]

Neuropsychological assessments are particularly insensitive to deficits in executive functioning.[101] These types of frontal lobe injuries frequently appear in situations that are complex, novel and highly unstructured.[102] If collateral witnesses say that after an accident the person experiences a dramatic change in personality, is unable to control their behaviour or regulate their emotions, has less social tact, poor impulse control, an inability to empathize with others, marked egocentricity, frequently uses 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, then these are red flags for frontal lobe damage.[103]

Given the deficiencies and insensitivity of 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.[104] The neuropsychologist should interview one or more of the collateral witnesses so that this information can be used in the formulation of the expert's opinion. 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 very minimal PTA, if there is sufficient credible collateral evidence on which the trier of fact can base their decision, a finding of an mTBI may be found.

The trier of fact does not know what the plaintiff was like before the traumatic event. The best way to tell the story is through the evidence of collateral witnesses. Pick witnesses who can testify to your client's abilities and accomplishments before the event. In *Wallman* plaintiff's counsel called a number of friends and colleagues who were able to provide the trier of fact with cogent examples of the changes they observed in the plaintiff following the collision. Weatherill J. held:

> To accept the defendants' submissions that the plaintiff is a malingerer and that the forces imparted on the plaintiff by the Accident could not have injured him, I would have to completely disregard the evidence of the plaintiff, Ms. Roth and all other lay witnesses called by the plaintiff (several of whom are physicians) who testified about

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[99] Sbordone, R., "Neuropsychological tests are poor at assessing the frontal lobes, executive functions, and neurobehavioral symptoms of traumatically brain-injured patients" (2010) 3 Psychological Injury and Law 24-35.

[100] 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).

[101] Sbordone, *supra* note 99, at 28; 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.

[102] Sbordone, *supra* note 199, at 33.

[103] Sbordone (2008), *supra* note 100, at 23.

[104] *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.).

> the plaintiff's sudden and dramatic change in character and personality in the hours, days, and months and years following the Accident.[105]

The compelling evidence from the lay witnesses appears to have been a significant factor in finding that the plaintiff sustained an mTBI with ongoing sequelae. Fact should always trump opinion.

VI. Conclusion

Trying an mTBI case is challenging and costly. For all the success stories, there are horror stories of cases lost and disbursements that are never recovered. The challenge remains to marshal the evidence necessary to educate the trier of fact so that the award of damages will justify the cost of proceeding to trial.

Each day new studies are published and more information is gained about the consequences of mTBI. Increasingly, research is confirming the words of Lezak regarding "recovery" from mTBI:

> 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.[106]

In the absence of positive neuroimaging, the most powerful evidence in an mTBI case is collateral witness evidence of significant cognitive, emotional, and behavioural changes following the traumatic event. It is the cumulative effect of these witnesses that will convince the judge or jury that your client is a member of the "miserable minority", the 10 to 20 percent of persons who never recover from an mTBI.

[105] *Wallman*, *supra*, at para 452.

[106] Lezak, *supra* note 82, at 162.