Traumatic Brain Injury

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Epidemiology

Traumatic brain injury (TBI) is a major public health concern and is a leading cause of death from injury. While the exact number of individuals suffering is unknown, some studies estimate an incidence of 91–430 per 100,000 per population year [1]. In the United States (US), there are nearly 1.6 million identified head injuries per year and approximately 16 % of those are admitted to a hospital [2]. The US mortality rate is 50,000–60,000 per year and an estimated 80,000–90,000 people per year have long-term disability as a result [2–4]. The bimodal age of distribution peaks between ages 0–4 and 15–19 [3]. The younger ages of injury may reflect injury from child abuse, and the older a predilection toward increased risky behavior. After peaking in the young adult years, the incidence of TBI declines into mid-adulthood [5]. Common causes of TBI include falls, motor vehicle collisions, pedestrian injuries, and assaults [3]. When considering hospital costs, rehabilitation costs, and loss of productivity, TBI costs the US health-care system approximately $100 billion per year [2, 6].

Classification and Types

Neurologic Severity Score

TBI includes a spectrum of brain injuries that can be classified in two ways: (1) by severity and (2) by anatomical location. Glasgow Coma Scale (GCS) is used to grade severity despite its original intent of classification for nontraumatic injuries (Table 4.1). Minor injury is defined by a GCS score of 13–15. Moderate injury is defined by a score of 9–12 and severe injury by a score of 3–8 (Table 4.2). When using GCS as a classification schema, the motor score most accurately predicts ultimate neurologic outcome [5]. In general, mortality is rare in patients with mild TBI. Moderate TBI portends a slightly worse prognosis but with a mortality rate of still <10 %. In severe TBI; however, mortality rates can approach 40 %, and those that survive commonly have lasting deficits [7, 8].

Table 4.1 The Glasgow Coma Scale (GCS) scoring mechanism

Category	Score
Eye opening	
Spontaneous	4
To voice	3
To pain	2
None	1
Verbal response	
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Motor response	
Follows commands	6
Localizes to pain	5
Withdraws to pain	4
Decorticate/flexion movement to pain	3
Decerebrate/extension movement to pain	2
None	1

Table 4.2 Severity of traumatic brain injury (TBI) by the Glasgow Coma Scale (GCS)

Glasgow Coma Scale score	Traumatic brain injury severity
13–15	Mild
9–12	Moderate
3–8	Severe

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Anatomic Location

Anatomically, TBIs can be focal or diffuse. Focal injuries are classified by anatomic location of injury.

Skull Fractures

Skull fractures are either basilar or confined in the cranial vault. Basilar skull fractures add additional potential complications by communicating with other structures such as the middle ear, nasopharynx, or sinuses. They are also frequently associated with cranial nerve injuries.

All skull fractures are either open or closed, depending on any overlying penetration of the scalp. They are further categorized as either displaced or non-displaced (which is also referred to as depressed or non-depressed). Specific treatment depends on the anatomic location of the fracture and its characteristics that are beyond the scope of critical care.

Intracranial Lesions

Intracranial lesions are also subdivided into focal or diffuse in nature. They are generally caused by disruption of the vasculature which presents as various types of hematomas or parenchymal hemorrhages depending on location. These are commonly direct injuries to the brain and are thus considered primary injuries.

Focal Intracranial Lesions

Intraparenchymal Hemorrhage

Intraparenchymal hemorrhage (IPH) is seen in 20–35 % of severe TBIs and approximately 8.2 % of all TBIs [3, 4]. Initial identification of IPH is critical to recognize as these lesions frequently evolve with resulting increases in cerebral edema and potential for mass effect. Additionally, delayed IPH can occur in up to 20 % of TBI cases but usually within the first 3 days of initial injury [3]. For these reasons, repeat imaging during the first 24 h post injury is recommended (Fig. 4.1a) [4]. The presentation and patterns of IPH are similar to that of cerebral contusions, which can be considered a less severe type of IPH.

Subdural Hematoma

Subdural hematomas (SDH) occur in approximately 30 % of patients with TBI [4]. Shearing forces in the subdural space cause tearing of bridging veins resulting in accumulation of blood between the dura and arachnoid. Radiographically, they follow the contour of the brain parenchyma (in a classically described concave fashion) and can change in appearance over time (Fig. 4.1b). These are generally high force impact injuries, where direct brain and axonal injury can also occur, which can result in a worse prognosis or greater neurologic injury than in the other focal lesions [2]. They are subdivided into hyperacute (<6 h), acute (6 h to 3 days), subacute (3 days to 3 weeks), and chronic (3 weeks to 3 months) timepoints [3].

Epidural Hematoma

Occurring in approximately 0.5–1 % of all head traumas, epidural hematomas (EDH) have a propensity toward males, young adults, and those at the extremes of age, as the dura and inner table of the skull (where EDHs occur) are more fixed [3, 4]. EDHs are impact injuries commonly associated with lateral (temporal) skull fractures that result in tearing of the middle meningeal artery. Only about 10 % of these injuries are due to a venous injury [3]. The classic presentation includes a brief post injury loss of consciousness followed by a lucid interval before a progressive loss of mental status again. Early diagnosis, evaluation, and intervention are essential due to the potential for rapid deterioration and permanent brain injury. Overall mortality rate lies between 5 and 12 % when unilateral and 15–20 % with bilaterality [3, 9]. Imaging studies of EDHs appear as hyperdense lenticular (convex) lesions adjacent to the area of injury. Up to 10 % can appear in a delayed fashion radiographically (Fig. 4.1c) [3].

Subarachnoid Hemorrhage

Traumatic subarachnoid hemorrhage (SAH) is characterized by bleeding between the arachnoid membrane and pia matter. 33–39 % of patients with a head injury have a traumatic SAH on CT imaging (Fig. 4.1d). They usually occur adjacent to the site of injury or impact. They are generally caused by scraping of a vein against a tentorial edge [10]. SAH portends a significantly worse outcome [11, 12]. A large European study showed these patients to be older (mean 45.7 years) than those without subarachnoid hemorrhage (mean 37.6 years) with a worse GCS on admission [12].

Diffuse Intracranial Lesions

Diffuse Axonal Injury

Diffuse axonal injury (DAI) is generally found on the severe end of the TBI spectrum. DAI typically results from an axonal shearing injury or stretch injury following an acceleration or deceleration event. Direct axonal damage can be mild and reversible but is often more severe and permanent. DAI is often not visible on conventional CT scans, which can appear normal in 50–80 % of cases or just have a parenchymal hyper-density in 20–50 % of injuries. MRI is typically used to reveal the loss of gray/white differentiation predominately in the frontal lobes and corpus callosum [3]. Additionally, small petechial hemorrhages can also present where the gray and white matter differentiates. These hemorrhages and their resultant diffuse edema can create brainstem compression [6, 13]. The prognosis of DAI is very poor, with both a high mortality rate and a high incidence of residual neurologic deficits in survivors [5].

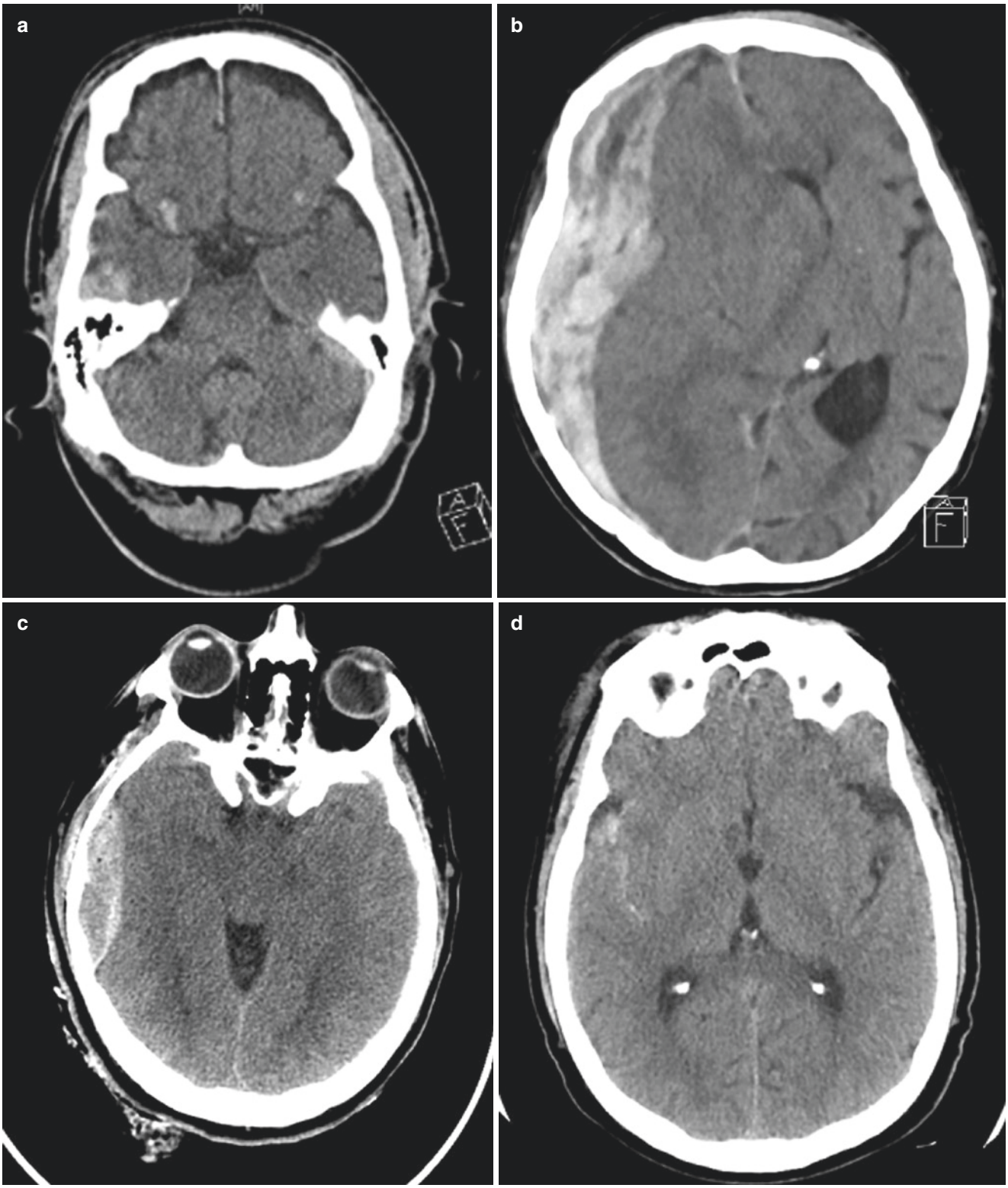

Fig. 4.1 (**a**) Intraparenchymal hemorrhage. (**b**) Subdural hematoma with midline shift. (**c**) Epidural hematoma. (**d**) Traumatic subarachnoid hemorrhage in the right sylvian fissure

Concussion

Concussions are defined as a transient event, often causing a brief loss of consciousness, with non-focal symptoms, and no permanent sequelae after an impact injury to the head. Signs and symptoms can be vague, often going unrecognized, and can include headaches, visual disturbances, dizziness, decreased attention/concentration, and amnesia [3, 14]. These symptoms can last from days to months. CT scans may be unremarkable or reveal mild diffuse swelling secondary to hyperemia. MRIs can show pathology in 25 % of the cases where CT scans show no abnormalities [14]. However, MRI is generally not indicated for diagnosis or treatment. Repetitive concussions are associated with worse long-term functional outcomes.

Primary and Secondary Brain Injury

Aside from treating the primary brain injury, preventing secondary progression is of the utmost importance to the intensivist when managing TBI. Secondary injuries are caused by the failure of cerebral autoregulation of blood flow and oxygen delivery; loss of this equilibrium can result in propagation of the ischemic penumbra [3]. These injuries progress after the initial impact and can be difficult to control [3]. Because of their insidious nature, watchful anticipation is needed on the part of the intensivist. Secondary injuries are also contributed to by edema, swelling, hypoxia, hypotension, electrolyte disturbances, hypoglycemia, infection, seizure, increased intracranial pressures, and hyperthermia [3, 6, 15]. Management is complex and will be discussed in the following sections.

Evaluation

Physical Examination

Although generally not present during the initial examination of a patient, it is important for the intensivist to be familiar with the initial trauma evaluation and its findings. There is a comprehensive physical evaluation, including a focused neurologic exam. If other injuries take initial precedence, it is important for a thorough follow-up neurologic exam on admission to the ICU. If the neurologic injury is the primary driver of the patient's pathologic condition, especially if it requires immediate treatment, the intensivist must triage for secondary and tertiary systemic traumatic injuries when the brain injury is controlled. This often occurs after the patient has been received in the ICU.

Intensivists should also be aware of commonly associated injuries with TBI. This most notably includes cervical spinal cord injuries. TBI patients often arrive in the ICU with a rigid cervical collar in place. Spine precautions should be continued until the cervical spine is cleared by a trauma or neurosurgeon.

Neurologic Examination

GCS is the most commonly used method of both initial and follow-up neurological evaluation. It evaluates cognitive function objectively and can be assessed by all levels of practitioner. Changes in GCS, even if small, can be an early sign of deterioration and warrant additional evaluation. The neurologic physical examination also includes a head to toe assessment of motor and sensory function, brain stem function, cranial nerve examination, reflexes, and pupillary reactivity.

Pupillary reactivity is a vital component of ongoing physical assessment in the ICU. Abnormalities in pupillary reactivity and size can indicate worsening TBI and is associated with poorer neurologic outcomes [16]. The most critical example is acute pupillary dilation, which can be the result of pending herniation. This is caused by direct compression of the third cranial nerve [17].

Pupillary changes also correlate with brainstem oxygenation and cerebral tissue perfusion and ischemia [16, 18]. Acute pupillary changes should be considered a neurologic emergency as timely interventions can improve outcomes [19].

Imaging

CT Scan

CT scan is the current gold standard for assessing TBI initially. It is quick, available in almost all centers, and can be interpreted expeditiously by both radiologists and intensivists [20]. It can also be obtained in serial fashion as prompted by changes in physical exam. CT brain imaging is obtained without contrast in the acute TBI setting so as to accentuate any acute blood products in the cranial vault. Guidelines for initial CT scan imaging for TBI patients include anyone with a GCS of 14 or lower. CT scans can also be obtained in any patient with risk factors for intracranial pathology including nausea, vomiting, severe headache, age <4 years or >65 years, amnesia, mechanism, neurologic deficits, and those on anticoagulation or antiplatelet agents [20]. Follow-up CT imaging is recommended following most lesions seen on initial CT scan and if any clinical change occurs. Progression usually occurs within 6–9 h after an injury and thus this is the typical window for re-imaging [20].

CT Angiography

Blunt cerebrovascular injury (BCVI) occurs in 1/1000 trauma patients in the United States [21]. Most of these injuries are diagnosed after the development of symptoms, at

which point the intervention window may have passed, resulting in significant morbidity (80 %) and mortality (40 %) [21]. Because of this devastating potential, it is important for intensivists to be diligent about screening patients, maintaining a high index of suspicion, and performing imaging studies where appropriate.

Basilar skull and petrous bone fractures are highly associated with BCVI. CT angiogram (CTA) is the most cost-effective means of evaluation, but if symptoms are suspicious despite a negative CTA, an MRI may be indicated to rule out a carotid or vertebral artery injury [20, 21].

Additional recommendations from the Eastern Association for the Surgery of Trauma's BCVI Guidelines include CTA screening in asymptomatic blunt head trauma patients with a GCS ≤ 8, cervical spine fracture (especially C1–C3 or through the transverse foramen, with rotational component or subluxation), and Le Fort II or III facial fractures.

Magnetic Resonance Imaging

Magnetic resonance imaging (MRI) has the highest sensitivity in revealing TBI, but because of the time constraints involved with obtaining an MRI, it is typically not done in the acute setting. MRI is also the most sensitive at detecting DAI and other nonhemorrhagic contusions [20]. Once the acute phase of resuscitation and stabilization is complete, MRI may offer additional information on primary and secondary lesions and may help to better neuro-prognosticate [20].

Monitoring

Appropriate monitoring for progression of TBI is essential in the ICU and is recommended by the Brain Trauma Foundation Guidelines [22]. Monitoring techniques have evolved over time with technological advances. Key monitoring techniques are briefly discussed below as an extensive review has been provided in that respective chapter.

Intracranial Pressure Monitoring

Cerebral perfusion pressure (CPP) is defined as the difference between mean arterial pressure (MAP) and intracranial pressure (ICP) [CPP = MAP − ICP]. CPP estimates the pressure gradient across vascular surfaces and is an important marker of cerebral blood flow [23]. ICP monitoring is recommended in all patients with severe TBI and an abnormal head CT scan. If the CT scan is normal, it is still indicated if two or more of the following is true: the patient age is over 40 years, there is unilateral or bilateral posturing, or the SBP is <90 mmHg [3, 24]. Monitoring can be subdivided into internal versus external.

Internal ICP Monitoring

Internal monitoring devices are designed to be introduced into different anatomical locations within the brain including the intraparenchymal, intraventricular, epidural, subdural, and subarachnoid spaces. The gold standard in invasive monitoring is via an extraventricular device (EVD) where a catheter is placed into the lateral ventricle percutaneously via a burr hole [23]. The advantage of an EVD is that along with ICP monitoring, cerebrospinal fluid (CSF) and hemorrhagic fluid can also be drained [23].

Microtransducers are also invasive monitoring devices with a very low profile. They are just as accurate as EVDs when it comes to measuring ICP, but they cannot drain fluid and cannot be manipulated once placed due to the fact that most models cannot be recalibrated [23].

External ICP Monitoring

External monitoring devices do not pose the bleeding or infectious risks that invasive monitors have; however, they may not be as accurate. Transcranial doppler (TCD) measures blood flow via the middle cerebral artery which is subject to changes in ICP. Its accuracy is limited by operator placement and interpretation and occasionally due to poor penetration through the skull [23]. Tympanic membrane displacement (TMD) utilizes the communication of CSF and perilymph via the perilymphatic duct. The reflex movement of the tympanic membrane correlates with ICP. Optic nerve sheath diameter (ONSD) also correlates with ICP as it expands with increased ICP. Motion-sensitive MRI is also another option [25].

Brain Tissue Oxygen (PbtO2) Monitoring

Several PbtO2 devices are available for neuromonitoring and frequently integrate with other invasive ICP monitoring devices. They add an additional dimension beyond pressure to evaluate cerebral perfusion and guide management.

Management

The management of TBI can be divided into medical versus surgical management. In the acute phase, medical management is instituted first with the rapid ability to deliver surgical therapy if medical management fails.

Medical Management

Pathophysiology of Cerebral Perfusion

CPP is an important factor in the neurophysiology of TBI as it represents cerebral blood flow (CBF) and oxygen delivery. Under normal circumstances, the brain can autoregulate to

maintain CBF when systemic MAPs range from 50 to 150 mmHg [4]. It is important for the intensivist to recognize that autoregulation is abnormal after a TBI and that tighter control of CPP (which the Brain Trauma Foundation recommends at 50–70 mmHg) is warranted. This is to prevent secondary brain injury, promote adequate oxygen delivery, and reduce the morbidity and mortality of TBI. Cerebral blood flow can be diminished for days to weeks after an injury [3, 4]. Physiologically, low cerebral blood flow can fail to meet cerebral metabolic demands and is thus associated with poorer outcomes [4, 24]. Conversely, high CPP (above 70 mmHg) can be associated with cerebral edema and can result in an increased risk of adult respiratory distress syndrome (ARDS) [24].

The space inside the skull is fixed and the volume of the intracranial contents remains generally constant. In severe TBI where a mass lesion like a hematoma can occupy space, venous blood and cerebrospinal fluid (CSF) can be displaced out of the cranium to maintain a normal ICP. This compensatory mechanism is known as the Monro-Kellie doctrine [4]. However, the limits of this displacement can be surpassed and the ICP can rapidly increase causing further injury. When this occurs, the triad of hypertension, bradycardia, and respiratory irregularities occur; this is Cushing's reflex and is often considered a neurological emergency [26]. Rapid measures to control ICP must be undertaken.

Reduction of ICP

Hypertonic Saline

Intravenous hypertonic saline (HTS) lowers ICP through mobilization of water from the brain tissue across an oncotic gradient. Additionally, CBF and oxygen delivery are improved through erythrocyte deformability and dehydration as a result of plasma dilution and volume expansion [3]. The HTS onset of action is within minutes and can last hours and thus is used in the acute and subacute setting. Optimal serum sodium levels are between 145 and 160 mEq/L [2]. Because of the potential for rapid hypernatremia and risk of central pontine myelinolysis, close serum sodium and osmolality monitoring is essential. Frequency of monitoring and dosing are often via institutional protocol.

Mannitol

Mannitol also uses hyperosmolar therapy to reduce ICP. Mannitol creates an osmotic diuresis. This diuresis similarly creates a reduction of cerebral edema, an expansion of plasma volume, reduced blood viscosity, and increased CBF. Mannitol has an immediate onset of action (minutes) and its effects can last up to 6 h [3]. In the acute setting, the usual dose is 0.25–1 g/kg. As it is an osmotic diuretic, it should not be given to hypotensive patients; in this setting, HTS may be more appropriate. Serum osmolarity must be monitored with a target level <320 mOsm [6].

Hyperventilation

Hyperventilation causes cerebral vasoconstriction and temporarily reduces cerebral volume, thus reducing ICP. However, the effect is short lived and prolonged vasoconstriction can lead to impaired cerebral perfusion [3, 4]. (Importantly, the converse is also true, allowing $PaCO_2$ to rise can cause cerebral vasodilation and an increased ICP [4].) Onset of action is rapid, within 30 s, and generally peaks at 8 min [3]. Hyperventilation strategies can be achieved by either bag valve mask ventilation or more precisely through ventilator manipulation.

When using the ventilator, the intensivist must be aware that positive end-expiratory pressure (PEEP) can increase ICP. Increased PEEP causes an increased intrathoracic pressure and cephalad transmission of increased central venous pressure (CVP) to the brain, disturbing CBF. By decreasing venous return and increasing intrathoracic pressure, PEEP may also cause a decrease in cardiac output thereby reducing MAP and subsequently CPP [27].

Elevation of the Head of the Bed

Elevating the head of the bed (HOB) significantly reduces ICP [28, 29]. The mechanism is twofold: (1) the result of displacement of CSF from the cranial cavity to the subarachnoid space and (2) extenuated brain venous outflow is enabled via gravity [29]. All patients with an increased ICP should have the HOB elevated between 30 and 45° [29].

Optimization of Systemic Blood Pressure and Oxygenation

A main goal in management of TBI patients is the prevention of secondary brain injury, which is often the result of systemic hypoxemia or hypotension. Both prehospital and in-hospital hypotension have a negative effect on severe TBI outcomes [30]. In some studies, a single prehospital systolic blood pressure (SBP) <90 mmHg was a factor associated with worse outcomes in TBI [22]. Similarly, in-hospital hypotension has also been found to be a predictor of increased mortality in TBI patients [31, 32]. It is prudent for the intensivist to prevent systemic hypoxemia and hypotension in order to minimize the effects of these secondary insults. Guidelines for numerical targets adapted from the Brain Trauma Foundation are listed in Table 4.3.

Pharmacologic Management of TBI

Increased pain and agitation can lead to increased ICP, so cautious pain control and sedation is an important aspect of the management of these patients. Appropriate sedation can improve hypoxia, hypertension, elevated ICP, and hypercarbia [1]. If sedatives and analgesics are adminis-

Table 4.3 Target values for management of elevated intracranial pressure (ICP)

	Target
ICP	<20 mmHg
Systolic blood pressure	>90 mmHg
Oxygenation	PaO_2 >60 mmHg or O_2 saturation >90 %
CPP	50–70 mmHg
PbtO2	>15 mmHg

tered, the patient should be carefully monitored to avoid the effects of hypotension, alteration of a neurologic exam, or rebound ICP elevation [3]. Short-acting and continuously infused agents are preferred as there is less disruption in the neurologic exam, and short-lived increases in ICP may be prevented [3]. Fentanyl is a preferred short-acting analgesic agent that is easily titratable and reversible. Propofol and midazolam are commonly used sedatives [1]. Propofol is a hypnotic anesthetic that is also easily titratable, is short acting, and has a rapid onset of action. Because it reduces cerebral oxygen consumption, it may have a neuroprotective effect on TBI patients as well [3, 24].

Barbiturates are central nervous system depressants that lower ICP and reduce cerebral oxygen consumption thereby conferring a protective effect. Because of their risks, their use is limited to cases of uncontrolled ICP refractory to initial medical and surgical therapies [24].

Seizure Prophylaxis

There is an upward of 50 % incidence of seizures in the TBI patient, especially those with a penetrating mechanism [24]. Post TBI seizures can be detrimental in that they can increase ICP, cerebral oxygen demand, and neurotransmitter release, all of which facilitate secondary brain injury. Risk factors for developing post-traumatic seizures include GCS <10, cortical contusions, depressed skull fractures, subdural, epidural, and intracerebral hematomas [24]. Seizures typically occur in two phases: immediately (<24 h) or within a week. For these reasons, the use of anticonvulsant agents in the first week following TBI is highly recommended [1, 24]. The most commonly used agents are phenytoin, levetiracetam, sodium valproate, and carbamazepine [1].

Venous Thromboembolism (VTE) Prophylaxis

TBI patients are at high risk for VTE events as they occur in up to 20–30 % of cases. Prophylaxis should be started as soon as possible, typically within 24 h of stable imaging. If there is an increased bleeding risk, an IVC filter should be considered. Institutional guidelines are highly recommended to defer inter-provider variances [33].

Therapeutic Hypothermia

Therapeutic hypothermia for neuroprotection has been shown to improve neurologic outcomes in post-cardiac arrest medical patients [34, 35]. Therapeutic hypothermia works by reducing cerebral metabolism, ICP, inflammation, lipid peroxidation, excitotoxicity, cell death, and seizures [3]. Active strategies to prevent fever in TBI patients are well proven. Further, prophylactic hypothermia has been associated with improved Glasgow Outcome Scale (GOS) scores when comparing to even normothermia controls [24]. Some studies suggest hypothermia to be 32–33 °C, but this must be balanced with the risks of electrolyte abnormalities, bleeding, and cardiac arrhythmias [24].

Nutrition

TBI patients have increased metabolic demands on the order of 120–250 % of basal caloric needs. Much of this increase is related to muscle tone [24]. An adequate and appropriate nutritional regimen in the ICU is paramount to meet these metabolic needs. For mortality reduction, the Brain Trauma Foundation recommends that full caloric replacement should be achieved, at the latest, by 7 days post injury. In order to accomplish this, feedings should be started, at the latest, within 3 days post injury [3, 24]. Enteral feeding is preferred [3, 33]. Regardless of enteral or parenteral delivery, protein supplementation is important [24]. Strict monitoring should occur to avoid derangements in electrolytes and glucose [36].

Surgical Management

Up to one third of TBI patients will become surgical candidates [37]. Because of this potential, and its often rapid decompensatory nature, all TBI patients should have access to neurosurgical care. Surgical evacuation is generally considered for any mass lesion causing a decline in the patient's level of consciousness, focal deficits, severe or worsening headache, nausea, or vomiting [37]. When patients are intubated or otherwise unable to communicate, indications include a decline in neurologic exam, sustained increase in ICP, or a change in the size of the mass lesion on serial imaging [37]. CT evidence of midline shift >5 mm and/or compression of the basal cisterns is an indication for surgical decompression [33]. In addition to mass lesions, TBI can also lead to cerebral edema which similarly encroaches on the limited space in the cranial vault.

Management of Hematomas

Epidural hematomas can rapidly expand placing direct pressure on the adjacent brain. Collections greater than 30 cc should be surgically treated independent of GCS. If they are less than 30 cc in volume, GCS or any midline shift should be taken into account with management decisions [37].

Hematomas in the middle fossa/inferotemporal lobe should have a lower threshold for surgical evacuation [37].

An SDH with a midline shift >5 mm or thickness >10 mm requires surgical treatment. A patient with a GCS <9 with a midline shift <5 mm or thickness <10 mm should be surgically treated if the GCS has decreased by 2 or more from injury to admission, if there are fixed or dilated or asymmetric pupils, or if the ICP is >20 mmHg [37].

Intraparenchymal hemorrhage (IPH) with neurologic deterioration related to the hemorrhage, refractory ICP elevation, or radiographic evidence of mass effect should be treated with surgical evacuation. Further, if the lesions are frontal or temporal, >20 cc in volume with a shift of >5 mm, or compression of the basal cisterns exists, surgical evacuation should also be considered [37]. If drainage of the lesion is not feasible based on location and/or depth, a decompressive craniectomy can be considered to relieve the elevated ICP.

Decompressive Craniotomy/Craniectomy

Decompressive craniotomy and opening of the dura allows areas of devitalized and injured brain to be removed as needed. In a decompressive craniectomy, the skull flap is not replaced, leaving only the dura and overlying scalp [3]. This allows for maximal pressure release from the cranial vault. When appropriate, this can be performed bilaterally and with substantially large bone flaps.

Burr Holes/Emergency Craniostomy

Emergency craniostomy, or burr holes, allows for drainage of subdural fluid. Although rarely performed, they can be a lifesaving technique, especially when definitive neurosurgical care is not readily available. These situations are more likely in rural areas or developing countries without access to advanced imaging or equipment [38].

Abdominal Decompression

Intracranial, intrathoracic, and intra-abdominal pressures are closely intertwined. Increases in intra-abdominal pressure (IAP) displace the diaphragm upward, leading to an increased intrathoracic (ITP) pressure, which in turn leads to an increased central venous pressure (CVP) and a decrease in cerebral venous outflow (CVO). Decreased CVO can directly elevate ICP ($\uparrow$IAP $\rightarrow$ $\uparrow$ITP $\rightarrow$ $\uparrow$CVP $\rightarrow$ $\downarrow$CVO $\rightarrow$ $\uparrow$ICP). Because of this relationship, decompressive laparotomy has been used as a means to reduce persistently elevated ICP when other measures have failed [39, 40].

Special Populations

Diffuse Axonal Injury

Diffuse axonal injury (DAI) results from a shearing injury of axons. There are no specific focal lesions apparent on head CT scan so MRI is typically employed as the more sensitive imaging modality. The severity of the injury is gauged more by clinical course than by visible injury. Patients with mild DAI have a 15 % mortality rate, while those with moderate DAI have a 25 % mortality rate [26]. Most patients with severe DAI succumb to the TBI [26]. No specific therapies for DAI exist and the treatment is largely supportive in nature.

Management of Skull Fractures

Depressed skull fractures are elevated surgically if the depression is greater than the opposing inner table. Open skull fractures are often similarly treated surgically to prevent infection. One exception is depressed skull fractures overlying the sagittal sinus. These should not be disturbed for fear of disruption of the sinus and the potential for massive, uncontrollable hemorrhage [37].

Management of Concussion

Concussions are generally managed by support care, recognizing the high incidence of headache, amnesia, confusion, and occasional loss of consciousness. Sports-related concussions are an important variant as a large part of their management strategy is prevention of subsequent injuries [3]. Early education and symptom management are also hallmarks of treatment [41].

Outcomes

Glasgow Outcomes Score (GOS)

Prediction of functional status and outcomes after the acute phase of TBI has widespread implications for cost, rehabilitation, and long-term care planning [42]. The GOS is the most widely used tool for measuring outcomes after TBI (Table 4.4) [43, 44]. Additionally, the GOS-Extended (GOSE) was also created to boost sensitivity to less prominent deficiencies in cognition, mood, and behavior (Table 4.5) [45].

Brain Death Exam/Determination

Brain death is characterized by no observable activity in the brain and cessation of all functions of the entire brain and

Table 4.4 Glasgow Outcome Scale (GOS)

Numerical score	Classification
1	Death
2	Persistent vegetative state
3	Severe disability
4	Moderate disability
5	Good recovery

Table 4.5 Glasgow Outcome Scale-Extended (GOSE)

Numerical score	Classification
1	Death
2	Persistent vegetative state
3	Lower severe disability
4	Upper severe disability
5	Lower moderate disability
6	Upper moderate disability
7	Lower good recovery
8	Upper good recovery

brainstem [3, 46]. When determining brain death, there should be no confounding variables like recent sedative, analgesic, paralytic or psychotropic medication administration, hypotension, encephalopathy, hypothermia, or other conditions that may obscure the neurologic exam [3]. The exact process and criteria for brain death determination is variable by state and institutional policies. An important adjunct of assessing for brain death or when suspecting brain death is to determine eligibility for organ donation. Intensivists are the frontline providers to engage their regional organ procurement organization and provide this opportunity to their patients and families.

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