FROM THE DESK OF ED GOOD

My materials regarding Dr. Bonnie Hayes, psychologist, are still in frequent demand and have seen more of British Columbia than I have. I welcome additions to the collection. I would also like to receive current information about Dr. Hayes' engagement by ICBC or by disability insurers.

I previously announced an information bank for materials related to Dr. H. Davis, psychiatrist. I have appointed a new custodian for this expert's documents. Please send any reports, transcripts or judgments regarding Dr. H. Davis directly to Joe Murphy.

Chris Temple has kindly volunteered to administrate materials at his Delta office with respect to Dr. G. D. Ponsford, orthopedist, and Dr. A. Posthuma, psychologist. If anyone has suggestions for other candidates, please contact me, especially if you are willing to be a bank manager!

On a completely unrelated topic, I have not read anything on the issue of bicycle helmets since the flurry of press which accompanied the Honourable Jackie Pement's June 15th announcement about a mandatory bicycle helmet law. The last I knew, proclamation of the legislation was set for September 1, 1996. As someone who frequently works with brain injured clients, I look forward to the likelihood that this initiative will result in fewer and less severe brain traumas. Although there were many people from many different organizations involved in lobbying for this new law, I would like to say a personal thank you to one of our members, Bernie Simpson, MLA, who worked extremely hard at bringing this legislation into being.

Edward P. Good
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Head Injury Update

By Michael J. Slater

TRAUMATIC BRAIN INJURY (TBI) WITHOUT LOSS OF CONSCIOUSNESS (LOC)

A review of Supreme Court decisions dealing with the issue of mild traumatic brain injury (MTBI) reveals a number of cases where medical experts have opined that a diagnosis of traumatic brain injury (TBI) cannot be made without a finding that there was a loss of consciousness (LOC). These opinions are not supported by the neurobehavioural literature. Internationally recognized specialists in the field of TBI acknowledge that TBI can occur in the absence of documented LOC.

Dr. Bryan Jennett, Professor of Neurosurgery at the University of Glasgow, the originator of the Glasgow Coma Scale (GCS) and co-author of *Management of Head Injuries*[1] published in 1981, stated:

> "Impairment of consciousness is indicative of diffuse brain damage, but there can also be marked local damage without either alteration in consciousness or amnesia."[2]

The most famous and striking example of a severe TBI with no LOC is the classic report of Phineas Gage whose unfortunate injury has been studied by every first-year medical student. Phineas Gage was a foreman of a railroad construction crew in the mid 1800's who sustained a severe frontal lobe injury when an explosive charge propelled an iron bar upward through the lower left side of his face with the point of the bar exiting the top of his skull after passing through the left frontal lobe.[3]

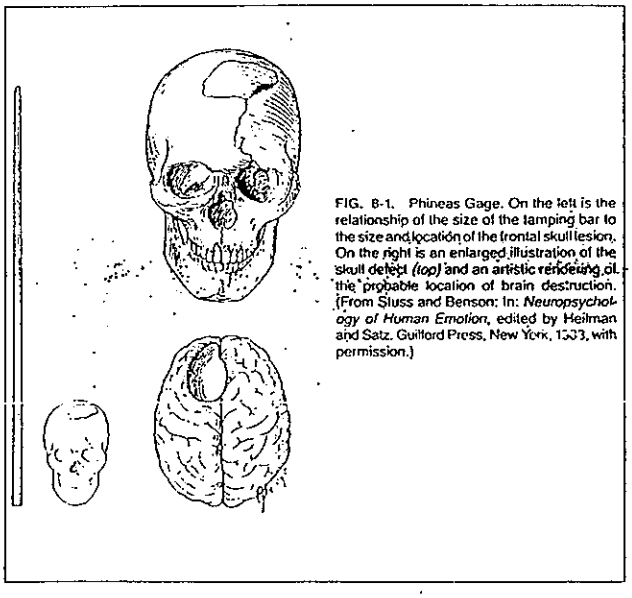

FIG. 8-1. Phineas Gage. On the left is the relationship of the size of the tamping bar to the size and location of the frontal skull lesion. On the right is an enlarged illustration of the skull defect *(top)* and an artistic rendering of the probable location of brain destruction. (From Sluss and Benson: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

Phineas Gage never lost consciousness and he was reported to be sitting up and talking with the iron bar protruding from his left temporal lobe. Physically Gage made a complete recovery, however, there was a significant change in his personality and emotional behaviour. He went from being a mild-mannered and

effective crew supervisor to being an impulsive, aggressive, and unreliable individual who was incapable of working in any capacity. The following description of Phineas Gage after the accident reveals a classic case of orbital frontal lobe injury:

> "His physical health is good, and I am inclined to say that he is recovered ... The equilibrium or balance, so to speak, between his intellectual faculty and animal propensities, seems to have been destroyed. He is fitful, irreverent, indulging at times in the grossest profanity (which was not previously his custom), manifesting but little deference for his fellows, impatient of restraint or advice when it conflicts with his desires, at times pertinaciously obstinate, yet capricious and vacillating, devising many plans of future operation, which are no sooner arranged than they are abandoned in turn for others appearing more feasible. A child in his intellectual capacity and manifestations, he has the animal passions of a strong man. Previous to his injury, though untrained in the schools, he possessed a well-balanced mind, and was looked upon by those who knew him as a shrewd, smart businessman, very energetic and persistent in executing all his plans of operation. In this regard his mind was radically changed, so decidedly that his friends and acquaintances said he was 'no longer Gage'."[4]

The definition of MTBI recently developed by the Mild Traumatic Brain Injury Committee of the Head Injury Interdisciplinary Special Interest Group of the American Congress of Rehabilitation Medicine makes it clear that LOC is not required in every case to define a MTBI:

> "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 (e.g. feeling dazed, disoriented, or confused);* and
> 4. focal neurological deficit(s) that may or may not be transient;" [emphasis added][5]

In a recent article published in *Neurology*, Neurologist Dr. Michael Alexander suggested that the reason medical experts fail to diagnose a MTBI is that:

> "Postgraduate teaching in neurology does not mirror the high prevalence of this disorder – i.e., most [medical] residents probably do not get proportionate instruction in the diagnosis and management of mild TBI. ...
>
> 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 15 probably represents true mild TBI. A score of 13 or 14 is due to confusion or disorientation and will be associated with a longer period of amnesia."[emphasis added][6]

In an earlier article entitled, "Neurobehavioural Consequences of Closed Head Injury", published in 1984 in *Neurology and Neurosurgery*, Dr. Alexander observed:

> "Unlike the patients with primarily DAI [diffuse axonal injury], the severity of a focal injury is not related to LOC and its duration; *many patients with severe focal lesions are never unconscious.*"[emphasis added][7]

Focal lesions which do not produce a LOC may be independent of the more diffuse injury that leads to a LOC or an alteration in consciousness.[8] Clinical neuropsychologist, Dr. Thomas Kay, coordinator of the Head Trauma Research Project at the New York University Medical Center describes the differences between diffuse and focal injuries to the brain.:

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

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 eletrochemical 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." [emphasis added][9]

Clinical neuropsychologist Dr. Robert Sbordone, author of over ninety books and articles in the field of TBI, provides the following description of a focal injury or cerebral contusion:

> "A cerebral contusion is a bruise on the surface of the brain which occurs as a result of the patient being either struck on the head, striking his or her head against a hard surface, or as a result of excessive acceleration or deceleration forces being exerted on the brain. When cerebral contusions occur as a result of blunt head trauma or the patient striking their head against a hard surface, the patient may only sustain a focal brain damage and not lose consciousness. Nonethless, these patients will typically report amnesia for the traumatic event itself. (e.g., striking their head)."[10]

WHIPLASH CAUSING TBI

It is also important to understand that focal injuries (cerebral contusions) as well as diffuse injuries (DAI) can result from an acceleration/deceleration movement such as occurs in some whiplash injuries without any direct external trauma to the head.[11] Dr. Muriel Lezak, in the 3rd edition of her text, *Neuropsychological Assessment* (1995), summarizes the literature:

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

1984; D. Pang, 1989).

When a moving head comes to a fast stop in an accident, the forward-moving energy (in a motor vehicle) or accelerating energy (in a fall) is translated into rapid acceleration/deceleration expanding and contracting wave-form movements of the brain matter, usually accompanied by the fast rotational propulsion of the brain within the skull. At the neuronal level, this rapid acceleration and deceleration, along with the rotational forces, results in damage to axons in cerebral and brain stem white matter and, in serious injuries, in the cerebellum too (Bostrom and Helander, 1986; R.L. Davis and Robertson, 1985; Gennarelli; Thibault, et al., 1982). This kind of axonal damage, called *diffuse axonal injury* (DAI), appears as torn axons, shearing of axon clusters, retraction balls consisting of sheared back axonal substance (*axoplasm*), and reactive swelling of strained and damaged axons. (J.H. Adams, Graham, and Gennarelli, 1985; J.H. Adams, Mitchell et al., 1977; Povlishock and Coburn, 1989). ...

Diffuse axonal injury can occur without any direct impact on the head, as it requires only the condition of rapid acceleration/deceleration such as takes place in whiplash injuries due to acceleration/deceleration forces resulting in rapid flexion-extension movement of the neck (Alves and Jane, 1985; R.W. Evans, 1992; C.M. Fisher, 1982b; Gennarelli, Thibault, et al., 1982; R.S. Parker, 1990; Yarnell and Rossie, 1988). [emphasis added][12]

CASE STUDY - EXAMPLE OF CEREBRAL CONTUSION OR FOCAL INJURY

The Plaintiff is a 20-year-old right-handed female, who was involved in a high-speed head-on collision. While it is unclear whether or not the Plaintiff was ever rendered unconscious, she was noted to be combative at the scene of the accident by the ambulance attendant and had a Glasgow Coma Scale (GCS) score of 14. She was taken by ambulance to the hospital where she was noted to be confused and have a low frustration tolerance. She was discharged with a diagnosis of closed head injury, forehead laceration, lip laceration, and possible basal skull fracture. Her mother and sister observed that while in the hospital and after she came home, she seemed confused. She returned to school and was able to complete senior high school while taking a reduced academic load. She was followed by an occupational therapist over the next year who observed that the Plaintiff continued to demonstrate cognitive, physical and social deficits characterized by impaired planning and organizational skills, difficulty getting along with others, poor frustration tolerance, irritability, cognitive inflexibility and a poor awareness of the effect of her behaviour on others.

Cerebral contusions typically produce significant and often relatively permanent alterations in personality and behaviour. The cerebral contusions may not be detected by standard neurological examination, CT or MRI scans, or standardized intelligence and/or neuropsychological tests. This may result in the failure to diagnose a cerebral contusion in addition to a MTBI arising from a cerebral concussion.[13] Accordingly, for a lawyer dealing with a TBI case such as this, it is important to retain an expert who is familiar with the pathophysiological mechanism and behavioural correlates of a cerebral contusion.

THE ROLE OF THE CLINICAL NEUROPSYCHOLOGIST

Several authors have commented on the importance of the experience of the neuropsychologist:[14]

> "A competent clinical neuropsychologist has a better chance of testing out the nature of a person's problems after minor head injury. ...
>
> Of course, like any other profession, the existence of the degree does not guarantee competence --and competence does not guarantee familiarity with the issues involved in minor head injury. Nevertheless, the availability of an informed and competent neuropsychologist is a tremendous asset in the stage of problem identification."[15]

In the above case study, neuropsychological testing demonstrated impairments in attention, cognitive flexibility, confrontational naming, verbal learning, visual and verbal memory (particularly under conditions of interference), higher order motor/executive functions, conceptualization, judgment, and organization/planning. At the emotional level, this patient was seen as moderate to severely depressed with a marked propensity for engaging in irresponsible behaviour, poor judgment, outbursts of anger, and the probable likelihood of suicidal behaviour. She was also seen as behaving in a very self-defeating manner, with little regard to the consequences of her behaviour.

The clinical neuropsychologist who examined the Plaintiff was experienced in the assessment of TBI, including cerebral contusions and provided the following opinion in this case:

> "A review of this patient's medical records, combined with her neuropsychological test data, indicates that this patient most likely sustained a cerebral contusion as a result of her motor vehicle accident. ... While cerebral contusions may not necessarily result in a loss of consciousness, they typically result in significant and often relatively permanent alterations in personality, cognitive functioning and behaviour. Research studies have demonstrated that the orbital frontal and inferior temporal areas of the brain are most likely to be damaged as a result of skull anomalies. Individuals who sustain damage to the orbital frontal lobes are likely to exhibit a pattern of disinhibition characterized by inappropriate social comments, inappropriate or antisocial behaviour, marked personality change, poor judgment and insight, and poor emotional control. Damage to the temporal lobes is likely to result in a pattern of brief and sudden outbursts of temper following relatively minor provocations or stresses, which is often followed by emotional distress or remorse. In addition, these patients are likely to exhibit memory disorders for recent events, as well as psychiatric difficulties, particularly, depression, violence, rapid mood swings and suicidal ideation.
>
> The disinhibition produced by contusion to the orbital frontal lobes is typically characterized by a dramatic change in the patient's personality, diminished frustration tolerance, irritability and impulsive behaviour. In addition, the patient is typically described by others as having become very egocentric and as having lost the ability to be affectionate toward significant others and also as being

unable to show proper concern for the welfare and feelings of others. These individuals will frequently exhibit rapid mood swings and engage in frequent inappropriate social behaviour, including antisocial acts, the use of crude or coarse language, and loss of social tact. In addition, these individuals are likely to engage in indiscriminate sexual activities with little or no regard for the consequences of such behaviour. Typically, individuals with orbital frontal lobe damage have little or no awareness of these changes and usually deny or minimize their problems. When these patients are administered standardized neuropsychological test batteries, they frequently show little evidence of cognitive abnormalities. However, neuropsychologists who have had considerable experience with these types of patients may detect cognitive abnormalities utilizing qualitative or specialized tests, as well as interviews with significant others.

The primary role of the orbital frontal cortex appears to be that of inhibiting the limbic system (the emotional center within the brain). Thus, damage to this region results in the patient's inability to regulate his or her emotions or behaviour. Unfortunately, many clinical psychologists are unaware of the specific role that the orbital frontal lobes play in the regulation of behaviour. Thus, while they may test patients in highly structured settings and administer a standardized neuropsychological test battery which contains clear instructions, they may report only relatively mild or even normal test performances. Thus, many psychologists are likely to erroneously conclude that the patient has only sustained a mild head injury. The large discrepancy between the patient's complaints and those of significant others are the hallmark of such injuries."

The following quote from the chapter "Behavioural Disorders Associated with Frontal Lobe Injury" in the book *Clinical Neuropsychiatry* supports the opinion of the clinical neuropsychologist:

"Orbitofrontal Syndrome

Behaviourally, the outstanding feature of the orbitofrontal syndrome is disinhibition. The patients lack social tact, make tasteless and socially inappropriate comments, may commit antisocial acts, and exhibit a general coarsening of interpersonal style. Emotionally the patients are irritable and labile with a tendency to an inane euphoria and inappropriate jocularity (witzelschut). The patients may be hyperactive, and hypomanic behaviour is not uncommon. Sexual preoccupations, inappropriate sexual jesting, and improper sexual comments are frequent, but overt sexual aggression is rare (Arseni et al., 1966; Blumer and Benson, 1975; Faust, 1966; Hunter et al., 1968; Jarvie, 1954; Reitman, 1946.)

Neuropsychological deficits are surprisingly difficult to identify in patients with orbitofrontal lesions. Impulsiveness, distractibility, and lack of concern for correct performance may interfere with intellectual assessment; however, when these can be contained,, basic language, memory, and cognitive skills are usually found to be intact. (Faust, 1966; Luria, 1980). Insight, especially into the social or emotional consequences of actions, is limited, and interpersonal judgment is poor.

Elementary neurological deficits are not prominent in patients with lesions limited to the orbitofrontal cortex."[16] [*This description of an orbitofrontal syndrome is remarkably similar to description of Phineas Gage published in 1868 and the clinical neuropsychologist's description of the Plaintiff in the case study.*]

Further support for TBI without LOC is found in an article published in a 1994 article in *Brain Injury*. Researchers compared CT, MRI and PET scans with neuropsychological assessment in the evaluation of nine MTBI cases. In all nine cases, the PET scan demonstrated cerebral pathology not visualized by CT or MRI scanning. The PET results corroborated the neuropsychological test findings. In four of the nine cases there was no documented LOC. In the remaining five cases, the LOC ranged from less than one minute to twenty minutes. The researchers found no differences between the four cases without LOC and the five cases with LOC and concluded:.

"The results of the present cases suggests that a minor TBI without LOC can cause significant functional brain damage. Furthermore, even though the patients in the present series do represent outliers, the fact that these mildly TBI patients show abnormalities and cerebral glucose metabolism, raises possibilities that PET data may prove useful in predicting which patients are at risk for 'post-concussive' syndrome."[17]

Further evidence in the literature for TBI without LOC can be found in studies that have reviewed neurodiagnostic test results in mild to moderate TBI cases. In the text *Neuropsychiatry of Traumatic Brain Injury*, the author noted:

"Injury does not always occur at the axonal level alone. Although cerebral concussion was the diagnosis in 80% of patients with mild brain injury in the San Diego County study (Kraus and Nourjah 1988), almost 5% had cerebral contusions, about 1% had intracerebral hemorrhages, and 14% had some other form of intracranial lesion. In Williams et al.'s study (1990) of 155 consecutive patients with mild brain injury, 32 had parenchymal contusions or hemorrhages (20%) and 27 (17%) had subdural or epidural hematomas.

The above suggests that brain injury considered trivial on the basis of the degree and duration of altered consciousness has demonstrable neuropathological effects, starting at the moment of impact and evolving over several hours to days and longer (Table 11-3). The types of injury seen, both macroscopically and microscopically, are similar in quality and location to those seen with moderate and severe degrees of brain injury. ...

Recent CT studies and studies comparing CT and magnetic resonance imaging (MRI) suggest that structural abnormalities are more common in mild brain injury than originally thought. Jenkins et al. (1986) found MRI abnormalities in all 8 patients with minor brain injuries *(no loss of consciousness in 4, GCS scores of 15 in the other 4)* in their study of mild

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> and severe brain injuries. Levin and colleagues (Eisenberg and Levin 1989; Levin et al. 1987a) compared CT and MRI in 11 patients with mild (GCS 13-15) and 9 patients with moderate (GCS 9-12) brain injury. Only 3 of the patients had no abnormality on either scan." [emphasis added][18]

CASES WITH NO LOC OR TRANSIENT LOC

Judges are now starting to recognize that TBI can occur with no LOC. The following cases are recent decisions of the Supreme Court of British Columbia where a finding of TBI has been made in circumstances where the Plaintiff suffered either a brief period of LOC or no LOC.

In *Chen* v. *Ruersatt*[19] the Plaintiff, a 59-year-old school custodian, was rear-ended in a motor vehicle accident on January 15, 1990. 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. Since the accident, his lifestyle changed significantly. He no longer had the physical or mental capacity to manage the family finances and household, to maintain employment, to look after rental properties, or to participate in physical and recreational activities. He also became sexually impotent and incontinent. The Plaintiff claimed that he suffered from a mild head injury with symptoms including headaches, slowness in movement and cognitive processes, reduced concentration, memory loss, difficulty with balance, vertigo, ringing in his ears, tremor in his right hand and a significant negative personality change (bad tempered and irritable). The Court found that the symptoms were consistent with a head injury notwithstanding an absence of evidence of 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." [emphasis added]

Damages awarded included non-pecuniary loss of $120,000.00, cost of future care of $221,184.00 and future income loss of $148,260.00. The Defendant appealed and the Plaintiff cross-appealed. The Court of Appeal dismissed the appeal and allowed the cross-appeal by reducing the contingency on future earning capacity from 30 percent 10 percent.

Jardine v. *Lend Lease Transportation Ltd.*[20] is an example of a whiplash case where the Court concluded that the Plaintiff's cognitive deficits were the result of subtle brain damage attributable to the accident. The Plaintiff was a 36-year-old woman who was enrolled in an M.A. Counselling Psychology Program at university at the time of the accident. She was also working part-time as a family therapist. The only significant event in her medical history was a minor concussion in 1978 (note: this is of some significance given the MTBI literature on the cumulative effects of concussive injuries).[21,22] The Plaintiff was involved in a rear end motor vehicle accident on June 1, 1985. The Plaintiff did not remember the crash, but remembered the "blackness", a sound like breaking glass, and her daughter crying. On the issue of LOC the Court found: *"It is not clear whether she lost consciousness completely. If she did so it was only momentarily."* She was taken by ambulance to a hospital where she was diagnosed as having a flexion-extension injury of the neck and back and discharged home. The hospital records did not reflect any loss of consciousness. Soon after the accident, she reported a number of symptoms including dizziness, blurred vision, headache, nausea, extreme fatigue, nightmares, change in her sense of taste, numbness on the left side of her body, impaired memory and concentration, inability to make sense of reading, difficulty explaining things to others, and difficulty in organization. A neurologist thought her symptoms were secondary to pain and discomfort. The Plaintiff returned to work but found that she had difficulty coping. Neuropsychological testing revealed a number of deficits including diminished intellectual capacity, impaired memory, word finding difficulty, problems in sustaining attention, slowed psychomotor speed, and impaired ability in logical analysis, abstract reasoning and new concept formation. The neuropsychologist was of the opinion that the Plaintiff had a postconcussion syndrome and was suffering from the effects of permanent brain damage. The defence neuropsychiatrist was influenced by the gap between the severity of the Plaintiff's symptoms and the lack of objective findings of structural brain injury on neurological examination or with CT and MRI scans, and diagnosed a major depressive disorder. The scales were tipped in favour of the Plaintiff by the collateral evidence of the Plaintiff's husband, friends and a fellow employee attesting to changes in the Plaintiff post-accident. The Court awarded damages for non-pecuniary loss of $60,000.00, past income loss of $25,000.00 and loss of future earning capacity of $275,000.00.

In *Haley* v. *Brown*[23] the Plaintiff, then 18, was injured in November, 1986 when the car in which he was a passenger struck a concrete barrier. The Plaintiff struck the right side of his head against the passenger window. The Plaintiff who had a GCS Scale of 14/15, was admitted to hospital and released within several hours. He was readmitted to hospital later that day complaining of severe head pain. A CT scan revealed a large right parietal epidural hematoma. A craniotomy was performed by a neurosurgeon and the hematoma evacuated. He was discharged from the hospital a week later. Prior to the accident he was a well-mannered, easy going, goal-oriented young man with a good sense of humour. He was a member of the military reserve and intended to become a police officer. After the accident, relatives, friends, and acquaintances noted personality changes in the Plaintiff. He was seen as nervous, high strung, and subject to drastic mood swings. He fought with his girlfriend and his family, his short term memory was poor, and he spent an enormous amount of time studying in order to graduate from Grade 12. He was unable to handle more than two courses at a time at the college level. He remained in the military reserve, however, there was conflicting evidence with regard to how well he performed in the reserve after the accident.

The Reasons for Judgment were handed down in two parts. The first part (230 pages) dealt with the issue of whether the Plaintiff sustained a MTBI. The Court reviews the evidence of collateral and expert witnesses in great detail. The second part of the Reasons deals with the quantum of damages.

The Court made the following findings of fact:

> 1. Prior to the injury, the Plaintiff had not suffered from any psychological or psychiatric problems;
>
> 2. After the accident there was a profound change in his personality. The personality changes

were seen shortly after he returned home from the hospital and grew worse over time.

3. The evidence of loss of consciousness at the scene of the collision was conflicting. *The Court found that the Plaintiff did not suffer loss of consciousness after the accident.* There was conflicting evidence with regard to the period of posttraumatic amnesia. The Plaintiff's evidence was that he remembered almost nothing for the three to four days after the surgery. The neurosurgeon recorded that the Plaintiff had significant amnesia for events half an hour prior to the accident and for most of the events up to the period of the surgery. The Court found that the period of posttraumatic amnesia was approximately 15 hours.

4. The majority of the Plaintiff's experts relied on a period of posttraumatic amnesia of three to four days which affected the weight to be given to their diagnoses.

5. The majority of the expert evidence was that personality changes and changes in cognitive function resulting from brain damage were subject to improvement over a two year period and possibly longer.

6. While there was conflicting evidence with regard to the existence and significance of left sided deficits noted by various medical examiners, the Court found that the Plaintiff did demonstrate left sided deficits after the accident which were consistent with an injury to the right side of the brain.

7. Notwithstanding a normal neurological examination by the neurosurgeon approximately one month post injury, the neurosurgeon was concerned about the possibility of future difficulties as he suggested that the Plaintiff should undergo neuropsychological testing which should be repeated at appropriate intervals.

8. The Court accepted the defence interpretation that the topographical brain mapping results were consistent with changes caused by the craniotomy rather than damage to the brain.

9. The Court also found that the findings of asymmetric frontal horns noted on some of the CT scans were not clinically significant.

10. The Court found that the MRI Scan identified two injuries to the brain: in the corpus callosum and in the cerebellum. While the Court found that both injuries were caused by the trauma of the accident, only the injury to the corpus callosum was deemed significant.

11. The Court accepted the evidence of the Plaintiff's neuropsychiatrist that the injury in the corpus callosum interfered with the transfer of information from the right to the left cerebral hemisphere, affecting visual memory and reducing the Plaintiff's ability to accurately transfer information between the two hemispheres.

12. The Court found that the Plaintiff had sustained an orbital frontal lobe injury notwithstanding the absence of any LOC or findings of a frontal lobe injury on the EEG, CT or MRI scans. The Court found that the Plaintiff displayed many of the characteristics

commonly associated with the orbital frontal lobe syndrome including irritability, low frustration tolerance, disregard for the welfare of others, rapid mood swings, distractability and egocentricity. The Court referred to the evidence of the defence neurologist Dr. Bratty, who testified in cross-examination:

> "The problem with respect to frontal lobe injuries is these people can get by if you structure things very well for them. If you lay down the rule and organize it properly for them, they might pass each of the milestones satisfactorily. It is when they are on their own and there are no rules, no structure, they fumble, they fail."
>
> ...their life is not satisfactory, they are not functioning satisfactorily and that's going to be apparent to people living with them and people who know them well."

13. The Court found that the "scatter" demonstrated on the Wais-R in the first neuropsychological testing was consistent with brain damage. The improvement in the neuropsychological test results was consistent with the improvement hoped for following brain injury. The Court found that the first neuropsychological testing was the most significant and that subsequent testing is confounded by practice effect and further, that the younger the subject, the more likely it is that their test performances will show practice effects.

14. The Plaintiff was able to achieve a number of successes in his various undertakings because he worked within his limitations. His life had been sheltered and very structured - the Militia, Douglas College, and some social activity. He always lived at home. The one job he had, in a fast-food restaurant, he found extremely stressful, both physically and emotionally and fared poorly.

The Court concluded that the Plaintiff suffered mild, permanent brain damage caused by the trauma of the accident. In addition, he suffered from a psychiatric/psychological condition resulting from his attempts to cope with brain damage. The Plaintiff was left with significant difficulties which will have an impact both in the work place and in daily living. The Court awarded damages for non-pecuniary loss of $90,000.00, future cost of care of $35,000.00 and future income loss of $340,000.00 based on a 35 percent loss of income earning capacity..

In *West* v. *Cotton*[24] the Plaintiff, a 40 year old program coordinator with the Red Cross, was rear ended in a motor vehicle accident. The force of the collision propelled the Plaintiff's vehicle into a telephone pole. The Plaintiff was able to exit his vehicle, but collapsed after taking a few steps. He was transported to hospital and released within two hours. The Plaintiff claimed that he had suffered a brain injury that resulted in significant permanent physical and behaviourial dysfunction with psychological symptoms that rendered him incapable of maintaining employment and enjoying any kind of normal life. The Third Party contended that the Plaintiff suffered soft tissue injuries. Two defence neurologists both concluded that the Plaintiff had not suffered brain damage on the basis that there was no recorded LOC at the scene of the accident. The Court did not agree and found that "the Plaintiff suffered some loss or interruption of consciousness at the time of the accident." An MRI scan and SPECT scan were positive and consistent with a head injury. The Plaintiff suffered a personality change, loss of planning and organizational skills, memory loss, reduction in concentration and verbal skills. The Court found that the Plaintiff sustained injuries to the left frontal lobe and the left temporal lobe of his brain. The Court awarded damages of $2,224,616.00 plus an award for tax gross up. Non-pecuniary loss was $150,000.00, cost of future care $814,800.00 and future income loss $854,285.00. The Third Party appealed the award of damages and the Court of Appeal reduced the damages by a total of $732,988.00 to $1,491,631.00 plus tax gross-up.

In *McKay* v. *Jenner*[25] the Plaintiff was involved in three motor vehicle accidents, none of which was particularly serious. There were almost no clear cut physical injuries or broken bones. ***The Plaintiff did not lose consciousness.*** The diagnosis included chronic pain, a closed head injury with mental impairment and TMJ. The basis for the claim of a closed head injury was the second accident in which the Plaintiff struck the left frontal area of her skull on the inside of her vehicle. The Plaintiff suffered from problems with short term memory, concentration, planning and organization, emotional control and use of language. Although the Judge commented that "the result of all this would not necessarily be apparent and were for the most part not apparent in her appearance before me on the stand", he was impressed with the evidence of the neuropsychologist. Dr. Catherine Mateer, observing that "her education, background, experience, accreditation and published materials place her in the highest rank in her chosen field." The Court found that the Plaintiff had suffered damage to the left frontal and temporal areas of the brain and awarded damages of $503,643.00 including non-pecuniary loss of $127,500.00.

In *Warder* v. *ICBC*[26] the Plaintiff was a 38 year old school teacher and Mayor of Esquimalt. He was involved in a single motor vehicle accident on March 1, 1989 when his vehicle struck a power pole to avoid colliding with an unidentified vehicle. The Plaintiff suffered soft tissue injuries, a fractured nose, PTSD and either organic brain damage or a psychological reaction to the accident leading to a depressive effect on his ability to function normally. Four years later at the trial, the Plaintiff had continuing difficulties with memory, expressing himself on paper, and initiating projects in the same way as he did before the accident. He was not as good a teacher, less effective as a politician, not as good a husband and father, had lost much of his self esteem, and had not regained the "spark" that he exhibited in all his activities before the accident. As the Plaintiff did not suffer any significant period of unconsciousness, defence experts maintained that he did not suffer organic brain damage. The Court accepted the interpretation of neuropsychological testing which tended to support the allegation of organic brain damage and found that the Plaintiff suffered a minor degree of brain damage which interacted with his depressed psychological state. This injury prevented the Plaintiff from continuing his climb up the ladder of success. He would be restricted in pursuing a political career and would be unable to cope with the demands of a school administrator. The Court found: "He has intelligence, but not the will." The award of damages included non-pecuniary loss of $80,000.00 and future loss of income of $300,000.00.

In *Datta* v. *Rowan*[27] the Plaintiff was a 48 year old child care worker who was injured in two motor vehicle accidents on November 3, 1989. She suffered what appeared to be soft tissue

injuries. She was taken to the hospital and discharged after an examination in the emergency department. She suffered from pain in her head, neck and down her left side. There was no indication that she had lost consciousness. She attended her family physician later that day. He found that she was confused and that her thinking processes were not clear. He diagnosed a possible concussion or contrecoup injury to the brain. Prior to the trial the Plaintiff had been examined by five neurologists, a psychiatrist, a psychologist, a physiatrist, three neuropsychologists, a rheumatologist, a dentist, a speech pathologist, an occupational therapist, a social worker and two physiotherapists. She was assessed by a head injury assessment team at University Hospital, who concluded that she had sustained a whiplash injury with possible concussion. The Plaintiff was forgetful, anxious, antisocial, irritable, depressed and suffered from an attentional deficit. The experts disagreed on whether the Plaintiff's problems were the result of a mild traumatic brain injury or of emotional overlay. The Court accepted the results of brain mapping and computerized evoked potential studies and found that the Plaintiff had sustained a mild brain injury that compromised her higher cognitive functioning. The Court also relied on the evidence of many witnesses who described the changes in the Plaintiff after the accident. The Plaintiff had received a Ph.D in physiology in 1974 from the University of Saskatchewan before deciding on a career change in 1984. The Court found that her intellectual abilities had been diminished. She had gone from the top category to a level at or below average. "She can still function normally but the sharp edge has been dulled." The Court awarded damages of $345,594.00, including non-pecuniary loss of $100,000.00 and future loss of income of $200,000.00.

In *Johnson* v. *Zenith*[28] the Plaintiff was a 33 year old social worker who was struck by a motor vehicle while crossing the street on October 12, 1989. She struck her head on the road surface. She did not think she lost consciousness but "felt like I was going to". Her physical injuries resolved and the issue at trial was whether, when the Plaintiff's head struck the road surface, she suffered organic brain damage causing such a permanent slowing down of her mentation that her wage earning capacity was substantially impaired. The Court quotes at length from many of the expert reports. Reports from a neurologist and a psychiatrist contain an excellent discussion of the mechanics of a mild traumatic brain injury. The Court found that the Plaintiff's cognitive deficiencies were the result of diffuse axonal injury to her brain. In her job performance she was described as a "shadow of her former competent self . . .: The change in her is neither imperceptible nor ephemeral but very substantial and has been enduring." The Court awarded damages of $623,975.00, including non-pecuniary loss of $90,000.00 and future loss of earnings of $490,000.00. An appeal by the Defendant was dismissed.

In *Perry* v. *Egilson*[29] the Plaintiff was injured in two motor vehicle accidents. The first accident occurred on November 2, 1986 when she was 14 years old. The second accident occurred on August 1, 1989. There is an indication of loss of consciousness in the first accident. The Court was impressed with the evidence of collateral witnesses who described the Plaintiff prior to the accidents as happy-go-lucky, friendly, talkative and reliable. The Plaintiff experienced a significant change in personality. She suffered from depression and cognitive deficits and dropped out of school after Grade 10. While not a particularly good student prior to the accident, evidence indicated she probably would have completed Grade 12. . A neurologist could not find any neurologic abnormality. The Court accepted the evidence of a neuropsychologist that the Plaintiff had sustained a minor traumatic brain injury. The Court considered the case "a thin skull situation" based on the evidence of the neuropsychologist that: "such injuries usually resolve - however, for individuals who have had prior learning or academic problems the sequelae of such `minor' injuries may become exacerbated. In reviewing her academic history, this appears to be Ms. Perry's case." The Court found that but for the accident there was a good possibility that the Plaintiff would have completed Grade 12, however, with professional assistance, she could rehabilitate herself to become employed in the work force. An award for future wage loss was based on lower wage rates and a lower probability of employment in the future. The Court awarded damages of $388,000.00, including non-pecuniary loss of $80,000.00 and future wage loss of $280,000.00.

In *Paananen* v. *McIntyre*[30] the Plaintiff suffered multiple injuries in an accident on July 17, 1990. The main issue at trial was whether or not the Plaintiff was suffering from the effects of a TBI. Evidence from collateral witnesses at the trial indicated that the Plaintiff's personality had changed after the accident. She was described prior to the accident as being an outgoing, positive, assertive, socially adept, vivacious, energetic and fun-loving person. She was successful in her profession as a realtor. After the accident, witnesses described her as depressed, withdrawn, timid, inattentive, forgetful, anxious and lacking in energy. There was an issue as to whether the Plaintiff suffered from a LOC. The defence relied on the opinion of a psychologist who testified that the Plaintiff's ongoing cognitive and emotional problems did not relate to TBI. The defence psychologist concluded that the Plaintiff was awake and oriented at the scene of the accident. The Court found that the Plaintiff had suffered an altered state of consciousness as a result of the accident and concluded that the Plaintiff had suffered a TBI. The Court placed reliance on the opinions of the Plaintiff's experts including a neurologist, Dr. Donald Cameron, and his opinion that based upon the Plaintiff's state of altered consciousness and very limited memory of the accident that she "suffered an organic brain injury as a result of an abrupt acceleration/deceleration of the brain within the cranium". The Court awarded damages of $775,500.00 including non-pecuniary loss of $120,000.00, past income loss of $181,167.00, impairment of future income-earning capacity of $400,000.00 and cost of future care of $359,331.00.

CONCLUSION

While these recent decisions suggest that experts in the medical profession and the Courts are beginning to accept that MTBI can result in brain damage with permanent residual sequelae, the same cannot be said for the lay public. Researchers at the University of Alberta investigated the laypersons' knowledge about the sequelae of MTBI and whiplash.[31] Their data suggest that the average person has limited knowledge of the extensive range of symptoms that can occur following a MTBI and shows little understanding or sympathy for cognitive symptoms such as memory problems and loss of concentration. Participants in the study believed a speed approaching 60 kph was necessary to cause brain damage to a driver who received a direct blow to the head following a rear end collision. Loss of consciousness was believed to enhance the likelihood of symptoms. Even common physical symptoms such as headache and neck pain following a direct blow to the head were thought to require speeds of 34 kph. Even higher speeds were believed to be necessary to produce symptoms following a

whiplash injury without any direct blow to the head.

The results of this study present a challenge for the lawyer faced with presenting a case to a Judge or jury involving largely subjective symptoms following a MTBI. Hopefully this conservative view will be revised by an awareness of the evolving medical and neuropsychological literature which have documented the potentially devastating cognitive, emotional and behavioural effects of a MTBI even in circumstances where there has been a transient or altered state of consciousness and in some cases no documented evidence of a disturbance of consciousness.[32]

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