LITIGATING A MILD TRAUMATIC BRAIN INJURY CASE

BY MICHAEL J. SLATER QC[1]
SLATER VECCHIO LLP, VANCOUVER, BC

I. INTRODUCTION

The 1984 decision in *Clark* v. *Tedesco*[2] is a reminder of how far we have progressed in the last 25 years in appreciating the potentially debilitating consequences of a traumatic brain injury (TBI). The plaintiff was a six year old boy who was injured in a motor vehicle accident. He hit his head and was unconscious for approximately four minutes. He vomited and was lethargic at the hospital. The neurological examination showed the presence of a jerky nystagmus to the right. A CT scan showed dilation in the right lateral ventricle indicating right-sided cerebral atrophy. The plaintiff's mother complained about his poor attitude and poor school work following the accident. He experienced balance problems. He tired easily and became frustrated as he was unable to finish his work. His mother was concerned that the brain injury would affect his future income earning capacity. The Court relied on the opinion of the neurologist who testified the plaintiff had suffered a blunt head injury and showed temporary, but definite disturbances of behaviour, intellect, and vestibular function. The Court concluded that the plaintiff "…has now fully recovered from his injuries, with a slight chance of an epileptic problem in the future." The award of damages was only $7,000.

Since the *Clark* decision the medical literature in the field of TBI and in particular mild traumatic brain injury (MTBI) has exploded. Lawyers can easily access this literature to understand the long term cognitive, behavioural, and emotional deficits that afflict people with TBI. Experts from behavioural neurology, neuropsychology, and neuropsychiatry can explain how a MTBI can significantly impair future earning capacity. Experts in rehabilitation science can explain why a MTBI victim may require substantial rehabilitation and funding for future care. Recently in *Danicek* v. *Alexander Holburn Beaudin & Lang*,[3] plaintiff's counsel effectively employed experts specializing in TBI to educate and thereby persuade the trier of fact that some MTBI victims do not recover and suffer significant long term consequences. The plaintiff, a young lawyer, was awarded $5.9 million in damages.

In deciding whether to invest time and resources in litigating a TBI case, keep in mind that there is an inverse relationship between the severity of the brain injury and the cost of litigation. Unlike moderate and severe TBI cases, MTBI cases share similar symptoms with other medical conditions. This leads to issues with diagnosis and causation. More experts are retained. The cost of litigation goes up and the likelihood of settlement goes down. A ten-day trial morphs into 20 or 30 days. Disbursements can exceed $200,000. The lawyer's investment in time and overhead may be even higher. The client is exposed to paying defence costs if unsuccessful. In *Danicek*, the defence attacked the diagnosis of MTBI and causation as well as the credibility of the plaintiff. The trial lasted 30 days. The upside in damages justified the risk in proceeding to trial.

In this paper I will focus on the MTBI case as it presents a far greater challenge in terms of difficulty and risk than the severe TBI case. What information do you need to know before accepting a retainer? Does the case meet the accepted definitions of MTBI? How do you dispel the common myths associated with MTBI? How important is evidence of collateral witnesses who can attest to cognitive, emotional, and behavioural changes following a traumatic injury?

II. ESSENTIAL INFORMATION

The following information is essential and should be obtained early in the litigation *before* referring the client to experts skilled in the diagnosis of MTBI:

1. **Client's full history pre- and post-accident.** 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 accident.** 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 (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. **Pre- and post-traumatic amnesia.** 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 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 a MTBI.
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 MTBI.
8. **School records** including any standardized test results.
9. **Employment records.** Contrast pre- and post-accident performance.
10. **Collateral information.** Family, friends, teachers, employers, and co-workers can confirm changes in cognitive, emotional, and behavioural functioning following the traumatic event.

Following a review of the essential background information, the next step is to ensure that the case meets a currently acceptable definition of MTBI.

III. 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."[4] A TBI can be mild, moderate, or severe. Historically, the terms minor head injury, concussion, postconcussive syndrome, posttraumatic syndrome, and traumatic head syndrome were used interchangeably.[5] 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.[6]

The term "head injury" and even "closed head injury" are no longer employed by knowledgeable MTBI experts to refer to brain injury.

A. American Congress of Rehabilitation Medicine (ACRM)

The first clear definition of MTBI was developed by the American Congress of Rehabilitation Medicine.[7] 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**
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> A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by *at least* 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
> - posttraumatic amnesia (PTA) not greater than 24 hours.
>
> **COMMENTS**
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> 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.

This definition gained widespread acceptance and is recognized by many neurologists, psychiatrists, physiatrists, and neuropsychologists. In *Reilly* v. *Lynn*,[8] the British Columbia Court of Appeal affirmed the ACRM definition. The Alberta Queens Bench also adopted this definition in *S.F.P.* v. *MacDonald*.[9] Despite these cases, some defence neurologists refuse to accept this definition and will not acknowledge the *Journal of Head Trauma Rehabilitation* as an authoritative source, notwithstanding it is a refereed journal and highly regarded in the field of TBI. For this reason it is important that one of the plaintiff's experts adopts this definition or another recognized definition of MTBI.

The ACRM definition is very similar to neurologist 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.[10]

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

The CDC Mild Traumatic Brain Injury Workgroup published a handbook "Facts for Physicians About Mild Traumatic Brain Injury".[11] It expands on the ACRM definition and is a convenient resource for the lawyer to determine whether a case meets the accepted diagnostic criteria for MTBI. An important point to note in the following excerpt from the handbook is 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.

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.

Signs and symptoms

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

Physical	Cognitive	Emotional	Sleep
• Headache • Nausea • Vomiting • Balance problems • Dizziness • Visual problems • Fatigue • Sensitivity to light • Sensitivity to noise • Numbness/ Tingling • Dazed or stunned	• Feeling mentally "foggy" • Feeling slowed down • Difficulty concentrating • Difficulty remembering • Forgetful of recent information or conversations • Confused about recent events • Answers questions slowly • Repeats questions	• Irritability • Sadness • More emotional • Nervousness	• Drowsiness • Sleeping less than usual • Sleeping more than usual • Trouble falling asleep

C. World Health Organization (WHO) definition of MTBI

Another authoritative definition of MTBI was published in 2004 by the WHO Collaborative Center Task Force. The Task Force formulated a standardized operational definition 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.[12]

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.[13]

IV. MYTHS COMMONLY ASSOCIATED WITH MTBI

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.[14] These myths include:

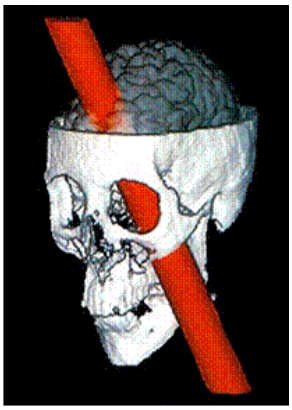
1. MTBI cannot occur without loss of consciousness (LOC);
2. A Glasgow Coma Scale (GCS) score of 15 means no MTBI;

3. Whiplash cannot cause a MTBI;
4. If neurodiagnostic imaging is negative, no MTBI has occurred; and
5. Everyone fully recovers from a MTBI.[15]

If defence experts rely on these myths the trier of fact must be informed that they are untrue. The lawyer can dispel these myths in direct examination with the testimony of experts familiar with the MTBI literature. The myths can also be dispelled by effective cross-examination of the defence's experts.

A. Myth 1: Loss of consciousness is necessary

The diagnosis of a MTBI does not require a LOC. The most famous and striking example of a severe TBI with no LOC is the case of Phineas Gage. 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.[16]



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. Phineas Gage made a complete physical recovery; however, his personality and emotional behaviour changed significantly. 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 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'.[17]

Focal injuries, such as that of Phineas Gage, may be independent of diffuse axonal injury (DAI) that leads to a LOC or an alteration in consciousness. Michael Alexander, wrote in *Neurology and Neurosurgery*:

> Unlike the patients with primarily DAI, the severity of a focal injury is not related to LOC and its duration; many patients with severe focal lesions are never unconscious.[18]

In diagnosing a TBI, the behaviour of an injured person at the accident 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 intelligence and 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.[19]

Over 17 years ago the British Columbia Supreme Court in *Chen* v. *Ruersatt* recognized that LOC was not a prerequisite for a MTBI.[20] 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, he lacked 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). Prowse J. found that the symptoms were consistent with a head injury notwithstanding an absence of evidence of LOC:

> 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.[21] [emphasis added]

More recently in *Cikojevic* v. *Timm*, Brown J. unequivocally stated that "a loss of consciousness is unnecessary for the diagnosis of MTBI."[22]

B. Myth 2: Glasgow Coma Scale score must be less than 15

Altered consciousness is the most consistent feature of diffuse brain damage resulting from acceleration/deceleration injuries. 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). 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]

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 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. Accordingly, when the GCS is administered multiple times, 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. Jennett and Teasdale recognized the limitations at the higher range of the scale:

> ... [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** ... [emphasis added] [26]

i. Post traumatic amnesia

The duration of post traumatic amnesia (PTA) - not the GCS score - is the best "yardstick for assessing severity of head injury."[27] 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. According to Jennett and Teasdale, any period of amnesia is evidence of diffuse brain damage:

> Altered consciousness soon after injury is the clue 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**.[28] [emphasis added]

Jennett and Teasdale expanded the original PTA scale for measuring TBI severity:[29]

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

PTA continues to be "the primary and most specific diagnostic indicator of injury."[30] 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.[31]

ii. Extended Glasgow Coma Scale

A MTBI may not be detected by the traditional GCS due to its insensitivity to milder brain damage. The greater sensitivity of PTA led to the creation of the Extended Glasgow Coma Scale (GCS-E).

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. This information can be important in accurately assessing the degree of TBI.

C. Myth 3: Whiplash cannot cause a MTBI

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

> 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).
> …
> 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).[32]

Thomas Kay wrote about the differences between focal and diffuse brain injuries 25 years ago, well before the clinical use of MRI scans. His comments are still relevant today:

> **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.

…

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]

In a jury trial 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, 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 the neurologist said he was getting ready for cross-examination. What he did not have in his file was Gennarelli's 1986 article titled "Mechanisms and Pathophysiology of Cerebral Concussion."[34] The lawyer asked the neurologist to read to the jury the following excerpts from the article:

> 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 nerve fibers and the consequent axonal damage that now appears to be the substrate of concussive brain injuries. [emphasis added] [35]

It did not matter whether the neurologist agreed with Gennarelli or not. If he agreed it became part of his evidence. If he did not agree his credibility was destroyed and the trier of fact was educated through the references to the literature in cross-examination.

D. Myth 4: Diagnostic imaging must be positive

The resolution of a CT scan is about one half a centimetre. An MRI scan is about one millimetre. Much of the tearing and shearing of axons in MTBI occurs at a microscopic level and would not be apparent on these scans. Every TBI places unique stress and strain on the brain and no two TBIs are identical in terms of how the brain is impacted.[36] However, the pathophysiological mechanism responsible for an altered state of consciousness is the same for both a MTBI and a more severe TBI.[37] This was recognized by Jennett and Teasdale who stated:

> 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.**
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> …
>
> 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]

Oppenheimer's findings were confirmed in a 1994 study where the authors examined the brains of five people who suffered a mild concussion (GCS 14 or 15) all of whom died 2 to 99 days post injury from other causes.[39] Diffuse axonal injury was found in all five cases.

Enduring pathophysiological effects are associated with MTBI. 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.[40] Individuals who have suffered a MTBI can have normal structural MRI and CT scan findings but magnetoencephalographic abnormalities that are significant.[41] Studies using diffusion tensor MRIs have shown white matter abnormalities following MTBI.[42] Further, 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.[43] These imaging and neuronal injury biomarker studies combined with the post-mortem studies provide indisputable evidence that structural pathology can be present in MTBI.[44]

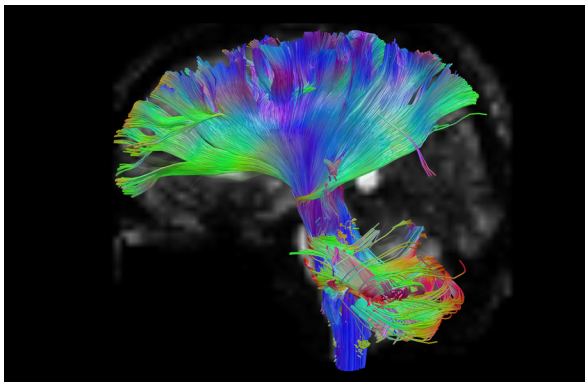
When structural pathology is not evident, even using the most sophisticated imaging techniques, this does not mean that a brain injury has not occurred.[45] CT and standard MRI scans depict brain structure and lack the resolution to visualize the microscopic damage which occurs in MTBI cases.[46] In 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.[47] [emphasis added]

In 2007, Macleod J. in *Labrecque* v. *Heimbeckner*[48] 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.[49]
>
> …
>
> [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 CT scan or even an MRI … 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.[50]

In addition to structural injury, MTBI produces metabolic injury.[51] 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 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.[52] PET has been used to explain why symptoms of MTBI can be present in the absence of any structural damage.



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 and thus may be more sensitive to milder forms of damage.[53] Research suggests that DTI may predict recovery in TBI patients, particularly with MTBI that causes axonal injury not identified in CT or MRI scans.[54] Further development of these newer imaging modalities may provide objective confirmation of MTBI and may ease the task of lawyers in educating jurors and judges.[55]

The present lack of objective evidence of MTBI has spurred researchers to focus on possible biomarkers of MTBI. Studies show promise for the identification and use of proteins and molecules that may be used to assess severity of injury and prognosis.[56]

E. Myth 5: Everyone recovers from MTBI

In 1995, 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.[57]

Studies demonstrate that persons with a history of MTBI from which they had supposedly clinically "recovered" developed dementia years later.[58] These studies also "support the presence of a permanent neuropathologic basis to MTBI, even though clinical 'recovery' has occurred".[59]

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.[60] 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.[61] In early 2009, the Boston University School of Medicine found evidence of CTE in the brain of a deceased 18 year old male who suffered multiple concussions.[62] In 2010, researchers were surprised by the discovery of evidence of CTE in NFL receiver Chris Henry, because he had not been a big hitter and was not known to have suffered any concussions.[63] Most recently researchers at Boston University discovered evidence of CTE in the brain of former NHL player Bob Probert who died at 45 years of age. Researchers have discovered evidence of CTE in more than 50 deceased former athletes.[64] Some former athletes who sustained MTBI demonstrate adverse effects over 30 years later even when they appear asymptomatic to friends and family.[65]

i. "Miserable minority"

Defence experts often point to articles stating that the majority of MTBI patients will fully recover within 6-12 months of the injury.[66] However, there are a growing number of studies indicating that damage from MTBI can lead to progressive degenerative changes.[67] MTBI has been described as the most robust environmental Alzheimer's disease risk factor in the general population.[68]

While there is a direct relationship between the severity of concussion and the likelihood of symptoms lasting more than three months,[69] concussion severity alone is a poor predictor of who will experience long term symptoms.[70] Approximately 10 to 20 percent of persons never completely recover,[71] and are left with one or more physical symptoms, cognitive deficits, behavioral changes, or alteration in degree of emotional responsivity.[72] These individuals have been referred to by Ronald Ruff as the "miserable minority."[73] In a 1993 article, Dikmen and Levin stated:

> Not all patients with mild head injury complain of post-traumatic symptoms, and most improve without further intervention. In a fraction of the cases, however, the post-concussion symptoms do persist and may evolve into the so-called postconcussional syndrome. **Owing to the high incidence of mild head injuries this fraction of cases translates into a sizable group of patients, who may be significantly disabled in resuming their preinjury lifestyle**.[74] [emphasis added]

This "sizable group of patients" has not changed dramatically. What has changed is the recognition by the medical profession, and the courts, of the potentially debilitating effects of MTBI. In 1981 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 the field …" In 1986, New York University Medical Center researchers reported:

> 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.[75]

This ignorance continued over the next decade as observed by Peter 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.[76]

There are still many experts who do not believe that concussions or MTBIs produce anything beyond transient symptoms. But the lawyer can educate the trier of fact through the use of informed experts and by effective use of the authoritative literature in cross examination to demonstrate that the MTBI victim is a member of the "miserable minority".

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 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.[77] 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. Research shows the following factors are associated with a longer period of recovery from a MTBI: history of MTBI[78]; headache history[79]; developmental history[80]; psychiatric history (such as history of depression/mood disorder, anxiety, and/or sleep disorder)[81]

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.[82] Physical injury that triggers personality change is compensable.[83] In *Canadian Tort Law*, Linden canvasses a number of cases where the courts have awarded full compensation for the "vulnerable personality."[84] 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.[85]

iii. Individual vulnerability

The term "individual vulnerability" was introduced by 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 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 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. [86]

Kay also cited the following personality styles as at risk for a dysfunctional response to MTBI:

1. 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 MTBI in which real cognitive problems persist.
2. Persons who suffered emotional deprivation as children, when they are injured in ways that retribution cannot be extracted may become extremely hostile and dysfunctional in the presence of permanent symptoms.
3. Persons with strong tendencies toward dependency are often immobilized by the symptoms of MTBI, especially in the acute state, and respond by decreasing activity and increasing their anxiety about their inability to function.
4. Persons with high levels of emotional rigidity and impaired capacity for deep human relationships, and who manifest "borderline" characteristics in a mild form, do quite poorly after MTBI when real neuropsychological deficits persist.
5. Persons with tendencies toward grandiosity, inflated self-belief, and other elements of a narcissistic personality style, 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.[87]

iv. Prior traumatic brain injury

The cumulative deleterious effects of concussion have been recognized since 1975.[88] Neurologist James Kelly reviewed the literature and 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*.[89]

A MTBI causes the fine thread-like nerve cells to 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. 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).[90]

More recently, Gennarelli commented 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.**[91] [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.[92]

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".

v. Malingering, secondary gain and accident neurosis

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.[93] Malingering is defined as the "intentional production of false or grossly exaggerated physical or psychological symptoms motivated by external incentives."[94] In the article "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.[95]

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) 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 later. **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**.[96] [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.[97] 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.

F. MYTHS DISPELLED

The decision of Brown J. in *Cikojevic* v. *Timm*[98] provides lawyers with a strong precedent for MTBI cases. In *Cikojevic* the plaintiff sustained a MTBI in a motor vehicle accident. Before the accident the plaintiff was happy, personable, and athletic. After the accident, she experienced problems with concentration, organization, and planning in all facets of her life. Brown J's reasons provides a concise summary of the current view of MTBI:

> **VII. Evaluation of Mild Traumatic Brain Injuries**
>
> **248** Experts testifying at trial substantially agreed on how to evaluate MTBI. They did not all comment on all the following points. Overall, however, considering the expert evidence viewed as a whole, the summary below represents a fair consensus.
>
> **A. Diagnosis**
>
> …
>
> **250** A loss of consciousness is unnecessary for the diagnosis of MTBI. The Glasgow Coma Scale measures levels of conscious to assess initial severity, not to rule out traumatic brain injury. However, some degree of altered consciousness must be present before diagnosing MTBI. How long the altered consciousness lasts is relevant, as is loss of memory of events before the trauma. Matthew Hogg, the other passenger in the accident vehicle, said the plaintiff was dazed, shaky and "out of it". She did not know what had happened. She could not undo her seat belt. The plaintiff described gaps in memory, and Mr. Cikojevic said the plaintiff was not making sense at the hospital. Altered consciousness clearly occurred in this case.
>
> **B. What does "Mild" Refer to?**
>
> **251** Although experts sometimes disagree on whether to call an injury a mild concussion or a MTBI, either term is suitable.
>
> **252** "Mild" describes the severity of the organic injury, not its effect.
>
> **253** Although the organic severity of an injury usually associates with the severity of symptoms, sometimes symptoms can be severe while the organic injuries to the brain are mild.
>
> **254** Upwards of 85% of people suffering uncomplicated MTBI recover within six months. The recovery range lies between 85% and 95%, depending on the expert's views and the literature they accept. I find that around 90% of people suffering uncomplicated MTBI recover according to scientific literature. However, as noted by Dr. Anton, such statistics are of no value when dealing with a patient who falls into the subset of people who never fully recover. Each case must be evaluated individually.
>
> **255** The cognitive and emotional effects of MTBI can severely disable and impact the injured person's life.
>
> **C. MRIs and Imaging for Brain Damage**
>
> **256** If Magnetic Resonance Imaging (MRI) produces a negative scan, it does not rule out brain injury. Despite much improved MRIs, resolution remains far too low to show cellular damage such as axonal shearing. As Dr. Anton explained in his November 5, 2009 report, mechanical forces that propel the brain back and forward and rotate it can cause shearing of cells. Shearing can produce small bloodstains (hemosiderin), which MRI scanning may show. However, damage at the level of the axon is too microscopic to show up on an MRI. Even if hemosiderin shows up at first, within 18 months the bloodstains may be absorbed and no longer show up on an MRI. The plaintiff's MRI was more than 18 months past the accident date.

D. Neuropsychological Testing for Brain Injury

257 As discussed later, the initial battery of neuropsychological tests on the plaintiff in 2007 produced test results consistent with brain injury. Dr. Cohen, who administered the first set of tests, thought the test results implicated the frontal lobes, though the defendant does not concede this. A second battery of tests given the same year also produced results indicative of brain injury, though the defendant does not concede this either. A third battery of tests two years later showed improvement and produced results falling within the normal range.

258 However, neuropsychological testing can produce normal scores in people with diagnosed MTBI. Neuropsychological tests are least sensitive to deficits in the brain's executive functioning, which the frontal lobes control. Executive functioning involves, among others, organization and planning, control of impulses and emotion, focus, initiative and judgment. These are some of the areas where the evidence shows the plaintiff still has trouble.

259 Neuropsychological testing measures a person's highest cognitive capacity. Testing takes place in a quiet room that allows the person to concentrate on the test, so the results are not a good predictor of how the person will function in the uncontrolled settings of everyday living.

260 Mental or physical fatigue can lower some scores, and test results can vary from one day to another. Similarly, conditions such as depression, the psychological effects of a traumatic experience, and chronic pain can influence test results.

E. Depression and Psychological Effects of MTBI

261 Depression and psychological problems can produce a collection of symptoms that mimic MTBI and depression and psychological problems commonly develop after brain injury. Neurologist Dr. Tessler expressed his own view on the possible reasons brain injury victims commonly suffer psychological problems, saying that people get psychological problems because they do not like being brain injured. As explained by Dr. Anton, the way an injured brain physiologically responds to injury can produce changes that predispose the injured person to depression. This cross-over or mimicking effect can become entangled with brain injury symptoms and make diagnosing a brain injury challenging.

Following this review Brown J. went on to note that the plaintiff had completed post secondary education and was employed at the time of trial. However, Brown J. held that the plaintiff managed these achievements only because of numerous supports that were in place to assist her. Brown J. awarded $251,525 for costs of future care.

Significant awards for costs of future care are becoming more common in MTBI litigation. Another example is *Lines* v. *Gordon* where Lander J. awarded the plaintiff over $1 million for future care costs.[99] The standard of proof for cost of future care is not what is "medically necessary." The test is what is "reasonably justified" on the medical evidence.[100] This is an important distinction because the type of future care required in a MTBI case may not be medically necessary but may be reasonably justified based on the nature of the deficits.

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".[101] 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.[102]

More recent statements on the value of collateral information from reliable sources can be found in the 2005 *Textbook of Traumatic Brain Injury*.[103] 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.[104] 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.[105]

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.[106] 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.[107]

Neuropsychological tests are particularly insensitive to deficits in executive functioning.[108] These types of frontal lobe injuries frequently appear in situations that are complex, novel and highly unstructured.[109] 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.[110]

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.[111] In *Warder* v. *Insurance Corp. of British Columbia*,[112] Bouck J. found collateral witness 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 plaintiff after the accident and therefore had little information of what the plaintiff was like before:

> 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]

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 a MTBI may be found.

In *Lines* v. *Gordon*,[113] the plaintiff suffered a blow to the head and experienced PTA as a result of a motor vehicle collision. There was no LOC. The defence neurologist stated that the symptoms the plaintiff experienced were entirely due to the medication he was taking, and in no way could be caused by a brain injury. Lander J. 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.[114]

In *Lines*, the judge awarded the plaintiff over $3.5 million in damages. This is an excellent example of the importance of collateral witnesses in MTBI cases.

More recently in *Cikojevic*, Brown J. stated:

> 262 The experts agreed that no test objectively measures MTBI. However, they agreed on the high value of credible testimony from witnesses who have seen the injured person performing in daily life, especially when they are doing something that strains their cerebral and emotional resources.

Remember that the trier of fact does not know what the plaintiff was like before the accident. 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 accident. Do this before introducing any medical evidence and before calling the plaintiff. In most cases, the plaintiff should not be called at or near the beginning of the trial. Wait until the collateral witnesses have testified how the plaintiff has changed following the accident and the experts have shown the judge or jury how this is consistent with a MTBI. The plaintiff should not be in the courtroom before testifying. Witnesses may not feel comfortable testifying about all of the post-injury changes of the plaintiff if the plaintiff is present in the courtroom.[115]

VI. CONCLUSION

MTBI litigation is challenging and costly. The lawyer must ensure that before accepting a retainer the case meets the applicable diagnostic criteria. 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. Remember 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.[116]

In the absence of positive neuroimaging, the most powerful evidence in a MTBI case is collateral witness evidence of significant cognitive, emotional, and behavioural changes following the traumatic event. If a client has sustained a MTBI, the lawyer should be able to find several compelling witnesses who 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 "miserable minority", the 10 to 20 percent of persons who never recover from a MTBI.

1 This paper is an update of "Traumatic Brain Injury: What the Lawyer Needs to Know" presented at TLABC's conference Traumatic Brain Injury Litigation: Medical and Legal Issues, March 28-29, 2008, Vancouver BC. I would like to thank Nicole Kelly and Simon Collins for their assistance with this paper.

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