Traumatic Brain Injury

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Dr. David Rowed graduated from the University of Western Ontario (B.A. 1962, M.D. 1966). After postgraduate training in internal medicine at The University of Alberta, and
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This medical discussion paper will be useful to those seeking general information about the medical issue involved. It is intended to provide a broad and general overview of a medical topic that is frequently considered in Tribunal appeals.

Each medical discussion paper is written by a recognized expert in the field, who has been recommended by the Tribunal’s medical counsellors. Each author is asked to present a balanced view of the current medical knowledge on the topic. Discussion papers are not peer reviewed. They are written to be understood by lay individuals.

Discussion papers do not necessarily represent the views of the Tribunal. A vice-chair or panel may consider and rely on the medical information provided in the discussion paper, but the Tribunal is not bound by an opinion expressed in a discussion paper in any particular case. Every Tribunal decision must be based on the facts of the particular appeal. Tribunal adjudicators recognize that it is always open to the parties to an appeal to rely on or to distinguish a medical discussion paper, and to challenge it with alternative evidence: see Kamara v. Ontario (Workplace Safety and Insurance Appeals Tribunal) [2009] O.J. No. 2080 (Ont Div Court).
TRAUMATIC BRAIN INJURY (TBI)

Traumatic brain injury (TBI) is a disturbance of normal brain function caused by the action of external mechanical forces. Force may be transmitted to the brain by an impact to the skull, the jaw or the body. Shearing stresses to the brain and blood vessels, caused by abrupt acceleration or deceleration of the head, frequently rotational in direction, are responsible for much of the damage.

Outcomes of TBI can range from spontaneous and complete recovery to severe disability or even death. The overall leading cause of TBI is falls, especially in the young (under 14 years of age) and the elderly (65 and over). Motor vehicle accidents are the second most frequent cause and the leading cause of death in young adults. Males account for two-thirds of TBI, though women may experience a poorer outcome than men with an injury of apparently similar severity. Sports and assaults are other frequent causes of head injury.

TBI in the workplace may be caused by falls, falling objects, motor vehicle and machinery accidents, etc. Construction, transportation, agriculture, forestry, fishing and emergency medical services are occupational areas at highest risk. Twenty percent of workplace injuries are reportedly attributable to falls on uneven or wet surfaces or involving out-of-place objects and, therefore, appear to be highly preventable.

Injuries to the head may be “closed” i.e. covered by intact scalp or they may be “open” or “compound” with laceration of the overlying scalp. There may be an associated fracture of the skull or a laceration of the dura mater (the outermost and strongest of the 3 membranes that lie between the skull and the brain). There also may be contusion (bruising) or laceration (cutting) of the brain itself. Closed linear skull fracture is of little importance except as an indication of the amount of force that has been applied to the head. Closed skull fractures, however, may be depressed and the depressed fragment may injure the brain. Compound injuries constitute a pathway for intracranial infection e.g. meningitis, subdural empyema and cerebral abscess. Compounding may be external or internal. In the latter case, a skull fracture may involve the paranasal sinuses or mastoid air cells at the skull base and, if associated with injury to the dura mater and the arachnoid membrane, may serve as a portal for bacterial contamination with resulting infection as described above.

Skull fractures may also injure blood vessels, causing a hemorrhage which may accumulate as a blood clot (hematoma). The clot may be within the brain itself (intracerebral), or beneath the skull but external to the brain and either beneath the dura mater (subdural) or external to the dura mater (extradural). Since the intracranial volume is fixed, an expanding hematoma or contusion may compress and distort the underlying brain, causing additional injury. Displacement of the brain may result in pressure on the upper brainstem which, among other functions, maintains a state of consciousness. Hematomas may cause acute (immediate), subacute or chronic (delayed) compression of the brain.
Contusions of the brain may occur at a point of impact, with or without an associated depressed skull fracture. They may also occur at a distance from a point of impact due to the surface of the brain coming forcibly into contact with a bony prominence on the inner surface of the skull due to a transmitted force. Injuries opposite a point of impact are called contrecoup (counter blow) injuries. The most common contrecoup injuries are cerebral contusions, subdural and epidural hematomas.

Swelling of the brain (cerebral edema) may follow TBI because the function of cells that line small blood vessels (blood-brain barrier) are impaired, allowing leakage of protein and water into the brain. This may involve all or most of the brain (generalized) or be more limited in distribution (focal) e.g. in association with contusion. Since the skull constitutes a closed box, edema compromises brain function by reducing blood flow by compression of blood vessels and also by causing displacement of parts of the brain in a fashion similar to the effect resulting from blood clots, with resulting neurological deficits. Neurological deficits attributable to an intracranial hematoma, contusion or cerebral edema may onset or progress in a delayed fashion with an asymptomatic interval.

Diffuse axonal injury (DAI) results from shearing forces which disrupt the long processes of nerve cells (axons) by means of which nerve cells communicate with each other and form networks. Severe neurological deficits may result from this type of closed head injury if the shearing forces involved are great.

True incidence of TBI is difficult to obtain with certainty, for many reasons. Many persons who sustain closed head injuries are not seen by a physician or admitted to hospital. Criteria for recording head injury vary. Other injuries may obscure an associated head injury. The size of the population at risk may not be accurately determined. The known incidence of traumatic brain injury in Canada is about 50 per 100,000 population per year and the true incidence is almost certainly higher.

Severity of TBI varies from mild (MTBI), with apparent full recovery in most cases, to severe, with resulting permanent neurological deficit, prolonged coma or death. An understanding of anticipated persisting disability resulting from TBI requires a classification of initial injury severity, which can be correlated with outcome at a time when maximum recovery has been attained. Injuries are classified as mild, moderate and severe.

Moderate and, more especially, severe head injuries which result in clear-cut neurological and radiological findings usually present little difficulty in being recognized as a cause of prolonged disability. The direct injury to the brain that occurs at impact cannot be modified, though some degree of recovery may occur over time. Improvement in outcome depends, in part, upon preventing or treating conditions that result in secondary injury to the brain by interfering with its blood or oxygen supply. Some of these injuries will require urgent neurosurgical management e.g. for evacuation of intracranial hematoma, while others may require intensive medical management e.g. for attempted control of posttraumatic brain swelling (cerebral
Traumatic Brain Injury (TBI)

edema). Measures such as controlled hyperventilation to decrease the carbon dioxide content of the blood in order to reduce intracranial blood volume or the use of diuretics to remove water from the brain have proven utility in reducing brain swelling or to buy time until an intracranial hematoma can be removed.

New treatment modalities are constantly being sought. Progesterone, for example, has recently reportedly shown promise by reducing death and disability in brain injuries. The current results are preliminary and further study is required.

Severe head injuries may result in prolonged coma and permanent disability or death, despite intensive treatment. In severe head injuries there is some data to support the belief that longer duration of posttraumatic amnesia (PTA), a partial or complete loss of memory for events following TBI, correlates with poorer outcome. It needs to be emphasized that this applies only to severe injuries. For example, in one study more than 60 percent of head-injured patients experiencing PTA lasting 2 to 4 weeks returned to productive activity at 1 year post-injury whereas those experiencing more than 70 days of PTA had a less than 20% probability of returning to productivity at one year.

Mild traumatic brain injuries (MTBI), however, are often more difficult to understand as a cause of prolonged disability and these will be considered at greater length.

The instrument most widely employed for standardized assessment of head injuries, since 1974, is the Glasgow Coma Scale (GCS). Since this will be encountered regularly in medical records and serves as the instrument for classification of TBI as mild, moderate or severe, it is reproduced below.

**Glasgow Coma Scale (GCS)**

<table>
<thead>
<tr>
<th>Category</th>
<th>Response</th>
</tr>
</thead>
<tbody>
<tr>
<td>Eye opening</td>
<td></td>
</tr>
<tr>
<td>Spontaneous</td>
<td>4</td>
</tr>
<tr>
<td>To speech</td>
<td>3</td>
</tr>
<tr>
<td>To pain</td>
<td>2</td>
</tr>
<tr>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Verbal</td>
<td>(Modified for Infants)</td>
</tr>
<tr>
<td>Oriented</td>
<td>Babbles</td>
</tr>
<tr>
<td>Confused</td>
<td>Irritable</td>
</tr>
<tr>
<td>Inappropriate words</td>
<td>Cries to pain</td>
</tr>
<tr>
<td>Moans</td>
<td>Moans</td>
</tr>
<tr>
<td>None</td>
<td>None</td>
</tr>
</tbody>
</table>

3
**Traumatic Brain Injury (TBI)**

<table>
<thead>
<tr>
<th>Category</th>
<th>Response</th>
<th>Score</th>
</tr>
</thead>
<tbody>
<tr>
<td>Motor</td>
<td>Follows commands</td>
<td>6</td>
</tr>
<tr>
<td></td>
<td>Localizes pain</td>
<td>5</td>
</tr>
<tr>
<td></td>
<td>Withdraws to pain</td>
<td>4</td>
</tr>
<tr>
<td></td>
<td>Abnormal flexion</td>
<td>3</td>
</tr>
<tr>
<td></td>
<td>Abnormal extension</td>
<td>2</td>
</tr>
<tr>
<td></td>
<td>None</td>
<td>1</td>
</tr>
<tr>
<td>Glasgow Coma Score</td>
<td>Best possible score</td>
<td>15</td>
</tr>
<tr>
<td></td>
<td>Worst possible score</td>
<td>3</td>
</tr>
<tr>
<td>If tracheally intubated then verbal designated with “T”</td>
<td>Best possible score while intubated</td>
<td>10T</td>
</tr>
<tr>
<td></td>
<td>Worst possible score while intubated</td>
<td>2T</td>
</tr>
</tbody>
</table>

**Mild TBI** (GCS 13-15), **Moderate TBI** (GCS 9-12) and **Severe TBI** (GCS 3-8)

Much of what follows will be devoted to MTBI and post-concussion syndrome. As noted above, workplace events, which result in more severe head injuries, are, in general, easily understood as causes of acute and continuing disability. MTBI, on the other hand, is less well understood, by physicians and lay persons alike.

**Mild Traumatic Brain Injury (MTBI) or Cerebral Concussion**

MTBI is defined as head injury giving rise to a Glasgow Coma Scale of 13-15 at initial post-injury assessment. It is estimated that at least 75% of TBIs are mild by this criterion.

Cerebral concussion consists of an **alteration of consciousness** as a result of closed head injury. **There need not be loss of consciousness.** It is often difficult to establish whether or not loss of consciousness occurred unless eye witnesses are available and, perhaps, not even then. The head injured person may not recall loss of consciousness. In one series of witnessed concussed athletes, loss of consciousness was reported by only 25% of injured subjects. This tends to lead to an underestimation of the true incidence of unconsciousness. Typical clinical features are vacant expression, delayed responses to questions or instructions, distractibility (lack of focus), disorientation to person, place or time, slurred or incoherent speech, incoordination, exaggerated emotional responses, impaired short term memory and, in many instances, observed loss of consciousness.

The apparent severity of concussion varies and, consequently, several grading scales and guidelines have been proposed. Some of the more popular of these are the American Academy of Neurology and the Cantu grading scales and the Vienna, Prague and Zurich guidelines. This literature is derived from sports injuries with the intent of establishing guidelines for safe return to play. Unfortunately, the grading
scales are not well supported by sufficient outcome data and are therefore not particularly helpful in predicting the consequences of concussion e.g. the duration of postconcussional symptoms. These scales and guidelines, at best, may be helpful in deciding when an athlete may return to play but are not particularly helpful in other contexts, such as the workplace.

Duration of unconsciousness and duration of either PTA or retrograde amnesia (RGA) - partial or complete loss of memory for events preceding TBI, are considered to be indicative of the severity of concussion. The underlying mechanisms of PTA and RGA differ slightly and PTA is probably a better predictor of outcome. Concussions followed by loss of consciousness for longer than 1 minute or followed by convulsive seizures or by prolonged cognitive impairment and also repeated concussions have been designated as “complex”. The term is not particularly helpful but does describe concussions that may reasonably be considered to probably be more severe than transient loss of consciousness. There is some statistical evidence to support the belief that initial cognitive impairment persisting at 48 hour follow-up correlates with longer duration of postconcussional symptoms onsetting hours to days after injury.

Cerebral concussion is not typically associated with abnormalities in either computed tomography (CT) or magnetic resonance imaging (MRI) of the brain. It is possible that certain newer MRI techniques may prove valuable in studying concussion and in evaluating post-concussion syndrome in the future (see below). Metabolic changes in the brain caused by concussion have been studied but thus far have not proven helpful in management of concussed patients.

Postconcussion Syndrome, (Postconcussive Syndrome, Postconcussional Disorder)

Postconcussion syndrome may result in prolonged disability in workplace injuries.

It has long been recognized that persons who sustain a cerebral concussion may experience a number of persisting symptoms in ensuing weeks or months. These include headache, dizziness, “lightheadedness” or imbalance, nausea, visual impairment (typically “blurring”), hearing impairment (tinnitus or “ringing in the ears”, decreased auditory acuity), impairment of concentration and/or memory or other cognitive impairment e.g. judgment and slow information processing, sleep disturbance, emotional lability, irritability or depression, easy fatigability, photophobia (abnormal sensitivity to light, pathological fear of and avoidance of light places) , phonophobia (pathological sensitivity to noise) and personality change. Many or few of these symptoms may be reported, along with a host of less common features.

Formalized diagnostic criteria have been developed for post-concussion syndrome. The two most widely accepted listings of diagnostic criteria are reproduced below.
The Diagnostic and Statistical Manual of Mental Disorders, 4th Edition (DSM-IV) criteria for Postconcussional Disorder are as follows:

A. A history of head trauma that has caused significant cerebral concussion.

B. Evidence from neuropsychological testing or quantified cognitive assessment of difficulty in attention (concentrating, shifting focus of attention, performing simultaneous cognitive tasks), or memory (learning or recalling information).

C. Three (or more) of the following occur shortly after the trauma and last at least 3 months:
   1. Becoming fatigued easily
   2. Disordered sleep
   3. Headache
   4. Vertigo or dizziness
   5. Irritability or aggression with little or no provocation
   6. Anxiety, depression, or affective lability
   7. Changes in personality (e.g., social or sexual inappropriateness)
   8. Apathy or lack of spontaneity

D. The symptoms in criteria B and C have their onset following head trauma or else represent a substantial worsening of pre-existing symptoms.

E. The disturbance causes significant impairment in social or occupational functioning and represents a significant decline from a previous level of functioning. In school-aged children, the impairment may be manifested by a significant worsening in school or academic performance dating from the trauma.

F. The symptoms do not meet criteria for dementia due to head trauma and are not better accounted for by another mental disorder (e.g., amnestic disorder due to head trauma, personality change due to head trauma).

I would note in relation to the DSM-IV criteria above that concussion consists solely of an alteration of consciousness as a result of closed head injury and would attach no particular significance to the designation “significant” in this context.

The International Classification of Diseases (ICD-10) diagnostic criteria for post-concussion syndrome are as follows:

A. A. History of head trauma with loss of consciousness preceding symptom onset by a maximum of 4 weeks.

B. Symptoms in 3 or more of the following symptom categories:
   - Headache, dizziness, malaise, fatigue, noise intolerance;
   - Irritability, depression, anxiety, emotional liability;
Subjective concentration, memory, or intellectual difficulties without neuropsychological evidence of marked impairment;

- Insomnia;
- Reduced alcohol tolerance; and
- Preoccupation with above symptoms and fear of brain damage with hypochondriacal concern and adoption of sick role.

The features of MTBI patients described by these 2 classifications differ slightly.

Most patients who experience postconcussion symptoms recover within 7-10 days. Systematic reviews and meta-analyses of neuropsychological functioning indicate that objective impairments can be measured in the initial days and weeks after MTBI, but that effects are not typically apparent beyond 3 months after injury. Neuropsychological test scores tend to improve rapidly during the first 6 months after injury. Although subjective cognitive complaints are common, the presence and significance of objective cognitive dysfunction in persistent PCS is frequently difficult to document.

Those who experience persisting postconcussion symptoms beyond 3 months have a higher incidence of comorbidities. Overall, the results of outcome studies indicate that patients with persisting postconcussion symptoms experienced higher levels of depression, anxiety disorders and posttraumatic stress symptoms and were more likely to suffer from substance abuse. They had fewer supportive persons and perceived a lower quality of support, greater difficulties in aspects of overall community integration, poorer health-related quality of life, both mental and physical, including the individual’s perception of his or her illness. These patients also experienced poorer global outcomes. Presence of persisting postconcussion symptoms beyond 6 months post injury predicts a probability of a poorer final outcome after MTBI.

There are potential motives for secondary gain related to postconcussion syndrome which include financial reward or drug-seeking.

A long-recognized paradox is the fact that the number and frequency of postconcussional complaints following minor head injury are disproportionately great compared with those following more serious head injuries that demonstrate clear-cut pathology, clear-cut neurological findings and abnormal findings on medical imaging.

Treatment of Postconcussion Syndrome

There is, at present, no clear evidence that therapeutic measures beyond supportive and symptomatic treatment are of benefit in reducing the severity or duration of postconcussion symptoms.

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MTBI, but that effects are not typically apparent beyond 3 months after injury. Neuropsychological test scores tend to improve rapidly during the first 6 months after injury. Although subjective cognitive complaints are common, the presence and significance of objective cognitive dysfunction in persistent PCS is frequently difficult to document.

Incidence of psychiatric comorbidity, as noted above, is high in individuals with persistent PCS. About two-thirds may meet diagnostic criteria for either DSM Axis I and/or Axis II disorders. After MTBI, residual direct effects of brain injury may contribute little to persistent symptoms lasting beyond 3-6 months. Comorbid depression may increase the reporting of both the number and severity of symptoms after MTBI. Depression may affect some PCS symptoms more than others. Impairment of psychomotor speed and sustained attention, for example, may be particularly affected by concomitant depression. Ensuring that pre-existing psychiatric conditions are optimally treated may be beneficial to patients experiencing persistent PCS.

Prophylactic educational programmes (in the initial hours, days and weeks after injury), emphasising the common nature of postconcussional symptoms and the expectation that the symptoms will improve over time, have achieved some positive results in reducing persistent symptoms of PCS. Systematic educational programs following MTBI might reduce the overall morbidity of TBI.

Involvement in a medicolegal process is recognised as a risk factor for persistent symptoms after MTBI. An ongoing medicolegal claim may inadvertently reinforce maladaptive responses, such as increasing focus on symptoms without associated management, or emphasising issues of blame and responsibility. Resolution of ongoing legal proceedings, if possible, may improve PCS symptomatology.

In general, athletes recover from PCS symptoms much more quickly than nonathletes. Preliminary therapeutic attempts to utilize this observation by using a controlled exercise program suggests that this approach may be beneficial in patients with persisting PCS symptoms but, at this point, there is no certainty that a more systematic approach to controlled exercise would be beneficial.

Cognitive-behaviour therapy (CBT) has provided models and methods to treat a range of problems associated with PCS including depression and anxiety, sleep problems, chronic fatigue and pain. There is, however, no certainty of overall efficacy of CBT in PCS patients.

There is an extensive literature on PCS and its probable causes and very little clear evidence regarding causes or effective therapeutic measures. It is clear that a large majority of those who sustain MTBI either do not experience PCS or experience it for only a short period of time, not exceeding 3 months. There has been a great deal of disagreement about whether PCS is predominantly attributable to organic brain injury or to psychological or psychosocial factors. There is undoubtedly an organic
basis for early postconcussional symptoms and there are frequently psychological or psychosocial factors which contribute to later ongoing symptomatology, thereby prolonging disability. There is no compelling evidence that any attempted therapeutic measures are consistently beneficial in relieving symptoms of PCS. One exception to this generalization is vertigo which may be arising as a result of inner ear dysfunction and may be susceptible to amelioration. An otolaryngologist should be consulted.

A current rational approach to the management of PCS that persists for longer than 3 months would appear to include provision of information, reassurance, management of psychiatric comorbidity, including drug addiction, resolution of secondary gain considerations if possible and, perhaps, a supervised physical exercise program.

Cumulative Effects of Concussion

Football players who have suffered one concussion are more likely to experience a subsequent concussion than are athletes who have never been concussed and are also more likely to experience confusion and amnesia on the field. There is also evidence that players who have experienced multiple concussions take progressively longer to recover. This suggests that some changes in the brain may persist after clinical recovery has occurred and that repeated concussions have cumulative effects on the brain. It is possible that additional factors may affect susceptibility to repeated concussion.

Chronic Traumatic Encephalopathy

Chronic traumatic encephalopathy (CTE) is a neurodegenerative disorder that is believed to be caused by repeated MTBI. CTE is manifested clinically by changes in higher mental function, mood (especially depression and apathy) and in personality and behavior (especially poor impulse control and lack of inhibition), and movement (including Parkinsonism and possibly signs of motor neuron disease). Neuropsychological tests reveal impairments in memory, attention and concentration, information processing, fine motor control, sequencing abilities, and executive functions. Associated neuropathological findings are similar to the changes seen in Alzheimer’s disease (AD). MRI changes have also been described, including loss of white matter (the connecting pathways of the brain), medial temporal lobe abnormalities and other features similar to those seen in AD. CTE appears to be rare in the workplace with the exception of its occurrence in professional athletes and military personnel deployed in combat roles. Where applicable in the workplace setting measures to prevent repeat concussion are relevant.
Medical Imaging in TBI

Skull X-rays

Skull X-rays are performed by passage of X-ray beams through the head onto either photosensitive film or a digital imaging plate. The image created depends on attenuation of the X-rays by all of the interposed tissues. The soft tissues - brain, blood vessels, cerebrospinal fluid, etc. cause little attenuation of the beam and do not result in useful imaging by this technique. The image is "negative" i.e. the darkest areas reflect least attenuation of the X-ray beam. Bone, calcifications, intracranial air and fractures of the skull vault are identified. Depressed fracture of the skull vault may injure the underlying dura and brain. Fractures that enter the paranasal sinuses may lead to complications such as, meningitis or brain abscess. Those crossing vascular structures like the middle meningeal artery may be associated with intracranial hematomas.

Skull X-rays need not be performed routinely in MTBI. They may be helpful in moderate and severe TBI, though computed tomography (CT) and magnetic resonance imaging (MRI) are now widely and rapidly available and provide much more information on the skull base and on the brain, blood vessels and blood clots and on other intracranial structures. If CT is not available in the acute setting, skull X-rays can provide information that may be relevant regarding mass lesions, which may cause displacement of calcified structures like the pineal gland or regarding a skull fracture that has an increased risk of being complicated by intracranial hemorrhage.

Computed Tomography (CT)

CT is the single most useful imaging technique for the investigation of acute head injury. A narrow beam of X-rays is projected through the head onto digital detectors. The beam and detectors rotate around the head, describing a circle, and the X-rays are attenuated to varying degrees by the interposed tissues - skull, brain, cerebrospinal fluid, etc. The computer-generated image obtained constitutes a virtual slice of the head. The advent of high-speed multi-slice spiral or helical scanners, which use multiple detectors to image several "slices", in conjunction with a motorized table which moves continuously and rapidly through the CT gantry, has revolutionized the management of severe head injuries and other acute neurological emergencies due to speed and accuracy of diagnosis.

CT utilizes an open gantry which can be tolerated by most patients, including those who are mildly claustrophobic, and is compatible with implanted metal devices (though images may be degraded to some extent by metallic artifact). CT imposes few limitations on the management of acute TBI patients. The presence and extent of intracranial bleeding (extradural, subdural or intracerebral hematoma), cerebral contusion (bruising), displacement of the brain by mass lesions, detection of basal
skull fractures and injury to blood vessels are some of the consequences of trauma which are important and easily detectable by CT.

CT images of the head can be “enhanced” by intravenous injection of radiodense contrast material. This is not often required in acute head injury but may be useful in detecting delayed complications such as brain abscess.

Computed tomography angiography (CTA) utilizes radiodense contrast material to demonstrate the arteries and veins of the brain. It is less invasive and more rapidly available than conventional angiography and may be useful in detecting injury to intracranial blood vessels.

CT of the head, despite its great utility, should not be performed routinely in all head injuries because there is an attendant financial cost and the examination does entail delivery of a moderate amount of radiation, giving rise to theoretical potential long-term risks of tumour induction, particularly in younger patients if multiple studies are required.

Magnetic Resonance Imaging (MRI)

The methodology of MRI is more complex. When body tissues are subjected to a strong magnetic field, free hydrogen nuclei (protons) align themselves with the direction of the field. A radiofrequency (RF) pulse is applied perpendicular to the direction of the magnetic field resulting in tilting of the force of the magnetic field away from the RF source and when the RF is removed, the direction of the magnetic field returns to its original orientation (relaxation). Energy is emitted in the form of RF during relaxation. Protons realign at differing rates in different tissues. This energy is detected and processed to create a three dimensional image of normal brain structures and of tissue abnormalities which vary with the physical makeup of the tissue substance being studied.

Advantages of MRI include absence of bone artifact, better definition of soft tissues and greater sensitivity to many disease processes. Disadvantages include incompatibility with ferrous metal implants or foreign bodies and with implanted medical electronic devices e.g. cardiac pacemakers. While MRI can visualize fine structures like cranial nerves and relatively small blood vessels it has a limited role in acute head trauma at present. As methodology evolves, shorter MRI sequences may increase the role of MRI in acute trauma.

MRI is, however, potentially very useful in studying the later effects of head injury e.g. diffuse axonal injury, chronic traumatic encephalopathy and possibly post-concussion syndrome, as well as delayed complications of head injury such as cerebral abscess.

Newer MRI sequences e.g. diffusion tensor imaging (DTI), functional MRI (fMRI), and magnetic resonance spectroscopy (MRS) are promising and may turn out to
be sensitive objective methods of assessing MTBI and PCS. DTI shows signal abnormalities by detecting restricted diffusion of water through tissues. Changes have been seen in the deep, periventricular white matter of the brain and in the corpus callosum (the white matter pathway which connects the cerebral hemispheres) and other white matter areas in patients with mild traumatic brain injury with normal MRI on routine imaging sequences. These changes may be transient and may only be detectable for a relatively short time after injury but may prove useful in the study of PCS. Changes of either diffuse axonal injury (DAI) or focal axonal injury (FAI) which were previously only consistently detectable at autopsy may be also revealed by this methodology.

Functional MRI (fMRI) studies brain function, as opposed to structure, and depends upon detection of loss of oxygen from hemoglobin in the blood in areas of the brain that are active when a particular cognitive task is performed. Functional MRI may be abnormal in patients with PCS.

MRS detects chemicals present in brain tissue. Quantitative estimates after TBI may indicate focal areas of brain abnormality and may give indications of the nature of the abnormality.

MRI and CT may both be useful in detecting late complications of TBI e.g. hydrocephalus- obstruction to the flow and absorption of cerebrospinal fluid related to bleeding into the subarachnoid space at the time of injury or chronic subdural hematoma.

**Single Photon Emission Computed Tomography (SPECT)**

Single photon emission computed tomography (SPECT) uses injected gamma emitting radiopharmaceuticals detected by rotating gamma cameras to create virtual 2 dimensional slices of the brain. Isotope concentration in areas of brain is proportional to relative blood flow and metabolism. Multiple slices can then be reconstructed to form a 3 dimensional image. The images are not high resolution. Co-registration with CT images in reconstruction improves anatomical localization.

Though abnormalities may be seen following TBI, their significance is uncertain. Co-existing psychiatric abnormalities may be a confounding factor.

Expert medical opinion recommends, with respect to medical evidence, that SPECT findings should be admissible only to support clinical history, neuropsychological test results, and structural brain imaging findings and not as stand-alone diagnostic data.
Traumatic Brain Injury (TBI)

Positron Emission Tomography (PET)

PET provides quantitative measurement of cerebral perfusion and metabolism with excellent spatial resolution using injected radiopharmaceuticals. Its role in TBI and PCS is unproven and the technique is, in general, neither readily available nor applicable in the acute TBI setting. The technique may provide useful research data in PCS.

Current Status of Functional Brain Imaging in TBI

Special MRI sequences and SPECT and PET are not particularly useful in TBI management at the present time. There is no doubt that clinical neurological abnormalities may persist for a time after brief loss of consciousness whether or not imaging abnormalities are identified. Therefore absence of imaging changes does not constitute evidence that concussion has not occurred. Caution is warranted at this time. Until acquisition, analysis and interpretation of newer imaging techniques are standardized, and the error rates of these techniques with respect to the diagnosis of MTBI are established and generally accepted by medical experts and until the significance of short-term signal changes are better understood, these imaging techniques serve only to potentially corroborate other evidence of MTBI. They may ultimately contribute to a better understanding of MTBI and PCS. At present, however, they do not have a defined role in routine patient management in MTBI and PCS and are not diagnostic of head injury without other supporting evidence.

References


