



# Head Injury Update

## Mild Traumatic Brain Injury: An Introduction

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*"We see what we look for. We look for what we know"*

Johann Wolfgang von Goethe

### INTRODUCTION

In a Supreme Court Trial several years ago, a medical expert was carefully explaining to the Judge the pathophysiological mechanism of a traumatic brain injury. The Judge turned to the expert and remarked, "the next thing you will be telling me is that you can have brain damage from a whiplash type injury." The expert responded, well, yes you can. The Judge, obviously not impressed by this evidence, awarded the Plaintiff \$20,000.00 in damages.

Minor head injury is the most common, yet least understood, of the three gradients of head injury (i.e., minor, moderate, severe).<sup>1</sup> As a result, victims of minor head injury have been largely ignored by both the medical and legal professions. Neurosurgeons, Jennett & Teasdale in their internationally recognized text, *Management of Head Injuries*, published in 1981 comment:

Our conclusion is that the damage done by, and the symptoms subsequently suffered after, mild head injuries are frequently underestimated. Several factors contribute to this. One is that many of the hospital doctors who deal with mildly injured patients are unfamiliar with recent work in this field, and in any event are not used to dealing with the largely subjective complaints that are the feature of these patients' persisting disability. On the other hand, those who are accustomed to dealing with severe head injuries are apt to view the mildly concussed patient as fortunate to have escaped serious brain damage - a comparison of little significance to the patient.<sup>2</sup>

A minor head injury is typically defined by one of the following criteria: a Glasgow Coma Scale (GCS) score of 13 or more; a period of altered consciousness or a period of posttraumatic amnesia (PTA) of less than one hour.

#### Glasgow Coma Scale (GCS)

The GCS is a 15 point scale for assessing levels of consciousness.

#### GLASGOW COMA SCALE

##### Eye Opening

spontaneous	E 4	to speech	3
to pain	2	nil	1

##### Best motor response

obeys	M 6	abnormal flexion	3
localizes	5	withdraws	4
exterior response	2	nil	1

#### Verbal response

orientated	V 5	confused conversation	4
inappropriate words	3	incomprehensible sounds	2
nil	1		

Coma score (E + M + V) = 3 to 15

#### POST TRAUMATIC AMNESIA

The period of PTA is the interval between the injury and the time when the patient begins to lay down continuous memory of ongoing events. The PTA includes the time during which the patient was awake but confused. The following scale was intended to provide a rough measure of the severity of a head injury:

##### PTA

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

The problem with these criteria is that some persons suffer from persisting debilitating deficits after receiving a minor head injury notwithstanding a high GCS and no loss of consciousness.

Researchers have concluded that the GCS may not be the most valid and sensitive indicator of a mild brain injury and have suggested that a more sensitive measure is required because of the potential long term deleterious effects in the mild head injured group of patients.<sup>3</sup> Dr. Brian Jennett, the originator of the GCS, makes the point that it was designed to classify severe injuries:

...it 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]<sup>4</sup>

#### POST CONCUSSION SYNDROME

The usual symptoms associated with a minor head injury include headache, impairment in attention and concentration, poor memory, depression and emotional instability, lowered frustration tolerance, sleep disturbances, reduced sexual drive and intolerance to alcohol.<sup>5, 6</sup> This constellation of symptoms has come to be referred to as postconcussion syndrome or posttraumatic syndrome. Medical assessment of minor head injured individuals will generally fail to detect any of the usual indicators of brain damage such as loss of consciousness, positive neurological findings or positive neurodiagnostic test results. Consequently, patients complaining of these symptoms weeks, months and years after trauma are usually dismissed as suffering from traumatic neurosis or even labelled as outright malingerers.<sup>7</sup>

Dr. H. Davis, a Clinical Assistant Professor in the Department of Psychiatry at the University of British Columbia, in an article published in the *Advocate*, makes the point that it is difficult for the physician to decide whether brain damage or psychogenic influences are responsible for the clinical picture presented. He suggests that while organic factors may be responsible for the early symptoms, the prolongation of symptoms is likely due to psychogenic factors exacerbated by medical and legal consultations when compensation claims are pending. He states:

Such ambiguities arise in minor head injuries where coma

is brief, if at all, physical disability absent and neurological investigations negative.

Covert causal factors, often not immediately obvious, may become apparent when the total situation is reviewed. Thus, when a reaction following an injury is disproportionately severe and runs counter to what would be expected in view of the patients premorbid stability, it may be tempting to postulate brain damage even though no hard evidence can be obtained in support. However, one often finds a hidden theme or motive which may more readily account for such disturbance and often iatrogenic effects are found to be responsible. Iatrogenesis implies the causation of symptoms by physicians and other well meaning persons with an overemphasis on special investigations and a resultant overemphasis of an injury. Repeated medical and, incidentally, legal consultations negatively reinforce the situation as, especially when litigation is operative, symptoms are kept on the boil, both physical and psychological. *It is thus highly recommended that any court proceedings be dispensed with as soon as possible and the patient allowed to continue with his or her normal life [emphasis added].*<sup>8</sup>

Investigators involved in a head trauma research program at New York University Medical Centre provide a different perspective. They concluded:

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.

We discovered, as others had reported, that these patients appeared fine until they attempted to resume their responsibilities at home, work, or school. When they did so, a significant number experience 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. Their doctors were unable to find anything wrong with them, and they were thought to be having psychiatric problems - or worse yet, to be malingering. They became the bane of neurologists, psychologists, psychiatrists and vocational counsellors, all of whose usual techniques did not produce positive results.

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 behavioural deficits that seriously interfered with their ability to lead fully functional lives.

...CAT Scans and neurological examinations reveal no observable damage to the brain that can be localized to a particular region. Yet there is evidence that the subjective and cognitive problems encountered by some persons after minor head injury may have an organic basis. This is important information because it means that treating such problems as if they were purely psychological in nature will not make the core problems disappear.<sup>9</sup>

In a discussion of the posttraumatic syndrome, Canadian neurologists William Pryse-Phillips and T.J. Murray, in their text *Essential Neurology*, support the conclusions of the New York

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#### University researchers:

There has been a medicolegal controversy for years as to whether this is primarily organic or psychogenic, but evidence is accumulating that it has an organic basis in damage to fiber tracts in the upper brainstem.

There are a few myths attached to the syndrome, including the widespread belief that it does not occur with severe injuries, that the prognosis is usually good, and that the patients are merely seeking compensation (as the symptoms may disappear when the claim is settled and it is less commonly seen after amateur sporting injuries). *Although long-standing, such opinions cannot be substantiated and these patients have been harshly treated by the courts and by our profession for many years. The development of the syndrome does not relate to possible compensation or insurance payments* [emphasis added].

The relationship between this syndrome and the previous personality of the patient is uncertain, but the personality change now incurred, with irritability and depression, does influence the other complaints. The prognosis is not necessarily good, and although many of the milder cases clear within 6 months, other patients may never completely lose their symptoms.<sup>10</sup>

Further support for an organic rather than a psychogenic explanation can be found in the recent text, *Rehabilitation of Post-Concussive Disorders*<sup>11</sup> and in a recent article by Evans, "The Postconcussion Syndrome and the Sequelae of Mild Head Injury. The Neurology of Trauma."<sup>12</sup>

#### MINOR HEAD INJURY OR MILD TRAUMATIC BRAIN INJURY (MTBI)

One of the reasons for this medicolegal controversy may be the terminology employed in the literature. In the last decade, researchers have often used the term minor head injury and mild traumatic brain injury (MTBI) interchangeably. The second issue of the *Journal of Head Trauma Rehabilitation* in 1986 was devoted to the topic, "Minor Head Injury". In 1993, the Journal devoted a second issue to the same topic but with the new title, "Mild Traumatic Brain Injury". Dr. Thomas Kay in his article "Neuropsychological treatment of Mild Traumatic Brain Injury" explains that the two conditions are not identical and that symptoms may emanate from either or both conditions:

Minor head injury refers to an injury to the head, face, and neck area with symptoms caused by damage to the skull, scalp, soft tissues, or peripheral nerves but where there is not necessarily injury to the brain. MTBI refers to a minor head 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.<sup>13</sup>

A new definition of MTBI has recently been developed by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine. This definition appears in a recent issue of the *Journal of Head Trauma Rehabilitation*<sup>14</sup>:

##### Definition

A patient with mild traumatic brain injury is a person who has had a traumatically induced physiological disruption of brain function, as manifested by *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

This definition includes: 1) the head being struck, 2) the head striking an object, and 3) the brain undergoing an acceleration/deceleration movement (ie, 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.

### DIFFUSE AND FOCAL DAMAGE

Two types of injuries can occur following MTBI: diffuse injuries and focal injuries.<sup>15,16</sup> Diffuse brain damage can result from a blow to the head or as a result of a whiplash type injury. In these injuries, it is not the blow to the head that is significant, but the acceleration or deceleration of the head which produces strains and distortions within the brain resulting in a shearing or stretching of nerve fibers throughout the brain. Thomas Gennarelli, Associate Professor of Neurosurgery at the University of Pennsylvania, and one of the leading researchers in this field, stated:

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 fibres and the consequent axonal damage that now appears to be the substrate of concussive brain injuries.<sup>17</sup>

Researchers have clearly demonstrated that whiplash type, mechanical acceleration-deceleration injuries in primates produce observable brain damage.<sup>18, 19, 20, 21</sup> Rotational acceleration appears to be the primary mechanism responsible for the production of diffuse brain injuries.<sup>22</sup> Similar brain damage has been observed in humans. Oppenheimer examined the brains of individuals who sustained minor to severe head injuries who died within several days of causes unrelated to the head injury. He found evidence of microscopic brain damage in cases where the cerebral injury was clinically trivial, consisting of a concussion which lasted for a few minutes. Oppenheimer concluded:

Following a head injury, diffuse microscopic lesions can be seen in the high proportion of human brains. ...They are believed to be mechanical in origin, and can be attributed to

- (1) surface sheering and contusions;
- (2) stretching and tearing of small blood vessels;
- (3) stretching and tearing of groups of nerve fibres;
- (4) tearing of nerve fibres by a crossing vessel.

They are seen, not only after severe trauma, but also in cases of "concussion". Detailed studies of their sights and distribution could throw light on the mechanics of acceleration injuries of the brain.<sup>23</sup>

In a recent article titled, "Mechanisms of mild traumatic brain injury", neurosurgeons from the Department of Neurosurgery at the University of Texas discussed the neuropathological mechanisms of diffuse axonal injury:

#### Axonal injury

Clinical studies suggest that diffuse axonal injury may be a factor in mild to moderate TBI in humans. These neuropathologic studies suggest a continuum of diffuse axonal injury severity in which the lesions are mechanical in origin and are caused by stretching and tearing of nerve-fibers and small blood vessels. This postulated continuum

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of severity is also supported by neuropathologic studies in animals with experimental mild head injury. Experimental data suggest two viewpoints regarding the onset and development of axonal injury. It may be that axonal injury occurs at the moment of trauma by the shear or tensile forces physically disrupting the axons, leading to membrane retraction, extrusion of axoplasm and formation of large reactive swellings. On the other hand, it may be that a subtle and progressive axonal change after mild TBI eventually leads to swelling of axons and disruption of the neuraxis without initial tearing of the axons at the moment of trauma.

Substantial clinical and experimental evidence exists to support the theory that axons are consistently damaged in head injuries ranging from minor to severe. Until recently, investigators have focused primarily on the biomechanical aspects of axonal injury and have suggested that axons are almost preferentially vulnerable to mechanical forces generated with head injury. Gennarelli et al, in their early studies of experimental head injury in nonhuman primates, argued that diffuse axonal injury is the principal morphologic feature of TBI. Findings by Jane et al suggest that in some instances minor head injury or concussion can be associated with axonal injury. In animals sustaining an acceleration-deceleration non-impact injury, degenerating axons were noted in the inferior colliculus, pons, and dorsolateral medulla at 7 days after TBI. The finding of immediate mechanical axonal damage was based upon the identification of axon retraction balls, which are believed by most to form through the physical shearing or tearing of axons with subsequent retraction, extrusion of axoplasm, and formation of large reactive swellings.

Although mechanical forces are necessary to produce axonal injury, such injury may result from a biochemical cascade of pathophysiological events initiated by the mechanical events rather than formed at the moment of injury. Povlishock et al demonstrated that, as in the case of severe and moderate head injuries, mild head injury results in the genesis of reactive swellings within 12 to 24 hours of the traumatic episode. When comparably injured animals were examined before 12 hours posttrauma, however, no reactive swellings were found. To assess better the significance of such an apparent discrepancy, detailed light and electron microscopic studies were conducted to evaluate the intra-axonal anterograde transport of horseradish peroxidase over a 24-hour posttraumatic course. Through such an approach, it became apparent that the traumatic event did not tear or shear the axons to form retraction balls immediately but rather induced an initial, subtle form of axonal changes that then, over time, became progressively severe. Recent data suggest that axonal injury in humans is an evolutionary process.<sup>24</sup>

In diffuse mild head injury cases, there is generally believed to be a reduction in the overall speed, efficiency, execution and integration of mental processes.<sup>25</sup> This has been described as "reduced information processing capacity" by Gronwall:<sup>26</sup>

After MHI, patients have difficulty in all areas that require them to analyze more items of information than they can handle simultaneously. They present as slow because it takes longer for smaller than normal chunks of information to be processed. They present as distractible because they do not have the spare capacity to monitor irrelevant stimuli

at the same time as they are attending to the relevant stimulus. They present as forgetful because while they are concentrating on point A, they do not have the processing space to think about point B simultaneously. They present as inattentive because when the amount of information that they are given exceeds their capacities, they cannot take it all in.<sup>26</sup>

There is evidence that the severity of these deficits increases with repeated MTBI.<sup>27,28</sup> This may explain why some persons who seem to recover after an initial MTBI develop permanent sequelae after a subsequent MTBI.

Focal injuries occur when the soft brain collides with the rough, bony inside surface of the skull. The most common area for contusions to occur is in the frontal and temporal lobes.<sup>29</sup> Deficits are primarily in the areas of learning and memory, planning and organization, attention and concentration, and emotional control.<sup>30</sup>

A blow to the head can also produce a contusion to the brain at the point of impact (coup) or directly opposite the point of impact (contrecoup). Depending upon the location of these types of the injury, they can produce specific deficits such as problems with language, perception, sensory functions, motor functions, sensory-motor integration, and sequencing.<sup>31</sup>

### NEURODIAGNOSTIC TESTS

Lawyers are continually searching for more objective measures of brain damage in order to convince a Judge or Jury that an individual has in fact sustained traumatic brain damage. Recent developments in neurodiagnostic technology are beginning to show some promise in the diagnosis of MTBI. Examples of current neurodiagnostic techniques include:

**Electronystagmography (ENG):** Many head injured patients complain of vertigo and dizziness which could indicate damage to the vestibular or balance mechanism. An Electronystagmography is a neurophysiological test which provides objective data concerning this vestibular function.<sup>32</sup>

**Electroencephalography (EEG):** A method of recording alterations in the electrical activity of the brain.<sup>33</sup> The EEG has limited diagnostic value in MTBI cases.

**Computerized Axial Tomography (Cat Scan, CT SCAN):** The CT Scan is a computer generated reconstruction of the brain. It is particularly useful in identifying swelling within the brain, the presence of a subdural or epidural hematoma and areas of gross structural damage.<sup>34</sup> The CT Scan will not show damage less than one-half centimetre in size and consequently it will not show diffuse or microscopic damage in MTBI cases.

**Magnetic Resonance Imaging (MRI):** This technology was developed in the early 1980's. It provides images similar to the CT Scan. While the MRI has a higher resolution than the CT Scan and can show small lesions of one or two millimetres in size, it is unlikely to be of much diagnostic value in cases of MTBI.<sup>35</sup>

**Evoked Potentials, (Auditory, Visual, Somatosensory):** Evoked potentials provide information about how the nervous system receives and transmits sensory inputs.<sup>36</sup> Repetitive stimuli are presented to the patient and electrodes record the resulting cerebral electrical activity. Auditory evoked potentials are recorded when clicks are presented to each ear separately. Visual evoked potentials are elicited with a visual stimulus such as a flashing light or an alternating black and white checkerboard. Somatosensory evoked potentials are elicited by a mechanical or electrical stimulus given peripherally.

*(Continued on page 52)*

**Topographic Brain Mapping:** Brain electrical activity mapping is the topographic analysis of scalp recorded EEG or evoked potential data. The BEAM and the Biologic are two examples of brain electrical activity mapping machines. Topographic mapping provides a colorized picture of electrical activity in the brain. The advantage of brain electrical mapping over the traditional EEG and evoked potential recording is that it utilizes the computer to break down the electrical activity and then provides a statistical analysis to compare the results to a normal population. The originator of the BEAM, Dr. Frank Duffy, Associate Professor of Neurology at Harvard Medical School, made the following comment with regard to the application of the BEAM to clinical practice:

The utility of BEAM and topographic analysis in clinical practice lies in its increased sensitivity over traditional EEG and EP analysis and its increased objectivity in establishing the presence or absence of brain-related disease and/or disability. As such, it is an added weapon in the clinician's arsenal of diagnostic tools. We consistently caution the users of topographic techniques that BEAM does not provide a "stand alone" diagnosis, but rather one of several inputs into comprehensive clinical analysis. We feel, however, that it can provide a vital and, in many cases, unique contribution to the diagnostic process.<sup>37</sup>

For a MTBI case in which brain mapping and computerized evoked potentials were admitted at trial, see *Datta v. Rowan*, [1993] B.C.D. Civ. 3385-07 (B.C.S.C.)

**Positron Emission Tomography (PET):** A computerized scanning technique that produces a picture showing the distribution of radioactivity in the brain after the injection of a radioactive isotope.<sup>38</sup> Whereas the CT or MRI scan provides a static picture of brain structure, the PET scan reflects brain function by showing blood flow and metabolic activity in different areas of the brain. It provides an illustration of disruptions in brain metabolism by monitoring the amount of glucose that brain cells consume.

PET scanning is presently employed in daily clinical practice at the Brain Imaging Centre at the University of California, Irvine, College of Medicine. PET scans from the Brain Imaging Centre include quantitative colour images reflecting a computer analysis of 89 cortical and subcortical regions in each hemisphere of the brain. Each area is statistically compared to a normative data base to yield an objective survey of metabolic rates in different areas of the brain. This technique enables the examiner to visualize brain function while simultaneously monitoring the performance of the patient on various cognitive and behavioural tasks. The cost of a PET scan is approximately \$2,500.00 U.S. PET scanning in British Columbia is currently not available for clinical applications.

In a 1989 research article comparing CT scanning, MRI scanning, PET scanning and neuropsychological assessment in the evaluation of six traumatic head injury cases, researchers found that the PET scan demonstrated cerebral pathology not visualized by CT or MRI scanning and was particularly sensitive in identifying reduced frontal lobe function.<sup>39</sup> The PET results also corroborated the neuropsychological test findings.

In a follow-up study published in 1994, the researchers studied nine cases of MTBI where there was little or no evidence of brain damage according to non-functional neuroimaging tests such as the CT scan or MRI, yet the neuropsychological examinations were positive.<sup>40</sup> The PET scans documented brain damage in all nine cases. In four of the nine cases there was no documented loss of consciousness (LOC). In the remaining five cases LOC ranged

from less than one minute to twenty minutes. The researchers found no differences between the four cases with no LOC and the five cases with LOC. The authors stated that these case studies suggest that head trauma without LOC can cause significant changes in brain function.

**Single Photon Emission Computed Tomography (SPECT):** This is a scanning technique that utilizes the tomographic reconstruction method of the CT Scan and MRI together with the detection of photons emitted when a tracer is administered to the patient.<sup>41</sup> The SPECT scan has been referred to as a "poor man's PET scan", but recent technological developments suggest that the SPECT will emerge as a powerful window into the function of the brain.<sup>42</sup> A state of the art multi-headed SPECT system has recently been installed and is in operation at the Nuclear Medicine Department at St. Paul's Hospital. A SPECT scan can be conducted at approximately one third to one half of the cost of a PET scan. For a case in which a SPECT scan was relied upon see *West v. Cotton*, [1993] B.C.D. Civ. 3389 - 52 (B.C.S.C.)

### NEUROPSYCHOLOGICAL TESTING AND ASSESSMENT

The discipline of neuropsychology developed in the 1940's because of the insensitivity of the standard neurological examination to diagnose many cases of brain damage. While advances are being made in the development of more sensitive neurodiagnostic tests, neuropsychological assessment is still considered to be the most sensitive measure of brain damage. In the majority of minor head injury cases, the success of the trial will depend upon the evidence of the neuropsychologist. Familiarity with the neuropsychological literature enables the lawyer to present the case to the Judge or jury through authoritative texts and articles from recognized journals put to defence experts in cross-examination. In this manner it can be demonstrated that the cognitive, emotional and behavioural problems experienced by the plaintiff are consistent with the MTBI literature.

Recent studies utilizing neuropsychological testing clearly demonstrate that a subgroup of victims of minor head trauma suffer from persisting debilitating deficits. Yarnell and Rossie<sup>43</sup>, in a study published in 1988 in *Brain Injury*, investigated 27 patients who suffered apparent whiplash injuries in motor vehicle accidents. None of the patients were more than initially dazed from the accident and periods of posttraumatic amnesia were brief. All of the patients were employed prior to their accidents. Typical symptoms included neck and back pain, headaches, dizziness, lightheadedness, paraesthesias, sleep alterations, poor memory, concentration and attention deficits, distractibility, difficulty learning and retaining new information, loss of drive and initiation, increased irritability, decreased frustration tolerance and depression. Neurological examination and neurodiagnostic testing failed to locate any abnormalities. However, neuropsychological evaluations showed impairments on tests of cognitive flexibility, non-verbal reasoning, new learning, memory, psychomotor agility and attention. Eighteen months post injury, none of the patients tested had returned to their previous level of occupational function. Fifty percent were unemployed and the other fifty percent were working at a reduced capacity in terms of hours or income level. Active involvement in a lawsuit did not correlate with return to work.

A study published in 1990 in the *Journal of Neurology, Neurosurgery, and Psychiatry*<sup>44</sup>, examined 53 patients who demonstrated postconcussive symptoms for at least one month post head injury. Thirty-one patients had sustained a brief loss of consciousness (concussion group). Twenty-two were only dazed

with no loss of consciousness (mild concussion group). Eight neuropsychological tests were selected for their sensitivity to brain dysfunction. The results demonstrated that minor head injury patients compared to control subjects demonstrated deficits on tests of reasoning, information processing, verbal learning, attention and organization. They found no evidence that injuries associated with a brief traumatic loss of consciousness were more debilitating than injuries that resulted in "dazing" but no loss of consciousness. The authors also noted the absence of any differences between the litigating and the non-litigating patients in their study.

#### FRONTAL LOBE INJURY (EXECUTIVE FUNCTION DEFICITS)

The area of the brain most susceptible to damage following MTBI are the frontal lobes.<sup>45</sup> Unfortunately, frontal lobe deficits are extremely difficult to identify with standard neuropsychological tests. Neuropsychologists have recently started to develop tests to assess "executive functions" which are linked to the frontal lobes.

Muriel Lezak, Professor of Neurology and Psychiatry at Oregon Health Sciences University and author of *Neuropsychological Assessment* made the following comments in a recent article "Newer contributions to the neuropsychological assessment of executive functions":

There is increasing awareness of the importance of executive functions to independent and responsible social behaviour and the vulnerability of executive functions to brain damage, particularly when incurred in motor vehicle accidents.

... The availability of tests of executive functions has become an important issue in neuropsychological assessment. Many head injury patients and other persons with frontal lobe disorders might "look good" on paper when their scores on traditional, structured tests are tabulated, but their worlds - and their family members - might be falling apart. This problem becomes frustrating and socially costly when patients' needs for rehabilitation are discounted or misinterpreted. It becomes poignant when those who are too impaired to hold a job - or even seek one appropriately - have their claims for workers' compensation or other benefits turned down because their real problems have not been documented.<sup>46</sup>

The insensitivity of neuropsychological testing to frontal lobe and executive function deficits means that the lawyer will have to establish through collateral witnesses that the plaintiff who has sustained a MTBI is not the same person as before the accident. Jennett and Teasdale recognized this problem in 1981 when they made the following observation in their textbook:

#### Personality Change

This is the most consistent feature of mental change after blunt head injury, but there is no way to measure it. In its more subtle form this may be noticeable only to relatives or close associates, and unless they are questioned systematically, the doctor may believe that the patient has made a complete recovery.<sup>47</sup>

Varney and Menetec, in an article titled "Psychosocial and executive deficits following closed head injury: Implications for orbital frontal cortex" discuss the importance of obtaining information from collateral sources:

The problem presented by "classic" patients with orbital frontal damage is that they have marked personality changes and related problems, but may perform well on standard psychological tests. Such patients are nevertheless disabled

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despite their normal physical appearance and psychological test profiles . . . Admittedly, TBI involves many different types of physical injury to the central nervous system (CNS), and many of the behavioural deficits shown by patients with orbital frontal damage are also seen in association with other lesion sites. The fact that orbital frontal cortex is at such risk in TBI and that assessment of deficits related to orbital frontal damage can be difficult makes for a useful jumping off point for the discussion . . .

Patients with TBI, particularly when mild, may perform normally on a wide variety of neuropsychological measures and may appear relatively normal within the structure of standard psychological interviews. At the same time, they are often substantially impaired in independent, self-determined "adult" behaviours and activities of daily living. Thus, there has been increasing recognition of the importance of obtaining information from collateral informants (eg, parents, spouses, siblings, coworkers) who are familiar with the patient. Collaterals are capable of elaborating on traditional cognitive deficits (eg, reliability of memory) and are also qualified to assess the social, behavioral, and interpersonal changes experienced by an individual as a result of a head injury because they interact with the patient on a day-to-day basis. Information obtained from collaterals may often differ markedly from information given by the patient. Patients with TBI may provide inaccurate histories, overreport or underreport symptomatology, and lack insight concerning their behaviour and its effect on others in their environment. Because these individuals are likely to fall within normal ranges on traditional batteries of neuropsychological tests and may appear normal during a psychological interview, psychosocial symptoms (which often render the individual ineffective in daily functioning) may be overlooked by the most astute observer without collateral information.<sup>48</sup>

#### RECOGNITION OF MTBI

In cases where a client has sustained a blow to the head or a significant whiplash injury and is suffering from emotional, cognitive or behavioural changes one or more months post accident, the lawyer should investigate the likelihood that the client is suffering from the effects of traumatic brain damage. In such cases, the lawyer should refer the client to experts skilled in the diagnosis of traumatic brain damage such as a neuropsychiatrist, behavioural neurologist and clinical neuropsychologist. In many cases of brain damage, it is often the strategy of the defence to claim that the Plaintiff has not sustained brain damage or that any cognitive, emotional or behavioural problems experienced by the Plaintiff are either the result of a pre-existing personality disorder or psychological factors arising from the accident, and as such unrelated to any head trauma. Familiarity with the literature dealing with the mechanics of traumatic brain damage, together with the utilization of more sensitive brain imaging techniques such as the MRI, PET scan, SPECT scan, and topographical brain mapping will hopefully assist the lawyer in convincing a Judge or jury that the cognitive, emotional or behavioural changes following head trauma are attributable to traumatic brain damage.

The lawyer should obtain the following information before consulting with the neuropsychiatrist, behavioural neurologist or clinical neuropsychologist:

1. Client's history pre and post accident. See Checklist below;
2. Confirmation of history from family, friends, employers and co-workers;

3. Circumstances of the accident. Did the client hit his head, lose consciousness, demonstrate confusion or disorientation;
4. Statements of witnesses to the accident;
5. 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 posttraumatic amnesia;
6. Ambulance Crew Report;
7. Hospital records;
8. Prior medical records. There may be evidence of a prior head injury;
9. School records including any standardized test results.

The major problem for the lawyer presented with a client complaining of persisting symptoms arising out of a minor head injury or MTBI is deciding upon the appropriate course of action. This becomes difficult when the lawyer is confronted by physicians who seem to fail to appreciate the potentially debilitating effects of mild brain damage or seem too eager to attribute ongoing symptomatology to non-organic or psychogenic causes. The lawyer has two alternatives. The first is to follow the recommendation of the medical examiner and settle the case as soon as possible to allow the client to continue with his or her "normal life". The second, and in my opinion more appropriate alternative, is to retain experts who are more in tune with recent developments in the field of MTBI so that the true nature and extent of the individual's deficits can be properly assessed. Dr. Nathan Zasler, Medical Director of the Concussion Care Center of Virginia, makes the following observation in a recent article in the *Journal of Head Trauma Rehabilitation*:

*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. Awareness of current advances in neurodiagnosis, including neuropsychological assessment, and in rehabilitative treatment is of paramount importance in providing adequate care to patients with postconcussive symptomatology [emphasis added].<sup>49</sup>*

#### CONCLUSION

While the majority of persons who sustain a MTBI recover without residual sequelae, a significant subgroup suffer permanent cognitive, emotional and behavioural deficits and may experience significant difficulty returning to their preinjury lifestyle. It is only in the last decade that the medical profession has started to appreciate the potentially devastating consequences suffered by some victims of MTBI. The microscopic brain damage sustained will generally not be detected by the standard neurological examination or current neurodiagnostic tests. While recent brain imaging techniques such as topographical brain mapping, the PET scan, and SPECT scan are gaining acceptance as clinical tools in the diagnosis of traumatic brain damage, the most sensitive measure of this type of brain damage is a comprehensive neuropsychological assessment supplemented by reliable lay evidence from family, friends and other persons in the community who can attest to the changes in the injured person following the traumatic event.<sup>50</sup>

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## EXPERT PERSONAL INJURY ASSESSMENT

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|---|---|
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  17. word finding difficulty
  18. slower thinking and motor abilities
  19. impaired ability to plan and organize
  20. impaired motivation and initiative
  21. change in sleeping patterns
  22. irritability
  23. poor frustration tolerance (impatient)
  24. inability to deal with stress
  25. impulsive behaviour
  26. mood swings, depression to euphoria
  27. suicidal ideation
  28. change in sense of humour
  29. lack of confidence and self-esteem
  30. egocentric
  31. change in libido (usually decreased sexual drive)
  32. increased sensitivity to alcohol
  33. diminished sense of smell and taste
  34. prior head injuries
  35. complex partial seizure - like symptoms (e.g., repetitive motor movements, visual distortions, auditory hallucinations, strange odours and tastes, *deja vu*, *jamais vu*, etc.)
  36. For a list of psychosocial symptoms often seen in association with frontal lobe damage following closed head injury, see the "Iowa Collateral Head Injury Interview" published in *Journal of Head Trauma Rehabilitation*, 1993, volume 8(1), 42 - 44

#### TRAUMATIC BRAIN INJURY (TBI) CHECKLIST

1. loss of consciousness
2. GCS (Glasgow Coma Scale)
3. PTA (Posttraumatic Amnesia)
4. headaches
5. vomiting or nausea
6. dizziness
7. blurred vision
8. fatigue
9. sensitivity to light
10. sensitivity to noise
11. impaired coordination
12. impaired memory, particularly short term memory
13. impaired concentration
14. impaired ability to attend to a task, easily distracted
15. impaired ability to learn new information
16. impaired ability to deal with multiple stimuli

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