ACS TQIP
BEST PRACTICES IN THE MANAGEMENT OF TRAUMATIC BRAIN INJURY

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COMMITTEE ON TRAUMA

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INTRODUCTION

Traumatic brain injury (TBI) is a disease process that carries major public health and socioeconomic consequences. In the United States alone, an estimated 2.5 million emergency department visits and hospitalizations are associated with TBI annually; and more than 50,000 individuals die from TBI. Moreover, a considerable proportion of TBI survivors incur temporary or permanent disability. The estimated annual burden of TBI on the United States economy is more than $76 billion, with the costs for disability and lost productivity outweighing the costs for acute medical care.

Data from well-designed, controlled studies on acute management of TBI are sparse. Evidence-based guidelines for TBI management have been compiled, but the paucity of high-quality studies limits the strength and scope of their counsel. The TQIP Best Practice Guidelines for the Management of Traumatic Brain Injury present recommendations regarding care of the TBI patients based on the best available evidence or, if evidence is lacking, based upon the consensus opinion of the expert panel.

USING THE GLASGOW COMA SCALE

Key messages:

- The Glasgow Coma Scale (GCS) provides a reliable tool for assessing disturbances of consciousness across care paths
- Standardized approaches to GCS assessment and reporting are essential
- The GCS should specify the score for each of the three components (eye, verbal, motor) when reporting on individual patients
- The sum of the component scores (GCS 3-15) is relevant for comparisons at the group level for purposes of classification and prognosis

The Glasgow Coma Scale (GCS) was introduced forty years ago by Teasdale and Jennett as a practical method for assessing the full spectrum of disorders of consciousness, from very mild to severe. It has been broadly adopted, and is internationally utilized as an integral part of clinical practice and research. The GCS aims to rate performance in three different domains of response: the eye, verbal, and motor response (Table 1). For individual patients, it is recommended that in that all three components be reported, e.g., E4V4M5, versus a sum score, e.g., GCS 13. The derived sum score of the GCS (3-15) is more relevant for comparisons at the group level and provides a useful tool for classification and prognosis.



A score of ≥13 correlates with a mild brain injury, 9 to 12 is a moderate injury, and ≤8 a severe brain injury.

If a GCS component is untestable due to intubation, sedation, or another confounder, the reason for this should be recorded. Although often done, a score of 1 should not be assigned because differentiation between a "true 1" and an untestable component is relevant. Graphical display of the three GCS components over time may facilitate earlier detection of changes.

Assessment requires either a spontaneous response or response following application of a stimulus. At more severely disturbed levels of consciousness, the motor score has better discrimination, but in milder injuries the eye and verbal components are more relevant. Thus, each component of the scale (Eye, Verbal, Motor) provides complementary information. Strengths of the GCS are that it covers a broad spectrum of disorders of consciousness, is widely applicable, and offers an important tool for monitoring changes in the level of consciousness. Standardized approaches to both its assessment and its reporting are required in order to be able to compare evaluations over time or when communicating with other health care professionals. Spontaneous responses are first observed without stimulating the patient in any way. First, verbal stimuli are applied, such as asking a patient to obey commands and at the same time observing whether, e.g., an eye opening occurs. If a patient is not responsive, a stimulus is applied to elicit a response. The location of the stimulus (central or peripheral) should be standardized and used consistently. To describe the motor response, only the reaction of the arms should be observed, not the legs.

Table 1. Glasgow Coma Scale

Eye opening (E)	
None	1
To pressure	2
To sound	3
Spontaneous	4
Untestable	Reason:
Verbal response (V)	
None	1
Sounds	2
Words	3
Confused	4
Oriented	5
Untestable	Reason:
Motor response (M)	
None	1
Extension	2
Abnormal flexion	3
Normal flexion	4
Localizing	5
Obey commands	6
Untestable	Reason:

TRIAGE AND TRANSPORT

Key Message

- Patients with a Glasgow Coma Scale (GCS) ≤ 13 should be rapidly transported directly from the scene to the highest level trauma center available in a defined trauma system to allow for expedient neurosurgical assessment and intervention
- Patients with a combination of TBI (GCS score ≤ 15) and moderate to severe extra-cranial anatomic injuries and Abbreviated Injury Score (AIS) ≥3 should be rapidly transferred to the highest level of care within a defined trauma system to allow for expedient neurosurgical and multidisciplinary assessment and intervention

Proper field triage is critical for patients with suspected TBI. Trauma patients with TBI require rapid resuscitation, definitive operative management, and critical care capabilities to prevent secondary brain injury. The US Center for Disease Control's (CDC) 2011 Field Triage Guidelines for Injured Patients direct EMS providers to transport all patients with a Glasgow Coma Scale (GCS) < 13, or those with any level of TBI (GCS ≤ 15) and extracranial injuries (AIS ≥ 3) to the highest level trauma center that has the expertise, personnel, and facilities to rapidly provide definitive care, usually a level I or II trauma center. Despite these guidelines, significant undertriage of TBI victims has been documented throughout the US in systems with and without trauma centers.

Providing the initial resuscitative care in lower-level trauma center centers (III, IV, or non-designated hospitals) may occasionally be rationalized in some rural settings with long transport times (≥ 1 hour). However, these hospitals should have predefined air/ground transfer protocols and agreements in place to provide for the immediate transfer of TBI patients to the highest level center available within a defined trauma system.

GOALS OF TREATMENT

These clinical parameters should be maintained as part of goal-directed TBI treatment. Some of these goals are more relevant for patients in the intensive care unit (ICU) setting (e.g., CPP, ICP, PbtO2) while others are applicable to all TBI patients. Adequate oxygenation and normocapnia should be maintained. Patients with significant pulmonary issues (e.g. Acute Respiratory Distress Syndrome) may require lung-specific parameters. Systolic blood pressure (SBP) and mean arterial pressure should be monitored closely to avoid hypotension. The goal for temperature management is normothermia. Core body temperature should be kept <38°C. The goal for electrolytes is to maintain within normal range. Specific attention to the sodium level is crucial in TBI patients. Hyponatremia must be avoided as this may worsen cerebral edema. TBI patients may also develop diabetes insipidus (DI) or the syndrome of inappropriate antidiuretic hormone (SIADH). Therefore patients should have frequent monitoring of the serum sodium and osmolality levels. Both



Table 2. Goals of Treatment

Pulse Oximetry ≥ 95%	ICP 20 - 25 mmHg	Serum sodium 135-145
PaO_2 ≥ 100 mmHg	$PbtO_2$ ≥ 15 mmHg	INR ≤ 1.4
$PaCO_2$ 35-45 mmHg	CPP ≥ 60 mmHg *	Platelets ≥ 75 x 10^3 / mm^3
SBP ≥ 100 mmHg	Temperature 36.0-38°C	Hemoglobin ≥ 7 g/dl
PH 7.35-7.45	Glucose 80-180 mg/dL	

PaO2: partial pressure of oxygen; PaCO2: partial pressure of carbon dioxide; SBP: systolic blood pressure; ICP: intracranial pressure; PbtO2: brain tissue oxygen tension; CPP: cerebral perfusion pressure; INR: international normalized ratio; *depending on status of cerebral autoregulation

hyperglycemia and hypoglycemia are detrimental to the outcome of patients with TBI. Serum glucose levels must be monitored closely in all TBI patients. More frequent monitoring is required following the initiation of nutritional support, particularly in patients with known or suspected diabetes mellitus.

Anemia and coagulopathy are common in patients with TBI and should be monitored closely. There is considerable practice variability in hemoglobin transfusion thresholds for TBI patients. A recent randomized clinical trial compared 2 hemoglobin transfusion thresholds (7 and 10 g/dl) after TBI. There were no differences in neurological outcome. However, the 10 g/dl threshold was associated with a higher incidence of adverse events, supporting the best practice recommendation of a 7 g/dl transfusion threshold. TBI patients should receive early evaluation for coagulopathy with assessment for direct and indirect coagulation cascades using INR to identify medical, iatrogenic, or early post-traumatic coagulopathy when present. Utilization of newer assays of coagulation capability (Thromboelastography or Rotational thromboelastometry, and/or platelet function assays) may provide additional information regarding the need for targeted therapy to reverse coagulopathy.

INTRACRANIAL PRESSURE MONITORING

Key Messages:

- ICP monitoring is important, but it does not replace careful neurological and radiographic examination

- ICP monitoring is indicated in comatose patients (GCS ≤ 8) and if there is evidence of structural brain damage on initial CT imaging
- ICP monitoring is generally not indicated in comatose patients without evidence of structural brain damage or elevated ICP (compressed/absent basal cisterns) on initial CT imaging. Patients may be observed with repeat CT imaging and forego ICP monitoring if there is no progression
- ICP monitoring should be considered in patients with a GCS > 8 who have structural brain damage with high risk for progression (large/ multiple contusions, coagulopathy)
- ICP monitoring should be considered in patients who require urgent surgery for extracranial injuries, who need mechanical ventilation because of extracranial injuries, or who evidence progression of pathology on CT imaging or clinical deterioration
- The preferred method for ICP monitoring is an external ventricular drain (EVD) because it is both diagnostic (measures ICP) and therapeutic (allows for drainage of cerebrospinal fluid (CSF)

Elevated ICP is predictive of poor outcome. Furthermore, cerebral perfusion pressure (CPP), a parameter derived from ICP (Mean Arterial Pressure – ICP), is an important marker of cerebral blood flow; augmenting CPP can help to restore cerebral perfusion and oxygenation. In addition to enabling CPP measurement, ICP monitoring can provide advanced warning of impending structural brain derangements such as contusion/hematoma progression, increased cerebral edema, and postoperative complications. The identification of ICP elevation can prompt further imaging, timely intervention, and definitive management..

ICP monitoring remains a critical component in the management of severe TBI. However, recent studies have highlighted the need to better define how ICP monitoring is used in the treatment of TBI. In the largest study of ICP monitoring to date, observational data from hospitals participating in the ACS TQIP demonstrate that ICP monitoring utilization was associated with lower in-hospital mortality. Other institutional practices not captured in the database, also appeared to contribute to improved outcome. The only randomized controlled trial compared treatment using ICP monitoring to maintain ICP ≤ 20 mm Hg to treatment based upon



imaging and neurological examination in TBI patients from South America. Although there was no difference in outcomes between the groups, this does not support the discontinuation of ICP monitoring in the treatment of TBI. Rather, it demonstrates the importance of aggressive treatment using ICP monitoring or frequent clinical and radiographic examination to identify intracranial hypertension. This study also challenges the currently accepted rigid ICP alert threshold of 20 mm Hg for all patients. The current accepted alert threshold is an ICP of 20 mm Hg, with a reasonable range of 20-25 mm Hg as a trigger for treatment of intracranial hypertension. Ongoing research may reveal that this threshold is dependent upon individual patient factors. An approach based on injury type and augmented by advanced neuromonitoring may lead to individualized treatment pathways.

The gold standard for ICP measurement is via an external ventricular drain (EVD), attached to an external strain-gauge transducer. The monitor, centrally placed within the cerebral ventricles, can measure global ICP and offers the therapeutic advantage of draining CSF to reduce intracranial volume. Intraparenchymal ICP monitoring is also a reliable method but does not allow for CSF drainage. Subdural and epidural monitors have been used, but these are the least accurate methods of ICP measurement.

MANAGEMENT OF INTRACRANIAL HYPERTENSION

Key Messages

- ICP is a global measure that cannot identify the specific mechanism(s) of pressure elevation. Additional neuromonitoring and assessment of cerebral autoregulation may help to individualize treatment
- The recommended "3-tiered" approach to ICP management utilizes various treatments to target different mechanisms. Higher tiers reflect more intensive management that is associated with increased complications
- Failure to control ICP/CPP within one tier, should prompt rapid progression to the next tier's treatment options
- Repeat CT imaging and neurological examination should be considered to rule out the development of surgical lesion and guide management

Because there is often no single pathophysiological pathway of ICP elevation, management is complex. Elevated ICP can be related to a variety of mechanisms, including: edema (cellular, extracellular), cerebral venous outflow obstruction, hyperemia (loss of autoregulation, vasodilation), mass effect (expanding hematoma), and disturbances in CSF circulation. ICP is a global measure that cannot distinguish among these mechanisms. Additional neuromonitoring of brain tissue oxygen tension (PbtO2), jugular venous oxygenation (SjvO2), cerebral blood flow (CBF), cerebral autoregulation, and other parameters may be helpful in identifying a more individualized approach to treatment. We have recommended a "tiered" approach to ICP management that utilizes various treatments to target different mechanisms. The higher tiers reflect more intensive management that is also associated with increased complications.



THREE-TIERED MANAGEMENT OF INTRACRANIAL PRESSURE

TIER 1

- Head of bed elevated at 30 degrees (reverse Trendelenburg) to improve cerebral venous outflow
- Sedation and analgesia using recommended short-acting agents (for example, propofol, fentanyl, midazolam) in intubated patients
- Ventricular drainage performed intermittently. Continuous drainage is not recommended unless an additional ICP monitor is placed, as when the drain is open, it does not accurately reflect the true ICP
- Repeat CT imaging and neurological examination should be considered to rule out the development of a surgical mass lesion and guide treatment

If ICP remains ≥ 20 - 25 mmHg proceed to Tier 2

TIER 2

- In patients with a parenchymal ICP monitor an EVD should be considered to allow for intermittent CSF drainage
- Hyperosmolar therapy should be given intermittently as needed for ICP elevation and not on a routine schedule
  - Mannitol should be administered in intermittent boluses (0.25 - 1 gm/kg body weight). Caution should be taken in the hypovolemic patient when osmotic diuresis is instituted with mannitol. The serum sodium and osmolality must be assessed frequently (every 6 hours) and additional doses should be held if serum osmolality exceeds 320 mOsm/L. Mannitol may also be held if there is evidence of hypovolemia
  - Hypertonic saline may be administered in intermittent boluses of 3% sodium chloride solution (250 ml over ½ hour) or other concentrations (e.g., 30cc of 23.4%). Serum sodium and osmolality must be assessed frequently (every 6 hours) and additional doses should be held if serum sodium exceeds 160 mEq/L
- Cerebral autoregulation should be assessed (see Advanced Neuromonitoring section). If the patient is not autoregulating, the CPP goal should be lowered to reduce ICP (to no less than 50 mm Hg). Additional neuromonitoring (e.g., PbtO2, SjvO2, CBF) may help determine optimal CPP

- PaCO2 goal of 30 - 35 mmHg should be maintained, as long as brain hypoxia is not encountered. Additional neuromonitoring (e.g., PbtO2, SjvO2, CBF) may help determine optimal PaCO2
- Repeat CT imaging and neurological examination should be considered to rule out development of a surgical mass lesion and guide treatment
- Neuromuscular paralysis achieved with a bolus "test dose" of a neuromuscular blocking agent should be considered if the above measures fail to adequately lower ICP and restore CPP. If there is a positive response, continuous infusion of a neuromuscular blocking agent should be employed (Tier 3)

If ICP remains ≥ 20 - 25 mmHg proceed to Tier 3

TIER 3

(includes potential salvage therapies)

- Decompressive hemi-craniectomy or bilateral craniectomy should only be performed if treatments in Tiers 1 and 2 are not sufficient or are limited by development of side effects of medical treatment
- Neuromuscular paralysis via continuous infusion of a neuromuscular blocking agent can be employed if there is a positive response to a bolus dose. The infusion should be titrated to maintain at least two twitches (out of a train of four) using a peripheral nerve stimulator. Adequate sedation must be utilized
- Barbiturate or propofol (anesthesia dosage) coma may be induced for those patients who have failed to respond to aggressive measures to control malignant intracranial hypertension, however it should only be instituted if a test dose of barbituate or propofol results in a decrease in ICP, thereby identifying the patient as a "responder." Hypotension is a frequent side effect of high dose therapy with these agents. Meticulous volume resuscitation should be ensured and infusion of vasopressor/inotropes may be required. Prolonged use or high dose of propofol can lead to propofol infusion syndrome. Continuous EEG may be used to ensure targeting of the infusion to burst suppression
- Hypothermia (<36 °C) is not currently recommended as an initial TBI treatment. Hypothermia should be reserved for "rescue" or salvage therapy after reasonable attempts at ICP control via the previous Tier 3 treatments have failed



ADVANCED NEUROMONITORING

Key Messages

- Advanced neuromonitoring and assessment of cerebral autoregulation may be helpful in identifying a more individualized approach to treatment
- Impaired cerebral oxygenation can occur in the face of normal ICP and CPP
- Cerebrovascular pressure reactivity index (PRx) and cerebral blood flow (CBF) monitoring can assess autoregulation status, which may help determine patient-specific CPP and ICP goals

TBI is a complex disease with substantial heterogeneity. ICP monitoring alone cannot detect all potential insults to the brain; ensuring adequate cerebral blood flow and oxygenation are important goals. Multiple studies have demonstrated an association between low brain tissue oxygen tension ($PbtO2 \leq 15$ mm Hg) and episodes of jugular venous oxygen desaturation ($SJvO2 \leq 50$ %) with poor outcome in TBI. Importantly, brain tissue hypoxia can occur even when ICP and CPP are normal. A recently completed Phase II prospective randomized clinical trial investigating PbtO2-based management of severe TBI compared treatment guided by ICP alone to treatment guided by both ICP and PbtO2 (BOOST, NCT00974259). The ICP+PbtO2 management group had statistically significant decreased duration and severity of brain hypoxia along with a 10% reduction in mortality and a trend toward reduced mortality and improved neurologic outcome at 6 months. This trial supports the value of advanced multimodality monitoring in TBI patients.

Cerebral pressure autoregulation is the brain's intrinsic ability to maintain constant CBF over a range of systemic blood pressures. This mechanism protects against cerebral ischemia due to hypotension and against excessive flow that can lead to elevated ICP. Cerebral autoregulation can be assessed at the bedside in the ICU with cerebrovascular pressure reactivity index (PRx) monitoring, CBF monitoring, and Transcranial Doppler (TCD) ultrasonography monitoring. The PRx is quantified as the slope of the regression line relating MAP and ICP and can be used to establish patient-specific CPP thresholds. For patients with impaired cerebral autoregulation ($PRx\ slope > 0.13$), a lower CPP (50 – 60 mm Hg) should be considered as an option for treatment. Patients with intact autoregulation ($PRx\ slope < 0.13$) may benefit from a higher CPP (50 – 60 mm Hg). When CBF is monitored directly, autoregulation status can be assessed with a hemodynamic challenge. In patients with intact autoregulation, CBF will change minimally in response to an increase in MAP. Conversely, CBF will rise with increasing MAP in patients with impaired autoregulation. Once determined, autoregulation status can be used to set CPP goals as described above. In a similar fashion, TCD ultrasonography and hemodynamic challenge can also be used to assess autoregulation in TBI patients.

SURGICAL MANAGEMENT

Key messages:

- A large traumatic hematoma should be evacuated before neurological deterioration develops, irrespective of the GCS
- A formal craniotomy is necessary to perform adequate resection
- TBI patients presenting to the ED in coma should be taken to surgery immediately upon arrival if a large hematoma is identified as the cause of the coma
- Decompressive craniectomy is effective in controlling intracranial pressure, but uncertainty exists as to its potential to improve outcome

Surgery for TBI patients is most commonly performed to evacuate epidural hematomas (EDH), subdural hematomas (SDH), cerebral contusions, or intracerebral hematomas (ICH) that are large enough to cause significant mass effect on the brain. Surgical evacuation of these hematomas should be performed as soon as possible. TBI patients presenting to the ED in a coma should be taken to surgery immediately upon arrival if a large hematoma is identified as the cause of the coma. Admitted patients who undergo neurological deterioration from delayed development or enlargement of a hematoma require prompt surgical evacuation to prevent further neurological worsening. A formal craniotomy is necessary to perform adequate resection; there is no role for attempted burr-hole drainage of these solid clots. Evidence-based guidelines for surgery have been compiled, but the paucity of high-quality randomized studies in this area limits the strength of recommendations. In general, CT evidence of raised ICP, such as midline shift of ≥5 mm and/or compression of the basal cisterns is an indication for surgical evacuation of a traumatic mass lesion. Even if a patient has a relatively high GCS score, a large traumatic hematoma should be evacuated before neurological deterioration develops from enlargement of the hematoma or swelling of the underlying brain. A lower threshold for surgical intervention may apply to posterior fossa lesions.

Decompressive Craniectomy (DC), in which a large bone flap is deliberately removed or not replaced, has witnessed a surge of popularity in recent years. Sometimes the flap is left off because massive cerebral swelling develops after evacuation of a hematoma, and at other times, the surgeon anticipates significant cerebral edema and pre-emptively leaves the bone flap off. In other cases, patients who would not normally undergo surgery may be taken to the operating room for DC if ICP begins to rise. A recent study casts doubt on the clinical benefit of a DC in patients with diffuse brain injury and raised ICP refractory to medical management. The randomized controlled DECRA trial demonstrated that although patients who received craniectomy achieved effective lowering of ICP, their neurologic outcomes at six months were worse than those of patients randomized to



maximal medical therapy. However, critics of this trial have highlighted unbalanced treatment groups, variability in medical treatments for the control group, high crossover rate to the surgical arm, and short-term follow-up (six months) as arguments against the conclusions of the study. The application of decompressive craniectomy for severe TBI remains a topic of lively debate.

Depressed skull fractures are commonly elevated if the depression is greater than the depth of the adjacent inner table, especially if located in a cosmetically important area like the forehead. Open depressed fractures are best treated surgically to prevent infection, but nonoperative management may be attempted in selected cases, limited to those without dural laceration, gross contamination or evidence of infection, or injury to the frontal sinus. In general, a depressed skull fracture over the sagittal sinus should not be treated surgically because of the high risk of uncontrollable hemorrhage.

NUTRITIONAL SUPPORT

Key Messages:

- Nutrition should begin early, as soon as the patient is hemodynamically stable, and ideally within 24-48 hours of injury
- Enteral nutrition is recommended over the use of parenteral nutrition
- Post-pyloric feeding methods are preferred as they are associated with a lower rate of pneumonia
- Full nutritional supplementation should be achieved within 7 days of injury

Patients with TBI demonstrate hypermetabolic and hypercatabolic activity lasting from 1 week to several months following their injury. Nutritional support should be initiated as early as possible, usually as soon as the patient is hemodynamically stabile and there are no significant gastrointestinal issues. Studies have demonstrated that early nutritional support is associated with fewer infections and lower mortality. “Early” is most commonly defined as within 24-48 hours of injury, and is adopted for these guidelines. This recommendation is made in conjunction with the Brain Trauma Foundation recommendation of achieving full nutritional support within 7 days of injury.

When considering nutrition support, enteral nutrition is recommended over the use of parenteral nutrition. If parenteral nutrition use is unavoidable, frequent glucose monitoring must be performed to insure that the patient remains euglycemic. A recent meta-analysis of post-pyloric vs. gastric feeding methods found that post-pyloric placement was associated with a significant reduction in the rate of pneumonia. The same analysis also demonstrated a trend toward reduced mortality and ventilator dependence.

TRACHEOSTOMY

Key Messages:

- If level of consciousness remains persistently depressed, TBI patients should undergo tracheostomy to facilitate liberation from mechanical ventilation; this can decrease risk of pneumonia and ventilator-induced lung injury
- Relative contraindications to tracheostomy include high intracranial pressure, hemodynamic instability, and severe respiratory failure
- All TBI patients deemed not likely to improve rapidly should be considered for early tracheostomy, within 8 days of injury

Patients suffering severe TBI require mechanical ventilation in intensive care units as a component of their initial post-injury care. If the level of consciousness remains persistently depressed, these patients should undergo tracheostomy to ensure a patent airway and thereby facilitate liberation from mechanical ventilation and, possible decrease in the associated risk of pneumonia and ventilator-induced lung injury. There are no absolute contraindications for this procedure. Relative contraindications include high intracranial pressure, hemodynamic instability and severe respiratory failure requiring high levels of FiO2 (>50%) and PEEP (>10cm H2O).

Benefits of performing tracheostomy for patients undergoing prolonged mechanical ventilation include improved patient comfort due to reduced oropharyngeal irritation and improved pulmonary toilet, which might also accelerate liberation from mechanical ventilation. A recent propensity-matched cohort study evaluated tracheostomy timing among patients with isolated severe TBI using data from hospitals participating in the American College of Surgeons Trauma Quality Improvement Program. In this observational study, early tracheostomy (≤ 8 days) relative to late tracheostomy (> 8 days) was associated with shorter mechanical ventilation duration and shorter ICU and hospital stays. This study also suggested that early tracheostomy is associated with lower risks of pneumonia, deep venous thrombosis, and decubitus ulcer.

TIMING OF SECONDARY PROCEDURES

Key Messages

- In patients with intracranial hypertension, consideration should be given to delaying trips to the operating room for non-intracranial procedures
- If patients with TBI require orthopedic operations, these should ideally be delayed 24 to 48 hours for initial stabilization of intracranial hypertension



- Laparoscopic procedures should be avoided
- Close monitoring is required during general anesthesia to avoid high ICP, hypotension, hypoxia, and hypo- or hypercarbia
- Intravenous anesthesia is preferable for severe TBI patients.
- Regional anesthetic techniques should be avoided in patients with intracranial hypertension

There are no large prospective studies defining the optimal timing of secondary extracranial surgery in patients with severe TBI. In making such decisions, close communication among the treating specialties is paramount. To avoid secondary brain injury, close monitoring during anesthesia is required to avoid hypotension, hypoxia, and hypo- or hypercarbia. While a single episode of hypotension doubles mortality, the combination of hypotension with hypoxia is associated with up to 75% mortality. If ICP is being monitored, CPP must be maintained at ≥ 60mm Hg. Because of the adverse effects of inhalational anesthesia on ICP, intravenous anesthesia may be preferable. Of note, regional anesthetic techniques are contraindicated in patients with intracranial hypertension.

Timing of orthopedic procedures (primarily long bone repair) does not appear to have an overall effect on outcomes in patients with severe TBI, with the following provisions. After initial stabilization, damage control orthopedics with early external fixation is favored, with delayed definitive treatment. This minimizes the "second hit" neurological phenomenon, triggered by the inflammatory response, hypotension, hypoxia, hyper- or hypocarbia, and intracranial hypertension, all of which are common occurrences with orthopedic procedures. Timing of spine fracture-dislocation surgery should depend on spine stability and the need for emergent spinal decompression in patients with spinal cord injury.

In patients with intractable intracranial hypertension, consideration should be given to delaying trips to the operating room unless life-saving procedures are required. Open laparotomy or open thoracotomy should be performed when needed, with adherence to the same general principles of avoiding secondary brain injury, as noted above. Laparoscopy should generally be avoided, especially early on, because it raises intra-abdominal pressure and also induces hypercarbia. The contribution of hypercarbia to long-term adverse neurologic outcomes is debatable, however. Routine ICU procedures, e.g., tracheostomy and percutaneous endoscopic gastrostomy may be performed once the patient's condition has stabilized.

TIMING OF PHARMACOLOGIC VENOUS THROMBOEMBOLISM PROPHYLAXIS

Key Messages

- Patients with TBI are at high risk for venous thromboembolism (VTE), with rates as high as 20-30%
- VTE prophylaxis should be considered within the first 72 hours following TBI in most patients. Earlier initiation of pharmacologic prophylaxis (<72 hours) appears to be safe in patients at low risk for progression of intracranial bleeding and have a stable repeat head CT scan
- Placement of a prophylactic inferior vena cava (IVC) filter should be considered in patients at high risk for progression of intracranial hemorrhage who cannot receive pharmacologic prophylaxis, including those with lower extremity long bone fractures or pelvic fractures in addition to TBI

Patients with TBI are at high risk for venous thromboembolism (VTE) with rates as high as 20-30%, even with appropriate mechanical prophylaxis. In spite of these risks, providers have traditionally erred on the side of withholding pharmacologic VTE prophylaxis, accepting a higher risk of a VTE event in order to prevent potential progression of intracranial hemorrhage following TBI. The challenge in deciding when to initiate pharmacologic prophylaxis lies in determining when the risk of progression of intracranial hemorrhage has become sufficiently low. Evidence suggests that delays in initiation of > 4 days after injury substantially increases the risk of VTE, so balancing these risks is critical. One approach is to ensure that the brain injury has stabilized on CT before initiation of prophylaxis. In several studies, pharmacologic prophylaxis is withheld pending a CT scan at intervals ranging from 24-72 hours post injury. In the absence of any changes on CT scan, prophylaxis with a low molecular weight heparin (LMWH) appears to be safe. Among patients who undergo evacuation of an intracranial bleed, it is advisable to wait for the head CT findings to stabilize before initiation of prophylaxis.

To provide some objective assessment of the risk of progression and to guide the timing of initiation of prophylaxis, Berne and others derived the Modified Berne-Norwood criteria (Table 3). Using this approach, it appears to be safe to initiate prophylaxis if the findings on head CT are stable (i.e., unchanged) after the first 24 hours. Prophylaxis should be withheld for at least 72 hours in patients who meet any of the moderate risk criteria or who demonstrate progression at 24 hours. If the head CT is stable at 72 hours, then prophylaxis may be initiated with low risk of progression. The high-risk group is perhaps the most challenging to manage. Because many such patients are excluded from observational studies, there are very few data upon which to base a strategy. A



Table 3. Modified Berne-Norwood Criteria

Low risk	Moderate risk	High risk
No moderate or high risk criteria	Subdural or epidural hematoma > 8 mm Contusion or intraventricular hemorrhage > 2 cm Multiple contusions per lobe Subarachnoid hemorrhage with abnormal CT angiogram Evidence of progression at 24 hrs	ICP monitor placement Craniotomy Evidence of progression at 72 hrs
Initiate pharmacologic prophylaxis if CT stable at 24 hrs	Initiate pharmacologic prophylaxis if CT stable at 72 hrs	Consider placement of an IVC filter*

**Consider alternate strategies as described in text*

retrievable IVC filter can be considered in these patients, particularly those who are very high risk for VTE (e.g., patients with lower extremity long bone fractures or pelvic fractures) and removed after the risk is reduced. Alternatively, surveillance duplex ultrasound of the lower extremity can be undertaken and if a DVT is identified, a IVC filter can be conconsidered. Finally, some centers initiate LMWH in patients with ICP monitors and following craniotomy after a stable head CT, although this practice has not been investigated.

MANAGEMENT CONSIDERATIONS FOR PEDIATRIC PATIENTS WITH TBI

Key Messages:

- Transferring children with TBI to a pediatric trauma center leads to decreased morbidity and mortality. If this is not possible, they should be transported to an adult trauma center capable of treating pediatric patients
- Pediatric TBI protocols should incorporate age appropriate physiologic parameters

All aspects of care of the pediatric patient with TBI should be optimized starting with pre-hospital management throughout transport and admission. Transferring children with TBI to a pediatric trauma center leads to decreased morbidity and mortality. If this is not possible, they should be transported to an adult trauma center capable of treating pediatric patients. For adult trauma centers that receive pediatric patients, the development pediatric TBI protocols are recommended. Hypoxia and hypotension should be prevented at all times during pre-hospital and in-hospital care. Because children of different ages have differing blood pressure and ventilation parameters, it is important to maintain meticulous adherence to age appropriate parameters.

Data from well-designed, controlled studies on acute management of TBI in the pediatric population are limited. The "Guidelines for the Acute Medical Management of Severe Traumatic Brain Injury in Infants, Children, and Adolescents" are aimed to be a comprehensive document reviewing the literature on all aspects of pediatric head injury management. These Guidelines were updated in 2012, and provide detailed TBI management algorithms for children supported by the current knowledge base.

With the exception of age appropriate parameters for blood pressure, the tiered approach for management of intracranial hypertension and operative management outlined in previous sections also apply to children.

MANAGEMENT CONSIDERATIONS FOR ELDERLY PATIENTS WITH TBI

Key Messages:

- Neurologic evaluation of the elderly patient with TBI can be complicated by pre-existing dementia, cognitive decline, or hearing/vision deficits; careful determination of pre-injury neurological baseline via family and caregivers is important
- Anticoagulants and anti-platelets medications can exacerbate the sequelae of TBI; reversal of these medications, if feasible, is an important early management goal
- Older age is associated with higher mortality and worse functional outcomes following TBI. However, age, in isolation, should not be considered a valid reason for treatment limiting decisions

History obtained from the patient or family can be very helpful, as comorbid conditions can profoundly affect the impact of a TBI on an elderly patient. In addition, medications that are frequently utilized in the elderly can exacerbate TBI (anticoagulants/anti-platelets) or confound evaluation. With the increasing use of Novel Oral Anticoagulants (NOAC) (for example, rivaroxaban/apixaban, dabigatran) the approaches to reversal are evolving. Case series have reported success with tranexamic acid and Factor Eight Inhibitor Bypass Activity (FEIBA)



to reverse dabigatran (with or without hemodialysis) and Prothrombin Complex Concentrate (PCC) for rivaroxaban/apixaban. It is suggested that each center develop its own protocol for rapid reversal of anticoagulants using local expertise. For more information about reversal of anticoagulants in the elderly, please refer to the ACS TQIP Geriatric Trauma Management Guidelines.

Neurologic evaluation of the elderly patient with TBI can often be complicated by pre-existing dementia, cognitive decline, or hearing/vision deficits. Family and caregivers can be invaluable sources of information when trying to determine a neurologic "baseline." Determining the appropriate level of diagnostic evaluation is important. One study found that in elderly patients with mild head injury, 14% of patients had evidence of traumatic lesion on head CT, with 20% of those lesions requiring neurosurgical intervention. Therefore, the American College of Emergency Physicians recommends that a head CT be obtained in any patient age ≥ 65 years who presents with mild head injury.

There is a paucity of information related to acute management of intracranial hypertension resulting from TBI in the elderly. Age-related changes in intracranial space are known to lower ICP significantly, with a concomitant rise in CPP. Further, cerebral autoregulation and pressure reactivity indices are known to decrease over time. These changes can be complicated by comorbid conditions and medications that are more common in the elderly patient sustaining TBI. Well-studied recommendations for optimal CPP thresholds in the elderly are lacking.

It is clear that as age advances, the risks of mortality and poor functional outcome from TBI increase. This is true for all types of brain injury, but most striking with a GCS < 9. Despite this grim prognosis, 30% of elderly TBI patients with severe TBI can survive to leave the hospital. There is tremendous variability in the aggressiveness of medical care following traumatic brain injury. This likely is due to local, regional, and cultural differences in how care is provided. Many of those deaths occur early after brain injury and likely reflect early decisions to withdraw life-sustaining therapy. At this time, due to the lack of sufficient prognostic tools, it is difficult to determine which patients may go on to have a meaningful recovery. Arbitrary age thresholds for limitations of care should be avoided. Rather, a detailed discussion with the family and decision-makers should center around the severity of injury, comorbid conditions, and respect for a patient's previously expressed wishes.

PROGNOSTITC DECISION-MAKING AND WITHDRAWAL OF MEDICAL SUPPORT

Key Messages:

- Severe TBI patients should receive full treatment for at least 72 hours post-injury
- Age alone should not be considered a valid reason for treatment-limiting decisions
- Caution is advised when using prognostic models in individual patients, in particular when considering treatment-limiting decisions
- It is strongly encouraged that each hospital develop a brain death determination policy that derives from accepted national standards

Patients with severe TBI are, by definition, severely injured and at high risk of death or long-term disability. Decisions regarding treatment approaches must be made rapidly and at the time of initial injury. Some physicians have advocated that care should be limited in those patients deemed to have a very poor prognosis for meaningful recovery as assessed by initial parameters such as GCS score, pupillary reactivity, patient age, and findings on neuroimaging. Mathematical models have been developed on populations of patients which may provide general guidance as to predicted outcome. The most extensively validated of these are the IMPACT and CRASH TBI models. Caution is recommended against using these outcome models for prognosticating on individual patients; all of these models are developed on specific populations of patients and produce point estimates with confidence intervals. Physicians using these models to discuss prognosis too often discuss the point estimates but do not include confidence intervals or explain the inherent uncertainty in these models (or in prognostication in general).

Numerous studies across various neurocritical care conditions, including intracerebral hemorrhage, global cerebral ischemia after cardiac arrest, and TBI, have found that reflexive default to early care limitations such as do-not-resuscitate (DNR) orders or withdrawal of medical support is associated with worsened outcome independent of other patient characteristics. Other studies have found that the ability to accurately and precisely prognosticate long-term outcome very early in a patient's course after severe TBI is limited and frequently incorrect (especially within the first day after injury). All of these findings have raised the concern of a "self-fulfilling prophecy" of poor outcome in those patients who do not receive aggressive care.



Given these concerns, the advocated best practice is to provide all severe TBI patients with a trial of aggressive therapy and not limit any interventions for at least 72 hours post-injury. While this time period is somewhat arbitrary, it represents a minimum period during which the effectiveness of initial interventions and the likelihood of patient survival can be assessed. Exceptions would be patients who are brain-dead or in whom a pre-injury Advance Directive states that such intervention is not desired. A longer period of treatment and observation is typically needed for prognosis of neurological recovery. Age, taken in isolation, should not be considered a valid reason for treatment-limiting decisions.

State law governs the criteria for the determination of brain death. However, standardized criteria for the determination of brain death have been developed and should be utilized. Specifically, patients must have no response to central pain, absent brainstem reflexes, and the inability to breathe independently. The clinical examination should be used rather than a confirmatory test, such as electroencephalography or cerebral blood flow assessment, unless prerequisites for using the clinical examination cannot be met. It is strongly encouraged that hospitals develop a defined brain death determination policy that derives from the accepted national standards.

OUTCOME ASSESSMENT AND QUALITY IMPROVEMENT IN TBI

Key messages:

- Outcome assessment is essential to benchmarking the quality of care in TBI patients
- A standardized and structured outcome assessment using the GOS-E at 6 months is recommended for TBI patients

TBI is a major cause of long-term change in functional, physical, emotional cognitive, and social domains. Assessment methods have different strengths and weaknesses, and few can be applied across the complete TBI severity spectrum. For a global assessment of function, the Glasgow Outcome Scale (GOS) or its expanded version, the Glasgow Outcome Scale-Extended (GOS-E) is broadly used to assess outcome of TBI. While the GOS/GOS-E may be appropriate for rating outcome in the long term, it is not suited for assessing outcome upon discharge. This is particularly notable for patients at the more severe end of the TBI spectrum who have been admitted to the intensive care unit. These patients are often in poor condition on discharge from the intensive care unit but improve over the weeks and months thereafter. Observing these changes and evaluating long term outcomes may provide

reinforcing evidence for establishing best practices to treat patients aggressively in the first days post-injury.

Improvement after TBI may occur over months or even years. Conversely, a minority of patients may show deterioration over time. A standardized and structured functional outcome assessment using the GOS-E) at six months post-injury is recommended for all TBI patients. The six-month point for standardized outcome assessments reflects a compromise between what is clinically feasible and eventual long-term clinical outcome. TBI predictive models developed on large clinical series (> 8000 patients), such as IMPACT and CRASH, provide individualized risk estimates and thus can enable establishment of baselines for clinical audits and benchmarking by permitting analysis of observed/ expected outcome. These models have been developed not only for mortality but also for functional outcome. The IMPACT model has been externally validated in multiple studies and is now being used to benchmark care in a growing number of trauma centers.

Process indicators should be identified and monitored regularly for TBI. ICP monitoring, DVT prophylaxis, nutrition, tracheostomy, time to withdrawal of support, and 6-month outcome assessment are recommended. Some system-wide process indicators, such ventilator associated pneumonia (VAP), may not be appropriate for severe TBI as these patients are at high risk for infections. Other routine measures of quality care, such as patient satisfaction surveys, may also be inappropriate for TBI patients due to cognitive and behavioural issues.



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