The issue of what constitutes a minor head injury, and what are the expected sequelae, is one which exercises expert witnesses and for that there is no consensus. This article seeks to extrapolate the interconnected issues of mild traumatic brain injury (MTBI), concussion and post-concussion, and to present the evidence base for these. The conclusion is that, if one follows the research evidence, accepts robust theoretical models and adopts the rule of thumb that the results of any assessment of these symptoms have to ‘make biological or psychometric sense’ (Iverson, 2006), it is possible to adopt a consistent position.

What is MTBI?
According to the World Health Organisation (2004), MTBI has a range of clinical indicators such as:

- Glasgow coma scale (GCS) score of 13 or above 30 minutes post injury;
- post-traumatic amnesia (PTA) of no more than 24 hours;
- loss of consciousness of no more than 30 minutes;
- temporary confusion or disorientation; and
- normal brain scan.

Larrabee (2005) reports that:

Cumulative research on the outcome of a single uncomplicated mild traumatic brain injury shows that neuropsychological deficits may persist for up to three months but the norm is full recovery with no long-term residual deficits.

The use of the term ‘uncomplicated’ is key. McCrea (2008) reports a wide range of research as to whether the TBI is complicated (structural brain damage visualized on acute neuroimaging) or uncomplicated (normal brain scan result) as having little predictive value in terms of outcomes for the majority of patients. There are brain changes in mild traumatic brain injury, but these are not a precursor to permanent cognitive deficits. Indeed, MTBI is classically defined as an essentially reversible syndrome without any detectable pathology (Williams et al, 2010).

Is concussion different to MTBI?
Concussion is defined as (Giza and Hovda, 2001):

... any transient neurologic dysfunction resulting from a biomechanical force. Loss of consciousness is a clinical hallmark of concussion but us not required to make the diagnosis.

There is a classification system (by Ommaya and Gennarelli, reported in McCrea, 2008), which places concussion on a continuum with all degrees of head trauma and incorporates the position of the American Academy of Neurology in 1997 (Larrabee 2005). This is presented in terms of level of alteration in mental status and associated characteristics where severity grade I is for confusion with normal consciousness and without amnesia. (This is what is often referred to as mild concussion and is typical in sports-field injuries.) The antithesis of this would be grade VI severity, which is associated with death. The intervening four grades of severity refer to differences in confusion, amnesia and consciousness with reference to the presence and degree of both PTA and retrograde amnesia.

Put more simply, following biomechanical force to the head, the severity of the concussion can be graded according to:

There are brain changes in mild traumatic brain injury, but these are not a precursor to permanent cognitive deficits. Indeed, MTBI is classically defined as an essentially reversible syndrome without any detectable pathology.
• nature of alteration in mental status;
• level of consciousness; and,
• type and degree of memory changes.

However, although these indicators have primary significance in terms of classifying the nature of the injury, they have been shown not to be predictive of ongoing sequelae (Ponsford et al 2012).

Concussion could be considered to be equivalent to MTBI. However, longstanding definitions and understandings of concussions still prevail in the literature, usually that referring to sporting injuries in professional athletes. For example, Gennarelli in 1986 (Lezak, 2004) refers to two types of concussion:

• Mild concussion: this is typified by no loss of consciousness and by symptoms such as ‘seeing stars’ and/or a short period of confusion or disorientation for a brief time after the event.
• Classic concussion: this is defined by a reversible coma ‘at the instant of trauma’, which may be accompanied by cardiovascular and pulmonary changes as well as neurologic abnormalities, all of which dissipate within 20-30 minutes of the triggering event.

In concussion and MTBI, it is accepted that there is a neurometabolic cascade that renders brain cells (neurons) injured but not destroyed (Belanger et al, 2007, in Williams et al, 2010). Giza and Hovda (2001) describe the processes in terms of abrupt neuronal depolarisation. (Typically, neurons pump out sodium and pump in potassium to maintain its resting state (polarised), ready for a stimulus to come along. With concussion, sodium channels are suddenly opened, positively charged sodium ions flood into the neuron making the inside of the cell positively charged thus opening the potassium channels allowing them to leave the cell, thus repolarising the neuron. In order to restore normal function, the sodium-potassium pump works in over-drive, triggering a dramatic jump in glucose metabolism, which, in turn, triggers a cellular energy crisis. At this point, the brain is vulnerable and temporary axonal injury is likely to occur).

There is a range of views within Clinical Neuropsychology as to the aetiology and effects of concussion and MTBI, all of which are supported by research evidence. There are those who believe that concussion, or MTBI, is on a continuum with all degrees of head injury and that the mechanisms of injury are identical, varying only in a degree of severity. Much of the evidence base for such views is taken from research using helmeted dummies (Finite Element analysis), or head models of real head injuries on the sports field replicated in a laboratory. This school of thought posits that there is cell-level damage even within the mildest of head injuries. There is evidence to suggest that significant changes in cerebral glucose metabolism can exist even in head injured patients with normal GCS scores (Giza and Hovda, 2001). However, such changes are unlikely to reach critical levels at which ischaemia (reduced blood supply to cells) can occur. It is more likely to effect a temporary cellular energy crisis.

The alternative view is propounded on the basis of research using HIT technology (Head Impact Telemetry) in college football players across seasons of play. This is considered to provide valuable insights into the lower limits of head acceleration that are associated with concussion and MTBI. It has been demonstrated that the average magnitude of sports concussive impacts is 95g. Interestingly (McCrea, 2008):

... the overwhelming majority of impacts greater than 80g did not result in concussion, suggesting that a minimum threshold of translational force in this range is necessary but not solely sufficient to cause MTBI.

The caveat is that where rotational forces are at play, the causative threshold for MTBI may be lower. Put simply, it is argued from the research evidence that a 100g linear translational force is approximately equivalent to a 25 mph motor vehicle collision with a brick wall, striking the head against the dashboard. McCrea is clear that impact of this magnitude can be observed without the occurrence of brain injury as this represents a minimum threshold at which injury can, but does not necessarily, occur.

From this it can be concluded that the pathology of mild traumatic brain injury (which includes concussion) is temporary and different to that of moderate and severe brain injuries. It is widely acknowledged that the vast majority of individuals recover from such events within one week to six months, with a faster recovery in young, healthy and fit individuals (Iverson, 2005).

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What is Post-concussion Syndrome (PCS)?
Recent research has cast doubt on the existence of Post-Concussion Syndrome as a clinically separate entity.

The difficulty is that the symptoms included in the standard diagnostic criteria for PCS are non-specific, or not exclusive to PCS. For example, headache, dizziness, fatigue, sleep disturbance, irritability, difficulty concentrating are included in both DSM-IV and ICD-10 diagnostic criteria for PCS. Bryant (2011) reflects that:

Symptoms described as PCS are common in many populations, and actually reflect a diffuse collection of frequently experienced sensations. In healthy individuals, headaches, sleep difficulty, irritability, and memory failures are relatively common in daily life.

Larrabee (2005) has earlier demonstrated that the symptoms referred to as PCS are also present, in similar configuration, in medical outpatient populations and in non-neurological litigants. Meares et al (2011) and others have shown that symptoms of PCS are not predicted by the presence of mild traumatic brain injury but by the presence of pain and post-traumatic stress disorder.

There is yet believed to be a ‘miserable minority’ of up to 15% of individuals who experience persistent, disabling symptoms following a
References


Walton, N (2011) ‘No Science Blues’ presentation at St John’s Chambers Conference (Bristol, November 2011)


### What is the evidence?

- The definition of uncomplicated TBI is consistent and consensual.
- MTBI is qualitatively and quantitatively different from moderate and severe TBI.
- There is widespread recognition that a diagnosis of MTBI is not predictive of persisting symptoms but that psychological factors (amongst others) are.
- PCS is a non-specific set of symptoms that are present in non-neurological patients and non-patients alike.
- The development of PCS represents a predominantly psychological response to transient neurological symptoms.
- Early, targeted and brief psychological interventions following MTBI (particularly to those at-risk of developing PCS) have been shown to be effective in preventing the entrenchment of symptoms.
- under-estimation of past (pre-concussion) problems and recovery (Iverson et al, 2010).

Iverson et al (2010) state that it would be a 'mistake to assume uncritically' that these symptoms are causally related to the initial concussion because most such injuries spontaneously resolve fairly quickly and because most symptoms associated with the diagnosis of PCS are non-specific. One has to consider the validity of symptoms in assessments of such clinical presentations (Iverson, 2006).

When assessing patients who lay claim to PCS symptoms, it is clear that the neuropsychological assessment results must make sense. It does not make sense that a small minority of individuals (who are often also pursuing compensatory litigation), develop a set of symptoms that are attributed to undetectable brain damage, usually after a significant passage of time since the initial injury. It does make sense to consider such symptoms through a psychological filter and to consider whether there is evidence of:

- misattribution of benign symptoms to the injury (in the absence of confirmatory medical evidence) thereby pathologising or overinterpreting them; and
- deliberate (or unintentional) exaggeration of symptoms.

Given the connection with 'expectation as aetiology' (Mittenberg et al, 1996), and the development of PCS, there is merit in the longstanding view that psychological therapy (which is based on a cognitive behavioural principles) achieves best effects when provided early in the development of symptoms, before they become entrenched or entangled with complicating factors (Bryant, 2011):

... communicating… that a range of non-specific symptoms are caused by brain damage communicates a cause with a poor prognosis. This expectation that common sensations are signs of permanent dysfunction can result in hypervigilance to every sensation, followed by catastrophic attributions about the adverse consequences of the sensations.

Patients need to be encouraged not to overinterpret the often benign symptoms that can occur. (Wood 2007):

What people know or believe about illness or injury will influence how they interpret bodily sensations. How a person attributes symptoms can determine how they will react to them.

Patients who attribute benign emotional and other changes to their head injury are more likely to develop and perpetuate post-concussive symptoms. Whittaker, Kemp & House (2007) evidence that a patient’s perceptions of their illness early after head injury play a part in the persistence of PCS.

Mittenberg et al (2001) indicate that single-session therapeutic treatments for those ‘at-risk’ of developing PCS can be effective in counterbalancing the person’s underlying perceptions or interpretations of their symptoms.

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concussive injury. However, this figure has recently been questioned and is most likely to actually be much lower, at around the 1% level (Walton, 2011).

King (2003) referred to a ‘window of vulnerability’ for the development of a cluster of symptoms which are referred to as post-concussion syndrome. The idea was that certain individuals were more likely to be pre-disposed to the development of a psychological response to a concussive injury and that if such symptoms are going to emerge they develop at this critical point, which is approaching and beyond the six-month point post-injury. There appears to be a complex interaction of organic and psychological factors. Key indicators for longer-term post-injury difficulties include:

- pre-existing psychopathology (Caroll et al, 2004);
- stress, anxiety, depression (post-injury) (Bryant, 2008);
- concurrent claim for compensation (Binder & Rolling, 1996);
- being female (Ponsford et al, 2000);
- avoidant coping styles (Wood, 2007);
- age over 40 (Thornhill et al, 2000);
- lower education (Stulemeijer et al, 2007);
- physical injuries, pain and medication (Caroll et al, 2004); and
- substance misuse (as an avoidant form of coping);
- under-estimation of past (pre-concussion) problems and recovery (Iverson et al, 2010).

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