ERABI EVIDENCE-BASED REVIEW of moderate to severe ACQUIRED BRAIN INJURY

Clinical Guidebook

1. Introduction to Moderate to Severe Acquired Brain Injury

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Introduction to Moderate to Severe Acquired Brain Injury

1.1 Evidence-Based Practice

The ERABI Clinical Guidebook has been developed through extensive collaboration with clinical experts, researchers, the Ontario Neurotrauma Foundation, St. Joseph's Healthcare London, the Lawson Health Research Institute, Western University, and our partners in Toronto and Ottawa.

The Evidence-Based Review of Acquired Brain Injury (ERABI) Clinical Guidebook is designed to include clinical information relevant to the identification of ABI sequelae, diagnosis of ABI deficits, outcome measures appropriate for diagnosis of specific deficits, and valuable evidence-based interventions for ABI rehabilitation. In addition to this framework, we have also included supplementary materials to support education in ABI rehabilitation such as clinical algorithms, key studies from the literature, quiz questions, and case studies to consolidate your knowledge. There are 14 Chapters contained in the ERABI Clinical Guidebook, focused around ABI specific deficits. Although we recognize research from other but similar disease states, such as stroke rehabilitation, may be relevant, we have restricted our content to ABI specific populations in order to maximize applications for ABI rehabilitation.

The ERABI Clinical Guidebook has been developed to provide a summary document of best evidence, and hopefully serve to help minimize the gaps that exist in evidence-based ABI rehabilitation clinical care. Some articles cite that the time lag from research to practice in healthcare may be as long as 17 years (Morris, Wooding & Grant, 2011; Hanney et al., 2015; Munro & Savel, 2016). Canada has recognized the need for high impact tools to support evidence-based care as stated in the Federal Mandate to the Minister of Health in 2017. This project also directly supports access to care, increased efficiency, and patient outcomes through technology, knowledge translation, and open access material.

Click HERE to access the INESSS-ONF Clinical Practice Guideline for the Rehabilitation of adults with Moderate to Severe TBI

1.2 What is an ABI?

For the purposes of this evidence-based review, we used the definition of ABI employed by the Toronto Acquired Brain Injury Network (2005). ABI is defined as damage to the brain that occurs after birth and is not related to congenital disorders, developmental disabilities, or processes that progressively damage the brain. ABI is an umbrella term that encompasses traumatic and non-traumatic etiologies. ABI typically involves a wide range of impairments affecting physical, neurocognitive and/or psychological functioning. A person with an 'ABI' might therefore refer to an individual with a traumatic brain injury (TBI) of any severity, or a non-traumatic brain injury such as a person with Herpes encephalitis, viral meningitis or acute hypertensive encephalopathy. As opposed to an insidious developmental process, an 'ABI' infers that a person, previously intact from a neurological perspective, subsequently 'acquired' some form of brain pathology during their lifespan. Common traumatic causes include motor vehicle accidents, falls, assaults, gunshot wounds, and sport injuries (Greenwald, Burnett, & Miller, 2003). Non-traumatic causes of ABI include focal brain lesions, anoxia, tumours, aneurysm, vascular malformations, and infections of the brain (Toronto Acquired Brain Injury Network, 2005).

For the purposes of this Clinical Guidebook vascular causes of focal ABIs, such as stroke, will be excluded from our evidence synthesis. Included and excluded aetiologies of ABI for the purposes of this Guidebook can be found in Table 1.1.

Click HERE to access the complete methodology for ERABI

Table 1.1 Definitions of ABI used in the ERABI Clinical Guidebook.

Included in ABI definition	Excluded from ABI definition
Traumatic Causes • Motor vehicle accidents • Falls • Assaults • Gunshot wounds • Sport Injuries **Non-traumatic Causes** • Tumours (benign/meningioma only) • Anoxia • Subarachnoid hemorrhage (non-focal) • Meningitis • Encephalitis/encephalopathy (viral, bacterial, drug, hepatic) • Subdural Hematoma	**Vascular and Pathological Incidents** • Intracerebral hemorrhage (focal) • Cerebrovascular accident (i.e., stroke) • Vascular accidents • Malignant/metastatic tumours **Congenital and Developmental Problems** • Cerebral Palsy • Autism • Developmental delay • Down's syndrome • Spina bifida with hydrocephalus • Muscular dystrophy **Progressive Processes** • Alzheimer's disease • Pick's disease • Dementia • Amytrophic Lateral Sclerosis • Multiple Sclerosis • Parkinson's disease • Huntington's disease

The vast majority of ABIs result from trauma and the two are essentially synonymous. ABIs are one of the leading causes of lifelong disability in North America (Greenwald et al., 2003; Pickett, Ardern, & Brison, 2001; Thurman & Guerrero, 1999). In the United States between 1.4 and 1.7 million people sustain a TBI every year (Faul, Xu, Wald, & Coronado, 2010; Zaloshnja, Miller, Langlois, & Selassie, 2008), with more than 120,000 people expected to develop long-term disabilities (Zaloshnja et al., 2008). In the province of Ontario, more than 80,000 individuals sustained a TBI between 2002 and 2006 (Colantonio et al., 2010). Although the majority of individuals who sustain a TBI have mild impairment, the frequency of moderate severity injury is increasing; moderate TBIs accounted for 19% of injuries in 1992 compared with 37% of injuries in 2002 (Colantonio, McVittie, Lewko, & Yin, 2009).

It is important to know which factors are significantly related to outcomes post ABI. Prognostic indicators can include such variables as injury severity, etiology of injury, age, rehabilitation length of stay, duration of post-traumatic amnesia, etc. Table 1.2 summarizes the most common ABI prognostic indicators identified in the literature.

Table 1.2 Common Prognostic Indicators for ABI

• Age • Sex • Presence of prior brain injury • Injury severity • Length of coma • Initial Glasgow Coma Scale (GCS) score • Injury etiology	• Rehabilitation length of stay • Duration of post-traumatic amnesia • Timing of rehabilitation • Intensity of rehabilitation • Nature of injury (TBI versus nTBI) • Presence of comorbidities

The majority of TBIs are related to motor vehicle or transportation accidents and falls, together accounting for approximately 75% of TBIs (Colantonio et al., 2009). Based on literature, falls account for approximately 35% to 42% of TBIs whereas MVAs are responsible for 12% to 17% (Colantonio et al., 2010; Faul et al., 2010). These trends have been consistent for approximately ten years (Roozenbeek, Maas, & Menon, 2013).

The rates of TBIs are typically higher among males, with males in the United States being 1.4 times more likely to sustain a TBI than a female (Faul et al., 2010). Data from Ontario, Canada is consistent with this finding and also shows greater rates of TBI among males (Chan, Zagorski, Parsons, & Colantonio, 2013a). This may be due to greater participation in risk-taking behaviors by males compared to females. A cohort study has shown that females have higher rates of TBIs due to falls, while TBIs in males are more likely to be caused by being struck by or against an object (Colantonio et al., 2010).

Another significant demographic factor when evaluating ABI incidence and outcomes is age. Evidence suggests that etiology of TBIs and outcome varies with age. For children aged 0-14, 50.2% of TBIs in the United States were caused by falls (Faul et al., 2010), however rates of abusive head trauma can be as high as 66% (Greenwald et al., 2003). For older adults, aged 65-85, falls make up approximately 60.7% of TBIs, with falls being the cause of 90% of TBIs for those over 85 (Chan, Zagorski, Parsons, & Colantonio, 2013b, 2013c).

As might be expected, the prognosis of good outcomes long term is also associated with age. Evidence suggests that age influences the trajectory of one's recovery following injury. Individuals in the older age bracket generally had poorer outcomes when compared to younger individuals (Marquez de la Plata et al., 2008). Older age at the time of injury has also been associated with poorer performance in various cognitive domains (Senathi-Raja, Ponsford, & Schönberger, 2010). A study by Ashman and Mascialino (2008) noted that deficits in encoding and retention of verbal information as well as inattention were more common and more serious post TBI in those over the age of 65 years. It has been postulated, for those who are older at the time of injury, that less neuronal plasticity may negatively affect the brain's ability to compensate or adapt in the same way a younger brain does post injury (Senathi-Raja et al., 2010). Mosenthal et al. (2002) found older subjects (>64 years of age) had a significantly higher mortality rate than their younger peers at all levels of TBI severity ($p<0.001$). Study authors suggested this increase in mortality may be attributable to multiple factors including pre-existing comorbidities, post injury complications, and the intrinsic challenges of aging itself (Mosenthal et al., 2002).

Click HERE to see the ERABI Module on Epidemiology and Long-term Outcomes

1.3 Grading ABIs

Q1. List three widely used indicators of severity in acute TBI

1. The Glasgow Coma Scale (GCS) (best score within 24 hours of injury).
2. Duration of unconsciousness.
3. Duration of posttraumatic amnesia (PTA).

TBI severity is usually classified according to the level of altered consciousness experienced by the patient following injury (Table 1.3). The use of level of consciousness as a measurement arose because the primary outcome to understand the severity of an injury has been the GCS. Consciousness levels following ABI can range from transient disorientation to deep coma. Patients are classified as having a mild, moderate or severe ABI according to their level of consciousness at the time of initial assessment. Various measures of altered consciousness are used in practice to determine injury severity. Common measures include the GCS, the duration of loss of consciousness (LOC), and the duration of PTA.

Table 1.3 Definitions of Traumatic Brain Injury Severity

Mild:	**Moderate:**	**Severe:**	**Very Severe:**
• PTA <1 hour • GCS 13-15 • LOC <15 minutes	• PTA 1-24 hours • GCS 9–12 • LOC <6 hours	• PTA 1–7 days • GCS between 3-8 • LOC 6-48 hours	• PTA >7 days • LOC >48 hours

The GCS is one of the most widely used measures of altered consciousness. Developed by Teasdale and Jennett (1974, 1976) it is comprised of three subsections: eye opening, best motor response, and verbal response (Table 1.4). Higher scores on the GCS are indicative of an increased level of consciousness. The total score is determined by adding the three sub scores. The total score can range from 3-15, with scores of 13-15 indicating a mild injury, 9-12 indicating a moderate injury, and 3-8 indicating a severe injury (Campbell, 2000; Murdoch & Theodoros, 2001). Module 17 of the ERABI provides more in depth information regarding the reliability and validity of this test.

For moderate to severe TBI, the **duration of LOC** appears to be a valid measure of injury severity. LOC of less than 15 minutes, up to 6 hours, and between 6-48 hours represents a mild, moderate, and severe injury, respectively. When LOC exceeds 48 hours, the injury is considered very severe (Campbell, 2000).

Table 1.4 The Glasgow Coma Scale

Response/Item	Points
Eye Opening	
Spontaneous	4
To speech	3
To pain	2
None	1
Motor Response	
Obeys commands	6
Localizes pain	5
Withdrawal (from painful stimulus)	4
Abnormal flexion	3
Extension	2
None	1
Verbal Response	
Oriented	5
Confused	4
Inappropriate	3
Incomprehensible	2
None	1

PTA is the time period post trauma for which the conscious patient has no recall for events. PTA is formally defined as the period following emergence from coma in which the patient may appear confused, disoriented, or agitated (Campbell, 2000). Research indicates a dose-response relationship, with the length of PTA frequently being proportional to the severity of injury. Injury severity is defined as mild if the duration of PTA is less than 1 hour, moderate if between 1–24 hours, and severe if PTA is between 1–7 days. PTA exceeding 7 days is considered to represent a very severe injury (Campbell, 2000; Russell, 1932).

Q2. Describe the GCS including its strengths and limitations

Description

- The GCS is a quick, simple and objective tool used during the initial examination to estimate TBI severity.
- The GCS consists of 15 items in three basic categories: (1) eye opening – 4 items, (2) verbal response – 5 items, and (3) motor response – 6 items.
- Minimum score of 3
- Maximum score of 15

Strengths

1. Simple, straightforward and brief bedside assessment.
2. Most familiar and most widely used instrument in the assessment of level of consciousness.
3. Established categories related to depth of coma and severity of injury.
4. Significant predictor of outcome following head injury.
5. Can be used by various groups of healthcare professionals regardless of level of education or ICU experience

Weaknesses

1. The application of painful stimulus is controversial.
2. Assessment can be compromised by early interventions such as intubation and sedation.
3. Use of a global score may result in loss of information that adversely affects the predictive accuracy of the GCS.
4. The motor response sub score has the greatest influence on the total GCS score.
5. Individuals with the same GCS scores in varying permutations can have significantly different survival outcomes.
6. Lack of experience may result in inaccurate assessment.
7. It is an ordinal scale whereby, for example, the difference between scores of 3 and 4 Is not the same as the difference between scores of 13 and 14

The GCS score has been shown to have a significant correlation with outcome following severe TBI, both as the summary score (Choi et al., 1994; Choi, Narayan, Anderson, & Ward, 1988) and as the motor sub score (Beca et al., 1995; Bhatty & Kapoor, 1993; Choi et al., 1988; Michaud, Rivara, Grady, & Reay, 1992). In a prospective study, the positive predictive value for a poor outcome (dead, vegetative, or severely disabled) was calculated to be 77% for patients with a GCS score of 3-5 and 26% for a GCS score of 6-

Table 1.5 Prediction of mortality based on initial GCS score

GCS Score	Mortality
3	65%
4	45%
5	35%
6	24%
7-13	10-15%

8 (Table 1.5) (Narayan et al., 1981). As is commonly done, this study grouped GCS measurements versus outcome. In a larger study each GCS level would have its own predictive value. For example, in a series of 315 patients with TBI from Australia, a significant inverse correlation was demonstrated between the initial GCS score (obtained 6-48 hours after injury) and mortality (Fearnside, Cook, McDougall, & McNeil, 1993).

1.4 Neuroanatomy Review

As might be expected, the location of the ABI can play a significant role in the deficits observed afterwards, recognizing that many of these injuries are diffuse in nature or a combination of focal and diffuse injuries. Figure 1.1 provides an overview of the types of deficits that can be seen given the location of the insult. Although this list is not exhaustive, it provides the basic functions and concepts (singular or multiple) that can be negatively impacted by a brain injury.

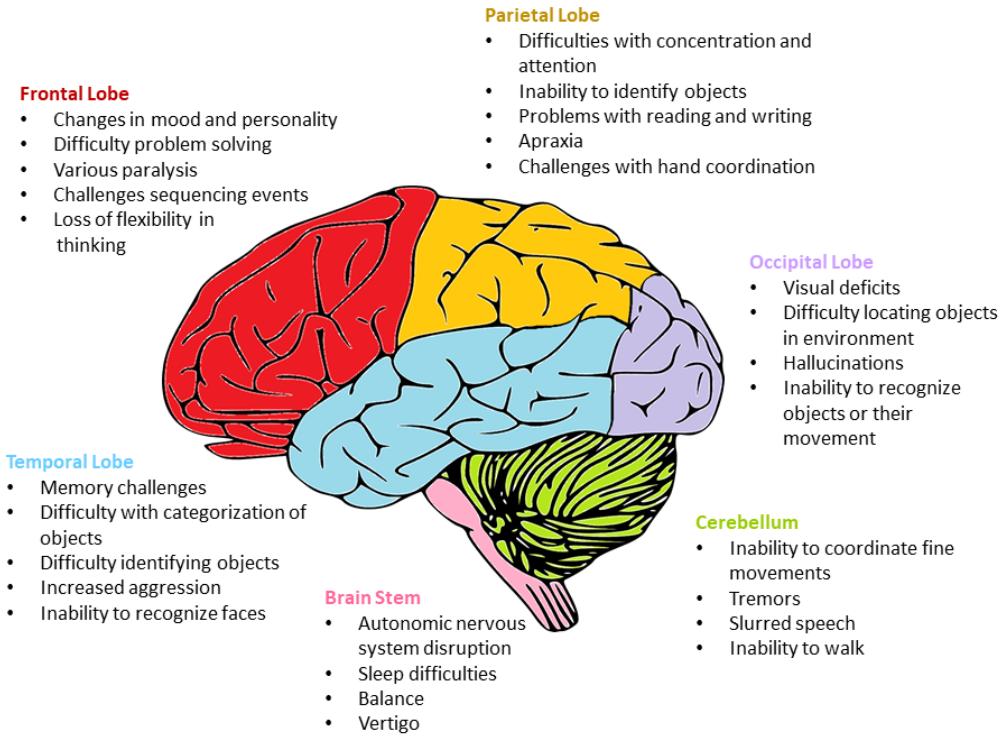
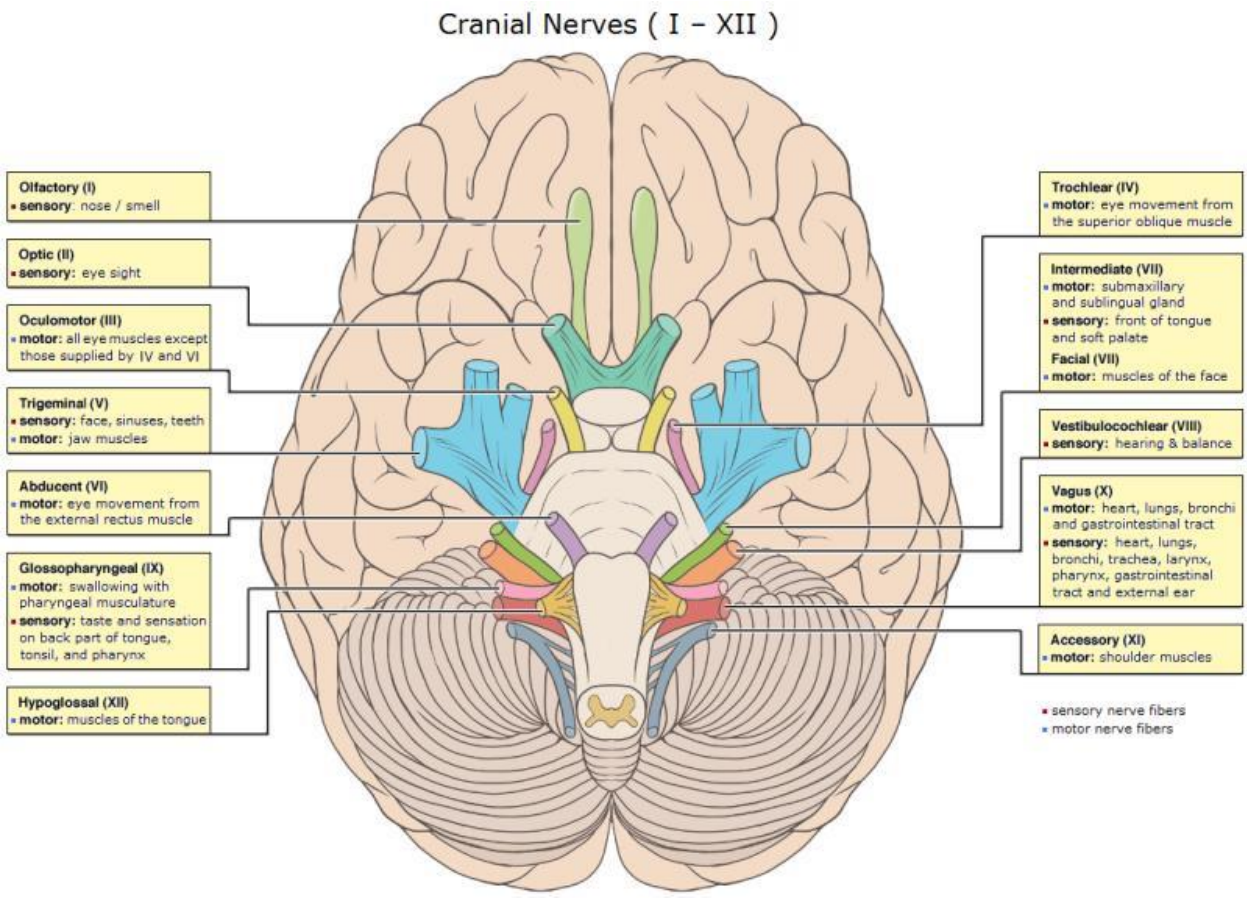


Figure 1.1 A sagittal view of the human brain which identifies the major regions of function as well as the common observed deficits which can arise from insult to each specific region of the brain (Adapted from Excel Care Nurse Case Management, 2019).

Overall, there are 6 regions of the brain which can be impacted by a brain injury. These are the frontal lobe, the parietal lobe, the occipital lobe, the temporal lobe, cerebellum, and the brain stem. Each of these regions is characterized by the functions that they are responsible for. The frontal lobe (red) of the brain is responsible for higher level cognition, motor skills, expressive language, and other functions. It terminates at the central sulcus and includes the motor cortex. The parietal lobe (yellow) includes the somatosensory cortex and is primarily responsible for processing tactile and sensory information such as

pain, pressure, and touch. The temporal lobe (blue), located at the bottom of each hemisphere, controls the comprehension of language, auditory functions and memory. The occipital lobe (purple) located at the posterior aspect of the brain, is associated with interpreting visual stimuli and information. The cerebellum (green), which is part of the hindbrain, is responsible for coordinating gross and fine movements, walking, while damage to this region can result in tremors, dizziness, and slurred speech. Although these are not complete descriptions of what each area of the brain is responsible for, it is sufficient background knowledge to put the existing ABI literature and relevant rehabilitation interventions into context.

In addition to these regions of the brain, there are internal structures consisting of cranial nerves, ventricles, and specialized structures which can also be damaged with specific consequences. Damage to one of the 12 cranial nerves can result in pain, tingling, or loss of sensation. Damage to the pituitary can result in neuroendocrine dysfunction, while damage to the hippocampus can result in memory deficits. For more details if a deficit is linked to the dysfunction or damage of a specific region of the brain it will be discussed in the chapter related to that specific deficit (ex. The ERABI module on Neuroendocrine Dysfunction discusses damage to both the anterior and posterior pituitary). Figures 1.2 and 1.3 show the neuroanatomy of the 12 cranial nerves as well as the internal specialized structures of the brain for reference.



(Original image attribution goes to Patrick J. Lynch; and was taken from https://my-ms.org/anatomy_brain_part4.htm)

Figure 1.2 Ventral view of the brain with each of the 12 cranial nerves identified along with their primary functions.

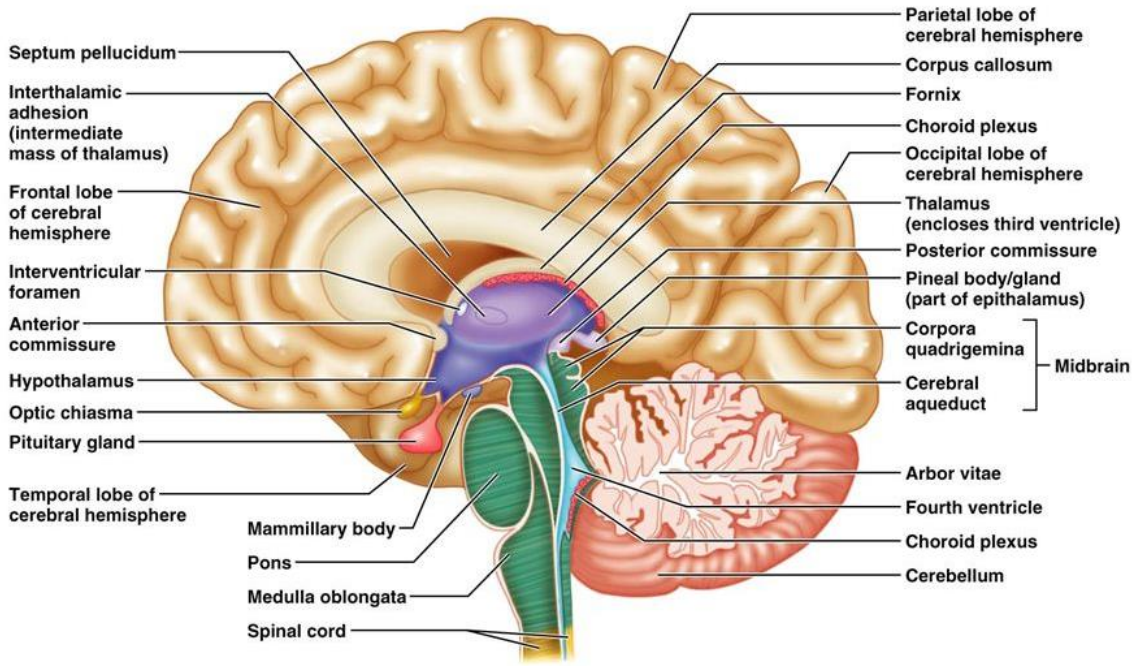


Figure 1.3 Sagittal view of the brain showing the internal structures of the brain including the ventricle, hypothalamus, and pituitary gland.

1.5 Mechanism of Injury

The two important factors when considering an ABI are the mechanism of injury (contusion or rotational forces) and being aware of, and managing, both primary and secondary injuries. The mechanism of the injury refers to the circumstance of the acquired damage. The two primary categories of injury mechanisms are presented in Table 1.6. As you can see the majority of mechanisms of injury are derived from traumatic instances, such as motor vehicle accidents or by making contact with an object.

Table 1.6 Mechanisms of Injury for ABI

Mechanistic Category	Description
Contact/Contusion	- Injury to scalp - Fracture of skull with or without associated extradural hematoma - Surface contusions, lacerations, and associated intracerebral hematomas
Acceleration/Deceleration/Rotational Forces	- Tearing of bridging veins with the formation of subdural hematoma - Diffuse axonal injury, tissue tears and associated intracerebral hematomas - Diffuse vascular injury

When discussing primary and secondary injuries, the primary injury refers to the damage caused as a direct result of the ABI; this can be the forces at impact, vascular injury (Subarachnoid hemorrhage; SAH), benign tumors, cortical disruption or other aetiologies. Secondary injuries encompass the evolution of the primary injury to include additional damage. Four categories of secondary injuries have been described by Kochanek et al. (2013). The first three categories are independent and include 1) "ischemia, excitotoxicity, energy failure", 2) secondary cerebral swelling, and 3) axonal injury, with the fourth category of inflammation and regeneration, contributing to the evolution of other secondary injury categories. The most common resultants from secondary injury are increased intracranial pressure (discussed in Module 16 of ERABI) and excitotoxicity leading to eventual apoptosis cascades (Kochanek, 2013). Regardless of the severity of the ABI both primary and secondary injuries can play a significant role in determining the outcome of an individual with an ABI.

1.6 Types of ABI

1.6.1 Diffuse Axonal Injury

Q3. Describe diffuse axonal injuries

1. Diffuse axonal injury (DAI) is seen exclusively following TBI and results from acceleration-deceleration and rotational forces associated with a high-velocity impact (ie. motor vehicle accidents).
2. Physical shearing of axons results in hemorrhage, tissue tears, axonal swelling and the formation of axonal bulbs acutely. Subacute clusters of microglia and macrophages are seen. Chronically Wallerian degeneration occurs.
3. DAI can be responsible for the initial loss of consciousness seen in acute TBI.
4. Damage is most often seen within midline structures and at interfaces between gray and white matter. In particular, the corpus callosum, the parasagittal white matter, the interventricular septum, the walls of the third ventricle and the brainstem, in particular the midbrain and the pons.

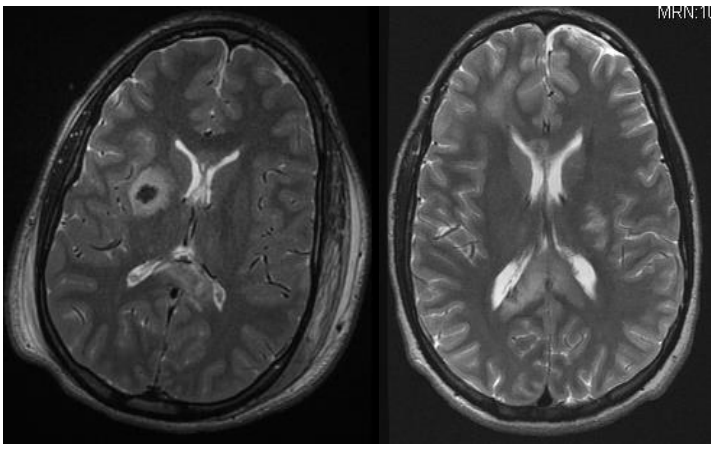


Figure 1.4 Two scans showing diffuse axonal injuries in the subcortical regions.

DAI is the ***distinguishing feature of traumatic brain injury due to high-speed accidents***. The predominant causes of DAI include:

1. High-speed motor vehicle collisions above 15 mph or 24 km/h.
2. Shaken baby syndrome.
3. High-speed collisions in sports (i.e. football, hockey, soccer, rugby) (see figure 1.5).

Rotational forces can cause diffuse tearing of neural processes and blood vessels throughout the white matter resulting in diffuse axonal injury (Figure 1.5). Hemorrhagic changes involving the midline structures are often associated with rotational acceleration and tend to involve the parasagittal white matter, corpus callosum, structures in the walls of the 3rd ventricle and striatum (basal ganglia) (Goldberg, 2001). Strain tends to be concentrated at the interfaces between gray and white matter, at the midbrain juncture between the brainstem and diencephalon, and at the juncture between the corpus callosum and the cerebral hemispheres (Goldberg, 2001).

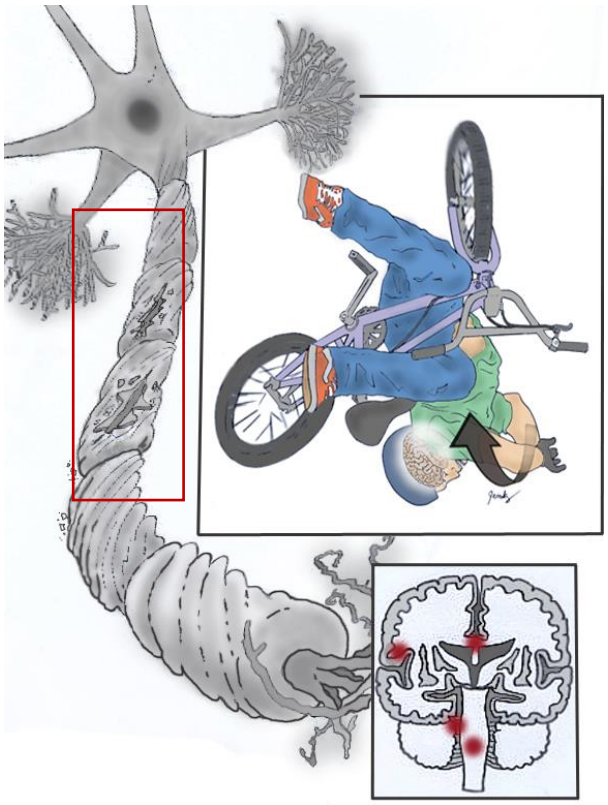


Figure 1.5 Image showing the axonal tearing and shearing that can occur as a result of rotational forces on the cranium during a traumatic brain injury.

Q4. Describe some of the clinical features seen following diffuse axonal injuries.

1. Rostral brain stem involvement results in initial loss of consciousness, poor attention and concentration.
2. Corticospinal tract involvement results in hemiparesis.
3. Shearing of the grey-white matter junction results in slowed mental processing and fatigue.
4. Cerebellar peduncle involvement results in ataxia.
5. Brainstem injury involvement results in dysarthria and dysphagia.

Given the breadth of symptoms that can result as a consequence of DAI, specific considerations must be made in order to compensate for deficits in an appropriate manner during early recovery.

Q5. How does diffuse axonal injury impact recovery and rehabilitation?

1. Disrupted connections between nerves results in slowed mental processing, fatigue, poor attention and concentration.
2. Rehabilitation must be organized in a manner that compensates for these difficulties such as by providing a structured environment.
3. Physical and cognitive stamina may be reduced and proper pacing will need to be implemented.
4. Poor attention combined with memory difficulties and behavioural concerns may require attendant care.

1.6.2 Focal Injury

Cortical Contusions

Q6. What are cerebral contusions? Where do they tend to occur most often?

1. Cerebral contusions are cortical “bruises”
2. They occur at the crests of gyri and extend to variable depths.
3. They occur commonly at the inferior frontal, anterior temporal, and inferior occipital lobes.

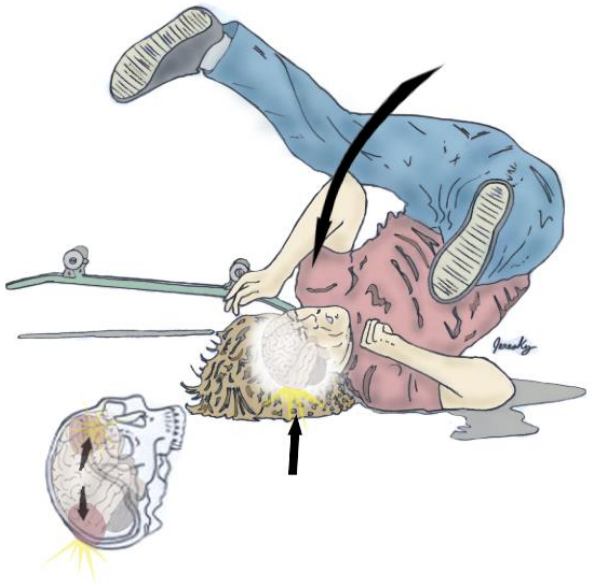


Figure 1.6 Example of a coup-contrecoup injury that might occur where an individual strikes their head twice on opposite poles of the cranium.

Cortical contusions are quite common following traumatic brain injury and in some cases can be quite involved, extending through the cortex and into the subcortical white matter (Burke & Ordia, 2000). Cortical contusions tend to occur in characteristic areas, in part because of the movement of the brain in the skull with acceleration-deceleration and rotational forces and because of the location of bony protrusions where the brain can strike inside the skull (Burke & Ordia, 2000). ***One common combination of contusions is the coup-contrecoup injury*** (Figure 1.6). In this instance the patient strikes the front of their head and the brain accelerates forward in the direction of the impact striking the skull. The brain then rebounds in the opposite direction and strikes the back of the skull, creating the countercoup injury. ***Contusions usually occur bilaterally in the frontal poles, the anterior tips of the temporal lobes and in some cases the lateral parts of the temporal lobes and the occipital regions*** (Burke & Ordia, 2000).

Intracranial Hemorrhages

Intracranial hemorrhage, regardless of whether it is epidural, subdural, intracerebral, intraventricular, or subarachnoid in location, is a major concern post TBI (Burke & Ordia, 2000). A hematoma can exasperate damage to the brain by: 1) placing direct pressure on the underlying brain structures, or 2) causing a portion of the brain to herniate leading to secondary compression of the brainstem (Burke & Ordia, 2000).

Intracranial Hemorrhages: Epidural Hematomas

Q7. What is the general prognosis of epidural hematomas?

1. The underlying brain injury is often not severe if treated promptly.

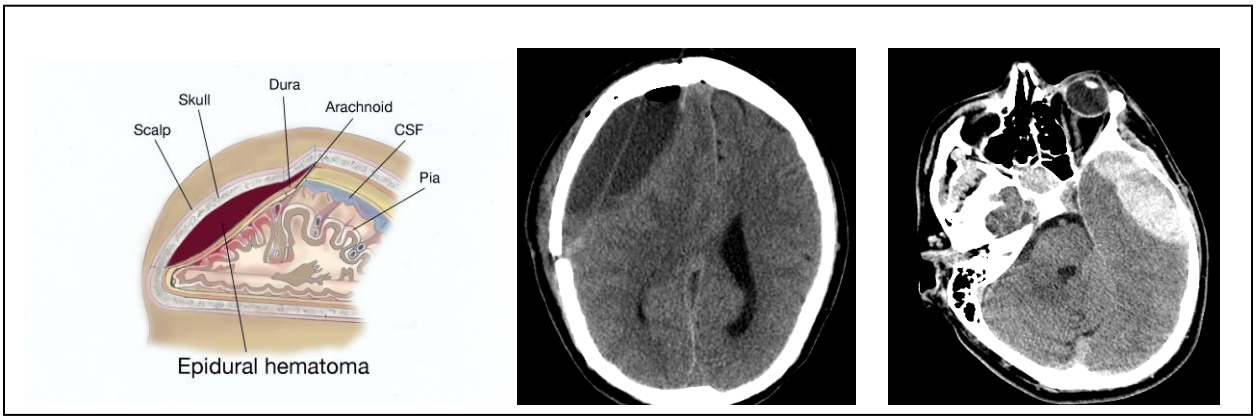


Figure 1.7 An illustration and two scans which exemplify both the nature of epidural hematomas as well as their observed pathology.

Epidural hematomas (EDH) are often the result of an impact to the head that causes disruption of the middle meningeal artery and/or its branches (Burke & Ordia, 2000). Less commonly an EDH can occur due to a dural venous sinus injury. Blood collects between the dura and the skull. On imaging, an EDH has a characteristic "lens-shaped" appearance that crosses the midline but does not cross the skull's suture lines. Symptom presentation can be delayed and may only become apparent 5 days post injury (Zasler, Katz, & Zafonte, 2007). However, if the EDH is related to an arterial laceration, it may evolve quickly resulting in rapid deterioration and even death (Burke & Ordia, 2000). If treated promptly the underlying brain injury may not be that severe (Burke & Ordia, 2000). Signs of an EDH include a decreased level of consciousness or "clouding". If lesions occur in the frontal lobe, onset of symptoms may be slow and vague (Zasler et al., 2007). Patients must be monitored carefully.

Intracranial Hemorrhages: Subdural Hematomas

Q8. What is the general prognosis of subdural hematomas (SDH)?

1. Prognosis is poor with a high mortality because of the severity of the underlying brain injury

SDH are common in severe brain trauma and often occur in individuals who are on blood thinners or who are older. SDH occur due to injuries of the cortical bridging veins or rarely the pial artery (Burke & Ordia, 2000). On imaging, a SDH has a crescent shape that does not cross the midline (it is confined by dural reflections) but can cross skull suture lines. Due to the severity of injury to the underlying brain tissue, the prognosis is poor and mortality rates are high.

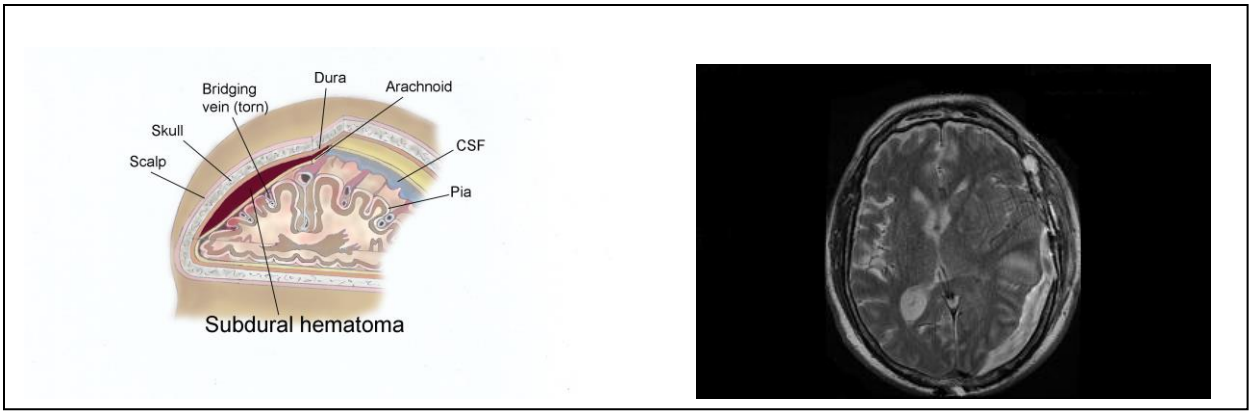


Figure 1.8 An illustration and a scan which exemplifies both the nature and the observed pathology of subdural hematomas.

Intracranial Hemorrhages: Intracerebral Hemorrhage

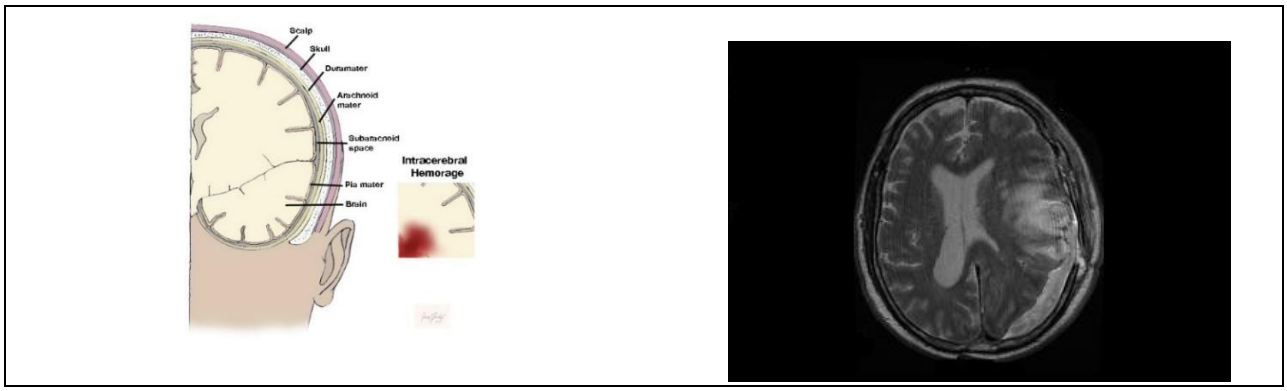


Figure 1.9 An illustration and scan showing the nature and pathology of intracerebral hemorrhages.

Intracerebral haemorrhages may result from ruptured blood vessels in association with a penetrating or non-penetrating head injury. Although they often occur in the frontal or temporal lobes, they may also be found in the cerebellum and brain stem.

Intracranial Hemorrhages: Subarachnoid Hemorrhage

The subarachnoid space is located between the arachnoid membrane and the pia mater that covers the surface of the brain. This space contains CSF, originating from the choroid plexus. The CSF flows from the ventricular system to the basal cisterns, and to the surface of the brain. Small arachnoid granulations within the dural venous sinuses absorb the CSF.

SAH can be atraumatic or traumatic in etiology. SAH most often occurs secondary to a ruptured aneurysm of the Circle of Willis causing blood to accumulate near the site of aneurysmal rupture. In contrast, SAH related to traumatic cerebral contusions is variable in terms of its location and is the result of microvessel shearing in the subarachnoid space. SAH can be associated with complications such as intraventricular haemorrhage, vasospasm, arachnoiditis, seizures, and communicating hydrocephalus because blood products obstruct the arachnoid granulations. Although rare, communicating hydrocephalus can progress to non-communicating hydrocephalus.

SAH presents on CT scan as bright hyperdense blood within the sulci and/or basal cisterns. CT is very sensitive for the detection of acute SAH. MRI is less sensitive for the detection of acute SAH, however it is very good for identifying chronic blood products or hemosiderin deposits.

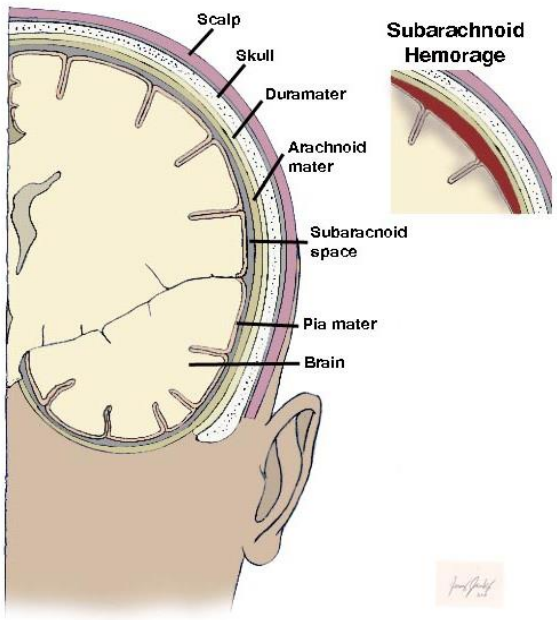


Figure 1.10 Illustration of a subarachnoid hemorrhage.

Intracranial Hemorrhages: Intraventricular Hemorrhage

Intraventricular hemorrhage (IVH) can be classified as primary or secondary. Primary IVH occurs due to injury of the supependymal veins surrounding the ventricles. Secondary IVH occurs due to extension of adjacent SAH or Intracerebral haemorrhage. On CT, IVH appears bright and is often layered within the dependent portions of the ventricles. IVH can obstruct cerebrospinal fluid flow resulting in hydrocephalus.

1.7 Disorders of Consciousness

Q9. Which part of the brain determines consciousness?

1. Consciousness is a function of the ascending reticular activating system (RAS) and the cerebral cortex
2. The cell bodies of the RAS are located in the central reticular core of the upper brainstem (primarily midbrain) and connect to the cerebral cortex via thalamic and extra-thalamic projections.

Consciousness can be defined as an awareness of self, and one's environment (Plum & Posner, 1982). Both arousal and awareness are required to be in a conscious state. ABI can significantly impact the brainstem and the cortex which are responsible for these processes. Severe injuries result in disorders of consciousness (DOC) defined as a coma, a vegetative state (VS) (Ashwal et al., 1994), or a minimally conscious state (MCS) (Giacino et al., 2002). The American Academy of Neurology establishes a ≥28 day loss of consciousness in order to diagnose a DOC. To confirm a diagnoses of either a VS or a MCS the American Academy of Neurology Guidelines on Disorders of Consciousness (2018) state that electromyography, evoked potentials, and functional MRIs are able to discriminate between a MCS and VS and assist in the diagnosis of DOC. According to the American Academy of Neurology only 35% of individuals with a DOC recover consciousness within the first three months, however this increases to 67% of individuals recovering consciousness six months post-injury (Giaicino et al., 2018).

Click here to see the AAN Practice Guidelines for Disorders of Consciousness

Q10. Describe the clinical features of coma.

1. There is no evidence of self- or environmental-awareness.
2. Eyes remain continuously closed.
3. No sleep-wake cycles on EEG
4. No spontaneous purposeful movement
5. Inability to discretely localize noxious stimuli
6. No evidence of language comprehension or expression

The criteria for diagnosing a coma has remained relatively unchanged since first described by Plum and Posner in 1982. The key features of a coma include a) the complete loss of spontaneous or stimulus induced arousal, b) no detectible sleep/wake cycles on electroencephalography (EEG), c) no evidence of intentional motor activity such as those in response to a command, d) no language ability, e) eyes remain closed, even in the presence of adverse stimuli.

Q11. Define what is meant by the term vegetative state.

1. Loss of capacity to interact with the environment despite the preserved potential for spontaneous or stimulus-induced arousal

Q12. Describe the clinical features of the vegetative state.

1. Patient opens eyes (either spontaneously or with noxious stimuli)
2. Intermittent wakefulness with sleep-wake cycles
3. No evidence of purposeful behaviour; may startle to verbal/auditory stimuli but will not localize
4. No evidence of intelligible verbal or gestural communication
5. Visual tracking is considered a sign of patient transitioning out of the vegetative state

VS occurs when the capacity for stimulus-induced or spontaneous arousal is maintained while awareness surrounding oneself or one's environment is lost (Giacino, 2013). A diagnosis of VS is made with the re-emergence of spontaneous eye opening, and the loss of intentional behavioral responses, as well as the ability to engage in language expression or comprehension. A VS lasting more than one month can be described as a persistent vegetative state.

Q13. Define MCS.

1. Severely altered consciousness with minimal but definite behavioural evidence of self- or environmental awareness (Giacino, 2013)

Q14. Describe the clinical features of the MCS.

1. Sleep-wake cycles
2. The patient demonstrates minimal evidence of self- and/or environmental-awareness
3. Purposeful movements are reproducible but remain inconsistent: Simple command following, object manipulation, and gestural or verbal yes/no responses
4. Eyes will open spontaneously. The patient may also show visual fixation, smooth pursuit tracking and emotional or motor behaviours contingent upon specific eliciting stimuli (ie. patient may respond best to voices of family members)

MCS typically reflect a period of recovery from comas or vegetative state. In a post-mortem analysis of those with TBI and confirmed MCS, typical lesions were found to be grade 2 or 3 axonal injuries (Jennett, Adams, Murray, & Graham, 2001). To diagnosis a MCS one or more of the following behaviors must be present and reproducible (Zasler et al., 2007):

1. Simple command following
2. Intelligible verbalization
3. Recognizable verbal or gestural "yes/no" responses (without regard to accuracy)
4. Movements or emotional responses that are triggered by relevant environmental stimuli and cannot be attributed to reflexive activity. Examples of the fourth criterion include (a) smiling or crying following exposure to emotional (e.g., family photographs) but not neutral stimuli (e.g., photographs of objects), (b) vocalizations or gestures that occur in direct response to specific linguistic prompts, (c) accurate reaching toward objects laced within the immediate visual field, (d) manipulation of objects placed in the hand, and (e) sustained visual fixation or pursuit eye movements.

Emergence from a MCS is typically identified by functional object use as well as increased interactive communication. These represent the criteria to recover from a MCS as both features require widely distributed cortical connectivity. Socially, these behaviors also represent a significant increase in personal autonomy, and therefore are used to determine if an individual has left a MCS.

1.8 Post Traumatic Amnesia

Q15. What is Post Traumatic Amnesia (PTA)?

1. PTA is defined as the "immediate and dramatic loss of memory that TBI can cause"
2. PTA is the period during which individuals with a TBI "are unable to effectively encode and retain new information and experiences"
3. PTA varies in duration from a few moments to a few months, either retroactively, proactively, or both.
4. Resolution of PTA clinically correlates with the period when incorporation of ongoing daily events occurs in the working memory.

If an individual has post-traumatic amnesia they may appear confused, lack self-awareness of their injuries, and even be agitated or combative. Individuals may even seem alert during the acute phases of their injury and engage normally, however, later they may report that they were unconscious for this period of time and have no memory of it (Eslinger, 2013).

Table 1.7 The relationship of PTA to likely outcomes in individuals with ABI.

Duration of PTA	Likely Outcome
1 day or less	Expect quick and full recovery with appropriate management (a few patients may show persisting disability)
More than 1 day and less than 1 week	Recovery period is more prolonged lasting weeks or months. However, for most patients full recovery is possible with good management.
1-2 weeks	Recovery occurs over many months. Many patients will be left with residual problems even after the recovery process has ended, but one can be reasonably optimistic about functional recovery with good management.
2-4 weeks	Process of recovery is very prolonged – 1 year or longer is not unusual. Permanent deficits are likely. There must be increasing pessimism about functional recovery when PTA reaches these lengths.
More than 4 weeks	Permanent deficits, indeed significant disability, are now certain. It is not just a matter of recovery but of long-term retraining and management.

(Adapted from Brooks DN and McKinlay WW, Evidence and Quantification in Head Injury: Seminar notes. Unpublished material, 1989).

PTA gradually improves over time in the majority of cases. The Galveston Orientation and Amnesia Test (GOAT) is typically used to diagnose and track PTA (Eslinger, 2013). Overall, researchers conclude that recovery can be predicted from early daily screening and the GOAT can be a moderately strong predictor of length of stay, functional independence, and short-term memory (Eslinger, 2013).

Q16. Describe the GOAT including advantages and disadvantages.

Description

The GOAT consists of 10 items regarding orientation to:
1. person: name, address and birth date
2. place: city/town and building they are in
3. time: current time, date, month year and date of hospital admission
4. memory of events both after and prior to the injury (Bode, Heinemann, & Semik, 2000).

Advantages
1. The GOAT provides an objective rating of early cognitive recovery eliminating the need for sometimes ambiguous terminology used to describe mental status, such as "confused" (Levin, O'Donnell, & Grossman, 1979).
2. Due to its design, the scale has been shown to be useful for assessing patients with a wide range of cognitive impairments (Salter, Jutai, & Teasell, 2008).
3. Can be used to guide timing of neuropsychological testing which should not be attempted until GOAT score consistently >70.

Limitations
1. The standard GOAT response format makes administration difficult with nonverbal patients (Novack, Dowler, Bush, Glen, & Schneider, 2000). It is important to note that A-GOAT has been developed for use in aphasic patients but requires further evaluation.
2. The requirement for oral or written expression may result in penalizing patients who are experiencing deficits of expression but not in orientation or in the retrieval or consolidation of memory (Jain, Layton, & Murray, 2000).
3. While the GOAT does contain items intended to provide an assessment of memory, it is primarily a measure of orientation. Eight of the 10 GOAT items evaluate orientation while only two examine memory (Forrester, Encel, & Geffen, 1994).

The GOAT provides an objective assessment with a standardized cut-off for the presence of PTA. However, in its original form, the GOAT is not well suited to assessment of patients with aphasia. It may be too lengthy for a simple, repeated bedside assessment of mental status. However, it is freely available and can be used by any healthcare professional (Salter et al., 2008).

Assessment consists of 10 items regarding orientation to person (name, address & birthdate), place (city/town and building they are in) and time (current time, date, month, year & date of hospital admission) as well as memory of events both after and prior to the injury (Bode et al., 2000). Oral questions are posed directly to the patient who may respond either orally or in writing (Jain et al., 2000; Levin et al., 1979). Error points are awarded for each incorrect response and are summed and deducted from 100 to arrive at the total score. Both the scale and instructions for assigning error points are available in Levin et al. (1979).

The GOAT was intended to evaluate orientation to time, place and person and to provide an estimation of the intervals prior to and following the injury for which there is no recall (Levin et al., 1979). *The duration of PTA is defined as the period following coma in which the GOAT score is 75 or less* (Levin et al.,

1979). ***PTA is considered to have ended if a score greater than 75 is achieved on 2 consecutive administrations (Novack et al., 2000; Wade, 1992; Zafonte et al., 1997)***. In the initial standardization study of Levin et al. (1979) using patients with mild head injury as a reference group, it was determined that a score of 75 represented a level achieved by 92% of the standardization group. No patients with mild head injury scored less than 65 on the GOAT. Scores between 66 and 75 are considered borderline-abnormal while scores above 75 fall into the range considered normal within the reference group (Levin et al., 1979; van Baalen et al., 2003).

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