Traumatic Brain Injury

An Overview of Epidemiology, Pathophysiology, and Medical Management

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KEYWORDS

• Traumatic brain injury • TBI • Head injury • Acquired brain injury • Concussion • Disorders of consciousness

KEY POINTS

- Traumatic brain injury (TBI) is gaining more attention because of long-term effects as well as increasing rates of brain injury driven by emergency department visits.
- Understanding the severity of TBI helps both with prognosis of functional recovery and in anticipating patients' rehabilitation needs.
- Medical complications are common after moderate and severe TBI and should be considered and addressed by the treating physician.
- There is a high rate of misdiagnosis in patients with severe TBI and disorders of consciousness; therefore, it is imperative to refer these patients to specialized multidisciplinary rehabilitation teams to optimize diagnosis, prognosis, and management.

The goal of this article is to provide a general review of the epidemiology, acute care, and chronic management of adult patients with traumatic brain injuries (TBI). This text was created for providers practicing outside of physical medicine and rehabilitation (PM&R). The medical and rehabilitation management of moderate to severe TBI is the focus of this article, with a brief discussion of the management of mild injuries.

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DEFINITIONS

According to the Centers for Disease Control and Prevention (CDC), a TBI is caused by a bump, blow, or jolt to the head, or a penetrating head injury that disrupts the normal function of the brain. Traumatic impact injuries can be defined as closed (nonpenetrating) or open (penetrating).[1,2]

EPIDEMIOLOGY

In 2014, the CDC documented 2.53 million TBI-related emergency department (ED) visits. There were approximately 288,000 TBI-related hospitalizations and 56,800 TBI-related deaths. These data include both adults and children. Older adults aged 75 years and older had the highest rate of TBI-associated ED visits (1682 per 100,000 people) followed by young children 0 to 4 years old (1618.6 per 100,000 people), and last, followed by adolescents and young adults 15 to 24 years old (1010.1 per 100,000 people).[1]

Emergency Department Visits and Deaths

TBI-related ED visits and deaths have increased steadily from 2006 to 2014.[1] This increase may be partially attributed to improved brain injury awareness among providers and more accurate reporting and surveillance methods (**Table 1**).

Table 1
Most common reasons for traumatic brain injury–related hospitalization

Age Range (years)	0–17	15–44	55+
Mechanism of injury	Falls	MVC	Falls

Modified from Centers for Disease Control and Prevention (2019). Surveillance Report of Traumatic Brain Injury-related Emergency Department Visits, Hospitalizations, and Deaths—United States, 2014. Centers for Disease Control and Prevention, U.S. Department of Health and Human Services.

Deaths

According to 2014 CDC data, the most common causes of TBI-related deaths in descending order are intentional self-harm (32.5%), unintentional falls (28.1%), and motor vehicle crashes (MVC) (18.7%).

Trends in TBI-related deaths tracked from 2006 to 2014 suggest intentional self-harm and unintentional falls are the only monitored categories with increasing incidence (**Table 2**).[1]

Table 2
Most common cause of death by age groups

Age Range (years)	0–4	15–24	25–34	45–64	65+	≥75[a]
Cause of death	Homicide	MVC	MVC	Intentional self-harm	Falls	MVC

[a] Highest rate of death.

Modified from Centers for Disease Control and Prevention (2019). Surveillance Report of Traumatic Brain Injury-related Emergency Department Visits, Hospitalizations, and Deaths—United States, 2014. Centers for Disease Control and Prevention, U.S. Department of Health and Human Services.

Mechanisms of Injury

The most common mechanisms of injury, in descending order of frequency, include unintentional falls, being unintentionally struck by an object, MVC, assault, other (no mechanism specified), and intentional self-harm.[1]

The Department of Defense identifies TBI as the signature injury of Operation Enduring Freedom and Operation Iraqi Freedom veterans. Blast injuries are a common mechanism of injury associated with this war period.[3]

Severity

Understanding brain injury severity helps with both prognosis of functional recovery and anticipating patients' rehabilitation needs (Fig. 1, Table 3).

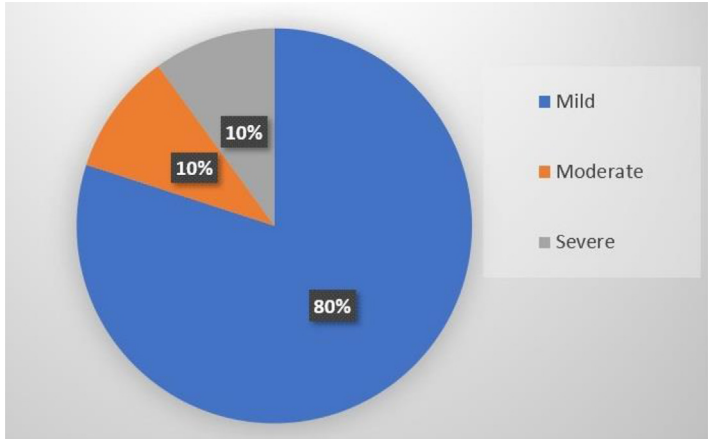


Fig. 1. Severity of TBI in the United States. (*Data from* Wagner AK, Arenth PM, Kwasnica C, et al. Traumatic Brain Injury. In: Cifu DX, editor. Braddom's Physical Medicine and Rehabilitation, 5th edition. Philadelphia: Elsevier; 2016.)

Table 3
Classification of traumatic brain injuries

	GCS[a] (First 24 h)	Loss of Consciousness	Alteration of Consciousness	Imaging	PTA
Mild	13–15	0–30 min	Up to 24 h	Normal	0–1 d
Moderate	9–12	>30 min and <24 h	>24 h	Normal or abnormal	>1 d and <7 d
Severe	3–8	>24 h	>24 h	Normal or abnormal	>7 d

Note: Some institutions use the term "mild complicated TBI" for patients who meet the mild classification by GCS, loss or alteration of consciousness, and posttraumatic amnesia but have abnormal imaging findings, such as a subdural hematoma or depressed skull fracture.[4]

[a] See Table 4.

Adapted from Veterans Affairs/Department of Defense. VA/DoD Clinical Practice Guideline for the Management of Concussion-Mild Traumatic Brain Injury. Washington, DC: Veterans Health Administration; 2016, with permission.

Table 4
Glasgow coma scale

Score	Eye Opening	Verbal	Motor[a]
1	None	None	None
2	To pain	Incomprehensible speech	Extension (decerebrate posturing) to pain
3	To speech	Inappropriate speech	Flexor (decorticate posturing) to pain
4	Spontaneous	Confused	Withdraws to pain
5	–	Oriented	Localizing response
6	–	–	Follows directions

[a] The motor score is the only part of the GCS with prognostic value.
Reprinted with permission from Elsevier (Teasdale G, Jennett B. Assessment of coma and consciousness. A practical scale. Lancet 1974;2(7872):83).

Traumatic Brain Injury in Sports

Current literature suggests TBI makes up 10% to 15% of all sports-related injuries. Collision sports (American football followed by women's soccer) report the highest incidence of TBI.[5] However, adequate reporting systems in other growing sports in the United States may be lacking, potentially leading to overrepresentation or underrepresentation within the current data sets.

Gender

There are gender differences in the epidemiology of TBI. According to the TBI Model System National Database Statistics from 2017, male cases greatly outnumbered female cases, accounting for more than 73% of all TBIs reported.[6] Conversely, in sports-related concussion, female cases outnumber male cases at about a 2:1 ratio.[5] The discrepancy in gender reporting for sports-concussion may be due to cultural differences (women being more willing to report injury than men) or physiologic differences, such as the difference in head-to-neck ratio between men and women.[5] Among older individuals (>65 years), the frequency of TBI is about the same for men and women.[7]

Long-Term Implications

TBI is a leading cause of long-term disability among children and young adults in the United States.[7] Current literature suggests more than 1.1% of the US population is living with a TBI. Of that population, more than 40% of patients with moderate to severe injuries will have long-term disability.[8] Cost estimates are varied, ranging from $56 to $221 billion annually.[2,4,5,7,8] The variation reflects difficulty measuring hidden costs associated with long-term disability, such as lost wages and caregiver expenses.[2,5]

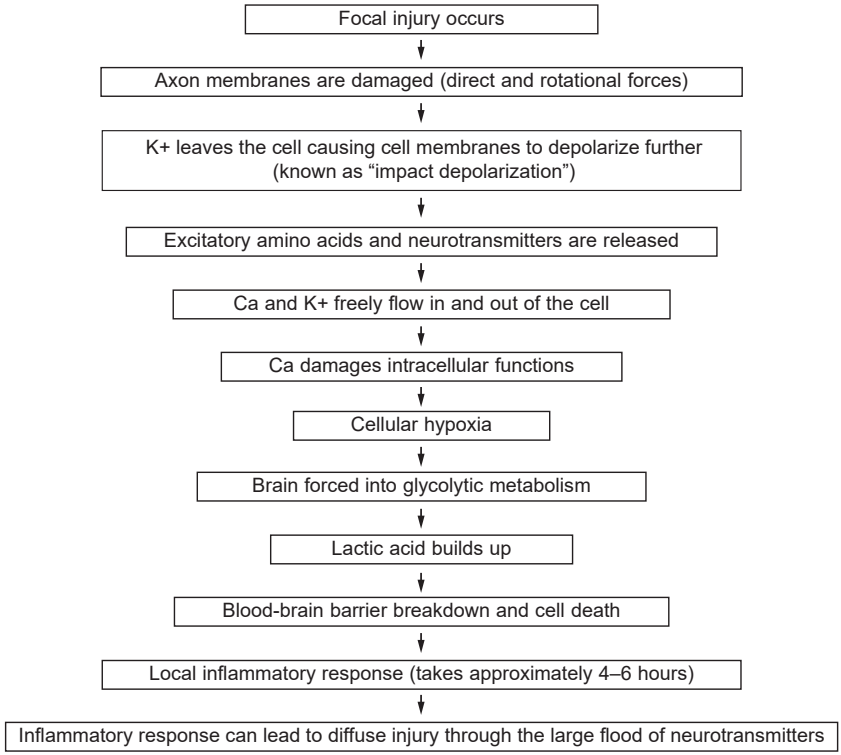
PATHOPHYSIOLOGY

Traumatic injuries disrupt normal cellular function within the brain through direct, rotational, and shear forces. These forces may be present in all severities of injury. Rotational forces disrupt axons within the white matter tracts of the brain, which can lead to diffuse axonal injury. A special MRI technique known as diffusion tensor imaging can evaluate white matter tract damage.[4] In addition, axonal injury results in local swelling, which slows signal transmission. Traumatic injury is also associated with changes in cerebral blood flow, resulting in an initial decrease in blood flow followed by

unresponsive vasodilation thought secondary to nitric oxide release in the tissue. This vascular phenomenon is best documented in cases of mild traumatic brain injury (mTBI) in rodent studies.[5,7,9]

The diagram in **Box 1** is a simplified flowchart of the pathophysiology at the cellular level after TBI.

Box 1
Pathophysiology of traumatic brain injury



Data from Refs.[5,7,9]

Both focal and diffuse injury can occur within the same patient. Focal injury can result from direct or indirect impact. Indirect impact is considered secondary to acceleration-deceleration force. As the brain is surrounded by a layer of cerebral spinal fluid (CSF), force from the direct impact will translate the brain to the opposite side of the skull, resulting in a second impact. Focal injuries are most associated with frontal and temporal lobe damage. Damage to these areas is linked to problems with executive function, impulsivity, and disinhibition (**Table 5**).[5]

Table 5
Primary versus secondary injury

Primary Injury	Secondary Injury
• Direct hit can produce indirect damage through acceleration-deceleration (coup contrecoup) mechanism • Penetrating (open) vs nonpenetrating (closed) • Considered the period of focal injury, which can progress to become diffuse through secondary injury mechanisms	• Damage at the cellular/molecular level • Ischemia causes cell death • Vasogenic edema = extracellular edema, associated with cerebral contusion • Cytogenic edema = intracellular edema, associated with hypoxic and ischemic injury

Data from Wagner AK, Arenth PM, Kwasnica C, et al. Traumatic Brain Injury. In: Cifu DX, editor. Braddom's Physical Medicine and Rehabilitation, 5th edition. Philadelphia: Elsevier; 2016; and Elovic E, Baerga E, Galang GF, et al. Traumatic Brain Injury. In: Cuccurullo SJ, editor. Physical Medicine and Rehabilitation Board Review, 2nd edition. New York: Demos Medical Publishing; 2010.

Focal Injury

This type of injury can occur via multiple mechanisms. It is important to understand that, unlike some other neurologic disorders, a focal injury to the brain due to trauma may not produce predictable clinical symptoms.[4] Examples of intracranial pathology resulting from focal injury includes epidural hematoma, subdural hematoma, subarachnoid hemorrhage (in the case of an isolated aneurysm rupture) and intraventricular hemorrhage are presented in **Boxes 2–5**.[2,4]

Box 2
Epidural hematoma

- Bleeding outside the dura of the brain (Fig. 2)
- Does not cross suture lines
- Classically associated with middle meningeal artery damage
- Clinically may involve a period of loss of consciousness followed by a "lucid" interval with subsequent cognitive decline due to increased intracranial pressure, which can lead to herniation
- Lentiform hyperintensity noted in right (R) frontal region on the left side of the image

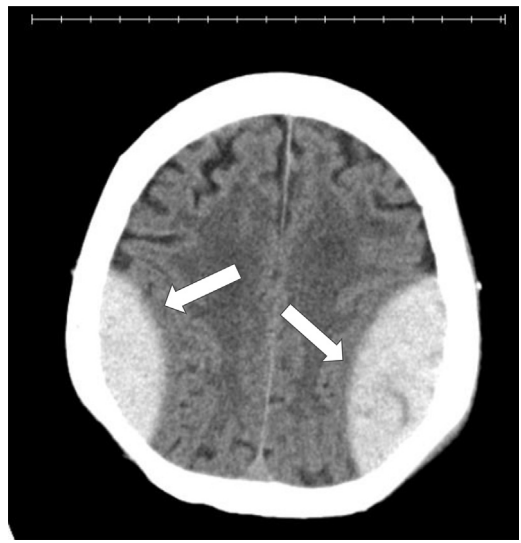


Fig. 2. Epidural hematoma (*white arrows)*are pointing at two hyperdense lentiform (lense-shaped or lemon-shaped) areas. In the acute phase, blood appears hyperdense (white) on a CT scan. Epidural hematomas are classically associated with injury to the middle meningeal artery and are considered extradural, as such, they do not typically cross the suture lines of the skull.

Box 3
Subdural hematoma

- Bleeding inside the dura (Fig. 3)
- Blood can cross suture lines
- Classically associated with damage to bridging veins
- Crescent-shaped region of hyperintensity shown via arrow on the left side of the image

Fig. 3. Subdural hematoma (*white arrow*) is pointing at a crescent shaped hyperdense (white) area demonstrating a subdural hematoma. Subdural hematomas are classically associated with damage to bridging cortical veins and as such they are able to cross suture lines. In this image, you can clearly see compression of the right lateral ventricle and right to left midline shift resulting from the subdural hematoma.

Box 4
Subarachnoid hemorrhage

- May be traumatic or nontraumatic (Fig. 4)
- Ruptured aneurysm is common cause
- Blood collects in the subarachnoid space, seen as areas of hyperintensity after acute injury (arrows)

Fig. 4. Subarachnoid hemorrhage. Arrows are pointing to hyperdensities within the subarachnoid space which, in the acute phase, indicates subarachnoid bleeding.

Box 5
Intraventricular hemorrhage

- Bleeding into the ventricles (Fig. 5)
- Clinically may be associated with hydrocephalus
- Acute hemorrhage and hydroce-phalus seen by hyperintensity in all ventricles on the left side of the image (arrows)

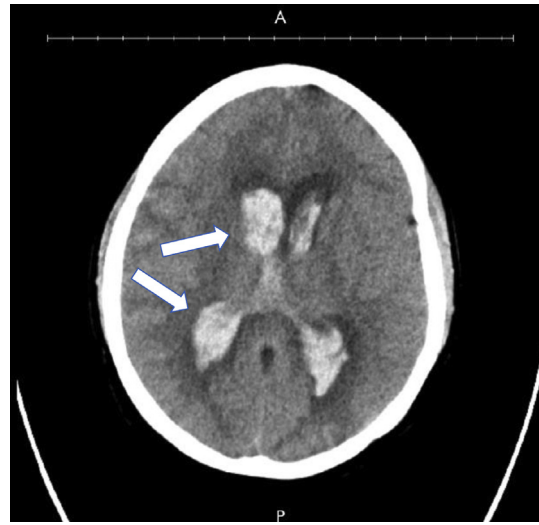


Fig. 5. Intraventricular hemorrhage. Arrows are pointing to the lateral ventricles of the brain which typically appear hypodense or black on a CT scan. The diffuse hyperdense region within the ventricles demonstrates acute intraventricular hemorrhage.

Diffuse Injury

The presence of diffuse axonal injury on diffusion-weighted imaging studies is associated with a poorer prognosis for recovery.[8] *Diffuse injury, as opposed to focal contusion, is associated with disorders of consciousness* (DOC) (Table 6).

Table 6
Grading of diffuse axonal injury

Grade I	Grade II	Grade III
Affects gray-white matter interface Frontal/ temporal > parietal/occipital	Involves frontal, temporal, parietal, occipital lobes, and corpus callosum	Includes damage to the brainstem as well as damage to structures mentioned in grade I and II

Data from Armstrong M, Chung K, Himmler M, et al. TBI Classifications and Rehabilitation Intensities. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018.

ACUTE MANAGEMENT

On-Scene Assessment

Following an acute TBI, the patient should be evaluated and medically stabilized as quickly as possible. As in other emergency situations, evaluating the airway, breathing, and circulation is the first step. Problems with circulation, as in a cardiac arrest, may lead to nontraumatic anoxic brain injury.[5]

Acquiring an initial Glasgow Coma Scale (GCS) score will help guide further treatment. It is important to repeat the GCS frequently because mental status can decline in a short period of time, necessitating rapid interventions, such as intubation and transfer to a higher level of care.[5]

Moderate to severe cases of TBI may require intubation and mechanical ventilation for airway protection. Many patients with TBI present with polytrauma. Although other injuries may require emergent attention, the TBI workup should include a prompt complete neurologic examination as well as a computed tomographic (CT) head scan without contrast to evaluate bleeding and CT scan of the cervical spine to evaluate fracture.[5]

Head CT scans are the first-line imaging assessment to reveal an intracranial pathologic condition, which may require surgical intervention. Common intracranial findings include depressed skull fracture, subdural hematoma, epidural hematoma, subarachnoid hemorrhage, and intraventricular hemorrhage. Neurosurgery should be consulted for an intracranial pathologic condition because patients may develop increased pressure inside the brain, causing herniation and death.[5]

A craniotomy is a decompressive surgery in which the skull is replaced immediately. In contrast, a craniectomy is a decompressive surgery in which the bone flap is removed and left off until swelling resolves in the brain and it can be replaced at a later time (replacing the bone is called a cranioplasty). Patients who have a bone flap removed should wear a custom-fitted helmet when upright or out of bed.[5]

Surgeons may place an intracranial pressure (ICP) monitoring device. Examples of this include the extraventricular drain, intraparenchymal catheter, or combination catheter, which allows real-time information about ICP. Neuro–intensive care unit providers often consider additional medical or surgical management once ICP is 20 to 22 mm Hg (normal ICP is 10–15 mm Hg). Practice guidelines exist for ICP management and may involve changing the elevation of the bed, draining CSF, hyperosmolar fluids like mannitol or hypertonic saline (3%), hyperventilation, barbiturate coma, or return to the operating room for further decompression.[5]

Other metrics, such as cerebral perfusion pressure and brain oxygenation, are important in the acute hospitalization phase. Electroencephalogram (EEG) may also be performed to assess sleep/wake cycles and evaluate possible seizure activity.[5]

Although this article focuses on moderate and severe injuries, not all patients who sustain a TBI require advanced head imaging or hospitalization. mTBIs (GCS 13–15) and concussions constitute the vast majority of all TBI cases (80%–90%). The Canadian Computed Tomography Head Rule for Minor Head Injury was developed to help guide clinicians regarding appropriateness of imaging for mTBI and sports-related concussion (**Box 6**).[10]

Post-traumatic Amnesia

Posttraumatic amnesia (PTA) is the period of time between a TBI event and recovery of active memory. Patients in PTA may be ambulating and carrying conversation, but they will be unable to recall details from the conversation soon after. This interval of "lost memory" is one of the most common metrics used to determine severity of injury and prognosis for recovery. PTA is described as retrograde or anterograde. Retrograde amnesia means an inability to retrieve past memories. Anterograde amnesia means an inability to make new memories. For patients with TBI, severe disability is unlikely if PTA lasts less

Box 6
Canadian computed tomography head rule for minor head injury

High risk (for neurologic intervention)
- Failure to reach 15 on the Glasgow Coma Scale within 2 hours
- Suspected open skull fracture
- Any sign of basal skull fracture
- Two or more vomiting episodes
- 65 years or older

Medium risk (for brain injury on CT scan)
- Retrograde amnesia (before impact) more than 30 minutes
- Dangerous mechanism of injury

From Magee DJ. Head and Face. In: Magee DJ, editor. Orthopedic Physical Assessment, 6th edition. St Louis: Saunders; 2014; with permission.

than 2 months. Conversely, functional recovery is unlikely if PTA persists past 3 months.[4]

Several validated tools are available to assess emergence from PTA. These validated tools include the Galveston Orientation and Amnesia Test, the Children's Orientation and Amnesia Test, and The Orientation Log.[2,5,11,12]

MEDICAL MANAGEMENT

Additional medical complications of TBI not discussed in detail within this article include cranial nerve damage, visual disturbances, spatial neglect, balance disorders, and movement disorders.[5]

Posttraumatic Seizures

The International League Against Epilepsy defines seizures as "a transient occurrence of signs and/or symptoms due to abnormal excessive or synchronous neuronal activity in the brain."[13] Posttraumatic seizures are a possible complication of TBI. Seizures following TBI are classified as immediate, early, or late. Focal seizures, formerly known as simple partial (pure motor) seizures, are the most common type to occur late in this population, although others (complex partial, generalized, and so forth) are documented.[7,13] More than 80% of patients who develop seizure activity will present in the first 2 years after injury. Currently, there is no evidence to suggest prolonged prophylaxis with antiepileptic drugs (AEDs) will prevent late seizures.[7] There is evidence that an initial AED loading dose followed by 7 days of AED therapy after an injury can help prevent early seizure. Patients who have documented early seizure activity often will remain on AED therapy for longer than 7 days per Neurosurgery or Neurology recommendations. Although AED therapy may be necessary, sedation is a common side effect that can negatively impact cognition (**Tables 7** and **8**).[5]

Table 7
Posttraumatic seizure classification

Immediate	Early	Late
<24 h	24 h to 7 d	>7 d

Data from Elovic E, Baerga E, Galang GF, et al. Traumatic Brain Injury. In: Cuccurullo SJ, editor. Physical Medicine and Rehabilitation Board Review, 2nd edition. New York: Demos Medical Publishing; 2010.

Table 8
Incidence of posttraumatic seizure by severity

Mild	Moderate	Severe
1.5%	2.9%	17%

Data from Elovic E, Baerga E, Galang GF, et al. Traumatic Brain Injury. In: Cuccurullo SJ, editor. Physical Medicine and Rehabilitation Board Review, 2nd edition. New York: Demos Medical Publishing; 2010.

Posttraumatic Neuroendocrine Disorders

The pituitary gland is sensitive to acceleration and deceleration injuries. Mechanisms of damage include mechanical owing to sella turcica location, interruption of fragile vascular supply, and systemic stress response.[8]

Although neuroendocrine abnormalities are rare in mTBI, hypopituitarism after severe TBI has a prevalence of 50% to 80%. In the acute postinjury phase, hyperprolactinemia was the highest reported complication followed by diabetes insipidus (DI), syndrome of inappropriate adrenocorticotropic hormone (SIADH), human growth hormone (HGH) adrenocorticotropic hormone deficiency (ACTH) deficiency. Addressing these issues can assist both cognitive and physical recovery.[5]

In the absence of clinical suspicion, current guidelines do not suggest routine monitoring of GH, ACTH, thyrotropin, and gonadal axes in the acute phase. It may be helpful to evaluate these hormones in patients with DOC because treatable neuroendocrine disorders may impede emerging consciousness.[5,8]

Posttraumatic Hydrocephalus

Posttraumatic hydrocephalus (PTH) is important to identify because treatment can affect functional outcome. Ventricle enlargement owing to brain tissue atrophy or ex vacuo dilation after craniectomy makes PTH difficult to distinguish on imaging. Considering clinical signs and symptoms is helpful in conjunction with imaging. Treatment will most often involve ventriculoperitoneal shunt placement (**Table 9**).[5]

Table 9
Posttraumatic hydrocephalus

- Risk factors: Intracranial bleeding, meningitis, postsurgical decompressive craniectomy, coma duration, advanced patient age
- Early shunting predicts better outcomes

Communicating Ventricular system is connected	Noncommunicating CSF flow is blocked
CSF flows from ventricles to subarachnoid space This is the most common type seen in TBI Blood products/dead tissue can block flow Often results in normal pressure hydrocephalus Signs/symptoms: 1. Lack of progress/plateau in therapy sessions 2. Ataxia 3. Urinary incontinence 4. Poor initiation 5. Decreased attention 6. Forgetfulness	Signs symptoms: Most are associated with increased ICP 1. Nausea 2. Vomiting 3. Lethargy 4. Headaches 5. Papilledema 6. Gait disturbances

Data from Greenwald BD, Hampton S, Jasey N, et al. Neurologic Complications After Traumatic Brain Injury. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018; and Wagner AK, Arenth PM, Kwasnica C, et al. Traumatic Brain Injury. In: Cifu DX, editor. Braddom's Physical Medicine and Rehabilitation, 5th edition. Philadelphia: Elsevier; 2016.

Post-traumatic Agitation

Posttraumatic agitation is defined by Braddom's *Physical Medicine and Rehabilitation* as "an excess of one or more behaviors that occurs during an altered state of consciousness." The altered state of consciousness this text alludes to is known as PTA, a common time period during which patients with TBI are unable to form new memories.[2]

Agitation is common in the acute phase after TBI (35%–96% of cases), but can persist long term.[8] Posttraumatic agitation is described as a subset of delirium. Behavioral issues are a major source of morbidity in patients with TBI and cause barriers to reintegration into the home and community. Agitation encompasses behaviors such as irritability, anger, and aggression (verbal or physical). It is important to identify specific behaviors to create a targeted treatment plan.[8]

Common and modifiable risk factors for agitation include an overstimulating environment or unpleasant experience, pain, infection, disrupted sleep patterns, and frontal lobe damage.[5,8]

How to Measure Agitation

Several objective scales exist to track a patient's agitated behaviors. These scales are meant to standardize the way health care providers describe agitation in an effort to provide targeted, successful interventions.

Rancho Los Amigos Scale–Revised

This scale is named for the location where it was developed, an inpatient rehabilitation center in Downey, California. The Rancho scale is meant to describe the typical process of emergence from a coma for patients with severe TBI. This scale is especially helpful for families trying to understand a loved one's recovery (**Table 10**).[14]

Table 10
Rancho Los Amigos Scale–revised

Level	Clinical Examination	Functional Independence Measure
I	No response	Total assist
II	Generalized response	Total assist
III	Localized response	Total assist
IV	Confused and agitated	Maximal assist
V	Confused and inappropriate, nonagitated	Maximal assist
VI	Confused and appropriate	Moderate assist
VII	Automatic and appropriate	Minimal assist
VIII	Purposeful, appropriate	Stand-by assist
IX	Purposeful, appropriate	Stand-by assist
X	Purposeful, appropriate	Modified independence

Adapted from Centre for Neuro Skills (CNS). Rancho Los Amigos Scale – Revised. Available at: https://www.neuroskills.com/education-and-resources/rancho-los-amigos-revised/. Accessed Oct 18 2019; with permission.

Common Neuropsychiatric Disorders Following Traumatic Brain Injury

General rules to successfully interact with a patient after acute TBI (**Table 11**) include[2] the following:

1. *Patients may be easily confused and have impaired memory:* Reorient the patient frequently. Use simple language. When providing an explanation, try to avoid giving

Table 11
Neuropsychiatric disorders: common chronic complications associated with traumatic brain injury

Major depression	• Most common mood disorder associated with TBI • Prevalence range 6% to 90% • Premorbid depression associated with depression postinjury
Anxiety	• Second most common disorder behind depression • Associated with cognitive fatigue • Selective serotonin reuptake inhibitors (SSRIs) can be effective in patients with TBI
Posttraumatic stress disorder	• Increased severity of injury may be protective against this • Patients with TBI do not need to recall the event to develop this condition
Psychosis	• Ensure not caused by medication side effects (amantadine) • Can be well managed with atypical antipsychotics • Avoid typical antipsychotics caused by dopamine-depleting properties
Paranoia	• Associated with PTA • Commonly persists long term after TBI • Reports of success with atypical antipsychotics
Pseudobulbar affect	• Characterized by inappropriate emotional response, such as random outbursts of laughing/crying • Treatment options include dextromethorphan HBr and quinidine sulfate, SSRIs, tricyclic antidepressants (TCAs)
Aggression	• Associated with emergence from PTA but can present at any time after TBI • Multimodal management with behavioral, environmental, and medication strategies • Beta-blockers, mood stabilizers, SSRIs, and atypical antipsychotics are medications most often used

Data from Ripley DL, Driver S, Stork R, et al. Pharmacologic Management of the Patient With Traumatic Brain Injury. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018.

too much information all at once (1 concept at a time). Allow extra time for explanations. Patients may be disinhibited and come across as rude; do not take this behavior personally.[2]

2. *Overstimulation can lead to irritable or agitated behavior; instead, provide a low stimulation environment:* Limit distractions and noise in the room (ie, turn off the radio or television, dim the lights, limit the number of people in the room to one or two, only allow 1 person to talk at a time). When giving instructions, provide simple step-by-step directions; depending on the patient's cognitive function, it may be appropriate to provide a written copy. Redirect patients when they become frustrated with an activity; allow rest breaks as needed. Consider that a patient may be experiencing pain or fatigue and may have trouble expressing this. Consider addressing these underlying issues to help improve their behavior.[2]
3. *Patients with TBI may be impulsive and lack safety awareness:* When addressing a patient, get their attention before you speak. Approach a patient slowly and always from the front, not from behind. Speak slowly, clearly, and softly. Use the patient's name frequently in conversation. Use demonstration, not just verbal instruction, and provide written material if relevant.[2]

First-Line Treatment for an Agitated Patient: Behavioral Interventions

Because of impaired cognition and communication, patients with TBI often have a hard time expressing an underlying problem, which can then manifest as agitated behavior. Consider possible medical reasons for agitation. Common medical triggers for agitation include urinary tract infection, respiratory infection, constipation, urinary retention, pain, and dehydration. It is reasonable to consider a basic workup for infection, including a urinalysis, a metabolic panel, and a complete blood count, to assess electrolytes and inflammatory markers. Occasionally, patients experience posttraumatic hydrocephalus or shunt malfunction if a shunt was previously placed, and obtaining a CT head scan without contrast may be reasonable depending on the case and presentation.[2]

Once medical causes for agitation are ruled out or treated, nonpharmacologic interventions are considered first line in behavioral management. Address overstimulation as described earlier, and providing a low stimulation environment is often a key for an agitated patient. Ensure the patient is protected from harming themselves and other people. Tolerate restlessness when possible because physically restraining a patient often leads to more anxiety and agitated behaviors. Interventions, such as using a floor bed with side panels, having a 1:1 sitter, video monitoring, and use of a locked ward, may be helpful. Restraints should only be used when absolutely necessary. If needed, consider using unrestrained hand mittens to prevent pulling at lines/tubes rather than wrist and ankle restraints. If able, keep health care providers consistent so the patient is able to see familiar faces daily.[2]

Pharmacologic Agitation Management

There is little evidence to support or refute use of medications for agitation in TBI based on the current literature (**Tables 12** and **13**).[15] The following is a list of mood stabilizers commonly used for agitation management.

Table 12
Pharmacologic management of agitation

Medication Class	Examples
Nonspecific beta-blockers	Propranolol, pindolol, metoprolol
Neuro stimulants	Methylphenidate, modafinil, amantadine, donepezil
Antipsychotics	Olanzapine, quetiapine, risperidone
SSRIs	Fluoxetine, sertraline
Anticonvulsants	Valproic acid, carbamazepine
TCAs	Amitriptyline, nortriptyline, desipramine
Benzodiazepines	Diazepam, lorazepam
Norepinephrine and dopamine reuptake inhibitors	Buspirone
Alpha-2-agonist	Clonidine
Opiates[a]	Morphine, oxycodone, and similar

[a] If pain is triggering agitation.

Data from Ripley DL, Driver S, Stork R, et al. Pharmacologic Management of the Patient With Traumatic Brain Injury. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018.

Table 13
Dopamine-promoting agents may assist in cognitive recovery after severe traumatic brain injury

Medication	Mechanism of Action	Common Side Effects
Methylphenidate	Amphetamine	Tachycardia
Amantadine	Dopamine agonist	Lowers seizure threshold Orthostatic hypotension Visual hallucinations
Levodopa/Carbidopa	Dopamine agonist	Orthostatic hypotension, dizziness, nausea, Headache (HA)
Bromocriptine	Dopamine agonist	Nausea, HA, Dizziness
Donepezil (adults)	Acetylcholinesterase inhibitor	Insomnia

Data from Neurobehavioral Guidelines Working Group, Warden DL, Gordon B, et al. Guidelines for the Pharmacologic Treatment of Neurobehavioral Sequelae of Traumatic Brain Injury. J Neurotrauma 2006;23(10):1468-1501.

Carbamazepine

Mechanism of Action: Activates K+ channels, Na channel blocker, regulates limbic kindling
Common side effects: Aplastic anemia, hyponatremia, ataxia, nausea, agranulocytosis, sedation, toxic epidermal necrolysis[5,16]
Food and Drug Administration (FDA) indications: Trigeminal neuralgia, seizures

Lamotrigine

Mechanism of Action: Glutamate antagonist, inhibits Na channels
Common side effects: Headache, dizziness, diplopia, toxic epidermal necrolysis, fatigue

Valproic acid

Mechanism of Action: Delays repolarization of Na channels, increases GABA activity, controls limbic kindling, NMDA antagonist
Common side effects: Hepatotoxicity, somnolence, thrombocytopenia, weight gain
FDA indications: Neuropathic pain, alcohol withdrawal
In a randomized controlled trial, this drug did not demonstrate damage to cognition.

What to avoid and why

When treating a patient with TBI with behavioral disorders, avoid dopamine antagonist medications when able. Current literature suggests these medications may prolong PTA and inhibit cognitive recovery. Haloperidol, a commonly used medication for agitation, is a dopamine antagonist. Alternatives, including olanzapine and risperidone, likely have a similar risk, and there are studies with conflicting results, although data are difficult to interpret because of small population sizes. Medications commonly used to treat behavioral disorders after TBI can be associated with side effects (**Table 14**).

Table 14
Common adverse effects reported in patients with traumatic brain injury with standard pharmacologic interventions

Beta-Blockers	Hypotension, bradycardia, fatigue
TCAs	Seizures
Clozapine	Weight gain, drooling, seizures
Desipramine	Mania
Fluoxetine	Dysarthria, aphasia
Sertraline	Akathisia
Paroxetine	Akathisia
Lithium	Cognitive impairments, narrow therapeutic index, neurotoxicity

Although these medications can help in certain instances, impaired cognition may be a side effect due to the ability to cross the blood brain barrier resulting in sedation.[5]

Data from Neurobehavioral Guidelines Working Group, Warden DL, Gordon B, et al. Guidelines for the Pharmacologic Treatment of Neurobehavioral Sequelae of Traumatic Brain Injury. J Neurotrauma 2006;23(10):1468-1501.

Agitated Behavior Scale

This instrument was published in 1995. The goal of the Agitated Behavior Scale is to assess patients with agitation after an acute acquired brain injury, such as TBI. The tool is meant to allow frequent documentation for behaviors to help providers identify a trigger or temporal relationship to agitation. The Overt Agitation Severity Scale (not specific to TBI) and the Neurobehavioral Rating Scale (monitors agitation and PTA) are 2 additional tools developed more recently.[2]

Paroxysmal Sympathetic Hyperactivity ("Storming")

After a brain injury, patients may experience paroxysmal sympathetic hyperactivity. Common synonyms used include paroxysmal autonomic instability and dystonia syndrome and "storming." A hyperadrenergic state leading to posttraumatic agitation is the underlying theory behind this process. Storming features are seen in multiple types of acquired brain injuries, including traumatic, anoxic, and stroke,[17,18] and include the following:

- Occurs in 15% to 33% of patients with severe TBI (GCS <8)
- Typical time course: 24 hours to weeks after injury
- Signs: Tachycardia, hyperthermia, dystonia, posturing, diaphoresis, hypertension, pupillary dilatation, tachypnea
- Common mimics: Neuroleptic malignant syndrome, serotonin syndrome, sepsis
- Common triggers: Infection, pain, overstimulation, constipation, urinary retention, insomnia
- Nonpharmacologic treatments: Repositioning, massage, cool cloths, soothing music
- Pharmacologic treatments: Propranolol, clonidine, bromocriptine, gabapentin

Spasticity

Spasticity is defined as a velocity-dependent, involuntary resistance to a passive stretch. It is a known complication of moderate to severe TBI. The mechanism is attributed to central injuries, which cause loss of inhibitory signals, impairing the normal stretch reflex. The result is an increase in muscle tone, which can be painful and impaired motor function and range of motion. However, tone may be used functionally

to help patients transfer or walk, in some cases; this principle becomes important when developing a treatment plan. Spasticity is not isolated to TBI and is seen in several neurologic disorders.[5]

Spasticity management requires a multimodal approach. Initial management includes range-of-motion exercises, stretching, splinting, and bracing. Oral medications, focal injections, and intrathecal baclofen pump placement are other options. Although oral medications are often trialed first for spasticity, nearly all are sedating, which can impair cognition and overall function. Focal injections with botulinum toxin or phenol may be performed for targeted areas, depending on functional goals. Tone can impair ambulation as well as the ability to dress, transfer, and perform hygiene.[5]

Spasticity can be a symptom of an underlying noxious stimulus (such as a full bladder, constipation, infection, or a pressure wound). If spasticity becomes markedly worse without explanation, it is reasonable to evaluate the patient for an underlying trigger (**Table 15**).

Table 15
Pharmacology of spasticity management

Commonly Used Medications for Spasticity Management		
Medication	**Mechanism of Action**	**Side Effects and Special Considerations**
Baclofen (oral)	Centrally acting GABA analogue that binds to GABA-B receptors to inhibit muscle stretch reflex and decrease motor neuron activity at the spinal cord level	Somnolence, fatigue, muscle weakness, xerostomia, urinary retention, constipation, elevated liver function tests (LFTs) Abrupt cessation is associated with withdrawal, including altered mental status, hallucinations, seizures, increased muscle tone, and spasms
Baclofen (intrathecal)	Same as above	Reduced systemic side effects when compared with oral because intrathecal delivery allows for higher concentration at a lower dose Withdrawal symptoms are related to a malfunction with the baclofen pump device or damage to the catheter
Tizanidine	Alpha-2 agonist that inhibits the release of excitatory neurotransmitters (glutamate, aspartate) from spinal interneurons	Somnolence, dizziness, hypotension, xerostomia, elevated LFTs
Dantrolene	Inhibits the release of calcium from the sarcoplasmic reticulum of muscle, interfering with skeletal muscle contraction	Muscle weakness, drowsiness, diarrhea, hepatotoxicity; often preferred for TBI-induced spasticity because it acts peripherally
Gabapentin	GABA analogue, although mechanism of action is not well understood	Drowsiness, dizziness, edema

(*continued on next page*)

Table 15
(*continued*)

Commonly Used Medications for Spasticity Management

Medication	Mechanism of Action	Side Effects and Special Considerations
Diazepam	Binds to GABA-A receptor, facilitating chloride influx and inducing neuronal inhibition	Sedation, cognitive impairments; abrupt cessation can lead to withdrawal symptoms
Clonidine	Centrally acting alpha-2 agonist that decreases sympathetic outflow	Hypotension, rebound hypertension, bradycardia, xerostomia, drowsiness, constipation, depression
Botulinum toxin	Inhibits presynaptic acetylcholine release by cleaving the SNAP-25 protein in the SNARE complex	Weakness, fatigue, flulike symptoms, dysphagia, complications associated with the procedure, such as infection, bleeding, and pain; short-term effect (3–6 mo)
Phenol	Neurotoxin that denatures proteins in the area surrounding the injection site	Dysesthesias, hypotension, prolonged pain, complications associated with procedure, such as infection, bleeding, and pain Longer lasting than botulinum toxin (6 mo to 1 y)

Adapted from Eapen BC, Hong S, Subbarao B, et al. Medical Complications After Moderate to Severe Traumatic Brain Injury. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018; with permission.

Heterotopic ossification

Heterotopic ossification (HO) is a phenomenon seen in several neurologic and orthopedic injuries. Lamellar bone forms in soft tissue and can severely limit a joint's range of motion and function. The mechanism is poorly understood. The frequency of occurrence has a wide range with reports as low as 4% and as high as 23%. In patients with TBI, the hips, knees, and shoulders are the most common areas of HO formation. HO is challenging to detect. The presentation is often similar to a bone fracture, septic joint, cellulitis, or deep venous thrombosis (DVT). Laboratory workup with inflammatory markers is nonspecific. Diagnostic tools include bone scans and ultrasound, although these are also nonspecific. Radiographs will not show HO until it is advanced, making treatment difficult. Treatment options include nonsteroidal anti-inflammatory drugs, bisphosphonates, and radiation therapy in the early stages and surgical resection in the later stages. Despite treatment, HO often recurs.[5]

Hypercoagulability

Patients with moderate to severe TBI requiring hospitalization and subsequent immobilization are at increased risk for DVT and pulmonary embolism. In the absence of any prevention measures, 20% to 25% of patients with TBI develop a DVT. However, many hospitalized patients with TBI have evidence of recent intracranial bleeding, and thus, the benefit of chemoprophylaxis must be weighed against the possibility of bleeding. No clear guidelines exist to determine timing of starting chemoprophylaxis.[5]

Malnutrition

Patients with TBI, particularly those with severe injuries, have increased caloric needs following injury. These metabolic changes are attributed to an inflammatory state.

Although TBI is associated with a 75% to 200% increase in energy expenditure, the use of sedatives and presence of disorders of consciousness (DOC) decrease metabolism. Because many hospitalized patients with TBI are unable to eat orally, it is important to start enteral feeding as soon as possible with a focus on delivering a high-protein diet (2–2.5 g/kg/d). There is evidence to suggest early feeding (within 48 hours) can decrease neuroendocrine complications.[5]

CONCUSSION

Concussion has been used synonymously with the term mTBI. Emerging classification mechanisms specific to concussion promote separation of these terms. Mild injuries likely account for more than 80% of all TBI, although the true incidence is difficult to ascertain because many patients who sustain these injuries do not seek medical attention, and therefore, they are not documented or tracked. Most research and subsequent assessment tools are focused on sports-related concussion.[19]

The 2017 fifth edition of the Standardized Concussion Assessment Tool is validated for patients 13 years and older; a child version exists for younger patients. This tool is a comprehensive evaluation and includes orientation questions, a GCS, a neurologic screen, a cervical spine test, a cognitive screen, balance testing, and a symptom checklist. Several other postconcussion patient self-assessment tools are available for patients experiencing persistent symptoms.[19]

When evaluating a patient after a concussion, consider these important red-flag features, which may warrant further workup and imaging. Red-flag signs include loss of consciousness greater than 30 seconds, posttraumatic amnesia greater than 30 minutes, seizure activity, vomiting, severe headache, focal neurologic findings, and limited or painful neck range of motion.

The vast majority of patients with mTBI will recover within 1 to 2 weeks. Current literature suggests up to 15% of patients will experience persistent postconcussive symptoms, although the term "postconcussion syndrome" is typically reserved for patients with multiple symptom complaints that persist for many months to years after their injury.[20] Although the mechanism for development of postconcussion syndrome is not well understood, several factors, including social, biological, and psychological, likely play a role. Linking symptoms directly to mTBI can be challenging because the associated symptoms are common in the general population. Postconcussion headache is the most common symptom. Other symptoms include sleep dysfunction, cognitive dysfunction, vestibular disorders, visual/spatial dysfunction, irritability, and emotional lability.[19,20]

DISORDERS OF CONSCIOUSNESS (DISTURBANCES OF CONSCIOUSNESS)

Consciousness is a function of the ascending reticular activating system and the cerebral cortex. The term DOC describes a state of prolonged altered consciousness, which is categorized into coma, vegetative state (VS), and minimally conscious state (MCS) depending on the presence of arousal and awareness of self and environment (**Table 16**).[21]

Table 16
Disorders of consciousness

	Coma	VS (Unresponsive Wakefulness Syndrome)	MCS
Arousal	Absent	Present	Present
Awareness	Absent	Absent	Present

Data from Giacino JT, Fins JJ, Laureys S, et al. Disorders of consciousness after acquired brain injury: the state of the science. Nat Rev Neurol 2014;10(2):99-114.

A coma is a state of unconsciousness with no evidence of arousal and awareness. There is no eye-opening or sleep-wake cycle on EEG. Those who survive this state will transition to either VS or minimally conscious state (MCS) within 2 to 4 weeks.

A VS, also known as unresponsive wakefulness syndrome, reflects the dissociation between wakefulness and awareness. In a VS, sleep-wake cycles are evident on EEG. Patients may arouse with external stimuli as demonstrated by intermittent eye opening, but they do not show signs of perception or purposeful movement. Patients in VS may show stereotyped gestural movements, such as yawning, chewing, auditory/visual startle, vocalization, crying, smiling, and moaning without contingence, but they do not indicate the presence of awareness.[22]

An MCS is characterized by a severe impairment of consciousness with evidence of wakefulness and preservation of awareness. Awareness refers to the ability of an individual to respond to both external and internal stimuli. These patients can demonstrate inconsistent, but reproducible command following, nonreflexive movement, object manipulation, localization of pain, visual pursuit, verbalization, contingent affective response, and so forth.[5]

One is considered to have emerged from MCS (eMCS) once he or she performs *functional* object use (eg, bringing a cup to his or her mouth) or *functional interactive* communication (eg, accurate response to yes or no questions).[22]

Assessment and Diagnosis

Correctly determining the level of consciousness for DOC patients is often challenging, especially when confounding factors, such as sensory, motor, and cognitive impairments that mimic DOC, are present (**Box 7**). Also, it is important to address and treat reversible causes for impaired consciousness, such as sedating medications, concurrent medical issues, and unrecognized intracranial abnormalities before the assessment (**Box 8**). Making a correct diagnosis is important for several reasons. Access to specialized rehabilitation services is much more limited for individuals thought to be in VS than for someone in MCS. Rehabilitation goals also differ based on the perceived level of consciousness (eg, enhancing arousal for VS vs establishing a communication system for MCS).

Box 7
Examples of mimics and confounding factors of disorders of consciousness

Mimics
- Locked-in syndrome
- Catatonia
- Akinetic mutism

Confounding factors
- Widespread paresis or paralysis (eg, critical illness myopathy, critical illness neuropathy)
- Profound sensory deficits (eg, blindness, deafness)
- Bilateral cranial nerve III palsy
- Diffuse spasticity and contracture
- High-order cognitive deficits (eg, aphasia, apraxia)

Adapted from Kothari S, Gilbert-Baffoe E, O'Brien KA. Disorders of Consciousness. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018; with permission.

Box 8
Reversible causes of impaired consciousness

- Seizures (eg, subclinical seizure, nonconvulsive status epilepticus)
- Neuroendocrine abnormalities (eg, growth hormone deficiency, thyroid function abnormalities)
- Infection (eg, urinary tract infection, pneumonia, meningitis)
- Metabolic abnormalities (eg, hyponatremia, hypoglycemia)
- Intracranial abnormalities (eg, hydrocephalus, progressive intracranial bleed)
- Sedating medication (eg, anticholinergic, GABAergic, antidopaminergic)
- Disrupted sleep-wake cycle

Adapted from Kothari S, Gilbert-Baffoe E, O'Brien KA. Disorders of Consciousness. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018; with permission.

Although bedside evaluation can help diagnose a patient's level of consciousness, these subjective evaluations need to be supplemented by formal assessments because observed responses are often subtle. One study discovered 41% of patients diagnosed with VS based on the clinical consensus were actually in an MCS following standardized behavioral assessment (see later discussion).[23]

Clinicians should keep in mind that the level of consciousness may continue to fluctuate during recovery and can yield inconsistent behaviors. For that reason, formal evaluations should be performed multiple times with different modes of assessment by multiple examiners at various times of day under optimal environmental conditions.[5]

In general, behavioral assessments are considered the "gold standard" for determining the presence and level of consciousness. Specialists use the Coma Recovery Scale Revised and/or the Individualized Quantitative Behavioral Assessment, which are comprehensive scales useful in detecting subtle presence or changes in consciousness. Nonbehavioral assessments are performed using various diagnostic tools, such as pupillometry, surface electromyography, functional MRI scan, and transcranial magnetic stimulation coupled with EEG, to detect consciousness that is not behaviorally evident. It is important to note that there is a high rate of false negative results with nonbehavioral assessments. Negative responses should not be used to exclude the possible presence of consciousness.[5]

Treatment

Once reversible causes and confounding factors are identified and addressed, interventions to enhance the level of consciousness should be considered. Consciousness assessments should continue during this phase as patients may transition to different states (eg, VS to MCS, MCS to eMCS). Treatment modalities can be divided into pharmacologic and nonpharmacologic categories as listed in **Box 9**.[5]

Applied energy therapy modalities, such as transcranial direct current stimulation (tDCS), repetitive transcranial magnetic stimulation (rTMS), deep brain stimulation (DBS), and right median nerve stimulation, are currently being investigated. In order

Box 9
Interventions to enhance the level of consciousness

Pharmacologic Treatment	Nonpharmacologic Treatment
Hypoarousal • Amantadine • Bromocriptine • Modafinil • Levodopa • Zolpidem Attention/processing speed • Methylphenidate • Dextroamphetamine Memory • Donepezil • Memantine	Mobilization • Sitting program • Standing program • Body-weight-supported therapeutic gait Sensory stimulation • Tactile, auditory, visual, vestibular, and Interpersonal interaction Applied energy therapy • tDCS • *r*TMS • DBS • Right median nerve stimulation

Modified from Kothari S, Gilbert-Baffoe E, O'Brien KA. Disorders of Consciousness. In: Eapen BC, Cifu DX, editors. Rehabilitation after Traumatic Brain Injury. St. Louis: Elsevier; 2018; with permission.

to provide accurate diagnostic evaluation, prognostication, and subsequent management, the new American Academy of Neurology guidelines recommend that clinicians should refer DOC patients to multidisciplinary rehabilitation teams whereby the patients will be treated with the goals listed in **Box 10**.[24]

Box 10
Goals of a disorders-of-consciousness program

- Assess current level of consciousness
- Address reversible causes of impaired consciousness
- Initiate interventions to enhance consciousness
- Establish a system of communication
- Identify and magnify residual voluntary movement
- Address restrictions in range of motion
- Intensive mobilization and environmental enrichment
- Prevent and manage secondary medical complications
- Optimize respiration/nutrition/elimination/integument
- Provide family education/training/support
- Establish a plan for aftercare

From Kothari S, Gilbert-Baffoe E, O'Brien KA. Disorders of Consciousness. In: Eapen BC, Cifu DX, editors. Rehabilitation After Traumatic Brain Injury. St. Louis: Elsevier; 2018; with permission.

Prognosis and Outcomes in Disorders of Consciousness

In general, patients in MCS have a more favorable prognosis than those in VS.[25] Within the VS group, traumatic cause carries a better prognosis than nontraumatic cause.[26]

Recent studies show long-term recovery is possible beyond 1 year after injury. One study found that approximately 20% of patients in traumatic VS admitted to a

comprehensive inpatient rehabilitation program were functionally independent and capable of returning to employment at 1, 2, or 5 years.[27] Another study noted 88% to 100% of people who regained command-following within 28 days after injury and 50% to 75% of patients who did not were independent on the cognitive, mobility, and self-care functional independence measure scores by 10 years.[28] These findings suggest individuals with DOC may continue to benefit from ongoing functional monitoring and care plans for years after injury.

Ethical Considerations in Disorders of Consciousness

In the United States, individuals on mechanical ventilation with a GCS score of 5 or less are considered for organ procurement.[29] In-hospital mortality for patients with severe TBI (including DOC) is as high as 32%, with 70% of those deaths associated with withdrawal of life-sustaining therapy.[30] Given the diagnostic and prognostic uncertainty in DOC, health care providers may be at risk of starting discussions about limiting or withdrawing medical treatments prematurely while the patients may have the potential to recover, or have already recovered, consciousness. Another common ethical issue arises from patients' limited ability to consent for medical treatment, procedures, and research. In any ethically complicated situations, consulting an ethics committee is recommended.

SOCIAL CONSIDERATIONS

Disposition for Patients After Traumatic Brain Injury

Several options exist for rehabilitation services following acute hospitalization. The level of rehabilitation depends on several factors, including the patients' functional deficits, rehabilitation goals, and therapy participation. Current literature demonstrates improved outcomes for patients who complete an intensive rehabilitation program after TBI. High-quality evidence suggests patients with severe TBI who complete intensive inpatient rehabilitation in an inpatient rehabilitation facility (IRF) demonstrate earlier gains in independence, reduced length of acute hospital stay, and substantial cost savings.[5,8]

IRFs offer a resource-intensive program completed in a hospital setting. Patients must have nursing, medical, and rehabilitation needs that necessitate this level of care. They must require at least 2 therapy subspecialties (between physical therapy, occupational therapy, and speech therapy). In addition, they should be willing and able to participate in 3 or more hours of therapy per day, 5 out of 7 days per week. Physiatrists are generally the primary providers for patients at IRFs. Physiatrists manage rehabilitation-related medical needs, evaluate splinting and bracing needs, and oversee therapy services and durable medical equipment ordering with the goal of maximizing patient function. Interdisciplinary rounds are held once per week on each patient involving the physical, occupational, and speech therapists, physicians, nurses, and other staff members, which may include a neuropsychologist, recreational therapist, dietician, social worker, and case manager. These rounds are tailored to addressing the patient's progress toward functional goals, assessing barriers, and determining an appropriate discharge plan.[31]

For medically complicated patients unable to tolerate 3 hours of therapy per day, a long-term acute care hospital (LTACH) may be appropriate. LTACH patients receive about 1 hour of therapy per day, 5 days per week, and are still seen daily by a physician.[5,31]

Subacute rehabilitation in a skilled nursing facility (SNF) may be an option for patients who do not qualify for acute inpatient rehabilitation at an IRF, but are unable

to discharge home. SNF patients are typically more medically stable than those in LTACHs. SNFs and LTACH facilities offer regular therapy services, generally including physical, occupational, and speech therapy. The rigor of therapy will vary, but patients typically receive 5 hours of therapy per week in these centers.[5,31]

If a patient is medically stable enough to discharge home, they have a capable caregiver, and/or they are physically able to navigate their home environment, outpatient therapy may be an option after hospital discharge. Outpatient therapy services in a clinic familiar with neurologic injuries can offer high-quality rehabilitation.[5,31]

Home health services are an option for home-bound medically stable patients not requiring acute or subacute level rehabilitation. Home health services may include a comprehensive home-safety evaluation as well as nursing care, physical, occupational and speech therapies.[5,8]

Primary Prevention

In the 1990s, the Federal Government noticed the rising incidence of TBI and TBI-related disability in the United States. Several programs were enacted through the CDC in an effort to track TBI outcomes, promote brain injury awareness, and identify ways to prevent TBI.[32] At that time, MVC was the number one mechanism of injury. A decrease in MVC as the mechanism of injury is attributed to improved motor vehicle safety features. Growing use of these features, including seatbelts, airbags, car seats, motorcycle helmets as well as improved driver's safety standards, are attributed to a decrease in MVC-related TBI. Although sports helmets do not appear to prevent concussion, they are attributed to preventing more severe injuries.[33] In addition, growing concussion awareness in sports may help identify TBIs earlier, and treating them appropriately may improve long-term morbidity and mortality.[32,33]

SUMMARY

TBI is a prevalent condition in the United States. This prevalence highlights the need for increased awareness of the unique characteristics of this population across medical specialties. Patients with TBI are often misunderstood and misdiagnosed, particularly in the DOC population whereby several confounding factors may be at play. Although PM&R brain injury specialists are important in addressing the sequelae of TBI, an interdisciplinary approach is essential to patients' success considering the medical, surgical, and psychological effects of this diagnosis.

DISCLOSURE

The authors have nothing to disclose.

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