Workplace Safety and Insurance
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Tribunal d'appel de la sécurité professionnelle
et de l'assurance contre les accidents du travail

Traumatic Brain Injury and Concussion

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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 Discussion papers are not peer reviewed.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). For more information about these papers, please consult the *WSIAT Guide to Medical Information and Medical Assessors*.

Introduction

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 head (impact injury). 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 (impulse injury). Impact and impulse injuries often co-exist; for example, the impact injury, as the head strikes the ground, results in acceleration and deceleration (impulsive injury) as the soft brain is pushed and pulled against the unyielding hard surface of the skull and intracranial fibrous septations. Generally speaking, impulsive injury tends to have worse outcome than impact injury because the injury that it causes is diffuse, deeper, and tends to involve more areas that when injured cause symptoms.

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 and/or flying objects, motor vehicle and machinery accidents, etc. Construction, transportation, agriculture, forestry, fishing, education (especially concussion), 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. Helmet, even properly fitted and worn, do not prevent head injuries, but can prevent scalp laceration and skull fracture. Blast overpressure (pressure caused by a shock wave from an explosion that is above normal atmospheric pressure) can result in traumatic brain injury from industrial explosions, military exercises and combat. Associated injuries often include inhalation injury, radiation exposure, chemical/thermal burns and shrapnel injury. Secondary impact and impulsive injuries can occur as the victims fall and hit the ground.

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 (tearing) 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. TBI is a leading cause of disability globally. In Canada, 2% of the population lives with a TBI, and there are 18,000 hospitalizations for TBI each year. One-third of individuals with a TBI are women. Women with TBI are more likely than men to experience mental health problems post-injury. TBI occurs at an annual rate of

500 out of 100,000 individuals. That is approximately 165,000 in Canada. This equals 456 people every day, or one person injured every 3 minutes in Canada.

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 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.

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)**. The utility of the GCS resides with its objectivity, reproducibility, ease of use, universal applicability, predictability of the force to the brain, and ability to prognosticate. For example, a low GCS is most likely associated with a high energy and more serious brain injury. Furthermore, GCS of < 5 on admission is associated with up to 78% mortality. Since GCS 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

Response	Scale	Score
Eye Opening	Eyes open spontaneously	4 Points
	Eyes open to verbal command, speech, or shout	3 Points
	Eyes open to pain (not applied to face)	2 Points
	None	1 Points
Verbal Response	Oriented	5 Points
	Confused conversation, but able to answer questions	4 Points
	Inappropriate responses, words discernable	3 Points
	Incomprehensible sounds or speech	2 Points
	No verbal response	1 Points
Motor Response	Obeys commands for movement	6 Points
	Purposeful movement to painful stimulus	5 Points
	Withdraws from pain	4 Points
	Abnormal (spastic) flexion, decorticate posture	3 Points
	Extensor (rigid) response, decerebrate posture	2 Points
	No motor response	1 Points

Minor Brain Injury = 13-15 points; **Moderate Brain Injury** = 9-12 points;
Severe Brain Injury = 3-8 points

Much of what follows will be devoted to mTBI and post-concussion symptoms. As noted above, workplace events, which result in more severe head injuries, are, in general, easily understood as causes of acute and continuing disability. The complexity of mTBI and its sequalae, even though better understood with recent research, have remained an area of dispute and concern in workers' compensation. A long-recognized paradox is the fact that the number and frequency of post-concussion complaints following minor head injury are disproportionately greater compared with those following more serious head injuries that demonstrate clear-cut pathology, clear-cut neurological findings and abnormal findings on medical imaging.

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. There are about 200,000 concussions in Canada annually. The incidence is 493-653/1,000,000 in Ontario. This discussion will focus on non-sport-related concussion.

Cerebral concussion consists of an **alteration of consciousness** as a result of closed head injury. Blow to the head is often the mechanism, but force transmitted to the brain from a blow to the jaw and body can also result in concussion. Concussion has also been seen with Whiplash Associated Disorder. **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 Table below summarises the key definitions of mTBI and concussion:

Criteria (year)	Definition of Concussion	Injury Description
Ontario Neurotrauma (2018)	Acute neurophysiological event related to blunt impact or other mechanical energy applied to the head, neck, or body (with transmitting forces to the brain), such as from sudden acceleration, deceleration or rotational forces	Loss of consciousness < 30 min; any alteration of consciousness at the time of the injury; Post-traumatic amnesia ≤ 24 h; physical symptoms; normal standard imaging.
1st International conference of concussion in sport (2002)	A complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces.	(i) Direct blow to the head, face, neck or elsewhere on the body with an 'impulsive' force transmitted to the head. (ii) Rapid onset of short-lived impairment of neurological function that resolves spontaneously. (iii) May result in neuropathological changes, but the acute clinical symptoms largely reflect a functional disturbance versus structural injury. (iv) Results in a graded set of clinical syndromes that may or may not involve LOC. Resolution of the clinical and cognitive symptoms typically follows a sequential course. (v) Typically grossly normal structural neuroimaging studies.

Criteria (year)	Definition of Concussion	Injury Description
5th International conference of concussion in sport (2017)	Sports-related concussion is a TBI induced by biomechanical forces	Modifications to the above: (i) In some cases, signs and symptoms evolve over a number of minutes to hours. (ii) No abnormality is seen on standard structural neuroimaging. (iii) Sports-related concussion results in a range of clinical signs and symptoms. In some cases symptoms may be prolonged.

This definition literature is derived from sports injuries with the intent of establishing guidelines for safe return to play. Unfortunately, the definitions 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 post-concussion symptoms. These definitions, 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. Furthermore, these definitions fail to recognize the heterogeneity of the likely physical injury within the broad mTBI category.

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 post-concussive 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.

There is no convincing data to support concussion prevention with helmet, even properly worn and fitted. Helmets can decrease the incidence of scalp laceration and skull fracture.

The outcome of first concussion is generally good. Up to 80% of concussions resolve with no sequalae in 1-4 weeks. A short initial rest (physical and mental) of no more than 48 hours would result in improvement of symptoms. Once symptoms resolve, gradual return to work is encouraged.

Persistent Post-Concussive Symptoms/Postconcussion Syndrome, Postconcussive Syndrome/Postconcussional Disorder

1) Overview and Definition

About 15-30% of patients with concussions report symptoms, that have developed within minutes, hours and days after the concussion, persist after 3 months (Diagnostic and Statistical Manual Edition 5 (DSM-5); 1 month for the 11th edition of the International Classification of Disease (ICD-11)). Diagnosis of concussion is often based on self-reporting of symptoms; therefore, underdiagnosis, misdiagnosis and/ or delayed diagnosis are expected. The physical symptoms such as headache and dizziness usually develop within days of concussion; however, the neuropsychiatric and neuropsychological symptoms might have a delayed onset and/or might not be recognized/ diagnosed past the 3-month mark. 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. Syndrome of persistent post concussive symptoms may result in prolonged disability in workplace injuries. The long-term outlook is poor, with many experiencing ongoing negative impact on work and social functions.

2) Weakness of Terminology and Classification Systems

Symptoms of headache, dizziness and cognitive impairments are not unique or specific for head injury. Up to 75% of healthy population reports similar symptoms. Thus, it is not surprising that 59% of the general population who report having been “concussed” deny ever having had a brain/head injury. The terms post-concussive syndrome and post concussional disorder have been widely used to describe the persistence of these symptoms beyond 3 months following concussion. The lack of symptom specificity became apparent, when only 40% of diagnostic agreement was observed when applying the diagnostic criteria of the DSM-4 and ICD-10 to a large cohort. DSM-5 and ICD-11 have removed the category of post-concussive syndrome altogether and subsumed it under “neurocognitive disorders due to traumatic brain injury” and “mild neurocognitive disorder” (which can be secondary to trauma), respectively. Both reclassifications have been criticized for not reflecting the true nature of persistent post-concussive symptoms. The following Table summarises the current definitions of persistent symptoms post concussion:

Current Definitions of Persistent Post-Concussive Symptoms	Major/mild neurocognitive disorders due to Concussion (DSM-5)	Mild neurocognitive disorder (ICD-11)
Timing	>3 months	>1 month
Trigger	Head injury	Head injury with loss of consciousness or undetermined etiology
Interference with Activities	Severe vs modest interference with ability to be independent in daily living functions	Not severe to significant interference with independence or activities of daily living
Physical Symptoms	Headache, vertigo, sleep disorder, tinnitus, hyperacusis, photosensitivity, anosmia, hemiparesis, seizures, visual disturbance, orthopaedic injury, cranial nerve or neuromotor deficits.	Headache, dizziness, fatigue, insomnia, noise intolerance.
Emotional Symptoms	Irritability, reduced tolerance to psychotropic medication, loss of emotional control (e.g. aggression), inappropriate affect, apathy, anxiety, depressed mood, altered personality and/or social cognition	Irritability, reduced alcohol tolerance, depression, anxiety, emotional lability, preoccupation with symptoms
Cognitive Symptoms	Difficulty concentrating, learning and memory, executive function, slowed processing speed, reduced cognitive efficiency, decline in language, neglect, constructional dyspraxia.	Subjective decline in concentration, memory, or intellectual difficulties.

These definitions fail to recognize the heterogeneity of the likely physical injury within the broad mTBI category, and also, crucially, the other disorders can cause persistent symptoms after mTBI (e.g. PTSD, major depressive disorder, generalized anxiety disorder, migraine, functional neurological disorder) (see later discussion).

3) Common Persistent Post-Concussive Symptoms

Headache and dizziness are two of the commonest. Other common symptoms include light headedness 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.

4) Post Concussive Headache vs Pre-existing Headache Disorder

Post traumatic headache most commonly represents a migraine-type headache with associated migraine symptoms, including nausea, light and noise sensitivity, irritability and cognitive symptoms-symptoms that are also listed in persistent post-concussive symptoms. A pre-existing or family history of migraine are risk factors for persistent post-concussive headache. Even though the pathophysiology of post traumatic headache and primary migraine may overlap, treating all the same is bound to fail. Persistent psychological factors and medication overuse can prolong post traumatic headache. But early treatment of post traumatic headache is definitely warranted.

5) Post Concussive Dizziness

The commonest causes following concussion are benign paroxysmal positional vertigo (40%) and vestibular migraine (34%). Vestibular migraine is associated with other migrainous symptoms as discussed above; benign paroxysmal positional vertigo is also associated with cognitive impairments and heightened anxiety, especially if left untreated.

6) Seizure/Epilepsy and TBI

Patients with pre-morbid seizure disorder/epilepsy tend to have more persistent post-concussive symptoms, not just related to increased seizure frequency and difficulty with seizure control alone. The symptom persistence is most likely related to the susceptibility of the already structurally altered and inflamed epileptic brain to traumatic brain injury.

Early post traumatic seizures (PTS) occur within 7 days of injury; whereas, late PTS after 7 days. Post traumatic epilepsy (PTE) represents recurrent seizures greater than 7 days from injury. Early PTS and PTE are usually associated with more severe TBI with hemorrhages and laceration of brain, thus indicators for more severe injury and worst outcome. Anti-seizure prophylaxis does not decrease PTE, morbidity, mortality, and/or neurological deficits.

7) Predisposing Factors for Persistent Post Concussive Symptoms

Repeated concussions with or without complete healing increase the severity, duration and persistence of symptoms.

Pre-injury depressive or anxiety disorders are the strongest predictors of persistent symptoms. Other factors include pre-injury life events, social circumstances, personality traits (neuroticism and memory perfectionism), illness expectations and beliefs. Pre-existing anxiety and anxiety sensitivity are associated with more severe and prolonged symptom reporting, potentially related to negative illness beliefs. Overall, the results of outcome studies indicate that patients with persistent post concussion symptoms experienced higher levels of pre-injury depression, anxiety, disorders and post traumatic 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. Presence of persisting symptoms beyond 6 months post injury predicts a probability of poorer final outcome after mTBI.

Pre-injury neurodegeneration or even healthy ageing affect the outcome of the injury regardless of its severity. Neurodevelopmental disorders are also factors. Premorbid psychiatric illnesses including attention deficit hyperactivity disorder are seen in a higher proportion of those with mTBI than would otherwise be expected. This may relate to impulse control behaviours, including alcohol and substance misuse, which can predispose an individual to sustaining a TBI. Thus, the pre-exiting neurological condition on which the injury occurs interacts with the effect of the injury itself.

8) Persistent Post Concussive Symptoms and Psychiatric Disorders

Concussion increases the risk of developing a subsequent psychiatric condition nearly threefold. Women are more likely than men to experience mental health disorders after concussions.

However, trying to distinguish psychiatric conditions such as PTSD, generalised anxiety disorder, major depressive disorder, functional neurological disorder and somatic symptom disorder from persistent post concussive symptoms can be challenging due to significant symptom overlap. This overlap can obscure diagnostic clarity since 50% of depressed patients who have not had a concussion met the diagnostic criteria for moderate to severe post-concussive syndrome.

The following table summarises the differential diagnoses from DSM-5 or mimickers for persistent symptoms after concussion:

Differential Diagnoses or Mimickers	PTSD	Generalised anxiety disorder	Major depressive disorder	Functional neurological disorder	Somatic symptom disorder
Timing	1 month of persistent symptoms with delayed expression: Full symptom expression >6 months after event.	Persistent symptoms for more than 50% of the time for >6 months.	>2 weeks duration of new or clearly worsened symptoms, can be discrete episodes.	Acute: <6 months duration (may have similar previous episodes). Persistent: >6 months.	Any one symptom may not be persistent but state of being symptomatic >6 months.
Trigger	Actual/ threatened harm including head injury.	–	Can be traumatic/ stressful event, often on a background of adverse childhood experiences.	Onset may be preceded by injury (physical/ psychological)	Can be precipitated by stressful life events
General	Impaired social, occupational and other aspects of functioning.	Impaired social, occupational and other aspects of functioning	Impaired social, occupational and other aspects of functioning.	Impaired social, occupational and other aspects of functioning	Symptoms are distressing or result in significant disruption of daily life
Physical symptoms	Disturbed sleep (e.g. recurrent distressing dreams related to trauma), change in arousal, hypervigilance for potential threats, episodic physical symptoms can act as trigger for PTSD symptoms.	Disturbed sleep, fatigue, exaggerated startle, muscle tension or soreness, change in arousal including panic attacks, somatic symptoms e.g. sweating, diarrhoea	Sleep disturbance, fatigue, weight change, loss of libido, general heaviness of limbs, somatic symptoms especially pain, psychomotor agitation or retardation.	Disturbance of any neurological system, internal inconsistency on examination	May represent normal bodily sensations/ discomfort, may be specific (e.g. localized pain) or generalized (e.g. fatigue)

Differential Diagnoses or Mimickers	PTSD	Generalised anxiety disorder	Major depressive disorder	Functional neurological disorder	Somatic symptom disorder
Emotional symptoms	Irritability, outbursts, recklessness, flashbacks, intense/ prolonged distress related to cues with/without avoidance, persistent negative emotional state.	Irritability, anxiety/fear not related to traumatic event or specific triggers, overestimate dangers/ future threat with avoidance, worry about multiple events, situations or activities.	Irritation, outbursts, excessive guilt/ worthlessness, low/dysphoric mood or anhedonia, diminished interest, suicidal thoughts, anxiety, phobias, excessive worry.	Can be associated with dissociative symptoms at onset or during attacks, distress associated with loss of function	Persistently high levels of anxiety related to symptom and/or family history of disease, appraise bodily symptoms as threatening
Cognitive symptoms	Difficulty concentrating, inability to remember important aspects of event (not due to head injury), recurrent distressing memories (consider obsessive compulsive disorder criteria for obsession if unrelated to trauma)	Difficulty concentrating owing to worrisome thoughts, mind going blank.	Difficulty concentrating, thinking, distractibility, indecisiveness, obsessive rumination (compare PTSD where related to a specific event).	Absent from definition	Excessive thoughts related to somatic symptom (thoughts are less intrusive than obsessive compulsive disorder).

Functional neurological disorder is defined by complaint of abnormal function that can be demonstrated clinically and by investigations to be normal. Over 80% of patients with functional neurological disorder report a health event near the onset of functional symptoms. Therefore, it is highly possible that the immediate mTBI, often minor in nature, self-resolving and normally expected not to produce persistent symptoms, and the lasting physical and/or psychological consequences of the index accident could undoubtedly trigger the onset of functional neurological disorder and/or somatic symptom disorder.

There has been ongoing debate/disagreement about whether symptom persistency is predominantly attributable to organic brain injury (structural and/or biochemical) or to psychological or psychosocial factors. There is undoubtedly an organic basis for persistent symptoms and there are frequently psychological or psychosocial factors which contribute to later ongoing symptomatology, thereby prolonging disability.

Post-mortem studies of patients with a history of mTBI, but who died of other reasons, have found evidence of some white matter injury and persistent inflammation months after the injury. Secondary injury including psychiatric illnesses could therefore develop in minutes, hours or months, with possible long-term effects on symptoms and functions. It remains a scientific challenge to prove the physical head injury and the subsequent structural damage actually **cause** these psychiatric conditions, but not related to some undefined structural, genetic, biochemical, social and environmental factors that result in the development of these conditions independently and/or after head injury and/or after any body stresses.

9) Impact of Medicolegal and Workers' Compensation Issues

Medicolegal and workers' compensation processes appear to correlate with symptom persistence. This finding is often interpreted as evidence for psychological substrate as the cause for symptom development and symptom persistence. Medicolegal processes are adversarial in nature. There is often a party at fault, negligence and/or liability. Often the injured were not believed and/or the injury not acknowledged by the other party and/or the severity was minimised and/or the injured were even considered to be malingering, as the injured were subjected to repeated inquiries and assessments. These effects result in an increased likelihood of feigning behaviour as a behavioural response to express anger and revenge for trust-violation. Surprisingly, the financial implications are not necessarily the motivator. Resolution of ongoing legal proceedings, if possible, may improve symptomatology.

10) Role of Neuropsychological Testing after Concussion

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 post-concussive symptoms is frequently difficult to document.

Neuropsychological (NP) tests are an objective measure of brain-behaviour relationships and are more sensitive for subtle cognitive impairment than clinical exam. Most concussions can be managed appropriately without the use of NP testing. Computerised neuropsychological testing should be interpreted by healthcare professionals trained and familiar with the type of test and the individual test limitations, including a knowledgeable assessment of the reliable change index, baseline

variability and false-positive and false-negative rates. Paper and pencil NP tests can be more comprehensive, test different domains and assess for other conditions which may masquerade as or complicate assessment of concussion. NP testing should be used only as part of a comprehensive concussion management strategy and should not be used in isolation. The ideal timing, frequency and type of NP testing have not been determined. In some cases, properly administered and interpreted NP testing provides an added value to assess cognitive function decline and/or recovery in the management of sports concussions.

Concussion remains a clinical diagnosis ideally made by a healthcare provider knowledgeable in the recognition and evaluation of concussion. Therefore, NP assessment provides a helpful structure for the evaluation of concussion, although limited validation of this assessment tool is available.

11) Treatment of Persistent Post-Concussive Symptoms

There is no compelling evidence that any attempted therapeutic measures are consistently beneficial in relieving persistent symptoms. One exception to this generalization is benign paroxysmal positional vertigo which can be successfully treated by an otolaryngologist.

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

In general, athletes recover from persistent 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 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 persistent symptoms including depression and anxiety, sleep problems, chronic fatigue and pain. There is, however, no certainty of overall efficacy of CBT in these patients.

Special prescription glasses with binasal occlusion have helped with reading proficiency and visual-spatial perception; weighted compression body vest might be useful in re-training balance exercises.

Treatment with sex hormones has been attempted in small-scale trials, given the observation that mTBI symptoms might be affected by different phases of the menstrual cycle. But the effectiveness has not been proven. This is consistent with the lack of efficacy of progesterone in the treatment of severe TBI.

Involvement in a medicolegal process and workers' compensation 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 emphasizing issues of blame, liability and responsibility. Resolution of ongoing legal proceedings, if possible, may improve symptomatology.

A rational approach to the management of persistent post-concussive symptoms would appear to include provision of information, reassurance, management of psychosocial and psychiatric comorbidity, resolution of secondary gain considerations if possible, occupational therapeutic measures for symptom control and, perhaps, a supervised physical exercise program.

Long-term Sequalae of Concussion

1) Cumulative Effects of Concussion

Repeated concussions with or without complete healing increase the severity, duration and persistence of symptoms. Research from sport-related concussions showed 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. Furthermore, the force required for subsequent concussion is less. Moreover, multiple repeated sub-concussive hits (Injuries that normally would not have caused a concussion) would result in concussion symptoms in these athletes.

2) Chronic Traumatic Encephalopathy

Chronic traumatic encephalopathy (CTE) is a neurodegenerative disorder that is believed to be caused by repeated mTBI. CTE is a neuropathological diagnosis that can only be made after the deceased's brain has been examined. Often symptoms are retrospectively attributed to CTE after autopsy examination. Generally speaking, clinical CTE symptoms overlap those of several neurodegenerative disorders at the same time. These symptoms often became apparent years or decades after concussion events, which were often not reported, under-reported or misreported.

Clinical features of CTE may be manifested 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). CTE has been implicated in suicidality

and homicidality among some high-profile sport celebrities with history of multiple concussions. The Parkinsonism features are different from Primary Parkinson's Disease, since they tend not to respond to the usual anti-Parkinson's medications.

Lou Gehrig, the name sake for amyotrophic lateral sclerosis (ALS), reportedly had multiple concussions in his sports career. It is not clear whether he had ALS or CTE with signs of motor neuron disease. There is also no direct causation link between CTE and motor neuron disease.

Neuropsychological tests in CTE reveal impairments in memory, attention and concentration, information processing, fine motor control, sequencing abilities, and executive functions. Associated neuropathological findings showed strikingly similar changes to those found in Primary 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. There is, however, no direct causation link between CTE and Primary AD, even though they have clinical and neuropathological overlap.

CTE in workers' compensation is rare, since the dispute is often related to the shorter-term sequalae of concussions. However, awareness of this condition is relevant in the prevention of repeated concussions.

Medical Imaging in TBI and Concussion

1) 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.

2) Computed Tomography (CT)

CT is the single most useful imaging technique for the investigation of acute head injury. CT scan is readily available in most health care facilities, whether community or hospital-based. 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 a fairly wide 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.

3) 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 symptom persistence.

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 symptom persistence. However, DTI findings may not be specific for symptom persistence after mTBI, since similar signals were also detected in co-morbid conditions, such as migraine, depression, and post traumatic stress disorder. 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 symptom persistence. However, fMRI findings may not be specific for symptom persistence, since similar signals were also detected in co-morbid conditions, such as migraine, depression, post traumatic stress disorder and functional neurological disorder, irrespective of the presence of mTBI.

Magnetic resonance spectroscopy (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.

4) 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. Coexisting 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.

5) 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 concussion 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 concussion.

6) 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 is 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 symptom persistence. At present, however, they do not have a defined role in routine patient management in mTBI and symptom persistence and are not diagnostic of head injury without other supporting evidence.

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