Evidence-Based Review: Nursing Care of Adults with Severe Traumatic Brain Injury

Literature Review

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Abstract

Purpose: The purpose of this review of the literature is to provide nurses with evidence-based strategies to care for adult patients with severe traumatic brain injury (sTBI).

Methods: Neuroscience nurse experts performed a critical review of the published literature using Cochrane, PubMed, and the Cumulative Index of Nursing and Allied Health Literature (CINAHL) databases from January 2008 to November 2018, using a systematic, librarian-assisted search strategy. The National Guidelines Clearinghouse also was searched prior to July 16, 2018, to include a review of published guidelines from national and international professional organizations.

Results: The literature search yielded 123 articles that were included in the reference list. Evidence was used to develop a summary of the literature addressing key nursing management topics when caring for the adult patient with sTBI.

Conclusions: This review of the literature identifies evidence-based nursing practices when caring for adult patients with sTBI.

Keywords: Severe traumatic brain injury, primary brain injury, secondary brain injury, nursing care

Introduction

The purpose of this literature review is to provide an evidence-based appraisal of the literature to assist the registered nurse (RN) in providing quality nursing care to patients with severe traumatic brain injury (sTBI). Although this review targets the care of the adult patient with sTBI during acute hospitalizations, this review may be used for caring for patients with sTBI in a variety of healthcare settings and across the continuum of care. Because of the impact of caring for a patient with sTBI in terms of morbidity, mortality, cost, and the incidence and prevalence of people affected, new treatments continue to emerge. Adherence to the content provided in this literature review must be balanced with patient and family preferences, considerations of the healthcare team, additional practice-specific resources, and emerging research recommendations. This literature review is not intended to replace formal learning but rather to augment the knowledge base of clinicians and provide a readily accessible reference tool for nurses and other clinicians caring for patients with sTBI. In addition, the content presented within this document is not inclusive of all activities that might improve outcomes but reflects select nursing-centered interventions scientifically examined within the past decade. This review targets activities independently performed by nurses and those interdependent activities that nurses are responsible for implementing and monitoring to help achieve positive patient outcomes in this high-risk population.

Methods

To inform the literature review, computerized searches of PubMed and the Cumulative Index to Nursing and Allied Health Literature (CINAHL) were performed by a group of medical librarians using the keywords associated with 29 structured questions using the Population, Intervention, Comparison, Outcome, Time (PICOT) format. Specific search terms were used for all PICOT questions as follows:

For PubMed: ((((((((((((((((((TBI[TIAB]) OR ((“brain injuries, traumatic”[MeSH Terms] OR (“brain”[TIAB] AND “injuries”[TIAB] AND “traumatic”[TIAB]) OR “traumatic brain injuries”[TIAB] OR (“traumatic”[TIAB] AND “brain”[TIAB] AND “injury”[TIAB]) OR “traumatic brain injury”[TIAB]))) OR ((“brain injuries”[MeSH Terms] OR (“brain”[TIAB] AND “injuries”[TIAB]) OR “brain injuries”[TIAB] OR (“brain”[TIAB] AND “injury”[TIAB]) OR “brain injury”[TIAB]))) OR ((“brain contusion”[MeSH Terms] OR (“brain”[TIAB] AND “contusion”[TIAB]) OR “brain contusion”[TIAB]))) OR ((“brain concussion”[MeSH Terms] OR (“brain”[TIAB] AND “concussion”[TIAB]) OR “brain concussion”[TIAB]))) OR ((“brain injury, chronic”[MeSH Terms] OR (“brain”[TIAB] AND “injury”[TIAB] AND “chronic”[TIAB]) OR “chronic brain injury”[TIAB] OR (“chronic”[TIAB] AND “brain”[TIAB] AND “injury”[TIAB]))))) OR ((“diffuse axonal injury”[MeSH Terms] OR (“diffuse”[TIAB] AND “axonal”[TIAB] AND “injury”[TIAB]) OR “diffuse axonal injury”[TIAB]))) OR ((“brain”[TIAB] AND “laceration”[TIAB]) OR “brain laceration”[TIAB]))) OR ((“brain haemorrhage”[TIAB] OR “intracranial hemorrhages”[MeSH Terms] OR (“intracranial”[TIAB] AND “hemorrhages”[TIAB]) OR “intracranial hemorrhages”[TIAB] OR (“brain”[TIAB] AND “hemorrhage”[TIAB]) OR “brain hemorrhage”[TIAB]))) OR ((“cerebral haemorrhage”[TIAB] OR “cerebral hemorrhage”[MeSH Terms] OR (“cerebral”[TIAB] AND “hemorrhage”[TIAB]) OR “cerebral hemorrhage”[TIAB]))) OR ((“craniocerebral trauma”[MeSH Terms] OR (“craniocerebral”[TIAB] AND “trauma”[TIAB]) OR “craniocerebral trauma”[TIAB])))))) OR (((((((“brain”[MeSH Terms] OR “brain”[TIAB])

For CINAHL: (MH “Neuroscience Nursing+”) OR (MH “Critical Care Nursing+”) OR “neuroscience nursing” OR “Critical care nursing” OR “Critical care” OR “acute care” OR “neurointensive” OR “neuro ICU” OR “neuro-acute” OR “neurocritical” OR “neuro critical” OR “step-down”).

In addition, the search associated with each of the 29 PICOT questions was further refined based on the specific question and the quality of the literature. Each search was restricted to works published in English between January 2008 and November 2018 in which all or part of the sample included adults with sTBI. Although the searches focused on nursing care within the United States, they also included articles from other countries where resources and care may be similar. The target population was limited to adults. The reference lists of identified articles were used to identify other pertinent studies.

In addition, the writing group searched the electronic National Guideline Clearinghouse, an initiative of the Agency for Healthcare Research and Quality, for guidelines posted prior to July 16, 2018, as well as the Cochrane Library. Leading guidelines identified include Management of sTBI by the Brain Trauma Foundation (BTF),[1] Best Practices in the Management of Traumatic Brain Injury from the American College of Surgeons Trauma Quality Improvement Program (TQIP),[2] and the Emergency Neurological Life Support (ENLS) TBI algorithm published by the Neurocritical Care Society.[3]

The literature from articles meeting inclusion criteria for each topical area and PICOT question were summarized addressing the nursing management of patients with sTBI. Content was organized with the following topical headings: Prehospital and Emergency Department

Nursing Care of the Patient with sTBI, Nursing Management of the Patient with sTBI in the Intensive Care Unit (ICU), Acute Care Considerations, Rehabilitation Considerations, and Geriatric Considerations.

In alignment with BTF guidelines, *sTBI* is defined within this document as a brain injury incurred by a traumatic mechanism of injury with a resultant level of consciousness (LOC) categorized by a Glasgow Coma Scale (GCS) score of 8 or less.[1]

Introduction to sTBI

Background

sTBI is a leading cause of morbidity and mortality worldwide, particularly among young people.[4] Because sTBI is caused by an external injury to the head that affects how the brain normally works, it most often is quantified as an initial GCS score of 8 or less.[1,5-7] Patients with sTBI have significant structural and metabolic brain dysfunction and are at high risk of developing secondary brain injury and, therefore, further deterioration.[6] In addition, 56%–60% of patients with a GCS score of 8 or less will have one or more other injured organ systems, further complicating their care.[7] Because nurses care for patients affected by the impact of sTBI and have the ability to alter a patient's course of recovery, it is important that nurses have evidence-based resources to promote positive patient outcomes.

Epidemiology

Approximately 2.8 million people per year in the United States sustain a TBI.[8] Actual numbers may be higher because mild TBI cases are underreported.[8] Patients with sTBI represent between 6% and 8% of all TBI cases.[9] Falls (47%), blunt injuries (15%), unknown or other causes (15%), motor vehicle accidents (14%), and assaults (9%) account for the largest portion of TBI in civilian populations.[8] Blood alcohol concentration is positive in 56% of patients with TBI, not limited to sTBI.[10]

Of the total number of reported TBI cases, roughly 282,000 people are hospitalized and 56,000 die.[8,9] Falls among the elderly are responsible for the increasing number of hospital-related TBI admissions.[8] Although outcomes vary based on the mechanism of injury, the patient's age, concomitant injuries, and comorbidities, overall TBI mortality accounts for approximately one-third of all injury-related deaths.[11] The 30-day mortality for sTBI is 50%.[8] Mortality is highest in patients with penetrating TBI (such as those injuries caused by firearms), accounting for 34.8% of all TBI deaths, followed by motor vehicle accidents (31.4%) and falls (16.7%).[12] Civilian TBI rates are highest among people age 15 to 24 years and those older than age 65 years and occur 1.5 times more often in men than in women.[13,14] However, TBIs can affect anyone at any age and may be associated with ongoing complications and long-term disability. An estimated 5.3 million individuals are living with lifelong TBI-related functional, behavioral, and cognitive disability.[13,15] Medical costs for TBI exceed $76.5 billion per year, with approximately 90% of these costs being attributed to the cost of fatal TBIs and TBIs requiring hospitalization, many of which are severe.[14,15] Although recent estimates are not available, in the 1990s, the lifetime cost of caring for a person with sTBI exceeded $3 million.[13]

TBI Pathophysiology

The progression of sTBI occurs in two phases, known as primary and secondary injury. Primary brain injuries target the physical injuries occurring at the time of the traumatic event, which largely result in shearing and compression of the brain.[16] This includes injuries such as cortical and brain stem contusions; lacerations; bone fragmentation; skull fracture; diffuse and focal axonal injuries; torn cerebral blood vessels, such as in epidural hematoma, subdural hematoma (SDH), and traumatic subarachnoid hemorrhage; and focal and petechial hemorrhage.[16] Broadly, primary brain injuries represent the initial injuries that follow the translation of the kinetic energy into tissue damage.[17]

Secondary injuries occur within minutes, hours, or days of the primary injury. These are the biochemical reactions and cascades induced by the primary injury or injuries.[16] Secondary injuries include intracranial hematomas, vasogenic and cytotoxic cerebral edema, cerebral ischemia associated with increased intracranial pressure (ICP) and shock, hydrocephalus, metabolic abnormalities, neuroinflammation, meningitis, and vascular events such as vasospasm.[18,19] The majority of secondary injuries are ischemic in nature. Nursing and medical interventions in caring for a patient with sTBI primarily target the prevention and reduction of secondary injury, as these are thought to extend the amount of neuronal damage responsible for the severe physical and cognitive disabilities found in patients with sTBI.[18,19] A summary of common interventions to prevent secondary brain injury and promote positive patient outcomes is provided in **Figures 1** and **2**.

Figure 1. Intracranial and Cerebral Perfusion Pressure Management in sTBI

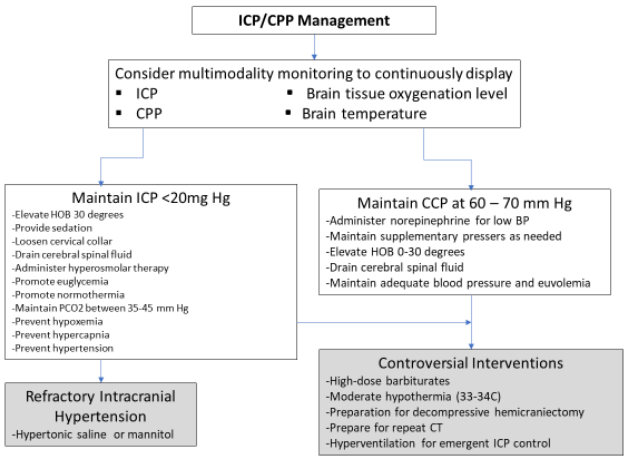
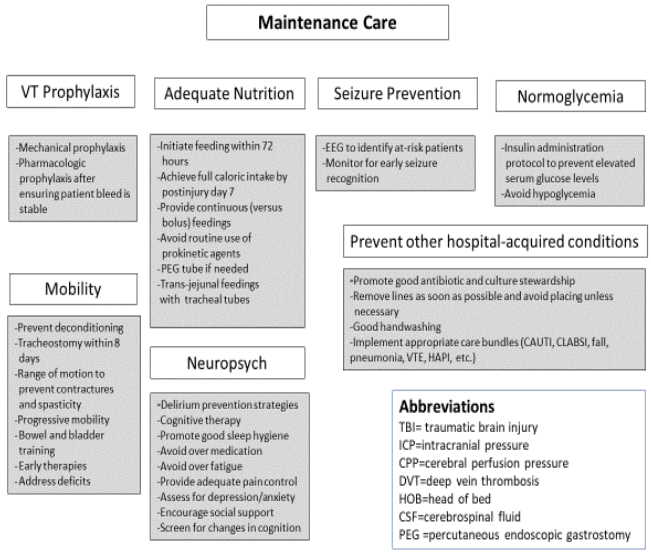


Figure 2. Maintenance Care in the Patient with sTBI



Prehospital and Emergency Nursing Care of the Patient with sTBI

Initial Assessment of the Patient with sTBI

Fifty percent of TBI-related deaths occur within the first few hours of injury.[9] Delays in appropriate care contribute to secondary injury corresponding to increased morbidity and mortality. Therefore, rapid and appropriate assessment of sTBI in the field and emergency department (ED) is essential in facilitating timely care management to prevent debilitating secondary brain injury and sequelae.

During the prehospital or ED phase, emphasis is placed on assessment of the airway maintenance with cervical spine protection, breathing and ventilation, circulation with bleeding control, disability/neurological examination, and exposure and environmental control (also known as the ABCDE mnemonic for the prehospital assessment of patients with trauma).[2,20–23] This assessment is further expanded in **Table 1**. These assessments should be performed during triage and transport.[22] The GCS score is a key component of the disability neurologic assessment, especially when evaluating patients with sTBI.[7,20]

Table 1. Initial Assessment of Patients Using the ABCDE Trauma Assessment[2,23]

Initial Assessment Priorities	Key Assessment Features and Thresholds	Rationale
Airway	• Check consciousness. • Assess whether the airway is clear. • Assess for security of the airway. • Airway should be monitored while maintaining spinal stabilization.	• Patients who have a decreased level of consciousness (LOC) (Glasgow Coma Scale [GCS] score < 8, not following commands) upon initial evaluation, rapidly declining mental status, or another severe extracranial injury that may compromise adequate oxygenation should receive rapid sequence intubation and assisted ventilation. • In trauma patients, 10% have associated cervical spine injuries; until radiographic imaging is completed to rule out spinal instability, the c-spine should be stabilized with a cervical collar or manual inline stabilization.
Breathing	• Oxygenation should be measured using pulse oximetry with a goal of SaO_2 > 90%.	• Hypoxia is associated with increased risk of mortality.

	• Assess maintenance of normocapnia—maintain normal breathing patterns. • Monitor end-tidal CO_2 ($ETCO_2$) with a goal of 35–40mmHg.	• Hyperventilation should be avoided ($ETCO_2$ < 35mmHg) unless there are signs of immediate herniation.
	• Assess respiratory pattern for irregular respirations (a component of Cushing's triad).	• Abnormal respiratory pattern may signal neurologic dysfunction, raised intracranial pressure (ICP), or injuries to the chest.
Circulation	• Examine for life-threatening hemorrhage. • Assess blood pressure with a mean arterial pressure (MAP) goal of at least 60 mmHg.	• Hypotension is associated with increased risk of mortality. • Avoidance of hypotension is especially important within the first 6 hours of injury. • Until ICP monitoring is in place, aim to reach a target MAP of 60 mmHg to ensure adequate cerebral perfusion pressure. • GCS exam may decline with hypotension and improve as blood pressure is corrected.
	• Assess for signs of herniation with Cushing's response: hypertension, bradycardia (components of Cushing's triad), and a widening pulse pressure.	• Cushing's response is a late compensatory mechanism to maintain cerebral blood flow in the presence of raised ICP. • Bradycardia is associated with increased ICP and cervical injury.
	• Tachycardia	• Tachycardia in the trauma patient can signal hypovolemic shock. • In severe traumatic brain injury, tachycardia often is associated with an autonomic response to injury of the hypothalamus and may signal a terminal event.
Additional Assessment/ Disability Neurologic Exam	• Verify mechanism of injury and assess LOC. • Verify GCS score at the scene. • Progressive decline in neurologic condition is consistent with a decrease in GCS score >2 points. • GCS score should be calculated following resuscitation but prior to receiving any paralytic or sedative agents.	• Presence of a GCS score <13 prompts evaluation transport to a designated trauma center for evaluation and possible neurosurgical consultation. • Severe extracranial trauma or rapid deterioration of mental status also may indicate the need for intubation. • According to Emergency Neurological Life Support guidelines, a GCS score ≤8 during the initial evaluation is an indication for endotracheal intubation. • Risk of intracranial complications increases as the GCS score declines. • The GCS should be performed repeatedly to identify worsening or improvement over time. • The GCS exam performed in the emergency department may be a more reliable assessment of TBI severity compared to a prehospital GCS assessment, if the patient is free of paralytics or sedative agents. • Pharmacologic sedation or paralytics can mimic or mask a neurologic deficit.

Table 1. Initial Assessment of Patients Using the ABCDE Trauma Assessment[2,23] *(continued)*

Initial Assessment Priorities	Key Assessment Features and Thresholds	Rationale
	• Check pupil size and functioning. • Pupil asymmetry is defined as >1mm difference in diameter. • A fixed pupil is defined as <1mm response to bright light. • Pupils should be assessed after resuscitation; note any evidence of orbital injury.	• Pupil size and reactivity is a useful indicator of expanding intracranial lesion or herniation. • Pupil asymmetry is a finding that prompts neurosurgical evaluation. • Orbital trauma may mimic or mask signs of herniation and should be considered in each patient.
	• Check seizure activity. • Verify whether the seizure was witnessed before or after the trauma.	• Presence of seizure activity should prompt neurosurgical consultation. • In some cases, a seizure may precede the trauma and may raise suspicion for a nontraumatic cause of decreased LOC.

Systemic Injury (Also Known as Exposure and Environment)	• Look for other nontraumatic causes of decreased LOC:
	  • Airway obstruction
	  • Hypoxia caused by tension pneumothorax
	  • Hypoglycemia
	  • Opiate overdose
	  • Neurologic event preceding the trauma, such as a spontaneous intracerebral hemorrhage, seizure, or stroke.
		• Confounding factors may limit the accuracy of the neurologic exam and impact the management.

	• Check for drug or alcohol intoxication. • Assess for severe extracranial trauma.	• American College of Surgeons Trauma Quality Improvement Program guidelines recommend transfer of a patient with any level of traumatic brain injury (GCS score 3–15) combined with severe extracranial injuries to a dedicated trauma center to allow for prompt neurosurgical and multidisciplinary assessment and intervention.

The Glasgow Coma Scale

The GCS was first presented in 1974 by Graham Teasdale and Bryan Jennett to assess the LOC of patients with an acute brain injury.[24] The GCS is widely used to record and trend the severity of brain injury based on neurological assessment.[25] As such, it also is used for clinical decision making, prognostication, and quantifying severity of injury in research studies. The GCS examination evaluates several responses for eye opening (scored 1–4), verbal ability (scored 1–5), and best motor response of an upper extremity (scored 1–6). In 2014, 40 years after its development, Teasdale updated the GCS examination scoring guidelines to address variations in technique.[25] These are further described in **Table 2**. Note, when scoring the motor response, the patient's score is based on the highest scoring response elicited in any single limb. If it is necessary to apply noxious stimuli, start with a central stimulus, such as sternal rub or supraorbital ridge pressure. Do not perform supraorbital ridge pressure in the presence of facial fractures. If there is no response to central noxious stimulus, apply peripheral stimulus, such as nailbed pressure.[26]

Table 2. Updated CGS Assessment Components[27]

The Glasgow Coma Scale (GCS) Exam			
Components of the GCS	**Term Used**	**Score**	**Key Points in Deciphering the Score**
Eye Opening	Spontaneous To sound To pressure None Not testable	4 3 2 1 NT	• Spontaneous opening should not be equated to alertness or awareness. • A response to a specific spoken command is not required when a patient is determined to open eyes to "sound." • To determine if a patient responds to a physical stimulus, graded pressure should be applied using: o fingertip pressure o trapezius pinch o supraorbital notch. • Factors that do not allow for eye opening, such as facial swelling or eye injury, are considered **not testable**.
Verbal Response	Oriented Confused Words Sounds None Not testable	5 4 3 2 1 NT	• An oriented verbal response requires that the patient must provide correct answers to the following: o person (their name) o place (their location) o time (the month). • The person is confused if any one of the three items is incorrect, even if communication is coherent. • If the patient's response lacks structured sentences or phrases, the classification words should be used. • Presence of an endotracheal tube, tracheostomy, or physical injury that limits the ability to produce a verbal response should be classified as **not testable**.
Motor Response	Obeys commands Localizing Normal flexion Abnormal flexion Extension None Not testable	6 5 4 3 2 1 NT	• Classifying a patient as obeys commands means that the patient has made a specific response to a request and not an automatic or reflexive reaction. The instruction should be complex and specify movement in two parts: o "Squeeze and release the examiner's fingers." o "Raise and lower your arms." • The standard for determining a localization response is to produce a connection between the location of the sensory input and specific movement made in response. • A patient's hand should be brought above the clavicle when a stimulus is applied to the head or neck. • Crossing of the hand over to the opposite side of the body is not sufficient to determine a localizing response. • **Normal flexion** of the elbow should be selected unless movement closely matches the features of an abnormal response. • **Abnormal flexion** is consistent with: o slow movement o repetition (the same response each time) o arm moves across chest o forearm rotates, thumb clenched o leg extends. • Straightening of the elbow is an example of an **extension response.** • Effects of other injuries (hemiplegia, spinal cord injury) or pharmacological agents such as paralytics should be classified as **not testable.**

Guidelines advocate using the GCS for assessing neurologic impairment across the continuum for patients with sTBI because it has high reliability, especially when performed in a consistent manner by trained healthcare professionals.[1,2] Repeated GCS examinations are important for detecting improvement or deterioration over time.

The GCS examination initially should be performed after airway, breathing, and circulation are assessed.[20] According to ENLS guidelines, a GCS score of 8 or less during the initial evaluation is an indication for endotracheal intubation.[23] When able, it is optimal to obtain a baseline GCS and neurological examination prior to intubation and the administration of sedative or paralytic medications. While under the effects of paralytic medication, patients should not have noxious stimuli or oculovestibular response tests performed because they will be unable to elicit a motor response as a result of muscle paralysis.[28]

When neuromuscular blocking agents (NMBAs) are given to block musculoskeletal activity, a peripheral nerve stimulator—referred to as the "train of four" (TOF)—is used as part of the assessment to determine the degree of nerve paralysis.[29] Placement on the wrist for ulnar nerve testing is preferred over the eyebrow for testing of the facial nerve or medial malleolus of the foot for testing of the posterior tibial nerve,[30] as it best reflects movement of the diaphragm. Limb edema may limit the usefulness of the device.[31] The machine delivers four electrical impulses, one after the next. The operator looks at the patient's pinkie finger (if the ulnar nerve is stimulated) or other corresponding stimulated anatomy and counts how many times the patient twitches.[30] The goal is to find the lowest electrical impulse at which the patient twitches four out of four times. This is used as the patient's baseline. The goal for an adequate level of paralysis is for the patient to twitch two of four times when the TOF is set at the patient's baseline.[30] Use of NMBAs are losing favor but still may be used in severely ill patients. Because NMBAs lack amnesic, sedative, and analgesic properties, simultaneous administration of analgesia or anxiolytics is mandatory.[30] In conjunction with the TOF, the clinical assessment (i.e., vital signs, synchrony with the mechanical ventilator) should be used to evaluate the extent of paralysis.[32]

Although the GCS is widely used, it is not always feasible to accurately assess the eye and verbal components. For example, patients may have injuries affecting their ability to open their eyes or speak (e.g., those who have aphasia, are aphonic, have trauma to their vocal apparatus or artificial airway, or have a language barrier in which the verbal score is difficult to ascertain), be intoxicated by drugs or alcohol, or have a brain stem injury. This has led to development of modified versions of the GCS, the most common being a version that focuses only on the motor component, the motor GCS (mGCS).[33] Outcome discrimination at 3 months and 12 months post-injury between the mGCS and full GCS also are similar, supporting the simpler tool (the mGCS) for predicting outcomes.[34] The highest mGCS within the first 24 hours post-injury has been shown to improve the performance of risk-adjustment models for predicting sTBI mortality.[35] Currently, the mGCS score collected on arrival to the ED is used in select risk-adjustment models for external benchmarking of sTBI mortality.[33] Other research supports using a binary assessment of the mGCS of less than 6 (indicating that the patient does not follow commands) versus 6 as a predictor of serious injury instead of the full GCS because it is easier to calculate, especially in the field, and therefore may simplify prehospital trauma triage.[36,37]

Despite evidence suggesting the superiority of using the mGCS as a simpler, effective tool for predicting injury severity, the mCGS has not been uniformly accepted, and there are concerns regarding inter-rater reliability and how to most accurately communicate physical examination findings. When using the mGCS, a decline in motor function should be considered a signal of worsening injury until determined otherwise. Because there are numerous scoring metrics for describing motor strength and function, institutions should be careful to adopt accurate and nonconflicting nomenclature for nurses to use when documenting physical findings to avoid confusion in how motor scoring is obtained and interpreted, regardless of which version of the GCS used.[38]

The Full Outline of UnResponsiveness (FOUR) Score

Evaluation of neurological status should drive clinical decision making and guide patient care–related goals. Although the GCS examination is extensively used in assessing the patient with an sTBI, there are shortcomings in its accuracy in certain types of patients, as mentioned in the previous section.

In 2005, Wijdicks et al. developed the Full Outline of UnResponsiveness (FOUR) score, an alternative assessment approach to address the limitations of the GCS exam (**Table 3**).[39] The FOUR score measures eye movement, motor function, brain stem reflexes, and respiratory pattern. Each of the four components is scored ranging from 0 to 4, with higher scores representing higher neurologic function. Unlike the GCS, each component of the FOUR score carries equal weight so the total score is not skewed by specific assessment parameters. In addition,

Table 3. FOUR Score Assessment[39]

Score	Eye Response	Motor Response	Brain Stem Reflexes	Respiration
4	Eyes open, tracking, or blinking on command	Thumbs-up, fist, or peace sign	Pupil and corneal reflexes present	Not intubated, regular breathing pattern
3	Eyes open but not tracking	Localizing to pain	One pupil is wide and fixed	Not intubated, Cheyne-Stokes breathing pattern
2	Eyes closed but open to loud voice	Flexion response to pain	Pupil or corneal reflexes absent	Not intubated, irregular breathing
1	Eyes closed but open to pain	Extension response to pain	Both pupil and corneal reflexes absent	Breathes above ventilator rate
0	Eyes remain closed with pain	No response to pain	Absent pupil, corneal, or cough reflex	Breathes at ventilator rate or apnea

the FOUR score measure enables evaluation of brain stem function in varying patient types, including those unable to provide verbal communication.

Evaluation of both eye opening and eye tracking provides additional insight into LOC (especially minimal consciousness) and may help clinicians differentiate between patients in a vegetative state versus those with cerebromedullospinal disconnection (also known as "locked-in" syndrome or pseudocoma). Cerebromedullospinal disconnection results in total paralysis of voluntary muscles, except for those responsible for vertical eye movement. It is important to ask patients who appear to be in a comatose state to look up and down (while holding their eyelids open). Patients with "locked-in" syndrome are conscious and aware of their surroundings, situation, and what is being said, although they are paralyzed and unable to talk. They hear, see, feel, smell, taste, and think. It is important that nurses and other members of the healthcare team acknowledge this and are cognizant about bedside discussions in the presence of the patient and avoid unnecessary application of noxious stimuli. Incorporating evaluation of brain stem function and respiratory pattern can help facilitate recognition of herniation syndromes.

A systematic review of the literature supports the usefulness of the FOUR score as an outcome predictor for many types of patients with altered LOC that has good inter-rater reliability between physicians and nurses.[40,41] The FOUR score is comparable to the GCS score in predicting short-term mortality, and a prospective cohort study found that both discharge GCS and FOUR scores correlated with long-term outcomes after hospital discharge.[42–44]

Although the FOUR score addresses many of the limitations of the GCS, it may not be used as often because of the additional training required to accurately complete the examination and decrease the variability in scoring among members of the healthcare team.

Nursing Assessment of the Patient with sTBI in the ICU

The neurologic examination remains fundamental in monitoring and guiding care for patients with sTBI. Basic components of the clinical neurologic examination in the patient with sTBI include serial evaluations of LOC, pupil evaluation, pertinent cranial nerve assessments, evaluation of sensory function, and assessment of best motor response. However, the neurological examination should be individualized to the patient and clinical circumstance.[18] A full assessment also should be completed because patients may have other injuries. The head and neck should be inspected for evidence of trauma with special attention to the ears (otorrhea), the nose (rhinorrhea), Battle's sign (retroauricular hematoma), or raccoon's eyes (periorbital ecchymoses)—indicators of potential basilar skull fracture. Prior to performing appropriate assessments, radiographic evaluation for spinal cord injury (SCI) should be performed because of the high rate of comorbid injury to the spine.

Upon admission to the ICU, the nurse should perform a neurologic examination, compare it with the assessment performed in the ED, and establish an assessment baseline for detecting neurologic changes. It is important to use a consistent approach to aid in the detection of changes during serial examinations.

During the initial treatment of a patient with trauma in the ED, placement of an ICP monitoring device may not occur. Therefore, upon arrive to the ICU, the RN should be prepared to assess for signs and symptoms indicating the need for invasive ICP monitoring.[23]

Pupil Examination

Assessment of pupil size and reactivity are essential clinical parameters in monitoring patients with sTBI. The pupillary light reflex assessment evaluates the functional ability of the optic and oculomotor cranial nerves (cranial nerves II and III, respectively). Clinical evaluation of the pupils focuses on four characteristics: size, reactivity to light, shape, and presence of anisocoria (unequal pupils). Of these, changes in pupil diameter or the development of anisocoria in the patient with sTBI often is the most concerning.

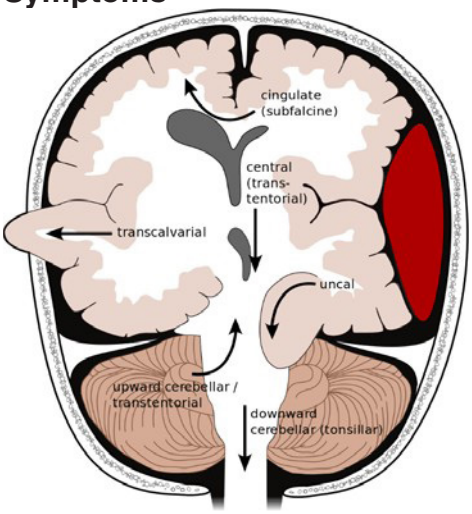
It is important to perform serial pupil examinations at the scene of the injury, in the ED, and at frequent intervals in the ICU. Pupil assessment findings may provide valuable information about the severity and progression of the injury, as well as information on the location of injury and brain function. For example, a dilated and nonreactive pupil requires immediate attention, because it signals compression of the third cranial nerve, signaling a potential midline shift or uncal herniation (**Figure 3**). Although pupillary abnormalities often indicate increased ICP associated with progression of hemorrhage or cerebral edema, several other factors may result in an abnormal pupil response, as outlined in **Table 4**. Acute pupil changes should be reported immediately because other diagnostic tools such as an emergent computed tomography (CT) scan or continuous ICP monitoring along with immediate interventions to correct the underlying problem may be warranted.

Table 4. Common Pupillary Finding in Patients with sTBI[45–47]

Pupil Abnormality	Assessment Findings	Pathophysiology
Asymmetric pupils	Often presents as a fixed and dilated pupil; however, it is important to determine whether the larger pupil is dilated or the smaller pupil is constricted. In mydriasis, the affected pupil is dilated and nonreactive to light.	• Asymmetric pupils usually indicate a structural lesion. In TBI, this is most commonly caused by compression of cranial nerve (CN) III as it passes between the posterior cerebral and superior cerebellar arteries, resulting in ipsilateral pupil dilation associated with herniation. It also can be caused by a posterior communicating aneurysm, a defect in the efferent pathway, or direct trauma to the nerve endings of the sphincter muscle of the iris. • The first clinical sign of CN III compression is pupil dilation, because the parasympathetic fibers are located on the outside of the nerve and are inactivated first by compression. This often occurs prior to any eye movement abnormality. As the compression continues, complete third nerve paralysis may occur. With further herniation progression, the contralateral oculomotor nerve may be compressed, producing bilateral pupil dilation. • A paradoxical unilateral dilation of the pupil on the side opposite the lesion may occur with subdural or intraparenchymal hemorrhage.
	With Adie's pupil, the affected pupil slowly constricts to prolonged light exposure and slowly dilates in the dark. Accommodation is sluggish.	• Adie's pupil usually is caused by parasympathetic denervation of the afflicted pupil.

Asymmetric pupils (continued)	In Horner's syndrome, the affected pupil is smaller than the other and does not immediately respond to direct light or to accommodation. Ptosis and, depending on the level of injury, loss of sweating may be present on the ipsilateral side of the face.	• Horner's syndrome is caused by a deficiency of sympathetic activity. This may be caused by (but is not limited to) cervical dissection, chest or cervical trauma, infection, ischemia to the medulla or hypothalamus, or cavernous sinus thrombosis.
	Varied	• Other causes of unilateral pupil dilation include medication effects or postsurgical pupil
Pinpoint (or constricted) pupils	In pinpoint pupils, also known as abnormal miosis, both pupils are like pinpoints and are too small for nurses to visually observe their reactions to light.	• This occurs as the result of parasympathetic stimulation or a disruption in the sympathetic pathway. This can be caused by pontine or intracranial hemorrhage, opioid use, organophosphate poisoning, clonidine overdose, pilocarpine eye drop use, and occasionally from mirtazapine or olanzapine. • Hypothermia also can cause small, unreactive pupils.
Nonreactive, dilated pupils	Both pupils are dilated with no direct or consensual light reflex and no response to accommodation. This is known as mydriasis.	• This often is an ominous sign of brain anoxia and brain death. • Midposition unreactive pupils result from lesions affecting both sympathetic and parasympathetic pathways, such as in central transtentorial herniation. • Abnormally shaped pupils suggest a midbrain lesion. • Bilateral large, unreactive pupils that display hippus or dilate with neck scratching suggest a tectal or pretectal (midbrain) lesion. • Drug-induced mydriasis may occur from hallucinogens, antihistamines, amphetamines, anticholinergics, dopamine, and barbiturates, as well as ophthalmic mydriatics administered in intraocular examinations (such as atropine or scopolamine). • Reactive pupils in coma help to distinguish metabolic from a structural coma.
Equal pupils with abnormal response	Characterized by spasmodic, rhythmic, but regular dilating and contracting pupillary movements	• Although hippus often is benign, in the presence of TBI, hippus can indicate frontal lobe injury, compression of CN III, a lesion on or injury to the midbrain, or barbiturate toxicity.
	Marcus Gunn pupil, also known as the relative afferent pupillary defect (RAPD), is seen during the swinging-flashlight test. The pupils dilate when a bright light is swung from the unaffected eye to the affected eye. The affected pupil will have a sluggish response to direct light with an intact consensual reflex. When the light is reshown into the affected pupil, the pupil will dilate.	• RAPD usually is caused by ischemia or infection of the optic or retinal nerves, retinal detachment, or severe macular degeneration.

Figure 3. Herniation Syndromes with Common Corresponding Signs and Symptoms



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Pupil size is measured in millimeters with the mean pupil diameter ranging from 2 mm to 6 mm.[48] A standardized measurement tool, such as a pupil gauge or automated pupillometer, should be used to decrease subjectivity, especially with serial measurements. Although most people have equal pupils, a discrepancy of less than 1.0 mm is considered within normal range.[48] Up to 20% of the noninjured population will have unequal pupils at baseline.[48] If in doubt about the cause of unequal pupils, asking family members about the patient's baseline may be helpful in determining causation and urgency.

When assessing the pupils, the nurse should instruct the conscious patient to open their eyes and focus on a distant and straight-ahead object. In a comatose patient, the nurse will need to gently lift the upper eyelids to evaluate the pupils. The nurse should inspect each pupil for size, shape, and reactivity to light (testing both the direct and consensual reflex), while comparing the two pupils for equality. Pupil size should be assessed before and after the pupil responds to direct light from a penlight. Reactivity to direct light is assessed by shining a low-beam penlight inward from the outer canthus of each eye, checking each eye individually. A penlight is preferred to use of a flashlight when manually checking the pupils. Do not shine the light directly into the pupil because the glare or reflection may obscure visualization. The speed of pupillary reactivity is recorded as brisk, sluggish, or nonreactive. A sluggish or slow pupillary response may indicate increased ICP. Nonreactive pupils often are associated with severe increases in ICP or severe brain damage. The consensual light reflex is when the opposite pupil constricts during the direct light assessment of the targeted pupil.

Although the pupillary assessment is an important component of serial neurological examinations, studies have demonstrated poor intra- and inter-rater reliability in manual examinations.[49–51] Clinicians may subjectively underestimate pupil size, miss anisocoria, or incorrectly classify pupil reactivity and often are inconsistent with their own assessments (poor intra-reliability).[51] Use of automated pupillometry is an alternative to manual assessment of the pupil light response.[52] This noninvasive tool is low cost, simple to use, and easy to interpret, and it may provide more objective and earlier measurements of clinical worsening, such as subtle pupillary changes. Automated pupillometry has been shown to detect a pupillary light reflex in 66.7% of pupils scored as "nonreactive" by practitioners.[53,54] These devices provide a variety of measures of pupil size and reactivity, including maximum and minimum size (measured in millimeters to the nearest 100th), constriction velocity (CV) (measured in millimeters per second and calculated as the amount of constriction divided by the duration during which the pupil remains constricted), latency (time from light stimulus until the start of constriction), and the neuropupillary index.[54] Derived by comparing output from a mathematical algorithm obtained from normal healthy volunteers, the neuropupillary index ranges from 0 to 5 and is a comparison of the response of the patient to normal responses.[55] A neuropupillary index value greater than 3.0 is considered normal, whereas neuropupillary index values less than 3 are considered abnormal and are associated with intracranial hypertension or increased ICP.[51,52] A neuropupillary index of zero indicates a fixed pupil (absent pupillary reflex).[55,56]

Initial neurological pupil index values have been shown to correlate with GCS score in patients with sTBI, with good sensitivity and specificity in predicting clinical outcome at 1 month post-injury, and may have potential as a prognostic indicator.[57] A single-site, prospective cohort study found that CV and neuropupillary index values correlate with ICP values, suggesting that automated pupillometry could serve as an adjunct to traditional invasive neuromonitoring, although replicated studies are needed.[44] This is further supported by a secondary analysis that examined the relationship between ICP values and serial pupillometer readings in the first 72 hours of ICU admission and reported corresponding trends in pupillometry readings and ICP elevations in the absence of invasive monitoring.[58] There is high inter-device and inter-rater reliability among individual pupillometers of the same brand, suggesting

that they can be used interchangeably and are not user dependent.[59]

Although several prospective studies have validated automated pupillometry as a superior method of accurately assessing pupil size and reactivity compared to manual assessments, studies demonstrating an impact on intermediate and long-term patient outcomes when using pupillometry compared to manual pupil assessments in sTBI are lacking.[54,60] Although using an automated pupillometer has its advantages, it is important to note that a systematic approach to implementation may be needed to support routine use,[56] and readings may be inaccurate or difficult to obtain in some cases, such as when a patient moves and in patients with periorbital edema, cataracts, or a prosthetic eye.[53]

In addition to a pupillary examination, the neuroscience nurse should assess extraocular movements and check for field deficits. It is not uncommon for patients with sTBI to have an interruption to the pathways innervating the extraocular muscles controlling eye movements, as well as areas of the pathways controlling the ability to focus and see. **Table 5** outlines abnormal eye movements in brain injury and coma. In addition, **Figure 4** includes the location and type of visual field cuts seen in brain injury.

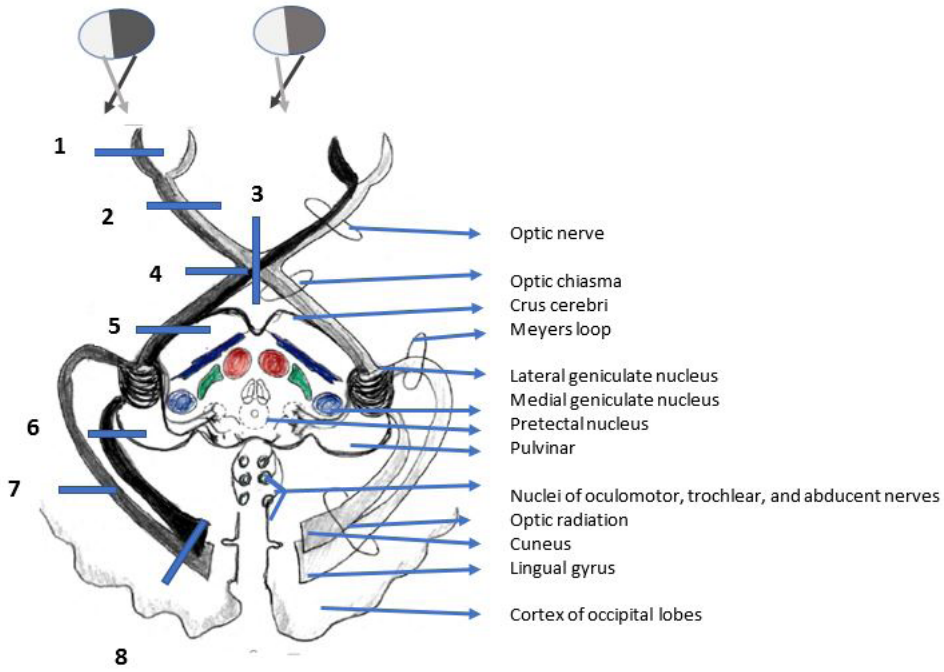
Table 5. Abnormal Eye Movements in Brain Injury and Coma[7,47]

Clinical Finding	Location of Lesion	Key Findings
I. Deviation at rest		
Bilateral conjugate deviation	• Frontal lobe (frontal eye fields or seizure)	• Reflex eye movements normal • Deviation toward side of injury • Deviation toward seizure foci (often accompanied by nystagmoid jerking)
	• Pons	• Deviation away from lesion • Calorics impaired on the side of lesion
	• Medial thalamic hemorrhage	• Medial thalamic hemorrhage
	• Brain stem	• Vertical gaze deviation, the most common is a sustained down-gaze with an up-gaze deficit associated with upper midbrain or caudal thalamic lesions
	• Hepatic encephalopathy	• Down-gaze deviation
	• Thalamic or midbrain pretectal lesion or post-seizure	• Downward deviation gaze with unreactive pupils, Parinaud's syndrome
Unilateral	• Uncal herniation • Cranial nerve (CN) III palsy	• Unilateral outward deviation on side of larger pupil (CN III palsy) • Pupil larger on the side of the palsy
	• CN VI palsy	• Unilateral inward deviation

Table 5. Abnormal Eye Movements in Brain Injury and Coma[7,47] *(continued)*

Clinical Finding	Location of Lesion	Key Findings
Skew deviation	• CN III or IV nerve or nucleus lesion	• Reflex eye movements normal • Deviation toward side of injury • Deviation toward seizure foci (often accompanied by nystagmoid jerking)
II. Spontaneous eye movement		
Random roving conjugate eye movement	• Metabolic/toxic encephalopathy most likely • Can occur with bilateral lesions above the brain stem.	• Indicates an intact CN III nucleus and medial longitudinal fasciculus • Also known as "windshield wiper eyes" • Roving eye movements in early coma cannot be mimicked and their presence excludes psychogenic unresponsiveness. • With depending coma, roving eye movement will disappear, followed by the oculocephalic response and the oculovestibular reflex.
Periodic alternating gaze	• Usually bilateral cerebral dysfunction	• Eye deviates side to side with frequency of approximately three to five movements per second (pausing 2–3 seconds in each direction)
Ocular bobbing	• Pons • Metabolic or toxic disorder	• Repetitive rapid vertical deviation downward with slow return to neural position • Note: If patient can close eyes or blink, the pons is intact.
III. Internuclear ophthalmoplegia (INO) and variations		
Disconjugate movements	• Medial longitudinal fasciculus (MLF) • CN III or VII pathway	• INO is associated with lesion in medical longitudinal fasciculus. • Eye ipsilateral to the MLF lesion does not adduct.
IV. Reflex eye movements (maneuvers to test brain stem)		
Oculocephalic reflex (doll's eye movements)	Brain stem reflexes	• The eyes should move conjugately in the direction opposite to the movement. An abnormal response (absent or asymmetric) implies brain stem disease. • Do not perform when neck instability is suspected. • Supratentorial lesions and metabolic processes usually do not affect this reflex, except for metabolic encephalopathy.
Oculovestibular reflex (caloric test)	Brain stem reflexes	• Normal response is tonic deviation toward the side of the irrigated ear (cold water). Warm water causes the opposite response. • Lack of response suggests sedative-hypnotic drug intoxication, structural lesion of the brain stem, or brain death. • Loss of reflex eye movement with preserved pupillary reactivity is diagnostic of drug toxicity.

Figure 4. Visual Field Cuts Associated with Brain Injury



Key	Visual field	Area of lesion	Clinical findings
1		Retina	Visual field defects of various patterns Scotoma
2		Optic nerve	Ipsilateral monocular visual loss Loss of pupillary light reflex in both eyes when light is shone in the left eye
3		Midline optic chiasm	Bitemporal May have relative afferent pupillary defect (RAPD)
4		Optic tract	Contralateral homonymous hemianopia May have RAPD
5		Lateral optic chiasm	Binasal hemianopia May have RAPD
6		Upper optic radiation – parietal lobe or cuneus	Contralateral homonymous inferior quadrantanopia
7		Lower optic radiation – temporal lobe or lingual gyrus	Contralateral homonymous superior quadrantanopia
8		Total optic radiation	Right homonymous hemianopia
8		Occipital visual cortex	Macular sparing contralateral homonymous hemianopia

Management of Patients with Increased ICP

Management of secondary brain injury in the ICU includes measures to avoid cerebral ischemia. Adequate cerebral blood flow (CBF) is a critical component to prevent cerebral ischemia and meet cerebral metabolic rate of oxygen ($CMRO_2$) demands.[7] CBF is difficult to quantify; however, invasive technologies in the ICU can provide indirect indications of CBF and perfusion. Measurement of ICP with invasive monitoring can provide timely information on factors impacting cerebral perfusion and facilitate early interventions in the setting of increased ICP to reduce the risk of secondary brain injury.[61,62] As ICP becomes elevated (usually defined as more than 20 mmHg), cerebral perfusion pressure (CPP) is reduced at any given mean arterial pressure (MAP).[7] In part, this is caused by impaired autoregulation, in which the brain becomes dependent on systemic blood pressure. The relationship between CPP, ICP, and MAP is as follows:

CPP = MAP – ICP.

Emergent ICP management of patients with sTBI is divided into two phases:

1. Acute management of the patient at high risk for or with impending brain herniation
2. Ongoing management of the patient with sustained elevated ICP.[56]

Current TQIP guidelines support ICP monitoring in patients who are comatose with a GCS score of 8 or less and if there is structural evidence of brain damage on CT imaging.[2] Monitoring of ICP is associated with lower in-hospital mortality rates and remains a standard of care for sTBI.[2] **Table 6** outlines other assessment findings associated with the potential need for ICP monitoring.

Table 6. Assessment Findings that Indicate the Potential Need for ICP Monitoring[7,63]

Any of the following indicate the potential need for ICP monitoring:
• Dilated and nonreactive or asymmetric pupils
• Extensor posturing or no motor response
• Progressive decline in neurologic exam
o Decrease in Glasgow Coma Scale (GCS) score >2 points
• Cushing's response
o Increased blood pressure (BP)
o Tachycardia followed by bradycardia
o Irregular respirations
• GCS score <8 and abnormal computed tomography (CT) scan
• GCS score <8 with normal CT scan and two or more of the following:
o Age >40 years
o Motor posturing
o Systolic BP <90 mmHg
• GCS score 9–15 with CT scan that demonstrates any of the following:
o Mass lesion
o Effaced cisterns
o Midline shift >5mm
• Following select surgical interventions
• Multiple systems injured with altered level of consciousness, especially where therapies for other injuries may have deleterious effects on ICP, such as high levels of positive end-expiratory pressure or the need for large volumes of intravenous fluids or heavy sedation
• With traumatic intracranial mass (such as epidural hematoma, subdural hematoma, depressed skull fracture)

Monitoring Tools and Devices

There are different mechanisms for monitoring ICP in the ICU. An external ventricular drainage (EVD) device with a dual lumen ventriculostomy catheter that allows for both ICP monitoring and cerebrospinal fluid (CSF) drainage often is the preferred device for managing ICP for most patients.[64,65] This device allows for CSF drainage to help control or manage increased ICP. If placement of an EVD device is not possible, insertion of a subarachnoid, subdural, or parenchymal bolt or catheter can provide ICP monitoring without drainage.

Although invasive ICP monitoring is important, it does not replace careful neurological and radiographic examination.[2] Therefore, patients with sTBI, including those requiring ICP monitoring, should be carefully assessed for signs and symptoms of increased ICP and impending brain herniation.[63] Cerebral monitoring devices may be inserted at the time of a surgical intervention (e.g., craniotomy, craniectomy, etc.) or in isolation.

Although not a universal standard of care, additional invasive cerebral monitoring may be available for advanced monitoring in some ICUs to measure cerebral autoregulation, cerebral blood flow, and cerebral oxygenation.[66] These methods are further described in **Table 7**.[1] Data obtained from cerebral monitoring devices (including ICP or EVD devices) should be evaluated in conjunction with the neurologic examination and not considered in isolation.[1,61,65] Astute nursing care is essential because invasive cerebral monitoring devices have the potential for serious complications, such as brain infection and sepsis.

To provide accurate readings, a consistent method for calibrating arterial pressure, MAP, and ICP is required, including standardizing the degree to which the head of the bed is elevated.[71,72] Maintenance of goal CPP may require meticulous titration of intravenous (IV) vasoactive medications, especially when cerebral autoregulation is impaired.

Other treatment goals include adequate oxygenation, normocapnia, normothermia, and avoidance of hyponatremia (sodium level below 135 mEq/L).[18] Besides potentially increasing cerebral edema, hyponatremia is associated with metabolic complications such as diabetes insipidus, cerebral salt wasting, and syndrome of inappropriate antidiuretic hormone.[2,73] **Table 8** provides insight into these disorders of sodium balance.

Related Metabolic and Physiological Parameters

Cerebral metabolism, ICP, and CPP also are affected by[16]

- hyperthermia (temperature > 37.5° or 38.0° C, although this number is controversial)[16,18]
- hypothermia (temperature < 35.5° C)
- hypocapnia ($PaCO_2$ < 35 mmHg)
- hypoxemia (PaO_2 < 60 mmHg; O_2 Saturation < 90%)
- hypotension (systolic blood pressure [SBP] < 90 mmHg)
- hypercapnia ($PaCO_2$ > 45 mmHg)
- hypertension (SBP > 160 mmHg, or MAP > 70 to 110 mmHg)
- anemia (hemoglobin < 100 g/L or hematocrit < 30%)
- hyperglycemia (blood sugar > 180 mg/dL)
- hypoglycemia (blood sugar < 80 mg/dL)
- hypo-osmolality (plasma osmolality < 290 mOsm/Kg H_2O)
- hyponatremia (serum sodium < 142 mEq/L)
- acid-base disorders (acidemia: pH < 7.35; alkalemia: pH > 7.45). Note: acute alkalosis increases the binding of calcium, which can lead to ionized hypocalcemia with tetany.
- seizures.

Therefore, nursing interventions should be aimed at maintaining normothermia, normoglycemia, targeted osmolarity, and serum electrolyte levels, as well as ensuring adequate oxygenation. There also should be a focus on achieving hemodynamic targets, treating anemia, maintaining targeted $PaCO^2$ goals, and assessing for seizure detection and prevention.

Throughout the ICU hospitalization, the neuroscience nurse should closely monitor the patient for signs and symptoms of increased ICP. **Table 9** outlines some of the most common signs and symptoms of increased ICP. Clinical signs of brain herniation or impending brain herniation include[63]

- unilateral or bilateral dilated pupil(s)
- loss of consciousness or decline in mental status
- abnormal posturing (involuntary flexion or extension of the arms and or legs)
- hypertension and bradycardia.

More specific signs and symptoms associated with the different types of brain herniation syndromes are further described and depicted in **Table 10** and Figure 3.

Table 7. Advanced Monitoring to Measure Cerebral Blood Flow, Brain Oxygenation, and Cerebral Metabolism Along with Electrophysiological Measures in the Treatment of sTBI[67–70]

Cerebral blood flow (CBF) monitoring	
Parenchymal thermal diffusion flowmetry	A probe with two thermistors set at different temperatures is placed; the rate of temperature dissipation from applied heat is then calculated. Increased heat dissipation indicates greater blood flow. Requires precise positioning of probe through a bolt.
Transcranial doppler	Noninvasively measures blood flow velocities of intracranial arteries via ultrasound. Most often used in the subarachnoid population.
Brain oxygen monitoring	
Jugular venous oxygenation ($SjVO_2$)	Catheter inserted into dominant internal jugular and then advanced superiorly to the jugular bulb. It provides a measure of global cerebral oxygen use. Normal values range between 55% and 75%. A $SjVO_2$ <55% indicates hypoperfusion or an increase in metabolic demand, thus suggesting cerebral ischemia.
Partial pressure of brain tissue oxygenation ($PbtO_2$) or brain tissue oxygen tension ($PbrO_2$)	Catheter inserted into brain parenchyma monitoring allowing for continuous real-time measure of brain tissue oxygen. PbtO is a marker of the balance between supply and consumption. Compromised PbtO (<20 mmHg) should be treated because it is associated with worsening brain injury.
Near-infrared spectroscopy (NIRS)	Used in the operating room to indicate global perfusion changes. Measures regional cerebral oxygen saturation by measuring the amount of light attenuation between an NIRS light source and receiver and comparing light spectra absorption from deoxyhemoglobin and oxyhemoglobin.
Cerebral metabolism monitoring	
Cerebral microdialysis	Catheter inserted into brain parenchyma to detect biochemical changes (e.g., glucose, lactate, pyruvate, glutamate, glycerol, pH, and more) in extracellular substrates or regional subcortical white matter. Consistently low glucose concentrations (<0.66 mmol/L) are associated with poor outcome. A lactate to pyruvate ratio >25 is a marker of metabolic distress.
Positron emission tomography	Noninvasive method to evaluate cerebral metabolism. Used mainly in research.
Magnetic resonance imaging spectroscopy	Noninvasive method to measure lactate content
Electrophysiological measurements	
Quantitative electroencephalogram (EEG)	Most useful when continuous, depicts brain electrical activity that is then converted to a digital form.
Intracortical depth electrodes	An invasive monitor inserted into the brain parenchyma. Useful in detecting seizures and cortical spreading depression not detected on scalp EEG.
Evoked potentials	Evoked potentials are the electrical manifestation of the brain's response to an external stimulus (such as an electrical stimulus applied to the median or tibial nerve) and can provide information regarding the functional integrity of sensory pathways.

Table 8. Syndrome of Inappropriate Secretion of Antidiuretic Hormone, Cerebral Salt Wasting, and Diabetes Insipidus in TBI

Parameter	Syndrome of Inappropriate Antidiuretic Hormone[74,75]	Cerebral Salt Wasting[75,76]	Diabetes Insipidus[77]
Serum sodium	<135 mEq/L	<133 mEq/L	>145 mEq/L
Urine sodium	>25 mEq/L	>40 mEq/L	<25 mEq/L
Serum osmolarity	<270 Osm/kg	>300 mOsm/kg	>285 mOsm/kg
Urine osmolarity	>300 mOsm/kg	>300 mOsm/kg	<300 mOsm/kg
Urine output	Decreased	Increased	Increased
Cerebral venous pressure (preload)	Normal to high	Low	Low to normal
Plasma antidiuretic hormone	High	Normal	Low
Treatment highlights	Fluid restriction Sodium Demeclocycline Vasopressin antagonist such as conivaptan	Give fluids Sodium Fludrocortisone	Drink to thirst or intravenous fluids (such as in coma) Desmopressin (DDAVP [central]) Vasopressin Diuretics such as hydrochlorothiazide (nephrogenic)

Table 9. Early and Late Signs and Symptoms of Increased ICP[23,56]

Early	Late
• Headache • Increasing confusion • Decreased mental abilities • Carphologia (nonpurposeful movement/psychomotor agitation) • Nausea • Vomiting • Double vision • Generalized weakness or fatigue • Intractable yawning, hiccoughing, and air hunger • Amaurosis, blurred vision, nystagmus, eye deviation, facial twitching	• Seizures • Unequal and or unreactive pupils • Loss of consciousness • Impairment of brain stem reflexes • Extensor posturing • Cushing's triad o Hypertension o Bradycardia o Irregular respirations or apnea

Table 10. Signs and Symptoms of Herniation Syndromes[56]

Type of Herniation	Description	Common Signs and Symptoms
Cingulate or subfalcine	Also known as a midline shift, this is the most common type of cerebral herniation pattern. Generally caused by unilateral mass effect in the frontal, parietal, or temporal lobe(s) leading to a medial shift of the ipsilateral cingulate gyrus beneath the free edge of the falx cerebri. Large lesions may lead to uncal or central herniation.	• Initial presentation can be benign and may include headache and apathy. • Signs of ipsilateral anterior cerebral artery ischemia such as contralateral leg weakness • If the contralateral arcuate fasciculus is involved, signs of expressive, receptive, or conductive aphasia may be present.
Central transtentorial	A downward displacement of medial brain structures through the tentorial notch by a supratentorial mass that exerts pressure on the underlying structures, such as the brain stem	• Early signs include increased confusion and headache leading to decreased level of consciousness leading to coma, including loss of reflexes and seizures, and eventual death. • Motor weakness leading to abnormal posturing • Fixed and dilated pupils
Uncal	A subtype of central transtentorial herniation related to the cerebral mass effect from increasing intracranial pressure. The uncus and the adjacent part of the temporal lobe slide downward across the tentorial incisura compressing the brainstem and the posterior cerebral arteries in the ambient cistern. May be unilateral or bilateral. Associated with poor prognosis because of the direct compression of vital midbrain centers.	• Initially presents with an ipsilateral dilated pupil that is unresponsive to light as a result of ipsilateral cranial nerve III (oculomotor nerve) compression. • May develop into bilaterally blown pupils. • Lateral or vertical gaze eye deviation and ptosis may occur. • Altered mental state leading to coma • Contralateral hemiparesis • If the ipsilateral posterior cerebral artery is affected, homonymous hemianopsia may occur
Upward transtentorial	Also known as ascending transtentorial herniation, this occurs where space-occupying lesions in the posterior cranial fossa cause superior displacement of superior parts of the cerebellum through the tentorial notch.	• Nausea or vomiting • Rapid progression toward a decreased level of consciousness and, eventually, death
Tonsillar	Also known as downward cerebellar herniation, this syndrome is caused by the inferior descent of the cerebellar tonsils below the foramen magnum.	• The brain stem is compressed against the clivus thereby altering the vital life-sustaining functions of the pons and medulla, such as the respiratory and cardiac centers, leading to respiratory and cardiac depression and death. • The most feared because of its rapid progression
Transcalvarial	Also known as an extracranial brain herniation, this occurs when brain tissue external to the calvaria herniates through a skull bone defect associated with surgery or trauma.	• Varies; the herniated brain tissue is at risk of ischemia and venous infarction.

Pharmacotherapy for Increased ICP

Both mannitol (0.5–1 g/kg) and hypertonic saline (HTS) (2%–23.4%) may be used to treat increased ICP and usually readministered as intermittent boluses.[1,78] It is important to use an inline filter with mannitol because the medication can crystalize, and to use a central line when infusing saline in concentrations greater than 3%–7.5% (depending on reference and local policy).[79,80] Both medications usually are given as a bolus and facilitate the movement of water into the vasculature. Nursing interventions include monitoring serum osmolarity (with goal of less than 320–340 mOsm/kg or per physician orders), fluid status, renal function, and serial serum sodium levels, especially with HTS administration.[18,81] Diligent monitoring is required when administering HTS to patients with low serum sodium levels (especially chronic hyponatremia), as an overcorrection that is too fast can lead to osmotic demyelination syndrome.[81] The osmolar gap (the difference between osmolality measured and osmolarity calculated) should be measured in patients receiving mannitol. The formula is as follows[82]:

Calculated osmolality = ([2Na] + [glucose/18] + [BUN/2.8] + [ETOH/4.6*])

Where Na = sodium

- BUN = blood urea nitrogen
- ETOH = blood alcohol level
- Normal range for calculated osmolality = 280 to 300 mOsm/kg H_2O
- A gap is equal to or greater than 10 mOsm/kg H_2O (suggesting exogenous osmoles), with normal being less than 10 mOsm/kg H_2O.

*Some versions of this equation do not include the correction for blood alcohol.

Mannitol should be stopped if the osmolar gap exceeds 20 mOsm/kg H2O. Mannitol can cause renal failure in high doses, whereas HTS may cause volume overload. Both can cause metabolic alkalosis hypokalemia and hypochloremia.

Head of Bed Elevation

Head-of-bed elevation and positioning of the head in a neutral (midline) position to facilitate venous drainage is a common, simple, and cost-effective intervention to decrease ICP and optimize CBF.[83–87] Initial management for patients with sTBI includes head elevation at a minimum of 30 degrees,[88] monitoring ICP and CPP, and maintaining established care goals.[22,64,65,86,87,89]

Changes in patient ICP and CPP often occur with patient repositioning, and can produce transient increases in ICP, particularly in the setting of increased ICP.[45,88,90,91] Sharp head rotation and prone positioning also may increase ICP.[85,92] Patient response to repositioning can be varied; therefore, individualized plans of care and clustering of activities should be evaluated for impact on ICP, CPP, and CBF response.[62]

C-Spine Support

ENLS guidelines currently recommend cervical-spine (c-spine) immobilization as a vital component of prehospital and initial hospital management prior to appropriate assessment and radiographic evaluation of SCI because of the high rate of comorbid injury.[22] It is important to ensure any collar used fits the sTBI patient appropriately because mal-fitting or tight-fitting collars may increase ICP by impeding cerebral venous outflow and decreasing CBF, which can exacerbate secondary brain injury.[84,89,93] Nurses should ensure the use of appropriately sized and correctly fitted cervical collars to lessen these deleterious effects.[87] Cervical collars and other types of neck braces also can cause skin injury, impact respiratory effort, and contribute to the patient's pain and discomfort.[94] Nurse interventions should include ways to lessen pressure on the skin that is exerted by cervical collars and aim to remove these devices as soon as it is deemed safe.[95]

General nursing care for the patient with a cervical collar includes removing and reapplying the cervical collar at regular intervals (with assistance in maintaining the neck and head in a neutral position) to provide skin assessments and skin care. Besides cleansing the skin, the pads inside the collar should be cleaned or replaced at this time. When performing this procedure, the nurse should pay close attention to brace tightness and monitor for changes in ICP and CPP with collar reapplication and patient repositioning.

In patients with low-velocity gunshot wounds to the head, routine cervical immobilization may not be necessary.[96] An individualized approach to selecting softer boards and vacuum mattresses for cervical spine immobilization may avoid ICP elevation and improve patient comfort.[97]

Noxious Stimuli

Although there is limited research on patient room dynamics, controlling the patient environment by limiting noxious stimulation has been an intervention aimed at minimizing adverse fluctuations in ICP and CPP values for sTBI patients.[49,89] Examples of noxious stimuli that are amenable to nursing interventions include[89]:

- uncomfortable or painful stimuli
- loud noises and voices
- sudden jarring of the bed

- sounds from bedside equipment
- bright overhead lights
- components of a neurologic assessment
- painful medical or nursing procedures.

It is important to note that limiting environmental stimulation may have varying effects on ICP, with some studies indicating minimal adverse effects.[49] However, it is important for families and healthcare professionals to assume patients have intact hearing and therefore avoid having disturbing conversations within hearing distance of the patient.[88] Nurse and family talking with the patient has rarely been shown to independently increase ICP or reduce CPP.[85] In fact, family member conversations and families talking to the patient (familiar voices) has been shown to more likely decrease ICP, where nurses talking to patients has been shown to have no significant impact on ICP.[49] When ICP is monitored, individual patient responses to each patient care intervention should be evaluated and used to guide subsequent care.[85]

Other Registered Nursing–Related Care

Key points for the neuroscience nurse caring for patients at risk for increased ICP include

- providing basic nursing care
- assessing for neurologic changes
- assessing for other injuries, including signs and symptoms of scalp, facial, spine, intra-abdominal, and long-bone injuries
- closely monitoring vital signs, ICP, and CPP[4,66,85]
- avoiding hypotonic fluids (such as dextrose 5% in water)
- prioritizing or altering patient care based on changes in ICP and CPP[62,86,98]
- carefully considering the impact of nursing care related to patient positioning, neck brace care, environmental noise, and stimuli on ICP and CPP
- maintaining invasive monitoring device systems
- standardizing or protocoling nursing interventions to improve compliance with neuroscience nursing care delivery, including the aseptic maintenance of invasive monitoring device systems[87]
- reporting findings and concerns to the appropriate healthcare provider
- early recognition and responding to paroxysmal sympathetic hyperactivity, also known as "sympathetic storming," a strong physiologic response that may be triggered by nursing care and often is characterized by agitation, clenching of fists, grinding of teeth, profuse sweating, sustained tachycardia, and marked hypertension. Initial treatment includes removing external triggers, such as noxious stimuli, body turning or movements, and bladder distention, before administering medications. The most useful pharmacologic agents are morphine sulfate and nonselective β-blockers (e.g., propranolol).[99]
- avoiding hyperventilation, which may exacerbate cerebral ischemia, especially in the first 24 hours.[18]

The nurse also should integrate and promote the use of evidence-based, standardized protocols for the care of the patient with sTBI because many are correlated with improved neurological outcomes and decreased mortality at 6 months post-injury.[100]

Neurosurgical Interventions

Neurosurgical interventions for sTBI patients may extend beyond the placement of monitoring devices and ventricular catheters and target both primary and secondary injury associated with sTBI. Common neurosurgical interventions include evacuation of intracranial hematomas, such as epidural and SDHs (note: any symptomatic posterior fossa mass lesion or those with mass effect on CT should be emergently removed); removal of foreign objects; correction of skull defects (such as depressed skull fractures); spine stabilization; correction of hydrocephalus; endovascular treatment of carotid or vertebral dissection as well as vasospasm; interventions for pneumocephalus; and decompressive hemicraniectomy with duroplasty (further described below).[7]

Ongoing neurological assessments and treatment of increased ICP remain top nursing responsibilities postoperatively. Occasionally, the patient may have a drain placed during surgery. Nursing care includes assessing and labeling the location of each drain, ongoing monitoring and measurement of drainage, and maintaining patency of the drain. In general, drains in the subdural space are either drained to gravity or to partial bulb suction, whereas subgaleal drains can be placed to full bulb suction. Do not place a brain drainage device to wall suction, as aggressive suction can tear vessels and cause hemorrhage. The provision of evidence-based routine postoperative nursing care is essential in preventing complications and maximizing positive patient outcomes.

Post-Hemicraniectomy with Duroplasty

Decompressive hemicraniectomy with duroplasty is an effective treatment for relieving severe refractory intracranial hypertension after sTBI.[101] During the procedure, a large portion of the skull is removed (may be unilateral or bilateral and usually involves the lateral skull), the dura is opened widely, and the scalp flap is closed over the

skull defect, thus allowing the injured brain to swell and expand through the cranial defect.[101]

The potential for short-term postoperative complications of this procedure requires astute and timely assessments by the neuroscience nurse. Potential complications include increased ICP, brain herniation through the bone window, surgical site infection, contralateral hematoma, ipsilateral subdural or subgaleal effusion, early seizure, CSF leakage, and hydrocephalus.[102] In addition, patients are at high risk for falls, which can be catastrophic because of the compromised skull and exposed cerebrum.[102]

Nursing interventions to promote recovery include protecting the exposed cerebrum through proper positioning, head protection, and helmet use when out of bed, as well as evidence-based fall prevention strategies.[89,102] In addition, routine wound assessment and care should include assessment of the wound for acute turgor and girth of the craniectomy site, indications of acute surgical bleeding, and CSF leakage.[31]

Longer-term complications of decompression craniectomy (DC) include sinking skin flap syndrome, also known as syndrome of the trephined.[102] Early symptoms can include a depressed mood, headache, behavioral disturbances, and seizures. Symptoms are related to cerebral cortex distortion under the skin flap, which can occur once cerebral edema subsides. More serious symptoms can include acute neurological changes and paradoxical herniation. Late complications may include ipsilateral effusion, late-onset seizure/epilepsy, and continued complications related to syndrome of the trephined.[102]

Bone flap replacement (cranioplasty) for DC typically is performed during a rehospitalization months after the initial surgery. Risks associated with replacement include infection, hematoma, hydroma, and bone flap resorption.[102] Targeted nursing care includes wound care, prevention of increased ICP, early detection of neurological changes, patient/family education, and care coordination.

Intrahospital Transport of Patients with sTBI

Patients with sTBI often require multiple intrafacility or intrahospital transports (IHTs) for diagnostic or interventional procedures, and can present potential patient hazards.[85] Clinically significant complications have been reported to occur in 36% of critically ill patients with brain injury during transport.[103] Complications include, but are not limited to, accidental extubation, equipment battery failure, increased ICP, and hyper- or hypotension.[103] Hemodynamic and respiratory instability in patients with sTBI are associated with elevations in ICP and decreased CPP, leading to extension of secondary brain injury, increased ICU length of stay, and increased mortality.[104]

Hazards encountered during IHT may result from inadequate planning prior to transport; the inability to maintain a consistent level of intensive care monitoring and assessments during transport; increased exposure to noxious stimuli; increased patient movement or repositioning; and ineffective means of treating changes in ICP, CPP, pain, or agitation levels. To prevent harm, a pretransport checklist prior to IHT is recommended.[105] Recommended items to include on a sample transport checklist are included in **Table 11**. During IHT, it is important to continue the same level of ICU patient monitoring and care.[105]

Transport staff should include nurses and/or clinicians trained in delivering immediate interventions for treating increased ICP and managing other adverse events.[103,105] ICP may increase during transport or during performance of the diagnostic or treatment modality itself.[85,106,107]

To prevent reflux of CSF during patient transport, EVDs typically are clamped during the transport period.[85] If the EVD is unclamped in the ICU prior to transport, the patient should be screened for ICP tolerance by clamping the EVD prior to transport to identify the potential risk of increasing ICP during IHT.[108] Increased ICP related to IHT is most often seen in critically ill patients with elevated baseline ICP values and in patients requiring continuous EVD diversion.[106] Premedication with analgesia or sedatives prior to IHT may help prevent or mitigate ICP elevation.[106]

Overview of Ventriculostomy Site Care

Healthcare-associated ventriculitis (HAV), also referred to as ventriculostomy-related infection or ventriculostomy-associated infection, has an incidence of up to 22%[109] or 11.4 infections per 1,000 EVD catheter days.[110] It is associated with increased morbidity, mortality, and treatment cost.[111] Infection may be introduced at the time of EVD placement[109] and during use of the device. Nursing interventions to prevent infection should include facilitating and monitoring adherence to an established evidence-based insertion protocol with use of a checklist to confirm compliance with insertion steps.[111-116] Several simple EVD insertion protocols with associated reductions in HAV have been published.[112,113,115,117-119] Three protocols published in the past 5 years are summarized in **Table 12**.

Post-EVD insertion, HAV may result from catheter and tubing contamination during manipulation, CSF sampling, or EVD colonization from skin or room contaminants.[109,120] Published insertion site care and maintenance procedures for ventriculostomy use vary greatly.[111,121] Not

Table 11. Intrahospital Pre-Transport Checklist[85,103,105,106,108]

Confirm the following prior to transport:
• Adequate battery supply for heart monitor and other transport equipment • Adequate oxygen tank levels for support • Adequate intravenous (IV) fluids, continuously infusing IV medications, sedatives, and analgesics • Consider a brief trial period of clamping external ventricular device (EVD) and laying the patient flat (simulates flat position during radiologic testing) to assess patient's tolerance (impact on intracranial pressure [ICP] and cerebral profusion pressure [CPP], oxygenation, ventilation, etc.). • Use transport staff familiar with the vehicle in which the patients with severe traumatic brain injury will be transferred and who have the ability to treat increasing ICP and decreasing CPP as needed.
During transport:
• Continue to monitor vital signs, ICP, CPP, end-tidal CO_2, and O_2 saturation levels at the same intervals as when in the intensive care unit. • Perform neurologic, pain, and sedation assessments regularly and treat as needed. • If EVD is in place, clamp during transport. Relevel and open if ICP increases. • Consider premedication with analgesia and sedatives prior to transport to avoid increasing ICP.

Table 12. Review of EVD Insertion Protocols Published Between 2014 and 2018[111,113,117]

EVD Insertion Protocol Summary	Chatzi (2014)	Hepburn-Smith (2016)	Sieg (2018)
Insertion outside of operating room	No	Yes	Yes
Hand hygiene and everyone in room has bouffant/mask on	Yes	Yes	Yes
Provider wearing full sterile attire	Yes	Yes	Yes
Site shaved to accommodate dressing size	Yes	Yes	Yes
Site cleansed with chlorhexidine and allowed to dry.	Not reported	Yes	Yes
Use of antibiotic-impregnated catheter	No	Yes	Yes
Use of antibiotic (intravenous [IV] or intraventricular)	IV and intraventricular	Normal	Low
Catheter tunneled	Not reported	Not reported	Yes (>5 cm)
Routine catheter exchange	Yes, every 7 days	No	No

all EVD insertion protocols address post-insertion site and dressing care, such as the use of a topical antimicrobial, type of dressing, timing of site care and dressing changes, or the role of site care in preventing site infections or ventriculitis.[112,113,115] Randomized controlled trials specifically comparing dressing practices for EVDs are lacking in the research literature.

Many sites report application of a dressing following ventriculostomy insertion,[111,113,116,122] although this practice is not consistent.[123] A summary of published ventriculostomy insertion protocols and infection rates (if available) from 2014 to 2018 is included in **Table 13**. Most recently published protocols do not include use of prophylactic antibiotics.[110,112] Two novel approaches to site care associated with decreased HAV are a single application of 2-octyl cyanoacrylate adhesive (Dermabond, Ethhicon, Inc.) at the time of ventriculostomy insertion[85] and the use of an alcohol impregnated cap attached to the proximal end of EVD tubing for access during CSF sampling.[123,124] Regardless of the approach, comprehensive EVD care should be standardized and based on evidence-based protocols.

ICU Bundle (ABCDEF Bundle)

The ABCDEF bundle is an interdisciplinary, evidence-based strategy that has been associated with improved patient outcomes in critically ill patients.[47,126] The components of the ABCDEF bundle include[47]

A. Assessment, prevention, and management of pain
B. Both spontaneous awakening trials and spontaneous breathing trials
C. Choice sedation/analgesia
D. Delirium assessment, monitoring, and management
E. Early mobility
F. Family engagement and empowerment.

Components are further described in **Table 14** and should be incorporated as part of routine care in the ICU.

Table 13. Review of the Literature on External Ventriculostomy Device Site Care Between 2014 and 2018[111,113,122,123,125]

Researcher	Bookland (2014)	Gozal (2014)	Ducis (2016)	Hepburn-Smith (2016)	Sieg (2018)
Type of dressing	None	Transparent occlusive dressing	None	Bio-occlusive dressing	Adherent dressing
Frequency of dressing change	Not reported	Replaced as needed	Not reported	Weekly or if loosened	Not reported
Site care with dressing change details	None	After chlorhexidine gluconate (CHG), skin painted with adhesive skin prep (benzoin)	Daily site assessments	None specified	Not reported
Other	Single application of 2-octyl cyanoacrylate adhesive at time of insertion	After dressing removal, scalp prepped with CHG, then 30 sec scrub with CHG (other side of stick)	Minimal hair removal at insertion site	Chlorhexidine-eluting patch	Chlorhexidine-eluting patch
Infection rate (pre- and post-study, if available)	18.6% pre to 3.5% post*	10.8% pre to 2.0% post	5.1% compared to national average of 8.8%	Not yet published	12% pre to 0% post*

*Statistically significant

Table 14. ABCDEF Intensive Care Unit Liberation Bundle[47,126]

Bundle assessments	Key points
A. Assessment, prevention, and management of pain	• Untreated pain can lead to delirium. • Vital signs alone may not be the best indicators of pain. • Assess for nonverbal signs of pain, such as facial grimacing, abnormal body movements, and ventilator dyssynchrony. • Premedicate patients before invasive procedures or turns. • Use a reliable and valid pain scale. If the patient cannot self-report, consider the Behavioral Pain Scale, Critical Care Pain Observation Tool, or Nociception Coma Scale-Revised. • Consider nonpharmacologic interventions, such as relaxation, massage/touch, music, and pet therapy.
B. Both spontaneous awakening trials (SATs) and spontaneous breathing trials (SBTs)	• Use standardized processes and procedures to promote daily team-driven initiatives to liberate patients from mechanical ventilation and sedation using sedation-weaning protocols, SATs, and SBTs.
C. Choice of sedation/ analgesia	• Treat pain before considering sedative therapy. • Use a valid and reliable sedation scale, such as the Richmond Agitation Sedation Scale or the Sedation-Agitation Scale, to assess the quality and depth of sedation in critically ill patients. • First-line medications, including non-benzodiazepine sedatives such as propofol or dexmedetomidine, are preferred. • If the patient remains agitated despite treatment with appropriate sedation/sedative, assess and treat for conditions such as hypoxia, hypoglycemia, hypotension, and drug or alcohol withdrawal.
D. Delirium assessment, monitoring, and management	• Use a valid and reliable assessment tool such as the Confusion Assessment Method for the ICU or the Intensive Care Delirium Screening Checklist to monitor for the development and severity of delirium. • To prevent delirium, try to optimize the patient's environment to allow for natural sleep-wake cycles, if able. This includes minimizing light and noise, clustering activities, and reducing stimuli at night, if able. • Early mobilization can help decrease delirium, although this may not always be possible in cases of severe traumatic brain injury with increased intracranial pressure. • Avoid medications known to provoke or further confound assessment, monitoring, or management of delirium.
E. Early mobility	• Assess for readiness for early and progressive mobility using a tool or protocol, such as the "MOVEN" (Mission Hospital Progressive Mobility Algorithm). • Be sure to consider the type of neurological injury prior to initiation.
F. Family engagement and empowerment	• Engage and encourage patient/family involvement in care. This can be accomplished through: o patient/family rounding o being open to family visitation polices o family presence during resuscitation o patient and family involvement in decision making, especially regarding goals of care and end-of-life decision making, as appropriate o providing daily updates o shared daily goal setting.

Acute Care Considerations

Prevention of Hospital-Acquired Conditions

Patients with sTBI are at high risk for hospital-acquired conditions that are sensitive to nursing care, such as hospital-acquired pneumonia, falls, pressure injury, central line–associated bloodstream infection, catheter-associated urinary tract infection, contractures, deep vein thrombosis, and other hospital-related infections. In a study quantifying complications during the first year after sTBI from hospital admission and throughout subacute rehabilitation, urinary tract infections (53%) and pneumonia (32%) were the most common complications. Less frequent were pressure ulcers (18%), joint contractures (18%), and deep venous thrombosis (4%).[127] **Table 15** outlines targeted strategies for prevention of these events.

Nutritional Support

Patients with TBI may have hypermetabolic and hypercatabolic activity lasting from 1 week to months post-injury, supporting early initiation of nutrition care. Enteral

Table 15. Targeted Strategies for Prevention of Hospital-Acquired Conditions[128-133]

Hospital-Acquired Condition	Targeted Strategies
Ventilator-associated pneumonia/hospital-acquired pneumonia	• Adhere to ventilator care bundle: o Daily awakening and breathing trials o Deep vein prophylaxis o Medications to prevent gastric ulceration • Head of bed at 30 degrees or higher if tolerated and no contraindications • Oral care every 4 hours with chlorhexidine gluconate including brushing of teeth • Monitoring of condensation in ventilator tubing • Swallow screen/evaluation prior to any oral intake • Speech language therapy evaluation if high risk for aspiration
Catheter-associated urinary tract infection	• Avoid placing urinary drainage catheters, unless necessary for patient care. • Prompt removal of catheters • Assess the need for continued use at least daily. • Consider alternatives such as condom catheters (and other types of external devices) or scheduled bladder scans with in-and-out catheterization for elevated post-void residuals and timed voidings. • Daily chlorhexidine baths • Limited insertion by trained individual and consider two-person insertion for females
Central-line associated bloodstream infections	• Remove central venous access devices (CVAD) as soon as no longer clinically indicated. • Avoid femoral line placement. • Aim to limit CVAD placement to the following: o vasopressor use o hypertonic saline administration o high-volume fluid resuscitation o hemodynamic monitoring. • Consider placement of a peripherally inserted central catheter in cases of o inability to obtain appropriate venous access o prolonged antibiotic administration.
Falls	• Pay attention to times of high patient activity and situations where there is decreased nursing capacity to observe patient. • Perform fall risk assessments at regular intervals using standardized tools. • Educate the patient and family about the risk of falls • Use fall wrist band, footwear, or signage to indicate a patient at high risk • Routine rounds and toileting • Environmental and geographical modifications to prevent falls

Table 15. Targeted Strategies for Prevention of Hospital-Acquired Conditions[128-133] *(continued)*

Hospital-Acquired Condition	Targeted Strategies
Hospital-acquired pressure injury (HAPI)	• Complete a thorough head-to-toe skin assessment on admission and then at least every shift. • A risk assessment should be done at least daily using a validated tool, such as the Braden Scale. • Nutritional assessment with intervention • Optimize nutrition/hydration, including early nutrition, calorie counts, and supplementation. • Turn and reposition the patient at least every 2 hours (although some patients may need more frequent turning) while in bed and at least every 1 hour when in a chair. • Pressure relief, a pressure-relief surface, or a special-care bed is recommended to help prevent pressure ulcers. • To prevent heel pressure ulcers, use a pillow or heel-lift devices to keep the patient's heels from touching the bed. • Keep the head of the bed at less than 30 degrees, unless contraindicated • Manage incontinence and moisture with a skin-care regimen, such as frequent cleansing and use of a moisture-barrier ointment. • Obtain a wound care/skin care nurse consult for high-risk patients.
HAPI related to a medical device	• Ensure the device is needed. • Use the correct device size for the patient. • Ensure proper application and removal of the device. • Protect the skin and tissue under the device. • When possible, do not place the device in an area that already is prone to breakdown. • If allowable, remove the device periodically (such as every shift) to assess the skin under the device. • Especially vulnerable areas include the head, neck, face, and ear from respiratory-related equipment such as oxygen tubing, tracheostomy times, and pulse oximetry probes. • Other high-risk devices include cervical collars, bedpans, endotracheal tubes/holders, face masks for non-invasive positive pressure ventilation, fecal containment devices, invasive lines, compression devices, splints, braces, urinary catheter tubing, external urinary catheters, wristbands, and orthopedic casts. • Use lift equipment to avoid shear/friction.
Venous thrombosis/pulmonary emboli	• Apply pneumatic compression devices and ensure that the patient is wearing them, especially while in bed. • Administer pharmaceutical intervention when safe. • Use early mobilization.
Contractures	• Early aggressive range of motion • Passive stretching of muscles and joints on a prescribed schedule • Positioning of the limbs to promote extension and oppose flexion • Splinting or serial casting when appropriate • When able to get out of bed, regularly prescribed periods of daily standing and/or walking • Pharmacological therapies (e.g., dantrolene, baclofen) may be considered by the rehabilitation team.
Infection due to invasive lines	• Ensure lines and drains (such as external ventricular devices) are placed under strict aseptic technique, including sterile field, sterile gowns/gowns, and mask use by all personnel in the room. • Assemble intracranial pressure equipment and other equipment as appropriate using a sterile technique. • Minimize access to invasive devices. • Use aseptic technique when managing drain/transducers/devices.

feeding is preferred and should be initiated within 24–72 hours in patients who are hemodynamically stable. At the latest, nutritional support should begin 5–7 days post-injury because early nutrition is associated with decreased mortality.[1,2] When enteral feeding is required, transgastric jejunal feeding is recommended over nasogastric feedings to reduce the incidence of pneumonia. Additional research is needed to determine the specific nutritional needs of the sTBI patient and how energy expenditure plays a role in nutritional support requirements.

Tracheostomy

Patients with TBI are at risk for developing ventilator-associated pneumonia or acute lung injury. Therefore, early tracheostomy often is considered within 8 days of injury.[2] Early tracheostomy may facilitate ventilator weaning, resulting in shorter ICU and hospital stays while reducing infection risk. Situations where early tracheostomy may be contraindicated include patients with increased ICP, hemodynamic instability, respiratory failure requiring high levels of positive end-expiratory pressure (greater than 10 cm H_2O) and FiO_2 administration greater than 50%.[2]

Management of Sleep Disturbances and Fatigue

Sleep disturbances and fatigue after TBI are common, although the pathophysiology is not fully understood. Between 30% and 70% of patients with sTBI report sleep disturbances.[134–137] Sleep disturbances are associated with increased rates of anxiety, depression, and fatigue. Sequelae of sleep disturbances include long-term behavioral challenges; impaired cognitive processing; inability to return to work; decreased quality of life; and higher incidence of antidepressant, antihypertensive, sedative, and narcotic use.[138] Fatigue, reported in 17%–40% of patients with TBI, may be experienced primarily as a problem with cognitive and physical aspects and feelings of weakness.[136,139,140]

Nurses should be aware of the impact of sleep disturbances among patients with sTBI and plan care aimed at facilitating sleep. Although sleep disturbances and fatigue commonly co-occur in TBI, it is recommended that nurses begin treatment of sleep disturbances prior to initiating specific treatments for fatigue.[141] In the management of sleep disorders and fatigue after TBI, nonpharmacological interventions aimed at the medical and neuropsychiatric causes or contributors should take precedence over pharmacotherapies.[141] Nonpharmacologic measures to aid in resolving sleep disturbances include cognitive behavioral therapy (CBT), blue-enriched white light therapy, acupuncture, and implementation of sleep hygiene protocols to promote a better sleep/wake cycle **(Table 16)**.[1,135,140,142–144] Commonly used pharmacologic agents for the treatment of sleep disturbances include benzodiazepines, tricyclic antidepressants, antihistamines, ramelteon, and melatonin **(Table 17)**.[1,134,136,140]

Table 16. Nonpharmacologic Sleep Interventions for Patients with sTBI[1,135,140]

Cognitive behavioral therapy	Combination of education, behavioral activation, behavioral experiments, problem solving and relaxation; found to result in decreased patient self-report of fatigue, depression, and sleep disturbances
Acupuncture	Nonpharmacologic, holistic therapy with few side effects; can promote regulation of neurotransmitters and humoral factors
Sleep hygiene protocol	Emphasis on maintaining sleep-wake cycle, exercise, avoiding naps, keeping daytime lighting in place, avoiding caffeine, and screens prior to bedtime
Blue light therapy	Helps retrain the circadian rhythm to promote better, more regular sleep

Table 17. Pharmacologic Agents Used in the Treatment of Sleep in Patients with sTBI[1,134,136,140]

Classification	Side Effects
Benzodiazepines	Found to have daytime sedative effects and adverse effects on motor and cognitive functioning; avoid use in older patients.
Tricyclic antidepressants	May reduce the seizure threshold
Antihistamines	May lead to physical dependence, daytime drowsiness
Ramelteon	U.S. Food and Drug Administration approved for treatment of insomnia with sleep-onset abnormalities; shorter half-life and less likely to become addictive. Ramelteon is reported to increase total sleep time and sleep latency with limited negative side effects.
Melatonin	Offers benefit of promoting sleep without sedative or addictive tendencies. Melatonin was associated with significant reduction in Pittsburgh Sleep Quality Index scores consistent with improved sleep quality.

Management of Neuropsychiatric Complications

Individuals who experience TBI are at increased risk of developing major depression, general anxiety disorder, obsessive-compulsive disorder, panic disorder, post-traumatic stress disorder (PTSD), antisocial behavior such as criminality and substance abuse, and suicide.[145] Individuals with preexisting behavioral and psychiatric problems may find that the brain injury exacerbates their condition and makes the management of day-to-day function more complex and difficult. Memory impairments and cognitive problems such as decreased concentration may further hinder a patient's recovery.

One of the most common disorders is PTSD, which often is associated with witnessing injuries or fatalities, suffering medical conditions resulting from the initial injury (such as from a blast injury, burns, or respiratory distress), and being a survivor of a traumatic event.[146]

Practice guidelines from the Department of Veterans Affairs Medical Center and Department of Defense include treatment of PTSD with appropriate pharmacologic and psychotherapeutic interventions and identification and treatment of comorbid neuropsychiatric conditions (e.g., depression), substance abuse, medical comorbidities, and cognitive issues.[146] The management of PTSD should include CBT as first-line therapy, antidepressant use, pain management, and suicide risk assessment.[146,147]

Patients with TBI are 10 times more likely to experience a depressive episode than the annual rate of depression in the general population (53%[148] compared with 6%[149]) and are at greater risk of suffering from recurring depressive symptoms for decades after the initial injury.[150] The first line of antidepressants for the treatment of PTSD is sertraline, paroxetine, and venlafaxine because they have fewer side effects and drug interactions compared with other antidepressant medications. Benzodiazepines and antipsychotics, in particular, should be avoided because of side effects. It is important to assess for untreated TBI sequelae in patients exhibit persisting symptoms of insomnia, irritability, difficulty concentrating, depression, or fatigue.[146,147]

Family caregivers of individuals with TBI-related disabilities may encounter challenges when caring for someone with cognitive, behavioral, and emotional changes associated with TBI. Caregivers may suffer from substantial stress as they attempt to meet their loved ones' long-term physical and emotional needs, as well the associated financial burdens. Therefore, the interdisciplinary team should include assessment of family member needs and align resources and support services when indicated.

Management of Visual Disturbances

Vision disturbances after TBI are common. Approximately 65%–79% of TBI patients report subjective visual complaints. Visual disturbances include defects in visual acuity, visual fields, eye movement, and the more complex aspects of vision such as visual perception.[151]

Results of a 2019 meta-analysis that looked at field loss, visual acuity, accommodative dysfunction, and convergence insufficiency found that in patients with moderate to severe TBI, 39.8% had visual field loss and 3.2 % experienced decreased visual acuity. In addition, 42.8% experienced accommodative dysfunction and 36.3% had convergence insufficiency.[152] Among patients who experience blast-related sTBI, estimates of visual disturbances may be higher, up to 50%–75%, and include blurred vision, photosensitivity, light-dark adaptation and accommodation, visual field cuts, and difficulty reading.[153]

As part of acute care management, patients with TBI should receive routine vision screenings that include an assessment of binocular vision and problems with convergence, which may present as headaches, fatigue, and disturbances in cognition.[154] Patients with chronic visual dysfunction after TBI may require occupational, vestibular, cognitive, and other forms of physical therapy. Environmental safety is a priority and should include adequate lighting and education about compensatory mechanisms for visual loss. Patching and use of prisms or corrective lenses may improve short-term outcomes.[155] Other techniques include visual-motor feedback training and computer-based oculomotor training.[156] The use of protocols for vision assessment is associated with better outcomes in patients with TBI.[2,154]

The use of protocols for evidence-based care has been studied and has demonstrated better outcomes for patients after TBI in the areas of vision screenings, motor and gait disturbances, anticoagulation therapy, cognitive training, brain imaging, nutritional support, and airway management.[2,146,154]

Management of Motor Disturbances

A major contributor to limited mobility after TBI, especially in older patients, are motor disturbances such as poor balance and spasticity. Decreased gait velocity can be present 48–72 hours post-injury with a return to baseline between 6 and 12 months.[157] Patients should receive fall risk assessments using standardized scoring tools at regular intervals throughout acute hospitalization and post-hospitalization. Based on the assessment, a targeted plan of care with evidence-based fall prevention strategies should be implemented.

Anticoagulation Therapy

Patients with TBI have an approximate 20%–30% risk for the development of venous thromboembolism (VTE).[2] Current studies support pharmacologic thromboembolism prophylaxis within 24–72 hours if the CT scan reads as stable and the patient is not at high risk for increased cerebral hemorrhage.[2,158,159] A prophylactic inferior vena cava filter may be considered in patients at very high risk for VTE who are not candidates for pharmacologic prophylaxis.[2]

Rehabilitation Considerations

Patients with sTBI-related impairments often need organized physical, occupational, speech, and other therapies to regain abilities or to develop new compensatory skills. This also may involve modifications to the home and other environments.

For survivors of TBI with disabilities, insurance coverage may impact the type, intensity, and duration of acute and post-acute services available.[160] Behavioral health services and cognitive and physical rehabilitation may be limited in some geographic regions or health systems, presenting challenges for sTBI patients who require ongoing therapies after hospital discharge.[160] As such, aligning needed services for patients often is a logistical, financial, and psychological challenge for caregivers and families.[160]

In the United States, the principal sources of funding and support for long-term TBI services are Supplemental Security Income (SSI), Social Security Disability Insurance (SSDI), Medicaid, and Medicare.[160] Eligibility for SSI and SSDI often is the critical path to Medicaid- or Medicare-sponsored health coverage.[160] It is important to include social and financial services in care planning.[160]

Cognitive Rehabilitation

Cognition in general is adversely affected by TBI, with attention, processing speed, memory, and executive function particularly impaired.[145] Specifically in cases of moderate to severe TBI, the level of cognitive deficit at 1 year may be predictive of persistent chronic deficits.

Goals of cognitive rehabilitation focus on improving functioning in one or more of the following areas: attention, vision, visual spatial functioning, communication, and memory of higher functioning.[146,161] Neurorehabilitation techniques, such as cognitive training, may improve participation, level of activity, recovery, and patient outcomes.

Stimulation to Promote Alertness

The use of sensory stimulation programs may promote better outcomes for patients with disorders of consciousness (DoC). Patients with DoC, including both vegetative state and minimally conscious state lasting 28 days or longer, have a better chance for recovery when managed by a multidisciplinary team and have reduced mortality if discharged to home or to an inpatient rehabilitation center.[162] In one study, 20% of patients with DoC admitted to inpatient rehabilitation were able to become functionally independent and return to work at one or more follow-up intervals (measured at 1, 2, and 5 years).[162]

Nursing staff and clinicians should take arousal state into consideration when performing serial neurologic assessments, while ruling out other issues that might hinder examination findings (e.g., sensory or neuromuscular impairments). It is important to assess for agitation, aggressive behavior, sleep disturbances, and urinary tract infections in this patient population because these issues may delay or disrupt rehabilitation.

There is limited evidence to provide guidance for the effectiveness of sensory stimulation in improving alertness and arousal in patients with DoC. However, LOC may improve if the stimuli is personalized to the patient's past experiences and preferences.[163,164] It is essential to include the family in the plan of care to provide a familiar voice, such as when asking the patient to follow commands or participate in therapy. Currently, amantadine is the most prevalent pharmacologic agent used to improve the level of arousal and alertness in patients with TBI, although there is limited research on its effectiveness in improving memory.[146] Active or passive music therapy may aid in rehabilitation of the patient with brain injuries. The auditory system has been shown to be more sensitive in identifying responsiveness, indicating awareness in patients with an absence of motor responses.[165,166] Careful attention to planning and assessing prior to, during, and after therapy sessions will aid in determining the patient's response (including signs of overstimulation and agitation).

Geriatric Considerations

Neuropathology in geriatric patients with TBI tends to be more focal than in the young, with SDHs and cerebral contusions occurring more commonly at any given level of injury severity.[141] To avoid missing important diagnoses, the American College of Emergency Physicians recommends that all patients age 65 years or older presenting with a mild head injury receive brain imaging.[2]

Although mortality rates vary for patients older than age 65 years with TBI, the highest death rate occurs within the first 48 hours. Mortality rates rise with increasing comorbidities (e.g., diabetes, heart disease, cancer, hypertension, and liver and renal disease) in older patients with TBI.[167] Despite the complications related to comorbidities, age alone should not guide treatment plans. Approximately 30% of older patients with TBI will survive and leave the hospital.[2]

Nursing interventions to support better outcomes in geriatric patients with sTBI include obtaining an adequate baseline assessment (e.g., mental, behavioral, and physical and psychosocial status), use of an interdisciplinary approach, and delirium assessment and treatment. An interdisciplinary approach with a geriatric team consultation is associated with a 25% increase in a patient's likelihood of being alive in their home at 1 year post-discharge, decreased delirium rates, a decreased number of falls with injury, and shorter lengths of hospital stay.[2] Geriatric screenings can explore a patient's ability for self-care and help identify deficiencies with eating, drinking, swallowing; visual disturbances; hearing loss; and the ability to perform activities of daily living. Motor and balance functions tend to recover more slowly following TBI in older adults, when compared to younger adults, because of premorbid limitations in cognition, sensation, strength, and balance.[141] Geriatric patients also can have decreased tolerance for intensive therapy sessions because of lower endurance and more joint and muscle pain and stiffness that must be considered when planning activities.[141]

Anticoagulant Use

A major concern for patients with sTBI, especially for geriatric patients, is the concomitant use of anticoagulants at the time of injury. These agents not only increase the risk for bleeding but present challenges with anticoagulation reversal in the setting of an acute injury. TQIP guidelines support warfarin (a vitamin K antagonist) reversal with vitamin K, fresh frozen plasma, or prothrombin complex concentrate (PCC)—with PCC (with simultaneous administration of IV vitamin K) being preferred over fresh frozen plasma because it is faster and requires less volume.[2] The targeted international normalized ration for warfarin reversal is less than 1.4.

For patients on direct thrombin inhibitors (dabigatran) or factor Xa inhibitors (apixaban, edoxaban, and rivaroxaban), specific reversal agents should be used. For dabigatran, idarucizumab is the preferred reversal agent and andexanet alfa is used for reversal of factor-Xa inhibitors. Activated charcoal also may be considered if the oral anticoagulant was taken within 2 hours of treatment. Patients receiving reversal agents should be monitored for thrombotic events, major side effects of these medications, and correction of clotting factors.[168]

Medication Management

Geriatric trauma patients are at high risk for adverse effects from medications. Older adults can have cognitive deficits that include delayed recall, reasoning, and fluency in communication.[140] Approximately 85% of patients with dementia are older than 65 years, and it can be challenging to determine the influence of age-related cognitive impairment compared to impairments sustained from the injury or from medications.[140] A multidisciplinary team should consider use of the Beers Criteria for Potentially Inappropriate Medication Use in Older Adults list to screen for potentially inappropriate medication use in older adults to guide medication use.[2,169] Narcotics should be avoided to reduce the risk of delirium and falls, which may increase morbidity and mortality.[2] Baseline medications such as statins and beta blockers, if indicated, should be restarted. Psychological concerns must be diagnosed and treated to avoid a negative impact on quality of life.

Summary

Caring for hospitalized patients with sTBI is both challenging and rewarding. Neuroscience nurses are integral members of the healthcare team managing this high-risk population. Integration of evidence-based information, along with clinical expertise and consideration for patient values and preferences, aids in the delivery of high-quality, safe nursing care that may optimize outcomes for patients throughout the continuum of care.

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