



TBI Update

By Michael J. Slater

MILD TRAUMATIC BRAIN INJURY (MTBI) AND THE THIN SKULL RULE

INTRODUCTION

The purpose of the *Traumatic Brain Injury (TBI) Update* is to inform those lawyers interested in TBI cases about recent developments in the neurobehavioural literature. The first *Update (The Verdict, December/94)* reviewed the literature in the field of Mild Traumatic Brain Injury (MTBI). The last *Update (The Verdict, January/96)* dealt with the specific issue of MTBI without loss of consciousness (LOC). This *Update* deals with some of the myths about MTBI that have been perpetuated by those individuals who are, in the words of Dr. Thomas Kay, Coordinator of the Head Trauma Research Project at New York University Medical Center, "guilty of gross ignorance and neglect of the long-term problems associated with mild head trauma."¹ The literature with regard to issues of malingering, secondary gain, and the effect of litigation or compensation claims is canvassed. The cumulative effect of repeated concussive events and the concept of "individual vulnerability" is discussed in relation to the "thin skull rule" and the "eggshell skull" or "eggshell personality."

THE SIGNIFICANT "WALKING WOUNDED"

The number of traumatic brain injuries in the United States each year is reported to be in the range of 750,000 to 3 million cases.^{2,3,4} MTBI accounts for approximately 75 to 90 percent of all traumatic brain injuries.^{5,6}

While the majority of persons recover from a MTBI within 6 months, a significant minority - approximately 10 to 15 percent - never completely recover⁷ and are left with one or more of the following problems: physical symptoms (e.g., dizziness, headache, sleep disturbance, quickness to fatigue or lethargy); cognitive deficits (e.g., involving attention, concentration, short-term memory or executive functions); and behavioral changes and/or alterations in degree of emotional responsiveness (e.g., irritability, quickness to anger, disinhibition or emotional lability).⁸ These individuals have been referred to as the "walking wounded."⁹

In a 1993 article published in *The Journal of Head Trauma Rehabilitation*, Professors Dykmen and Levin state:

Not all patients with mild head injury complain of posttraumatic symptoms, and most improve without further intervention. In a fraction of the cases, however, the postconcussion symptoms do persist and may evolve into the so-called postconcussional syndrome. Owing to the high incidence of mild head injuries *this fraction of cases translates into a sizable group of patients, who may be significantly disabled in resuming their preinjury lifestyle.*¹⁰ [emphasis added]

This "sizable group of patients" has not dramatically grown in number in the last 10 years. What has changed is the recognition by leading neurobehavioural experts in the medical profession of the potentially debilitating effects of MTBI. Neurosurgeons, Drs. Jennett and Teasdale, in their internationally recognized 1981 text, *Management of Head Injuries*, concluded that "the damage done by,

and the symptoms subsequently suffered after mild head injuries are frequently underestimated [as] ...doctors who deal with mildly injured patients are unfamiliar with recent work in the field..." Medical researchers involved in a MTBI program at New York University Medical Center reported in a 1986 article:

We found gross ignorance and neglect of the long term problems associated with 'mild' head trauma: those injuries where patients spent a brief time (if any) in the hospital, make quick medical recoveries, and were discharged directly home without any perceived need for formal rehabilitation... these patients appeared fine until they attempted to resume their responsibilities at home, work, or school. When they did so, a significant number experienced great difficulty. They complained of inability to remember, concentrate, organize, handle a number of tasks at once, and get as much work done as efficiently as they used to. The relationships with family, peers, and bosses often suffer and they develop psychological problems... In such cases, the unique problem of minor head injury readily became apparent despite swift and complete physical recoveries, and despite no obvious neurological basis for the problems, these persons were experiencing significant cognitive, emotional, and behavioral deficits that seriously interfered with their ability to lead fully functional lives.¹¹

A NEUROLOGIST'S VIEWPOINT

Neurologist Dr. Michael Alexander, in a recent article entitled "Mild Traumatic Brain Injury: Pathophysiology, Natural History and Clinical Management" published in the journal *Neurology*, refers to MTBI as one of the most common neurologic disorders. Dr. Alexander states that the reason medical professionals 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...[emphasis added]

Mild TBI is characterized by the following: (1) Head trauma may be due to contact forces or to acceleration/deceleration trauma. (2) The duration of unconsciousness is brief, usually seconds to minutes, and in some cases there is no loss of consciousness (LOC) but simply a brief period of dazed consciousness. (3) When the patient is evaluated in the emergency room or at the scene, the Glasgow Coma Scale (GCS) must be 13 to 15, by common definition. As discussed below, only a score of 13 or 14 is due to confusion or disorientation and will be associated with a longer period of amnesia.¹²

ORGANIC OR PSYCHOGENIC - REAL OR IMAGINED

The problem with the term mild head injury or MTBI is the connotation that the scope, effect or duration of symptoms will be trivial and of little consequence compared to moderate or severe TBI. This approach has been criticized by Dr. Sheldon Berrol, founding editor of the peer-reviewed *Journal of Head Trauma Rehabilitation* and respectfully referred to as the "grandfather of brain injury rehabilitation."¹³ Dr. Berrol commented:

The attempt to categorize the degree and importance of 'less than severe' injury into various subsets has led to terms that compare the injury, in terms of quality, with severe injury -- terms such as 'trivial head injury,' 'minimal head injury' and 'mild head injury.' One

cannot, however, assume that the scope, effect, or duration of symptoms will be trivial, minimal or mild. *Minor injuries – that is injuries with brief alterations of consciousness – may have associated focal patterns of considerable significance, with life-long effects for the individual.*¹⁴ [emphasis added]

This oversimplification of MTBI as a “trivial injury” (because the majority of individuals recover) has resulted in a number of observers to mistakenly characterize those victims of MTBI with persistent symptoms as suffering from psychiatric problems or worse yet, to be malingering. Dr. Kay made the following observation in the text *Rehabilitation of Post-Concussive Disorders*:

There exists a long-standing controversy over whether persistent symptoms and inability to function after concussions are due to organic factors or to purely psychological factors. This controversy often takes on the intensity of religious righteousness, with advocates and skeptics easily divisible into opposing camps, and with each side seeing and citing only the evidence that supports its position, while discounting conflicting accounts as suspect or bogus. Thus, Miller, when describing the post-concussional syndrome, sarcastically comments that its symptom complex is “graven on the heart of every claimant for compensation” (p. 257); he unsympathetically describes the “gloomy spectacle” of these “tense, miserable, unsmiling, evasive, defensive and disgruntled” patients; but he suggests that his colleagues “take heart” because “nearly all these patients recover completely and without treatment after their [legal] case is settled - win or lose” (p. 18). In our clinical and research experience, this attitude of skepticism bordering on hostility toward post-concussion patients is still extremely prevalent. Conversely, Taylor in a much more sympathetic vein, writes (p. 70): “Patients who have minor difficulties in concentration and performance which irritates them and make them act unusually are not ‘neurotic,’ they are ‘cerebrally disorganized,’ and for good organic reasons.” He then adds that we “do everything for the severely damaged 10% and neglect the non-serious 90%” (p. 70). Of note in this controversy is the fact that disagreement is not based on differences in scientific knowledge (both Miller and Taylor wrote in the 1960s); something about the dysfunctional symptoms of persons who appear physically normal arouses either suspicion or sympathy in other human beings, depending on the personality of the observer, differences in clinical experience, and on biases as to what constitutes “real” evidence of an underlying disorder.

If, however, all persons who become functionally disabled or struggle to maintain similar levels of functioning after a concussion are malingerers or neurotics, how is it that their symptoms of headache, dizziness, fatigue, concentration and memory problems, and slowness of information processing are so remarkably consistent? Patients with post-concussion syndrome who have never met other persons with similar symptoms report themselves being “thunderstruck” when they read descriptions of the syndrome, as if they were being personally characterized. This certainly suggests an external validation that concussion itself can lead to a well-defined set of persistent symptoms. Yet it is clear that not all

persons who suffer similar concussions have long-term problems. Why do some persons remain symptomatic, while others recover? This suggests that persons who do not “recover” are responding, at least in part, to something other than the damage done in the concussion itself.

The perspective taken in this chapter on this paradox is neuropsychological in nature and involves the concept of individual vulnerability: how completely a person “recovers” after a mild TBI and to what extent the ability to function is compromised depend on a complex interaction of organic and psychological factors, and the “diagnosis” cannot be dichotomized (i.e. the patient usually cannot be neatly categorized as “organic” or “neurotic”). More often than not there is in fact a combination of underlying neurologic and overlying personality, environmental, and psychological factors that determine functional outcome. Each person who suffers a concussion will respond differently, depending on the integrity - and therefore vulnerability - of his or her neurologic, personality, family, and vocational systems. The proper goal of the contemporary clinical neuropsychological evaluation is to sort out and describe the various factors that determine the person’s current level of functioning.

Neuropsychologist Dr. Catherine Mateer, Professor and Director of the clinical psychology program at the University of Victoria, is the author of over 60 articles published in the field of TBI as well as the 1989 text, *An Introduction to Cognitive Rehabilitation: Theory and Practice*. She is also the author of *Management of Mild Traumatic Brain Injuries* which is currently in press. In a chapter published in the 1992 text *Rehabilitation of Post-Concussive Disorders*, Dr. Mateer made the following observations with regard to MTBI victims:

These individuals, who by definition do not suffer any - or certainly not prolonged - loss of consciousness, often make swift and relatively complete physical recoveries, and usually demonstrate minimal or no “objective” neurologic basis for their problems. In many cases, there is little or no recognition, even on the patients’ part, of significant sequelae until they attempt to resume responsibilities at home, work, or school. It is often only then that problems with concentrating, remembering, and working efficiently, particularly on simultaneous tasks or in noisy environments, become apparent. The characteristic psychological problems often emerge only during or after this period, as self-confidence, energy, and productivity decline. It is also in this later stage that changes in emotional reactivity, often including irritability, frustration, depression, and anxiousness, begin to emerge, and when relationships with family, friends, and colleagues suffer.

In comparison to more severely head-injured patients, the individual with mild TBI is often more acutely aware of cognitive inefficiency and concerned about the ability to function. Historically, they do seek out medical assistance and treatment. But because they rarely demonstrate the classic ‘hard’ neurological signs, they are often told there is no organic reason for their disability and told to wait for further recovery, to learn to live with their symptoms, or to undergo psychiatric evaluation and treatment. Yet ‘waiting’ often does little

to alter the underlying problems these patients are experiencing. Iatrogenic problems secondary to *inappropriate* medication prescriptions may also lengthen the symptomatic period. The aforementioned factors and others may contribute to the feeling frequently experienced by these patients of 'going crazy' and feeling out of control. Reports of alteration in lifestyle, relationships and employment are often staggering, given what was initially viewed as a mild and certainly transient phenomenon.

MALINGERING, SECONDARY GAIN AND ACCIDENT NEUROSIIS

While it would be naive to believe that malingering and secondary gain are not factors in some cases of MTBI, the most current literature indicates that such cases of outright malingering are not as common as once believed.^{15,16} In an article entitled "Malingering Aspects of Mild Head Injury", the authors noted:

Miller contributed greatly to a controversy by claiming that accident neurosis occurs subsequent to head injury. With his views, he fueled a long-standing controversy between opposing attorneys, and his work has been quoted frequently. He reported that nearly all his patients (48 of 50) demonstrated substantial, if not complete, recovery 2 years after their claims were settled. Many others in subsequent studies have disputed this assertion, however. In a study of 500 patients with post-traumatic psychoneurosis, Thompson reported that financial settlement did not significantly alter the course of the illness. In an earlier study, Thompson found that of 190 individuals with posttraumatic psychoneurosis only 15% reported that their symptoms were better after litigation was finalized. More pertinent to the field of brain trauma, Kelly and Smith reported that few of their concussed patients who had not returned to work by the time of the settlement returned to work subsequent to their settlements. Mendelson suggests that the term *compensation neurosis* is invalid because it is not supported by criteria that typically are utilized to validate a disease entity. His study of 1992 demonstrated that 75% of those injured in compensation accidents failed to return to gainful employment, even 2 years after the settlements were finalized.

Canadian neurologists, Drs. Pryse-Phillips and Murray, in their 1985 text *Essential Neurology*, refer to several myths surrounding post concussion syndrome: that it does not occur with severe injuries; that the prognosis is usually good; and that the injured person is merely seeking compensation. The authors reject these myths as unfounded:

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. ...The prognosis is not necessarily good, and although many of the milder cases clear within 6 months, other patients may never completely lose their symptoms¹⁷ [emphasis added]

Dr. Muriel Lezak, in the 1995 edition of her text *Neuropsychological Assessment*, reviewed the literature with regard to the effect of compensation claims:

Insufficient or inappropriate behavioural examinations

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from Mission tells us that no self-respecting professional should be caught dead reading the newspaper or thinking about life, when a direct link-up to the internet or the office computer is there for the taking.

Speaking of techno-correctness, perhaps no one in the legal system is more put-upon by useless information overload than our judges. In the old days, judges had a reasonable case load, and no one looked upon it as being wrong for a judge to go off and have a game of golf once or twice a week after hearing a particularly difficult criminal case or sorting out a vicious custody dispute. After all, the whole business of judging commands that judges be given time to relax and ponder. Yet, while we demand that judges exercise the care and wisdom of Solomon in reaching their judgments, we expect them to take on more and more cases and absorb ever-increasing quantities of information, without respite. The public grows increasingly critical of the Bench, and much of the reason for this, from my point of view, can be found in the simple fact that judges are overworked and pressured to "produce" too many decisions. There is too much processing and too little pondering. Judging, like lawyering, is in danger of becoming too much a measure of quantity rather than quality.

Plainly put, judges need time to think. For my money, the number of judges in this Province should be doubled, and members of the Bench should be expected to sit in court for no more than 50% of a normal work week. They should be allowed (and expected, in most cases) to render their judgments within a few days following the conclusion of any case, but they should be relieved from the burdens imposed on the rest of our profession in terms of time and work pressures. These provisions should not be regarded as privilege or luxury. Rather, they should be regarded as recognition that we respect our justice system enough to commit the resources necessary to ensure that every litigant can expect only the best from the judge in whom he or she puts so much trust.

The recent collapse of a Whistler chairlift has once again raised the thorny issue of balancing the interests of resort operators against the safety of their patrons. The present state of the law regarding liability limitation waivers and exclusion clauses is such that it is probably safe to conclude that those injured in the chairlift collapse may not have any remedy against the resort operators, no matter how negligent, on the facts, those resort operators might have been.

We all recall the furor which arose during the tenure of our last government, in which legislation was introduced to provide wide-sweeping immunity for resort operators against all claims for personal injury arising out of the use of recreational facilities. The legislation proposed at that time would have codified the contractual exclusion clauses used to immunize resort operators against claims. Cogent opposition from this Association resulted in the abandonment of the proposed legislation.

In September, 1994, the Law Reform Commission released its Report on Recreational Injuries. The report dealt specifically with the abuses of liability limitation waivers, and proposed that commercial recreational operators should not be able to exclude or limit liability for personal injury or death arising from a specified list of risks which, by contractual limitation of liability, those operators are presently able to evade. Adoption of the Law Reform Commission's recommendations would not impose strict liability against resort operators. In fact, resort operators should not be burdened with responsibility for a wide class of sports injuries which are caused by the exigencies of participating in potentially hazardous activities. However, resort operators should not be able

to contract out their responsibility to provide safe products and facilities for use by the public, and the recommended legislation would provide a prohibition against such practices. I urge every member of this Association to carefully examine the recommendations contained in the Report on Recreational Injuries, and to ask their elected representatives in Victoria to enact legislation to ensure that the recommendations become law in the swiftest manner possible.



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of head trauma can lead to unjust social and legal decisions concerning employability and competency, can invalidate rehabilitation planning efforts, and can confuse patient and family, not infrequently adding financial distress to their already considerable stress and despair (Nemeth, 1991; Varney and Shepherd, 1991).

In this vein, it should be noted that patients seeking compensation for their injuries do not present more symptoms or deficits on testing than similar patients who do not have compensation claims (Rimel, Giordani, Barth, et al., 1981; Stuss, Ely et al., 1985), but the claimants may tend to complain more than other patients (McKinlay, Brooks, and Bond, 1983). A negative kind of support for the conclusion that litigation or compensation has little effect on patient behaviour was the finding that at three months post trauma, half of a group of mildly injured patients had not returned to work, yet none had compensation claims (R. Diamond et al., 1988). In fact, Shinedling et al (1990) reported not only no test differences between suing and nonsuing patients, but that both groups were deeply involved in denying their trauma-related deficits. Bornstein and his colleagues (1988) failed to find any differences in emotional status between patients involved in compensation issues and those who were not. However, Ruthford (1989) suggests that the stress of being in litigation could affect the duration of symptoms, noting that this effect would not be apparent at six weeks, but would become evident some time later. Yet L.M. Binder (1986) notes that "the effect of compensation claims and preinjury pathology is often secondary to organic factors," pointing out that patients with enduring symptoms are the ones most likely to sue.¹⁸

Dr. Berrol also criticized many of these outdated views of MTBI in a chapter published in the text *Rehabilitation of Post-Concussive Disorders*. The following comments of Dr. Berrol have been incorporated into the definition of mild traumatic brain injury developed by the Mild Traumatic Brain Injury Committee of the American Congress of Rehabilitation Medicine:

Post mortem neuropathological studies have, however, demonstrated that structural changes do occur in at least some patients who have had persistent symptoms as a result of a concussion, particularly when acceleration/deceleration mechanical forces have been present. ...

A concussion may result in rapid and complete recovery or in persistent symptoms; in the development of mechanical strain forces that are rapidly dissipated; or in the production of structural and electrophysiological

changes that persist. ...

The constellation of problems previously referred to as minor head injury, post-concussive syndrome, traumatic head syndrome, traumatic cephalgia, post-brain-injury syndrome, and post-traumatic syndrome should be identified under a common diagnostic category. The term *mild traumatic brain injury* identifies the degree of injury, the etiology, and the pathological substrate, and it is the preferred diagnostic category. ...

Some terminology has evolved to imply that persistent symptoms have no clinical basis, in spite of the literature described above. Persistence of symptoms has been suggested as having a direct relationship to the desire for compensation. ...Others who have attempted to establish a relationship between symptoms and desire for compensation have failed to do so. ...On the other hand, large multicenter studies in which unselected patient populations are evaluated have identified statistically significant numbers of mild head injured with longstanding symptoms that affect daily function and who have no potential for litigation. Others have argued that such pejorative terminology, which implies a preconceived concept of causation, not infrequently leads to inappropriate clinical management. Many investigators have actively sought to identify relationships between postinjury personality complaints and the issue of personal gain and compensation - or litigation and premorbid personality disorders - without success. ...

Although a small subset of patients with persistent symptomatology may have secondary gain on a conscious or unconscious level, or as a result of a pre-existing psychopathological condition, acceleration/deceleration injuries do lead to neuronal dysfunction of varying degrees, with established pathological alterations in a significant number of individuals. Mild brain injuries do result in neuronal dysfunction that may lead to persistent symptoms. Mild injuries to the brain may produce effects that are not 'minor' and may last for indeterminate periods of time. Terms that suggest the degree of impairment that may result from trauma, such as 'mild,' 'minor,' or 'trivial,' should be avoided in describing the consequences of injury. ...

It should be recognized that patients with mild traumatic brain injury can exhibit persistent emotional, cognitive, behavioral, and physical symptoms, alone or in combination, that may produce a functional disability even when the initial signs of injury are no longer present.

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.¹⁹

The new definition of MTBI was published in *The Journal of Head Trauma Rehabilitation*²⁰ and the full definition (including comments) was reproduced in the December/94 edition of *The Verdict*. A MTBI is defined as "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."

The mechanism of the injury is described in the definition and

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includes "1) the head being struck, 2) the head striking an object, and 3) the brain undergoing an acceleration/deceleration movement (i.e., whiplash) without direct external trauma to the head."

Clearly it is the subgroup of victims with persistent symptoms - the *walking wounded* - who find their way into the lawyer's office. Yet the perpetuation of the mythology surrounding MTBI makes it difficult for the lawyer involved in a MTBI case to demonstrate to a treating physician, Judge or members of a jury just how devastating the cognitive, physical and emotional symptoms can be to the daily functioning of the injured individual. Dr. Lezak, provides the following description of how just one symptom of MTBI, viz., "fatigue," can produce significant dysfunction:

Emotional distress and fatigue The third major problem category involves dysphoric emotional alterations in which fatigue may be the chief culprit, with both exquisitely acute awareness of deficits and compromised mental efficiency running close seconds to fatigue (Lezak, 1988d; Wand and Goltz, 1991). As a result of the slowed processing resulting from many microscopic sites of damage diffusely distributed throughout cerebral white matter and the upper brain stem, activities that were automatic now may only be accomplished with deliberate effort.

The activities that are normally automatic but become effortful after the injury, particularly during the first weeks or months, include many that are performed frequently throughout a normal activity day, such as concentrating, warding off distractions, reading for meaning, doing mental calculations, monitoring ongoing performances, planning the day's activities, attending to two conversations at once or conversing with background noise, etc. Some patients (44% in one study) complain of discomfort in bright light but, on testing, even more display a lowered threshold for luminance tolerance (Gronwall, 1991). It is little wonder that by late afternoon, if not by noon, many of these patients are exhausted. Making matters worse, as they get fatigued their efficiency plummets to even lower levels so that activities that were difficult when they were most rested and competent become extremely labored and even more error prone; e.g. they become more distractible, make more mistakes when speaking, become more clumsy, etc. Further compounding their burdens is heightened irritability, an experience with which everyone who has been ill or had surgery should recognize: when one's energy is depleted, patience and frustration tolerance drop and irritability emerges in their stead. No one's disposition is improved by fatigue; and severe fatigue can make the mildest person scratchy and short-tempered (Boll and Barth, 1983). Galbraith (1985) wisely points out that the frustrating experience of mental inefficiency may well contribute to irritability following mild injury. He also notes that it could result from direct damage to the limbic system although no site has been identified.

Unless specifically forewarned that these problems might occur and that they are natural consequences of an accident that may seem to have been an inconsequential event, patients experiencing the typical postconcussion symptoms, including fatigue and irritability, may become anxious, lose self-confidence, and be bewildered by the puzzling and unpleasant changes in themselves

(Conboy et al., 1986; Gronwall, 1974; McLean, Dikmen, et al., 1984). Many become acutely sensitive to them (Wang and Goltz, 1991). Some patients fear they may be going crazy. Many report a period of depression (J.D. Miller and Jones, 1990), and others develop the symptoms of depression and even suicidal ideation without spontaneously reporting their distress (Varney and Shepherd, 1991a). Depression typically does not set in until some time - usually about six months - after the injury, possibly because it takes that long for patients with enduring symptoms to become fully appreciative of their limitations and that these problems are not going to go away quickly (Fordyce et al., 1983; Prigatano, 1987b). Some head trauma patients remain depressed for a year and more (Varney, Martzke, and Roberts, 1987). That posttraumatic depression takes time to evolve strongly suggests a psychogenic etiology, perhaps akin to the grief that follows emotionally significant loss (H F. Jackson, 1988).

Those whose problems with mental inefficiency are relatively severe and enduring tend to become distressed by them, but these patients also may develop useful compensatory techniques such as working very slowly and double checking themselves to ensure correctness, concerns and traits akin to those obsessive-compulsive persons (Lezak, 1991; McKeon et al. 1984). Of course, personality predispositions can affect how the patient deals with these symptoms and may contribute to some patients' disablement (Kwentus et al., 1985; Rutherford et al., 1979).²¹

Why do some persons recover from MTBI while others continue to exhibit symptoms of the injury? Those examiners who have not stayed abreast of the literature fail to appreciate the potentially debilitating effects of a MTBI and seem anxious to attribute the persistence of symptoms to malingering and secondary gain. A typical example is the following characterization of a woman who suffered from significant physical, cognitive and emotional symptoms following an accident:

The injury seems to have wreaked havoc in her physical, emotional and everyday functioning, in a fashion far exceeding any demonstrable medical side effects. One can only conclude that there was a chronic pre-existing pattern of very inadequate inner resources and intelligence to cope and this incident served as an excuse to opt out of an unsatisfying life situation, or to capitalize on many secondary gains.

This opinion was provided by a clinical psychologist in a consultation report to a family physician. The case involved a woman who struck the back of her head against the rear window of her pickup truck after her vehicle was rear-ended. She was dazed and suffered only a transient LOC or altered state of consciousness. The case eventually settled for significant damages after a CT scan revealed damage to the frontal and occipital lobes of the brain consistent with *coup and contrecoup* brain injuries. However, in the vast majority of MTBI cases, neurodiagnostic imaging will fail to reveal any damage as the injuries to the brain are microscopic in nature and below the resolution of current brain imaging technology.²²

THE "THIN SKULL" OR "EGGSHELL PERSONALITY"

While there will be cases where this description by the psychologist applies, the recent literature in the field of MTBI, indicates that some individuals are simply more vulnerable to lasting injury than others. Many of the 10 to 15 percent of MTBI victims

who never completely recover may fall into the category of the "thin skull" or "eggshell personality" case. These individuals will have suffered long-term effects of a MTBI, not because they are all malingers or looking to capitalize on secondary gains, but because they have a greater susceptibility (physical and/or psychological) to this type of injury. This susceptibility can arise as a result of prior concussive injuries from which the individual made what appeared to be an uneventful recovery or as a result of a particular personality type rendering that individual more vulnerable. In terms of compensation, the appropriate question should be whether the physical and/or emotional consequences suffered by the individual are genuine and whether they arise as a result of the accident. If the answer is in the affirmative, then the law will provide compensation.

It is trite law that the tortfeasor takes his victim as he finds him and that the degree of damage suffered as a result of the tort will depend upon the characteristics and constitution of the victim.²³ A tortfeasor who injures another must accept the risk that his victim suffers from a frail skull or an unusual susceptibility to injury.²⁴ The thin skull situation arises where greater harm was caused because of the susceptibility to trauma.²⁵ Accordingly, a victim of MTBI who does not recover as quickly as one might expect or who suffers a more significant disability due to a prior concussive injury, is entitled to recover damages to the full extent of the injuries.

The thin-skull rule was first enunciated by Lord Justice Kennedy in *Dulieu v. White & Sons*,²⁶ in the following statement:

If a man is negligently run over or otherwise negligently injured in his body, it is no answer to the sufferer's claim for damage that he would have suffered less injury, or no injury at all, if he had not had an unusually thin skull or an unusually weak heart

In the just released 1996 edition of *Personal Injury Damages in Canada*, Cooper-Stephenson commented:

All that the thin skull rule does is to accept that 'the characteristics and constitution' of personal injury victims are different, and that this may lead to greater loss than might normally be anticipated. The rule applies even where the injury resulting from a relatively minor burn or abrasion is devastating.²⁷

The common sense rationale for this rule is self-evident when one considers the converse situation, viz., where a plaintiff with a thick skull, strong back, or unusually robust personality recovers less in damages than what would normally be expected as a consequence of a faster than anticipated recovery. The defendant benefits from a lower award of damages when he injures a plaintiff with a strong back or sturdy personality. However, "if the injury proves more serious in its incidents and its consequences because of the injured man's [pre-accident] condition, that does nothing but increase the damages the defendant must pay."²⁸

The authorities are clear that the thin skull rule applies to emotional as well as physical susceptibility, although there appears to be a need to differentiate between pre-accident susceptibility and post-injury mental attitude.^{29,30} Physical injury which triggers personality change is compensable.³¹ The question is whether the trauma was the "straw that broke the camel's back." In his text, *Canadian Tort Law*, Linden canvasses a number of cases where the courts have awarded full compensation for the "vulnerable personality."³² In the case *Malcom v. Broadhurst*,³³ Lane, J. said:

...there is no difference in principle between an eggshell skull and an eggshell personality...Exacerbation of her nervous depression was a readily foreseeable consequence of injuring her...Once damage of a particular kind, in this case psychological, can be foreseen,...the

fact that it arises or is continued by reason of an unusual complex of events does not avail the defendant.

The "eggshell skull" or "vulnerable or eggshell personality" is particularly applicable to cases of MTBI.

PRIOR TRAUMATIC BRAIN INJURY

The cumulative deleterious effects of concussion have been recognized since the research by Gronwall and Wrightson published in 1975.³⁴ Dr. James Kelly, Director of the Brain Injury Program at the Rehabilitation Institute of Chicago and Assistant Professor of Rehabilitation Medicine and Neurology at Northwestern University Medical School, is the author of "Concussion in Sports," published in *The Journal of the American Medical Association* in which he discusses the cumulative effects of multiple concussive events. He gives as an example the case of a 17 year-old who suffered a concussion without LOC in a high school football game. The only complaint was a headache during the next week of school. In the next game a week later, an analysis of game films showed that the player was stunned by a tackle, but mental functions appeared to clear quickly during a time-out. On the next play his helmet made light contact with another player. He arose under his own power and then lost consciousness. *He was pronounced dead 15 hours later.* The autopsy:

...found no notable brain hemorrhages, recent or remote anoxic damage, or diffuse axonal injury. Only the focal axonal swellings in the brain stem could be attributed to the original concussion 1 week before death. The most impressive findings were the massive swelling and vascular congestion of the brain...³⁵

In a 1995 paper presented at a recent conference, Dr. Kelly reviewed the literature confirming the potentially devastating effects of a cerebral concussion:

Secondly, repeated concussions that are spaced near in time to each other can lead to catastrophic neurologic injury. This has been reported in the literature as the "second impact syndrome," which is the development of brain swelling after a second concussion while an individual is still symptomatic from an earlier concussion. This form of brain swelling is due to cerebrovascular congestion and dilation of the blood vessels of the brain due to diminished vasomotor control.

And third, even repeated concussions spaced distant in time from each other can impart cumulative neurological damage reflected in documented neuropsychological decline in mental performance, atrophy on repeated neuroimaging studies, and the development of dementia (global intellectual decline) with Parkinsonian features first noted in boxers and termed *dementia pugilistica*.

There is a common misconception that an individual must be rendered unconscious to have suffered a concussion. In fact, the Congress of Neurological Surgeons stated in 1964 that concussion is an alteration in mental status produced by mechanical forces, with no mention of loss of consciousness in the criteria for diagnosis. Thoughtful articles appeared in the literature around the same time identifying confusion and amnesia as the most consistent clinical features of concussion. Loss of consciousness is a clear sign that TBI has occurred with sufficient force to affect bilateral hemispheric or brain stem functions.

The appearance of post-traumatic amnesia is even more convincing when combined with transient confusion or disorientation. Any period of loss of consciousness, no

matter how brief, is clear evidence for biomechanical injury to the brain, and there seems to be uniform agreement that this constitutes what had traditionally been called *classical concussion*.³⁶

... Furthermore, animal studies have shown that reactive axonal swelling is seen on electron microscopy after mild traumatic brain injury, calling into question the long held belief that no anatomical change occurs in concussion.³⁷ [This anatomical change in the form of microscopic brain damage was reported by Oppenheimer where he examined the brains of persons who sustained a concussion and died within several days of causes unrelated to the concussion].³⁸

Dr. Kelly also serves as a medical consultant to the Chicago Bears of the NFL and developed guidelines for preventing brain damage in athletes who experience concussive injuries. The increasing awareness and concern about the effects of concussion led to a meeting of the NFL Physicians Committee. Dr. Mary Chaeung of the National Institute of Neurological Disorders and Stroke indicated at the meeting that repeated concussions can cause bleeding at the site of the injury, death to brain cells and the tearing apart of the connections between brain cells.³⁹ Dr. Chaeung said "Its like a permanent dimming of the lights for the individual rather than a total black-out."

Further confirmation of the cumulative effects of concussive injuries can be found in the following review of the literature by Dr. Lezak:

Repeated head injuries tend to have a cumulative effect on cognition as a second, even mild concussion, leaves the victim somewhat more compromised than if this had been the sole injury (Gronwall, 1989b, 1991; with Wrightson, 1975). *Moreover, a single traumatic injury to the brain doubles the risk for a future head injury, and two such injuries raises the risk eightfold (Gaultieri and Cox, 1991).*⁴⁰ [emphasis added]

Thus, it is extremely important for the lawyer involved in a MTBI case to carefully explore the medical history of a client to ascertain whether a prior concussive event occurred. This may provide the medical basis to explain the failure to recover as expected from a subsequent concussive event or MTBI. The evidence that the effect of repeated concussions is cumulative is important in laying the foundation for the application of the "thin skull rule." Furthermore, the research supports more than just the proposition that cumulative concussions trigger a MTBI (the "straw that broke the camel's back). In a MTBI case, the fine thread-like nerve cells become stretched. Many of these stretched fibers either cease to function or function abnormally. It is the malfunction of these cells that provides the organic basis for the deficits experienced after MTBI.⁴¹ In a 1986 article, "Minor Head Injury: An Introduction for Professionals," Dr. Kay stated:

...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... even after "complete recovery," the occurrence of additional minor head traumas (especially common in sports) may eventually produce noticeable deficits, even though none is any worse than the first - *implying that the initial "complete recovery" was really a decrement in nervous system integrity too small to notice behaviorally...* many persons who later in life develop significant emotional,

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interpersonal, or behavioral problems, are found to have, upon careful interviewing, a history of minor head trauma at some time in their lives.⁴² [emphasis added]

INDIVIDUAL VULNERABILITY

Individuals may be differentially susceptible to identical trauma to the brain for a variety of reasons. Every brain and every personality is unique. The concept of "individual vulnerability" was introduced by Dr. Kay to explain the persistence of symptoms in a significant minority of MTBI cases:

The concept of "individual vulnerability" suggests that a large number of variables will influence how the injury will affect the person, and that each person has a given level of "vulnerability" on each of these dimensions. We know least about neurologic vulnerability. Individual differences in brain structure, hormonal and neurotransmitter balances, and other biologic systems may make one brain more susceptible to, say, an excitotoxic cascade than another brain. Other factors such as age, drug or alcohol abuse, or prior central nervous system (CNS) damage may also increase neurologic vulnerability, magnifying the functional effect of loss of a relatively small number of nerve cells. In addition, a wide variety of psychosocial and personality variables, including family dynamics, type of work, and many more, help determine how each individual person will uniquely react to the trauma of an accident, the presence of symptoms, and the persistence of subtle but real changes in cognitive capacity. (2)(3)(22). The interaction of these neurologic and psychological variables determines an individual vulnerability for each person who suffers a concussion and helps account for the inconsistency in outcomes after apparently similar neurologic events. ... Failure to medically diagnose mild TBI and anticipate the cognitive and behavioral sequelae exacerbates the psychological deterioration of the person. When a person with a genuine mild TBI suddenly finds him - or herself forgetting things, making errors, and taking longer and requiring more effort to do things that used to be automatic; when the person starts becoming disorganized, irritable, and getting into conflicts with friends, co-workers, and family; and when he or she is told by professionals that there is nothing wrong, that he or she should get on with life, then nothing exists to validate the experience that something is wrong, and the sense of self begins to erode. If subsequent medical follow-up fails to provide quick and useful diagnostic feedback on the post-concussive state, the person in danger of spiraling downward into failure, frustration, fear, avoidance, and loss of confidence and self esteem, and ultimately the person feels like he or she is "going crazy". If this psychological deterioration continues unabated, it can become more debilitating than the primary, neurologic deficits that fuel it. ...

No variable is more complex and important in understanding functional disability after mild TBI than personality. The situation is most clear in the extremes. A history of well-adjusted personality functioning in a flexible individual who has shown the ability to deal well with stress makes more credible the conclusion that true organic damage has occurred when there is a sudden and dramatic drop in ability to function after mild TBI. ...

While there is no established taxonomy of personality

styles at risk for a dysfunctional response to mild TBI, five examples can be cited from our clinical experience. First, we have noted that persons who are *highly driven - often obsessive-compulsive - overachiever*, whose sense of self is tightly bound up with intellectual pursuit and achievement, are greatly at risk for a catastrophic breakdown of the self after a mild TBI in which real cognitive problems persist. Often these persons have histories of success and achievement, but lack an inner self esteem. The "cognitive slippage" from a mild TBI is sufficient to "knock out" the high-performance mental engine that propelled them with sufficient speed to keep them emotionally afloat; the effects of a neurologically real mild TBI are psychologically devastating because they steal away the only source of self-esteem. The subtle neurologic event unmasks the underlying lack of sense of self. It is a mistake to write such persons off as having purely psychological problems; their psychological devastation must be seen as a secondary reaction to a primary neurologic event. They are not imagining or creating their problems, although their personalities do magnify the impact of the problems.

Second, we have noted that *persons who suffered emotional deprivation as children*, when they are injured in ways that retribution cannot be extracted (as is often the case in mild TBI) may become extremely hostile and dysfunctional in the presence of permanent symptoms.

... Third, *persons with strong tendencies toward dependency* are often immobilized by the symptoms of mild TBI, especially in the acute state, and respond by decreasing activity and increasing their anxiety about their inability to function. Even when symptoms actually resolve over time, the self-paralysis dynamic, set in motion early on, can take on a life of its own and continue to render the dependent person dysfunctional long after the primary sources of deficit have receded. Beside arguing strongly for an early interventional model (to counteract the self-isolating tendencies of the dependent person), such a possibility renders it imperative for the evaluator to weigh the contribution of a dependent personality style when determining the causes of functional disability.

Fourth, the term "*borderline*" personality has been widened beyond its strict diagnostic criteria to include a group of high functioning, often very intelligent persons, who appear to have high levels of emotional rigidity and impaired capacity for deep human relationships, and often manifest some "borderline" characteristics in a mild form, such as affective instability, intense anger, feelings of emptiness, and fear of rejection. Such persons, especially when they have poor social support systems, also do quite poorly after mild TBI when real neuropsychological deficits persist. Their coping threshold seems to have been exceeded by the increase in symptoms, and they become overwhelmed by the difficulties they face. Again, the identification of such a personality dynamic should *not* be the cause of negating the impact of the neurologic event; it simply means that in understanding the level of dysfunction and planning a program of rehabilitation, these personality factors need to be taken into account.

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Fifth, a different set of problems is presented by the *person with tendencies toward grandiosity*, inflated self-belief, and other elements of a narcissistic personality style. Such persons often minimize, deny, or hide the difficulties they are having, to the extent that their life must crumble around them before they will acknowledge to others the difficulty they are having. Unfortunately, when the reality finally hits, the narcissistic wound can be enormous and devastating, presenting a unique therapeutic challenge.

Again, these personality factors may not be in evidence during cursory history-taking, nor do they mean that real, organically based deficits do not exist. They do, however, complicate the neuropsychological diagnosis and present additional challenges to the process of rehabilitation.⁴³

CONCLUSION

The question for the trier of fact in cases of MTBI is whether the injured person is one of the "walking wounded" - the 10 to 15 percent of victims who for one genuine reason or another do not recover from their injury. The debate among various researchers as to whether the origin of these long-term problems associated with MTBI is organic or psychogenic may be interesting from a medical or scientific perspective, however, in the legal setting it has little practical significance. The law provides full compensation for physical and psychological injuries arising from the trauma regardless of the physical or psychological susceptibility of the injured victim. In a MTBI case this susceptibility can arise either because the injured person has a pre-morbid history of one or more concussive events or as a consequence of a "vulnerable personality." The only reason to distinguish between the physical and psychological effects or deficits arising from a MTBI is the extent to which this difference impacts on recovery.

Dr. Lezak provides a comprehensive review of the TBI literature in her 1995 text. She made the following observation with regard to "recovery" from MTBI:

I no longer use the term "recovery" when discussing brain damage. *Brain damage that is severe enough to alter the level of consciousness even momentarily, or to result in even transient impairment of sensory, motor, or cognitive functions, is likely to leave some residual deficits.* In cases where the damage is more than mild, the use of the word "recovery," which implies restoration or return to premorbid status, when discussing the patient's prognosis can give the patient and family false hope, delay practical planning, and cause unnecessary anxiety and disappointment.⁴⁴ [emphasis added]

The task for the lawyer representing a member of the "walking wounded" is to fully inform the Judge and jury, via credible expert evidence, about the devastating impairments that can arise as a result of a MTBI and to explain, with the assistance of reliable collateral evidence from family, friends and co-workers, the extent to which the *residual deficits* impact the functioning of the injured person.

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