To understand mild traumatic brain injury, one must first consider carefully its definition. As the name implies, brain injury involves some damage to the tissue of the brain. Damage however does not connote an irreversible structural change. Damage also does not require visible evidence of tissue damage, although of course as brain injuries increase in severity, this becomes more evident. The brain and its associated structures are very sensitive to changes in blood flow, mechanical injury, and to the presence of toxins. Blows to head or rapid acceleration/deceleration of the brain can result in any of the above, or all three.

Brain injuries can be graded in their severity. The severity of a brain injury, at least in terms of traumatic injury typically depends on the speed and force of the trauma involved. As physical forces increase on the structures of the skull, they are translated into physical forces that immediately impact upon the brain and its functioning. The brain is a jelly-like structure encased in hard bone and other brain coverings referred to as the meninges. It is bathed in the cerebral spinal fluid and is able to flexibly alter its shape to some extent because of the ventricular system, which is comprised of cerebral spinal fluid. When there is a blow to the head or when the head rapidly accelerates and decelerates such as in a whiplash type accident, the brain literally moves within the cranium and may stretch neuronal tissue. This movement of the brain within the cranium can also result in contusions on the surface of the brain arising from scraping of the interface between the brain and the bony surfaces. This is typically more pronounced in the frontal and temporal regions. This is because the bony surfaces there are rougher and lead to greater friction. As a brain injury increases in severity, one expects more contusions or bruises in these particular areas of brain. Damage of course, depending upon severity of injury, is not limited to that but may result in stretching and shearing of neurons in other areas as well.

Mild traumatic brain injuries are typically evaluated on the basis of the immediate signs and symptoms that occur to mentation subsequent to the injury itself. The severity of the brain injury is directly related to the degree to which consciousness is altered and the amount of time that consciousness is altered subsequent to the injury. This alteration of consciousness post-injury is of major significance in defining the severity of brain injury, or in this particular case, mild traumatic brain injury. For unknown reasons however, it may not always tell the entire story. In some instances, later investigations or imaging studies may demonstrate that in spite of relatively insignificant evidence of alteration of consciousness, there still have been significant lesions produced in the brain as a consequence of trauma. These so-called complications may result in consequences that are well beyond what might have been expected on the basis of levels of consciousness post-injury.

Attempts have been made to clarify the objective evidence for mild brain injury, at both the World Health Organization and the American Congress of Rehabilitation Medicine.
In each instance, diagnostic criteria have been established in order to develop a common nomenclature and assist in the development of more accurate prognoses. This then helps in the more immediate management of a brain injury and of course has implications for short range pathology and longer range outcomes.

I will now outline the definition as provided by the American Congress of Rehabilitation Medicine:

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

The World Health Organization Task Force has produced an analogous definition:

“MTBI is an acute brain injury resulting from mechanical energy to the head from external physical 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.”

In an ideal world, one would be able to rigidly follow the Task Force or Congress recommendations assiduously. In reality, these recommendations must be seen as guidelines, especially since there is some variability in what may actually occur at the time of an emergency or brain injury. There is obviously going to be a timeline between extending between the time that there is a blow to the head and the time that measurements of consciousness can initially be made.

The most widely used system to evaluate levels of consciousness following a brain injury is the Glasgow Coma Scale (GCS) score. This scale is based upon three observations made by a qualified attendant, usually an ambulance attendant at the outset. The first criteria has to do with eye opening, and this is scored on a scale of 1-4.
with 1 meaning the eyes are not open to 4 being spontaneously open. The second
criteria is the best verbal response, which is scaled from a low of 1 equaling none to 5
which equals normal conversation. The third element is the best motor response, which
is scaled from a level of 1 being none to 6 being normal. These three scores are then
added to render the full GCS score which has a minimum of 3 and a maximum of 15.
GCS scores may then be serially measured by ambulance attendants at given times
post-injury. Measurement may continue to be made in the Emergency Room and
through initial care in hospital. The tracking of the GCS score allows one to observe the
recovery from brain injury and to make further prognoses. GCS scores initially ranging
between 13 and 15 are thought to represent indications of mild traumatic brain injury or
concussion. Those below the threshold of 13 are thought to represent more moderate to
severe brain injuries, which have poorer outcome prognoses. It should be noted that
there can be confounding variables that might affect the GCS score such as sedating
medications and/or alcohol or other drugs in the blood stream of the victim in question.
These confounding factors must be considered when using the GCS score to
prognosticate.

The severity of any brain injury can thus be in part assessed not just as a consequence
of the absolute GCS score, but the chronicity of suboptimal scores. To the external
observer, an individual with a GCS score of 15 would appear conscious, but yet at the
same time, may still be in an altered state of consciousness. Individuals suffering brain
injuries including mild brain injuries typically will be amnestic for the event itself and
potentially for some period of time following the event. There may also be some
amnesia for the timeframe immediately preceding the incident referred to the period of
retrograde amnesia.

Retrograde amnesia is typically shorter than the typically longer period of post-traumatic
or anterograde amnesia. Post-traumatic amnesia (PTA) has also been demonstrated to
be a very sensitive predictor of outcomes in brain injury cases. Longer periods of post-
traumatic amnesia have been associated with brain injuries of greater severity.
Unfortunately, estimations of PTA have been typically unreliable often because of the
presence of sedating medications, and also because psychometric tools that could be
used for this purpose are typically not. It is thus the case that there is likely to be
disagreement among experts as to exactly what might constitute PTA in any particular
case, whereas GCS scores are typically seen as being more objective.

In the case of a mild traumatic brain injury, one should thus expect reasonably higher
GCS scores and shorter periods of post-traumatic amnesia with minimal if any
retrograde amnesia. Questioning of patients in the Emergency Room and by trained
personnel early on may help to establish a period of anterograde amnesia in a case
where there are otherwise observations of a conscious individual with high GCS scores.
It is thus conceivable based upon the definitions I have outlined for an individual to
sustain a mild traumatic brain injury in spite of normal GCS scores and the observation
that they are conscious. The key point in defining the evidence from mild brain injury in
my view stems from reliable evidence that there has been a breach in the train of
thought brought on directly by the injury in question. The depth of this breach and the
length for which it occurs correlates with the severity of the injury and the likely outcome.

MILD INJURY ASSUMPTIONS

I would now like to turn my attention to some of the assumptions conceptually underlying the notion of mild brain injury. The first of these has to do with the degree to which there has been tissue deformation in the event in question. As tissue deformation becomes more severe, one expects that neuronal axons will reach their breaking point and sheer. It is thought that the deformation involved in a mild traumatic brain injury is going to result in stretching of neurons, which has its own sequelae, as opposed to sheering which obviously results in permanent changes in the wiring of the brain and therefore the processing of information.

Mild traumatic brain injuries are also less likely to produce secondary sequelae such as hemorrhaging. Hemorrhaging can also have its own consequences such as increased intracranial pressure and reduced blood flow in the remainder of the brain resulting in anoxic effects. Motor vehicle accidents can be particularly problematic in that there may be injury to multiple areas of the body and there is always the possibility of fractures of long bones resulting in the release of fat emboli, which when travelling to the brain can do damage quite apart from whatever might have occurred arising from the blow to the brain itself.

In diagnosing a mild brain injury, one is thus looking for both inclusion and exclusion criteria. The diagnosis of mild brain injury becomes particularly important in litigation because it is the view of most experts that there will be full recovery from such a brain injury. Prognoses regarding mild brain injury become less certain, especially when there is evidence of complications which might become evident later on in imaging studies. As I have already indicated, a mild brain injury with complications, as suggested by visualized evidence of hemorrhaging in different parts of the brain may well lead to a different outcome than would a brain injury without such complications. In point of fact, an individual with these complications may demonstrate permanent deficits where one would not expect such an outcome in an individual with a pure mild traumatic brain injury. It is evident there might be debate about the significance of any particular small area of bleeding in the brain. What I believe is more important to recognize here is the fact that it is the presence of such bleeding and not the specific localization of that bleeding that is more important in developing the prognosis.

It is well understood that many areas of brain dysfunction may arise in the absence of bleeding. Thus, it is my view that the mere fact that there has been bleeding suggests to me a whole new level of severity of injury and a greater likelihood that there will be some evidence of permanent impairment, whether or not it is localized in line with the position of the evident blood products. It may well be the case that blood products (hemosiderosis) in a particular location of brain does not prognosticate major changes in mentation. However, the fact that those blood products arose from a particular injury
might well prognosticate more significant damage in many areas of brain than might have been thought possible by the particular localized complication.

CONCUSSION - ANOTHER TERM FOR MILD BRAIN INJURY

At the outset, I believe it should be recognized that the terms concussion and mild traumatic brain injury are synonymous. Nevertheless depending upon the medical or legal venue, the terms while being interchangeable are typically chosen depending upon the context in which they are being used. In the athletic context, concussion is a term that has been used in the long term rightly or wrongly, and in fact, it may be the case that as it came to be recognized that a concussion was in fact a brain injury, the seriousness of the condition came to be better appreciated.

According to the Colorado Medical Society guidelines, concussions may be graded on three levels noting severity of that concussion. According to the Colorado Medical Society guidelines, the following symptoms are associated with grades 1, 2, and 3 concussions:

- **Grade 1**
  - Transient confusion
  - No loss of consciousness
  - Mental status abnormalities resolve in less than 15 minutes
- **Grade 2**
  - Transient confusion
  - No loss of consciousness
  - Mental status abnormalities last more than 15 minutes
- **Grade 3**
  - Any loss of consciousness, brief or prolonged, seconds or minutes
  - One can see here the similarities between the guidelines from the American Congress of Rehabilitation Medicine and the World Health Organization. All of these systems of diagnosis have one thing in common, and that is that severity of injury is presumed to be greater as the level of consciousness becomes more impaired.

BIOMECHANICAL BASIS OF INJURY

At this point, I would like to delve more into the meat of the issue. As I have indicated earlier, deformation of brain tissue and the degree to which that deformation occurs is the key factor in understanding the severity of any brain injury. The greater that deformation, the more severe the injury. At some point, deformation will be so great as to result in the loss of tissue integrity. In between the time that there is deformation and complete loss of tissue integrity, there will be varying degrees of stretching and sheering. As the sheering becomes more prevalent, the longer term ramifications of the injury become more significant. In the absence of a direct blow to the head, shaking of the brain as already indicated can result in stretching of neurons and evidence for mild traumatic brain injury. The more stretching, the more problematic the biochemical
changes in the brain, which further result in more prolonged immediate brain injury sequelae.

In the personal injury context of law, many of the injuries arise from motor vehicle accidents. Significant rear end accidents where whiplash associated disorders are common can result in very significant acceleration/deceleration changes of the brain within the skull. These have been demonstrated to be most pronounced in animal models when the head is allowed to fully rotate to a position where the chin is on the chest. Minimizing the distance the head can travel through the use of headrest properly adjusted can help in reducing the consequences of such injuries. My point here is that mild brain injuries can be produced in the absence of a direct blow to the head, but in the presence of severe acceleration/deceleration forces (especially rotational forces) placed upon the contents of the cranium.

At this point, I think it is helpful to understand the morphological changes in neurons arising when unusual forces are applied to them. Neurons obviously have some flexibility and thus, every force will not necessarily result in stretching or sheering. The greater the force however and the rate at which that force is applied determines the degree to which there will likely be axonal damage. It is also the case that with enough stretching force, there might well be axonal damage that still results in the death of that axon. Consequences of axonal death can be viewed microscopically, but obviously this is not something that is done in living brain injuries to this point. It is thought that damage to axons occurs when forces have been sufficient to change the transport of axoplasm within the cell. Ultimately the degree of impairment will depend upon the number of axons that are damaged and/or destroyed.

NEUROPSYCHOLOGICAL METHODS

There are several key aspects to consider for neuropsychological investigations. Among these are: the history of the case; clinical interview; behavioural observations; observations of significant others; and finally neuropsychological test findings. The clinical history helps to put the entire case in perspective. From a neuropsychological standpoint, it is most critical to determine whether there is any history of abnormality in the individual in question prior to having sustained a brain injury. In addition, it also important to look at pre-injury psychological issues, if any, to ascertain how they might also play a role in current functioning. Patients can have brain dysfunction for a variety of reasons including genetic abnormalities, birth injuries, other traumatic injuries or toxic states arising from poisoning and/or other medical conditions. Medical historical data and information from the patient and significant others can shed light on the possibilities of preexisting dysfunction. Academic and work history information can also be helpful in establishing levels of function prior to any accident in question. It is also helpful at times to obtain the observations of significant others who have intimate knowledge of the client in question.

As a part of taking the history of course, one wishes to obtain the client's point of view as to how they were functioning prior to any incident and how they are now functioning
subsequent to it. A very key issue in gathering the history, especially in traumatic events is information with regard to changes in level of consciousness, if any, at the time of the incident in question. The history thus provides a context in which other information can be evaluated. Historical information can obviously be gleaned from various records including academic, medical, vocational, social service or whatever records might be available, but beyond this, information is gathered by direct questioning of the client in the clinical interview. The clinical interview serves not only as an information gathering tool, but also as a means of directly evaluating the behaviour of the client during a relatively structured and formal interaction. Throughout the clinical interview and during any testing, behavioural observations are made of the client in question.

The general affect and mood of the patient are noted and their engagement in the history taking and assessment process are carefully recorded. Obviously behaviours that are unusual in the testing context are also noted, and the degree to which behavioural factors may interfere with testing is a most important aspect of the evaluation. Lack of engagement and distractibility can clearly impact on multiple test findings leaving the examiner with potentially unreliable data. Individuals who have a direct and intimate knowledge of the client may also shed light on daily behaviour that is not necessarily seen during the office visit or testing. Collateral information from significant others can be obtained by interviewing them, and by the completion of questionnaires, which may have their own validity indices. It is thus always interesting to know how the patient sees themselves, how others see the patient, and then finally, how the neuropsychological testing might categorize the patient.

Neuropsychological testing may require 7 to 10 hours of face to face contact with the client. Testing includes measures to assess: basic arousal and attention, perceptual abilities, speech and language, memory, motoric abilities, abstract reasoning and problem solving abilities, general intellect, academic achievement and current emotional adjustment. The combination of tests in the test battery is designed to illustrate potential patterns of deficit and impairment that might arise from brain injury or other problems. As a result of the neuropsychological methods, a neuropsychologist attempts to meet a number of objectives in terms of basic referral questions.

In a litigation context, the most basic question is whether in fact there has been any evidence of a brain injury, and in many respects, this relies upon the history and clinical interview. Beyond this, another important question is whether or not the information we have been able to gather about the client represents reliable and valid indicia of either their current functioning or their capabilities. Assuming that the data are in fact reliable and valid, one can then proceed to quantify levels of impairment in the various areas of brain and evaluate emotional adjustment and/or the presence of psychopathology either related to or unrelated any injuries in question. When one quantifies impairments, one of course also needs to take into account whether or not the impairments appear to be proportionate to the injury as understood on the basis of history and patient self-report. It is the role of the neuropsychologist at that point to decide whether the observed test findings likely represent alterations in brain function and what the most probable cause for those changes in likely to be.
Individuals can be repeatedly tested in order to evaluate recovery or conversely, deterioration. In the assessment process, it must be borne in mind that medication and lesions may have differential effects that are noteworthy. Ultimately at a point where full recovery is expected, one assesses the ultimate levels of damage or impairment that might have arisen from any injury in question, and of course recommendations for care naturally follow from an understanding of the likely permanent limitations.

ASSESSMENT IMPRESSIONS

As a consequence of comprehensive neuropsychological testing, individuals may demonstrate entirely normal performance or may demonstrate impairments of function in a variety of specific areas. One of the key questions for the neuropsychologist is whether the impairments identified are likely neurologically based or of non-neurological origin. Neurologically based impairments are typically consistent in terms of the pattern of findings and proportionate to the severity of the injury. Disproportionate impairment raises questions as to the likely basis for that impairment and internally inconsistent findings raise questions as to reliability. Often times, individuals are tested more than once and this allows for a comparison to be made between test sessions again leading to impressions with regard to consistency of results. Typically, a consistent pattern of findings is more suggestive of a neurologically based impairment.

Non-neurologically based impairments are conversely more likely inconsistent and disproportionate to medical records. Non-neurologically based findings may also arise from various confounds such as medications, illicit drugs, and of course litigation. The state of mind of the client at the time of the assessment becomes very important in obtaining solid neuropsychological data where non-neurological factors are at a minimum. Agitated, unhappy clients will most probably produce findings that are not reliable and a consequence of their unhappiness. Individuals who are in a state of chronic pain and/or having chronic sleep deficits may also exhibit impairments that are not reliable and not necessarily related to any specific brain injury.

Unfortunately, it is also the case that individuals may at times engage in acts of deception in order to obtain secondary gain. I must state however that this is relatively rare. It is not rare however to find individuals whose test findings are not readily explained by neurological phenomena. Individuals can demonstrate impairments for a wide variety of reasons that are not neurologically based. It is also more common for individuals to present with greater deficits than are truly there without necessarily engaging in an act of deception. As a part of the assessment, it is the neuropsychologist's role to ascertain the likelihood that other factors are or are not contaminating findings.

In the litigation context, clients may potentially be malingering in terms of cognitive performance and/or psychopathology. Different measures are employed to look at these aspects separately, although there is some basis for recognizing that the odds are more likely of some overlap here. Malingering is a conscious act of deception. The motive for
that deception is really not important, and psychologists cannot really tell what the motive for malingering might be. When an expert states that a client is malingering, the basis for that judgment needs to be questioned. Since we can never truly know the motives of the client, it is my view that we need fairly strong evidence to draw the conclusion that they are engaging in a conscious act of deception.

Response bias measures that have normative standards can certainly assist us in looking at this issue but only in so far as this recognizes that the conclusions from such data are really limited. There are always a number of reasons that can be pointed to for why an individual will fail a normatively standardized response bias measure. One way to evaluate the possibility of malingering is to determine whether or not performance on any particular response bias measure falls significantly below chance expectations. When this occurs, one can argue that the client has likely known the correct answer and at the same time, chosen to provide an incorrect answer for any item in the response bias measure. For me, this constitutes objective evidence of malingering, but short of that, I am not really prepared to draw that conclusion. I am readily prepared to draw the conclusion that the results cannot be relied upon and disability cannot be assessed when in fact there is considerable evidence of response bias, regardless of whether the individual meets the threshold for conscious malingering.

RECOVERY FROM BRAIN INJURY

It is generally held that individuals suffering from mild traumatic brain injury or concussion will have a full neurological recovery from that brain injury. There are however significant differences in time course of the recovery and most typically, this is related to the severity of the injury in question. Injuries with more pronounced amnesia and lower levels of consciousness for more protracted periods would be expected to have longer recovery periods. There are a constellation of symptoms often associated with the head injury including headaches. Many individuals suffering from mild traumatic brain injury or concussion may appear to recover almost immediately, while others may take longer to recover. There appears to be less debate as whether there will be recovery, but more debate over the time course of that recovery.

It is thought that many individuals have recovery of symptoms within three months post-accident, but this is in many instances an underestimate. I have seen individuals with persisting symptoms of head injury for more than a year post-injury. Having said that, I would point out that it is most unlikely that an individual would suffer persisting brain injury sequelae beyond two years post-injury. There is however a small group of individuals, perhaps 5% of the brain injury population, that develops more intractable symptomatology. Nevertheless, it is difficult to separate many of the post-concussion symptoms from other physical symptoms arising from accident and injuries.

In point of fact, many of the same symptoms arising in a concussion may arise from whiplash syndrome alone without brain injury. Litigation and compensation issues may lead to prolonged symptomatology. It must be borne in mind that individuals involved in motor vehicle accidents where there is typically litigation are often injured in multiple
ways and additionally are often psychologically traumatized by the event in question. The persistence of pain symptomatology following these accidents and the emotional distress arising from trauma coupled with medications used to treat those problems often leads to accident related symptomatology that is in many respects similar to brain injury symptoms, but instead related to non-neurological accident/injury sequelae. Individuals may be symptomatic from a standpoint of behaviour, emotional distress and cognitive impairment in the absence of brain injury. Ultimately, one of the key questions in litigation is to determine whether the accident or event in question is the factor that results in changes in cognition and changes in psychological wellbeing. The typical question asked is whether were it not for the accident, these symptoms would or would not be ongoing. Individuals who suffer mild brain injuries and who have persisting cognitive symptomatology beyond the two year point are by and large likely having those symptoms for reasons other than the brain injury, but may be having them for reasons directly related to their accidents and injuries nonetheless.

The question at that point becomes the degree to which the symptoms are likely to remain chronic given they are not directly related to brain injury. There is really nothing to say that non-neurologically based chronic symptoms are less intractable than those that are neurologically based. Ultimately, this issue boils down to what is projected for this particular individual given the particular circumstances of the case and the levels of persisting symptomatology, in particular when they are occurring beyond two years post-injury. It should be obvious that the issues of mild brain injury, especially in the context of a motor vehicle accidents, is very complex and as a consequence, there are no formulaic answers readily applied to all cases. Individuals who are more vulnerable from a cognitive and/or emotional standpoint prior to their accidents are more likely to have pronounced and prolonged sequelae to accidents and injuries. The availability of treatments for these individuals and their ability to benefit from such treatments is also a key factor in understanding the chronicity of symptomatology following accident injuries, and in particular a mild traumatic brain injury.