Introduction to traumatic brain injury

A traumatic brain injury is often the result of one moment of mischance, but it may have long-lasting and far-reaching repercussions.

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A traumatic brain injury (TBI) is a ruthless leveller of the playing field, as is true of any neurological insult to the supremely powerful but paradoxically fragile organ that controls all our bodily functions and houses the intangible essence that makes each individual unique. Such injury can reduce the most erudite university professor to the ignominy of groping desperately for his own name, or the world's fastest sprinter to the level of a child learning to take his/her first steps once again. Some patients will walk away from a mild head injury with little concept of how much worse the outcome may have been. Others, more severely injured, may make what is termed a good recovery but be left with residual deficits that will leave a lasting imprint on their lives. Many sufferers will remain severely incapacitated and a lamentably large number will become part of the statistics on mortality after TBI.

This article discusses the aetiology, mechanisms of injury and effects of TBI. It also discusses some widely used tools for the assessment of patients with TBIs, and precedes an article on the rehabilitation of such individuals (p. 84 of this issue).

What is traumatic brain injury?

TBI, defined as brain damage caused by externally inflicted trauma to the head, may result in significant impairment of an individual's functioning – on physical, cognitive and psychosocial levels. Statistics on the incidence of TBI vary widely, but it is evident that the figures have increased steadily over recent years and that TBI is a leading cause of death and disability worldwide, particularly in young children and adults under the age of 40. Men are much more likely to incur TBIs, with the age group 15 - 30 years at highest risk.[1,2] Although trauma is a leading cause of brain injury as well as spinal cord injury, brain injury as a result of trauma is much more likely to occur than spinal cord injury.

The major causes of brain injury are motor vehicle accidents, violence (including gunshot wounds and assaults), falls, being hit by falling debris, and pedestrian vehicle accidents.[1] However, as the incidence of TBI has increased, so has the number of people surviving it, owing to faster and more effective emergency care, quicker and safer transportation to specialised treatment facilities, and advances in acute medical management. Because of the often irreversible and debilitating loss of function incurred as a result of severe brain injury, the increase in survival rates challenges medical and rehabilitation teams to treat and rehabilitate patients to the highest potential.

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Mechanisms of injury

A TBI is usually caused by a dynamic loading or impact to the head from direct blows or from sudden movements resulting from impacts to other body parts. The loading can result in any combination of compression, expansion, acceleration, deceleration and rotation of the brain inside the skull. Brain injuries may be diffuse, focal, or both. Motor vehicle accidents and falls involve acceleration and deceleration with rotation of the brain inside the skull. The brainstem is more stable than the cerebrum, which rotates around the brainstem during impact. The rotation places a stretch or shear force on the long axons that transmit information throughout the brain and brainstem. Such injuries are called diffuse axonal injuries (DAIs) and may result in coma due to damage to the axons in the midbrain reticular activating system.[3] The changes in the brain as a result of DAI are microscopic and may not visualise on computed tomography (CT) or magnetic resonance imaging (MRI) scans.

Focal lesions involve contusions or lacerations of the brain, resulting in extra-axial (within the skull) or intra-axial (within the brain) haemorrhage.[2] Although focal lesions can occur anywhere beneath the impact, they are usually seen at the anterior poles and inferior surfaces of the frontal and temporal lobes. They occur when the brain hits against the skull and scrapes over the irregular bony structures at these locations. The occipital and parietal lobes, which have smooth surfaces, are less likely to incur damage. Lesions of the brain and damage to the internal tissue and blood vessels can also be due to a mechanism called coup-countercoup. Damage directly related to trauma at the site of impact is called a

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coup lesion. As the brain is jolted away from the site of the original impact, it can hit the skull on the opposite side and cause internal damage referred to as a countercoup lesion. Cranial nerves can also be torn, stretched or contused as a consequence of TBI, resulting in particular clinical presentations. The skull – the first line of defence – is particularly vulnerable to injury. The skull may fracture as a result of the force of the impact in the area of, or at a distance from, the actual impact site. The type of fracture depends on the force of the blow.[3]

The injuries described above are termed primary injuries, as they are directly related to the trauma. However, secondary injuries may also occur owing to damage resulting from neuronal death because of hypoxia, oedema and/or initiation of inflammatory cascades.[4] Secondary injury is a significant cause of deterioration of TBI patients after the initial injury and contributes significantly to overall morbidity and mortality. It is therefore vitally important to minimise these secondary effects in the medical and rehabilitative management of these patients.

Signs and symptoms

Some symptoms of TBI are evident immediately, while others may not appear until days or even weeks after the injury. Symptoms of mild TBI include:[5]

- brief loss of consciousness
- headache
- mental confusion
- light-headedness
- dizziness
- blurred or double vision
- fatigue or lethargy
- bad taste in the mouth
- a change in sleep patterns
- behavioural or mood changes
- memory, concentration or calculation problems.

With a moderate or severe TBI, the patient may demonstrate these same symptoms, but may also have:

- prolonged loss of consciousness
- personality changes
- a severe, persistent or worsening headache
- repeated vomiting or nausea
- seizures
- inability to waken
- dilatation of one or both pupils
- slurred speech
- weakness, numbness or paralysis of extremities
- loss of co-ordination
- increased confusion, restlessness or agitation.

Effects of TBI

Some brain injuries are mild, with symptoms disappearing over time. Others may result in a debilitating loss of function and permanent disability. Effects of TBI are extensive and are summarised in Table I.

Effects on consciousness

A brain-injured person may exhibit any of a number of states of altered consciousness, depending on the severity of the injury. These states of arousal are best viewed on a continuum from complete consciousness to coma and brain death.

Brain death

Brain death is the lack of measurable brain function due to diffuse damage to the cerebral hemispheres and the brainstem, with loss of any integrated activity among distinct areas of the brain. It is irreversible and the removal of external support will result in immediate cardiac arrest and cessation of breathing.

Coma

This term is used to refer to patients who are completely unconscious, whose eyes are permanently closed, who cannot be aroused by any sensory stimulation and who demonstrate an absence of any observable interaction with the environment. A coma results from the interruption of communication from the reticular activating system to the cerebral hemispheres.[6]

Stupor

Stupor is a state in which the patient is unresponsive, but can be aroused briefly by a strong stimulus, such as a sharp pain.

Vegetative state

Patients in a vegetative state are unconscious and unaware of their surroundings, but continue to have a sleep-wake cycle and can have periods of wakefulness. At times, when they seem to be awake, their eyes open and sometimes wander. They can make a variety of spontaneous movements such as swallowing, grinding their teeth, crying, smiling and grasping someone's hand, but these motions are always reflexive and not the result of purposeful behaviour. It is possible for a person to function in a vegetative state but without consciousness; the deeper brainstem structures that regulate breathing, reflexes and heart rate are intact, but the cortex is completely impaired. After being in a vegetative state for more than a month after having sustained a brain injury, a patient is said to be in a persistent vegetative state (PVS) and the probability of recovery diminishes with time. In 1994 the Multi-Society Task Force on PVS (a team of 11 researchers from various institutions) concluded that the chances of recovery are close to zero if a patient remains in a vegetative state 1 year after a TBI or 3 months (later revised to 6 months) after brain damage from lack of oxygen or other causes. These patients were referred to as being in a 'permanent vegetative state'.[7]

Minimally conscious state

In the minimally conscious state a patient exhibits deliberate, or cognitively mediated, behaviour consistently enough to be distinguished from the entirely unconscious, reflexive responses that are seen in the PVS. An important difference from the vegetative state is that patients who have remained in the minimally conscious state for years still have a chance of recovery. Making the distinction between the vegetative and minimally conscious states can be difficult and requires repeated examinations by well-trained, experienced clinicians.[8] Conscious awareness is a subjective experience inherently difficult to measure, which highlights the importance of developing effective, objective ways to determine whether a patient is in a PVS. It also raises a number of complex legal and ethical considerations with regard to the long-term management of such patients.

Locked-in syndrome

This is a rare condition in which a patient is conscious and aware, but is unable to move owing to complete paralysis of the body's voluntary muscles. Generally, vertical eye movement and blinking are preserved, this ability usually being the only means of communication with the surrounding world. If this condition is not adequately understood and diagnosed, the patient could be thought to be in lower level of consciousness than he/she actually is.

After brain injury that leads to coma, a patient's progress may follow one of several

Table I. Effects of TBI

Motor deficits

- Weakness/paralysis
- Changes in muscle tone
- Diminished balance
- Decreased endurance
- Inability to plan motor movements
- Delays in initiation
- Tremors
- Dysphagia/swallowing problems
- Poor co-ordination

Cognitive deficits

- Changes in consciousness
- Confusion
- Disorientation
- Shortened attention span
- Memory problems
- Problem-solving deficits
- Poor executive skills
- Inability to understand abstract concepts
- Decreased awareness of self and others
- Command-following difficulties
- Difficulties filtering extraneous information
- Poor stimulus resistance

Functional deficits

- Impaired ability to perform activities of daily living
- Problems with managing finances, domestic work, child care, shopping, etc.
- Vocational problems
- Inability to drive a car or operate machinery

Perceptual/sensory deficits

- Visual deficits – blindness, diplopia, hemianopia, lack of visual acuity, visual-field deficits
- Visual- and spatial-perceptual deficits
- Loss of/heightened sensation
- Left- or right-sided neglect
- Proprioceptive deficits
- Auditory problems
- Olfactory problems

Communication/language deficits

- Aphasia
- Apraxia
- Word-finding problems
- Dysarthria
- Anomia
- Problems with reading, writing, arithmetic

Social deficits

- Poor sociolinguistic sensitivity
- Inappropriate behaviour/conversation
- Poor social interaction
- Limited awareness of self and others

Personality or psychiatric changes

- Apathy
- Irritability
- Impulsivity
- Anxiety and depression
- Emotional lability
- Poor volition and motivation
- Disinhibition – mood swings, poor frustration tolerance, aggression, inappropriate sexual behaviour

Regulatory disturbances

- Fatigue
- Changes in sleep patterns
- Changes in eating habits/appetite
- Dizziness
- Headache
- Loss of bladder and bowel control

Traumatic epilepsy

Epilepsy occurs in 2 - 5 % of all people who sustain brain injury. While most seizures occur immediately after the injury, or within the first year, it is also possible for epilepsy to surface years later

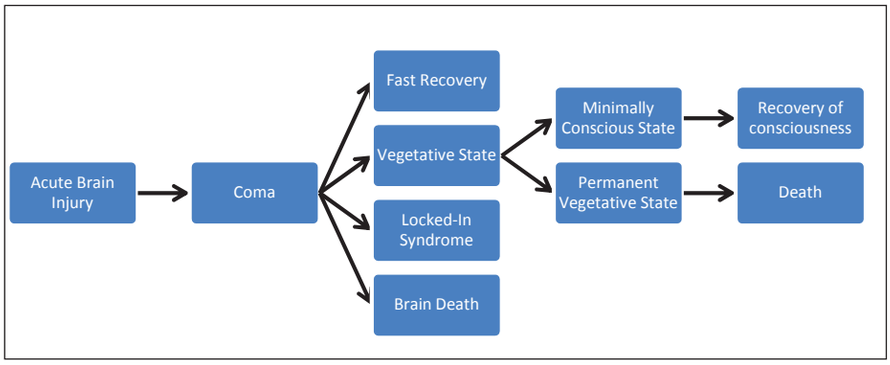

Fig. 1. Pathways of consciousness. (Modified from Laureys S. Eyes open, brain shut. Sci Am *2007; 296(5): 68 - 69.[9])*

paths. If the patient does not die or recover quickly, he/she will most likely make a transition to the vegetative state. The patient may then evolve to the minimally conscious state and often to further recovery of consciousness or remain in the vegetative state permanently.[9]

Assessment tools and outcome measures

As survival rates after TBI increase, so the objective measurement of functional impairment becomes more and more vital, both in predicting outcome and in justifying

Table II. Glasgow Coma Scale

Eye-opening response	Opens eyes spontaneously	4 points
	Opens eyes in response to voice	3 points
	Opens eyes in response to painful stimuli	2 points
	Does not open eyes	1 point
Verbal response	Orientated, converses normally	5 points
	Confused, disorientated conversation, but able to answer basic questions	4 points
	Inappropriate responses, words discernible	3 points
	Incomprehensible speech	2 points
	Makes no sounds	1 point
Motor response	Obeys commands for movement	6 points
	Purposeful movement to painful stimulus	5 points
	Withdraws from pain	4 points
	Abnormal (spastic) flexor response to painful stimuli, decorticate posture	3 points
	Extensor response to painful stimuli, decerebrate posture	2 points
	Makes no movements	1 point

Table III. Rancho Los Amigos Cognitive Scale

Level	Outcome
I	No response to pain, touch or sight
II	Generalised reflex response, patient reacts inconsistently and non-purposefully to stimuli in a nonspecific manner
III	Localised response. Blinks to strong light, turns toward/away from sound, responds to physical discomfort, inconsistent response to simple commands
IV	Confused/agitated. Alert but confused and disorientated. Frequently very active, aggressive or bizarre behaviour. Performs automatic motor activities (if not physically impaired), but behaviour is non-purposeful. Extremely short attention span. Unable to perform self-care
V	Confused/non-agitated. Gross attention to environment, highly distractible, requires continual redirection, difficulty learning new tasks, agitated by too much stimulation. May engage in social conversation but with inappropriate verbalisations. Can manage self-care with assistance
VI	Confused/appropriate. Inconsistent orientation to time and place. Begins to recall past, but retention span/recent memory impaired and interferes with new learning. Consistently follows simple directions, demonstrates goal-directed behaviour with assistance. Begins to be aware of self and others
VII	Automatic/appropriate. Performs daily routine in highly familiar environment in a non-confused but automatic manner. Skills noticeably deteriorate in unfamiliar environments. Lacks insight into own condition. Requires at least minimal supervision because judgement, problem solving and planning skills are impaired
VIII	Purposeful/appropriate. Alert and orientated, able to recall and integrate recent and past events. Can learn new activities and continue in home and living skills, although deficits in judgement, abstract reasoning, social, emotional and intellectual capacities may persist.

(Modified from: León-Carrión J. Methods and tools for the assessment of outcome after brain injury rehabilitation. In León-Carrión J, von Wild KRH, Zitnay GA, eds. *Brain Injury Treatment – Theories and Practice.* East Sussex: Taylor & Francis, 2006.[10])

and objectively evaluating the need for rehabilitation services after injury. The following are three widely used instruments for the assessment of patients with brain injury.

Glasgow Coma Scale

The Glasgow Coma Scale (GCS) was created as a standardised clinical scale to facilitate the reliable interobserver neurological assessments of head-injured patients who are in a coma. It is now used as a simple neurological scale in order to record the conscious state of a person. The value of the scale lies largely in using serial GCS scores as a predictor of outcome. A low GCS score that remains low, or a high GCS score that decreases, predicts poorer outcomes than high GCS scores that remain high, or a low GCS score that progressively improves.[4]

The GCS provides a score in the range of 3 - 15; patients with scores of 3 - 8 are usually said to be in a coma. The total score is the sum of the scores in 3 categories, as shown in Table II.

Rancho Los Amigos Scale

This scale was designed as an easy, useful tool to classify the cognitive functioning of patients after TBI. It is widely used in the acute phase and is currently used as an outcome measure during acute rehabilitation and on discharge. The various levels describe the level of cognitive functioning as patients emerge from coma and are often used for treatment planning. The Rancho Los Amigos Scale has 8 levels to which a patient can be assigned (Table III).

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Functional independence and functional assessment measures

The Functional Independence Measure (FIM) was developed as a tool to evaluate the functional status and rehabilitation outcomes in a variety of impairments, including neurologically impaired patients. The FIM is composed of 18 items with a 7-point rating scale (a score of 1 indicates complete dependence; a score of 7 indicates complete independence). The FIM evaluates self-care, sphincter control, mobility, communication, psychosocial adjustment and cognitive function. However, the FIM measures mainly motor and self-care tasks, which are the focus of early acute rehabilitation, while cognitive and social deficits, which form the focus of later acute and post-discharge rehabilitation, are underrepresented. As a result, the Functional Assessment Measure (FAM) was developed, which contributes

Table IV. FIM/FAM scale

7 Complete independence (timely, safely)
6 Modified independence (with devices, extra time)
5 Supervision (cueing, prompting)
4 Minimal assistance (performs 75% or more of the task)
3 Moderate assistance (performs 50 - 74% of the task)
2 Maximal assistance (performs 25 - 49% of the task)
1 Total assistance (performs less than 25% of the task, or requires assistance of 2 helpers)

Self-care
- Eating
- Grooming
- Bathing
- Dressing upper body
- Dressing lower body
- Toileting
- Swallowing

Sphincter control
- Bladder management
- Bowel management

Mobility
- Bed transfer
- Toilet transfer
- Bath transfer
- Car transfer

Locomotion
- Walking/wheelchair
- Stairs
- Community access

Communication
- Comprehension
- Expression
- Reading
- Writing
- Speech intelligibility

Psychosocial adjustment
- Social interaction
- Emotional status
- Adjustment to limitations
- Employability

Cognitive function
- Problem solving
- Memory
- Orientation
- Attention
- Safety judgement

an additional 12 new items to the original FIM scale. The new items evaluate more complex cognitive, behavioural, emotional, communicative and psychosocial aspects of neurological impairment.[10] Table IV lists the 30 FAM items as well as the 7-point rating scale.

Conclusion

It is evident that a TBI can result in a myriad of physical, functional, cognitive and psychosocial deficits in a person who previously functioned normally. Significant morbidity and mortality rates are associated with moderate and severe injuries. Mild injuries produce far less morbidity, but are still a serious problem because of their extremely high prevalence. While many patients will demonstrate some form of recovery, even after severe brain injury, the nature and extent of the recovery depend on a multitude of intrinsic and extrinsic factors. Predicting the outcome after a TBI is challenging and, as survival rates increase steadily, it is becoming increasingly important to evaluate the effectiveness of treatment and justify the cost of further rehabilitative services.

As the long-term effects of TBI are evaluated and the significant economic and social consequences are understood (particularly with the high incidence of TBI in childhood and young adulthood), the efficacious rehabilitation of patients after TBI is of vital importance.

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In a nutshell

- TBIs are a leading cause of death and disability worldwide.
- The incidence of TBI is highest in children and young adults.
- TBI can result in significant impairment of an individual's physical, cognitive and psychosocial functioning.
- The leading causes of TBI are motor vehicle accidents, violence, falls, being hit by falling debris and pedestrian vehicle accidents.
- Fast, effective medical care is required after a TBI in order to prevent secondary brain injuries and long-term complications.
- Signs and symptoms of TBI may only appear days or even weeks after the initial injury.
- Accurate and objective assessment of a patient's functioning after a TBI is essential in predicting outcome and justifying the need for rehabilitation after injury.