Definition

Traumatic Brain Injury (TBI) occurs when an external blunt or penetrating force causes brain dysfunction. The damage to the brain can be separated into primary and secondary injury. Primary injury is the instantaneous damage resulting from mechanical forces (i.e. intracranial hemorrhage). Secondary injury is the subsequent damage that occurs over hours to days as a result of altered cerebral blood flow and inflammatory processes.

Incidence

- TBI is the leading cause of morbidity and mortality in children in the United States. According to the CDC, approximately 500,000 children sustain a TBI annually.
- More than half (55%) of all TBI in children from 0-14 years are related to falls.
- TBI-related deaths in children 0–4 years are primarily associated with non-accidental trauma (43%) and motor vehicle crashes (29%).
- Motor vehicle crashes account for a majority of TBI-related deaths (56%) in youth 5–14 years[24].

Etiology

- Management of traumatic brain injury is focused on the prevention of secondary brain injury to improve outcome. Standard neuro-protective measures are based on management of intracranial pressure (ICP) and cerebral perfusion pressure (CPP) to optimize cerebral blood flow and oxygenation and avoid or minimize secondary brain injury.
- This guideline is based on national pediatric guidelines from the Brain Trauma Foundation and is designed to be followed in a stepwise manner.

Guideline Eligibility Criteria

Patients from newborn through adolescence with moderate (GCS 9-12) to severe (GCS </= 8) TBI.

Guideline Exclusion Criteria

Increased ICP from non-traumatic etiologies such as near-drowning or CPR (anoxia) or space-occupying lesions (tumors) are excluded. This is a guideline only. Individual circumstances need to be considered, as there may be times when it is appropriate or desired to deviate from this guideline.

Diagnostic Evaluation

History

Assess for a history of blunt or penetrating head trauma. There may be no history of trauma provided in the setting of abuse, which is the leading cause of death from TBI in young children. History should include any loss of consciousness and altered mental status including Glasgow coma scale (GCS) when available. Assess for a history of seizures and focal neurological deficits such as hemiparesis, non-reactive or unequal pupils, and posturing. Clinical presentation in infants with abusive head trauma often includes non-specific signs and symptoms such as fussiness/irritability, lethargy, poor feeding, vomiting, apnea and respiratory arrest.

Physical Examination

A brief neuro exam including best GCS (using the pediatric coma scale) and pupillary response should be performed during the primary survey, per Advanced Trauma Life Support (ATLS) guidelines. An accurate GCS prior to administration of sedating medications is important for decision-making in early management of TBI. A thorough neurological evaluation is done during the secondary survey with a head to toe examination including assessment for focal deficits.

Laboratory Tests

There are several serum TBI-metabolites that represent disruption in the blood-brain barrier and may improve prediction of patient outcomes (Oresic). As with any critically ill trauma patient, laboratory studies including blood gas, type and cross, hemoglobin and hematocrit, metabolic profile and clotting studies should be obtained.

Critical Points of Evidence

Evidence Supports

Summary Updated National Guidelines

- The Brain Trauma Foundation (BTF) publishes recommendations for management of pediatric TBI with the most recent 3rd edition guidelines released in 2019. The 2019 publication is part of an effort to update a suite of 3 Brain Trauma Foundation Guidelines including similar acute care guidelines for adults and guidelines for prehospital management of all ages[24]. The bullet points are a summary of the updated guidelines.
  - Use of ICP monitoring may be considered in infants and children with severe TBI with treatment considered at a threshold of 20mmHg (Level III).
  - There has been insufficient evidence to support a recommendation for the use of a monitor of Pbro2 to improve outcomes
  - If brain oxygenation monitoring is used, maintenance of partial pressure of brain tissue oxygen (PbtO2) >/= 10 mmHg may be considered (Level III).
  - A minimum CPP of 40mmHg may be considered in children with TBI (Level II).
  - A CPP threshold of 40-50mmHg may be considered; there may be age-specific thresholds with infants at the lower end and adolescents at the upper end of this range (Level III).
  - Excluding the possibility of elevated ICP on the basis of a normal initial (0-6hr after injury) CT examination of the brain is not suggested in a comatose pediatric patient (Level III)[24].
  - In the absence of neurologic deterioration or increasing ICP, obtaining a routine repeat CT scan >24 hrs after admission and initial follow-up study may not be indicated for decisions about neurological intervention (Level III).
  - Bolus HTS (3%) is recommended in patients with intracranial hypertension. Recommended effective doses for acute use range from 2-5ml/kg over 10-20 minutes (level II) [24].
  - Effective doses as a continuous infusion of 3% saline range between 0.1 and 1.0ml/kg/hr, administered on a sliding scale; the minimum dose needed to maintain ICP < 20mmHg should be used.
  - Bolus of 23.4% HTS is suggested for refractory ICP. The suggested dose is 0.5ml/kg with a maximum of 30 ml (Level III) [24].
  - Although mannitol is commonly used in the management of raised ICP in pediatric TBI, no studies meeting inclusion criteria were identified for use as evidence for this topic. Mannitol continues to have no contemporary controlled studies versus placebo, other osmolar agents or other therapies in children. All studies were limited to the 1970s and is therefore still not considered standard of care for TBI pediatric patients [24].
  - Prophylactic moderate hypothermia (32-33℃) is not recommended over normothermia to improve overall outcomes (Level II) [24].
  - Moderate hypothermia (32-33℃)for up to 48 hours duration should be considered to reduce ICH only for refractory ICP not responsive to first therapies (Level II). If hypothermia is induced for any indication, rewarming at a rate of 0.5-1.0℃ every 12-24 hours or slower is recommended to avoid complications (Level II) [24].

- If phenytoin is used during hypothermia, monitoring and dosing adjusted to minimize toxicity, especially during the rewarming period are suggested (Level III) [24].
- With the use of multiple ICP-related therapies, as well as the use of appropriate use of analgesia and sedation in the routine ICU care, avoiding bolus administration of midazolam and/or fentanyl during ICP crises is suggested due to the risks of cerebral hypoperfusion (Level III) [24].
- Continuous infusion of propofol for either sedation or management of refractory ICH in infants and children with severe TBI is not recommended (Level III) [24].
- Etomidate use remains largely controversial due to the accompanying adrenal suppression but a single dose used for intubation remains widely accepted [24].
- CSF drainage through an external ventricular drain is suggested to manage increased ICP [24].
- Prophylactic seizure treatment in the setting of severe TBI is suggested to reduce the occurrence of early (within 7d) post traumatic seizures. There is insufficient evidence to recommend levetiracetam over phenytoin based on either efficacy in preventing early PTS or toxicity (Level III)[24] (see separate seizure section).
- High-dose barbiturate therapy is suggested in hemodynamically stable patients with refractory ICH despite maximal medical and surgical management. When high-dose barbiturate therapy is used to treat refractory ICH, continuous arterial BP monitoring and cardiovascular support to maintain adequate CPP are required (Level III).
- Decompressive craniectomy is suggested to treat neurological deterioration, herniation or ICPs refractory to medical management during the early stages of their treatment (Level III)[24].
- Prophylactic severe hyperventilation to a PaC02 <30mmHg in the initial 48 hours after injury is not suggested (Level III)[24].
- If hyperventilation is used in the management of refractory ICH, advanced neuro-monitoring for evaluation of cerebral ischemia is suggested (Level III)[24].
- The use of corticosteroids is not suggested to improve outcome or reduce ICP. This is not intended to circumvent use of replacement corticosteroids for patients needing chronic steroid replacement therapy, those with adrenal suppression and those with injury to the hypothalamic-pituitary steroid axis(Level III)[24].
- In the absence of outcome data, the specific approach to glycemic control in the management of infants and children with severe TBI should be left to the treating physician (Level III).
- Initiation of early enteral nutritional support (within 72 hours from injury) is suggested to decrease mortality and improve outcomes(Level III)[24].

Literature Review of Specific Topics

- The most recent edition of the updated guidelines for Management of Pediatric Severe Traumatic Brain Injury includes 48 new studies. Although some progress has been made, overall the level of evidence informing these guidelines remains low.

Guideline Adherence

- Outcomes including discharge disposition and mortality rates in children with severe TBI are improved with implementation of a standardized protocol for management of traumatic brain injury[31]. Clinical indicators of adherence to national guidelines were associated with significantly higher discharge survival and improved discharge Glasgow outcome scale (GOS)[47].
- For reasons that are unclear, there continues to be low adherence to TBI management guidelines cited in the literature, specifically in regards to ICP monitoring. Many children do not undergo ICP monitoring with variability lying between 7 & 60% of children with severe TBI[37]. Reasons cited in this study of 64 patients included rapid improvement, moribund status and reassuring CT scan. Another study demonstrated that children < 2 years with severe TBI (GCS < 8) were less likely to receive an ICP monitor or hyperosmolar therapy in the last decade (2010 cohort) compared with children from the previous decade (2000 cohort), again suggesting significant treatment variation and low adherence to national guidelines[15].

Prehospital Care

- Hypoxia is an independent risk factor for both increased morbidity and mortality after TBI[30]. Ventilation can be performed via bag-valve mask or via endotracheal intubation. There has been no demonstrated difference in morbidity or mortality between the 2 modalities[11]. Rapid sequence intubation with pre-oxygenation should be used in almost all patients because the sympathetic stimulus from endotracheal intubation can cause an increase in ICP[30].
- Arrival hypercapnia and hypocapnia are common and associated with worse outcomes in intubated adults with TBI. Increased survival and good outcomes were noted (after adjusting for multiple variables) in patients with arrival PC02 of 30-49mmHg and authors believe this is an optimal target range for capnometry-guided ventilation[14]. A retrospective study of 194 children showed higher survival with admission PaO2 >300mmHg and PaCO2 of 36-45mmHg (Ramaiah).
- Hypotension is the single most powerful prognosticator of poor outcomes in pediatric patients with TBI. Even transient hypotension decreases the discharge survival rate 4-fold with the first 6 hours being the most sensitive period[30].
- Hypoxia (PaO2 of < 60mmHg or an oxygen saturation of < 90%) is one of the primary parameters associated with discharge survival; the higher the initial partial pressure of oxygen is, the higher the rate of long-term survival[30].
- Hypotension and hypoxia are common events in pediatric traumatic brain injury. Approximately 1/3 of children are not properly monitored in the early phases of management. Intervention to manage hypotension and hypoxia significantly improves outcomes[50].

Advanced Neuro-monitoring

- There is insufficient evidence to support a recommendation for the use of a monitor of P02 in brain interstitium to improve outcomes[24].
- Use of advanced neuromonitoring should only be for patients with no contraindications to invasive neuromonitoring such as coagulopathy and for patients who do not have a diagnosis of brain death[24].

Decompressive Craniectomy

- Decompressive craniectomy (DC) for the management of severe TBI is controversial. In a retrospective study of 17 pediatric patients with severe TBI found no significant differences in survival between patients with DC and controls, however, among survivors at 4 years after the TBI, 42% of the DC patients had mild disability or Glasgow outcome score (GOS) of 5 versus none of the controls [29].
- In a retrospective review of 386 children with severe TBI with 14 undergoing DC, authors concluded that DC effectively reduces ICP with low mortality and good long-term prognosis of survivors. Complications related to surgery are frequent with the most common being hygroma and infection (Suarez).
- In a retrospective study of 23 children who underwent DC for severe TBI, authors examined outcomes at discharge, 6 months and 2 years. They reported that 82% of survivors had good functional outcomes, with only 9% of surviving patients demonstrating severe disability at the 2-year follow-up[21].

Seizure Prophylaxis

- Early posttraumatic seizures may contribute to worsened outcomes after TBI and evidence to guide the evaluation, prevention and treatment of seizures is limited. There is significant variation in EEG monitoring, seizure prophylaxis and management of post-traumatic seizures across institutions. Sites with treatment protocols reported a decreased number of medications used[25].
- Continuous EEG monitoring identifies a significant number of subclinical seizures following TBI. Children <2.4 years and victims of abusive head trauma are particularly vulnerable to seizures. Continuous EEG monitoring allows for accurate diagnosis and timely treatment of post-traumatic seizures, and may mitigate secondary injury[32].
- Overall the use of Levetiracetam and Phenytoin is considered safe to reduce Post Traumatic Seizures[24].
- Effective prophylaxis of early post-traumatic seizures reduces brain metabolic demands, thereby reducing intracranial pressure and neurotransmitter release, as well as the risk of secondary brain injury[22].

Evidence Lacking/Inconclusive

Seizure Prophylaxis

- At the present time, there is insufficient evidence to recommend levetiracetam over phenytoin based on either efficacy in preventing early PTS or toxicity[24].
- The optimal duration of EEG monitoring is unclear. In a survey of neurophysiologists and neurointensivists from 97 institutions, nearly all respondents utilize continuous EEG to monitor adult patients with TBI and altered mental status. The majority of physicians surveyed monitor comatose patients for 24-48 hours[16]. Adult studies suggest a minimum of 24-72 hours of continuous EEG to detect seizures after acute brain injury[13].
- Post traumatic seizure risk factors are not completely understood. According to Bennett et al, risk in children with severe traumatic brain injury is greatest with younger age, injury by abuse, and subdural hemorrhage[8].

Hyperglycemia

- Currently there is insufficient data to recommend for or against tight glucose control for children with severe TBI and persistent hyperglycemia[24].

Hypertonic saline and risks

- While hyperosmolar therapy is a standard therapy for controlling ICP, emerging literature suggests that a hyperosmolar state may lead to thrombotic complications. In one study, the total bolus volume of 3% hypertonic saline and sustained levels greater than 160 mmol/L were independently associated with deep vein thrombosis[48].
- A sustained level (>72hrs) of serum sodium greater than or equal to 170mEq/L was significantly associated with thrombocytopenia and the need for erythrocyte transfusion[24].

Bolus fentanyl/versed

- Avoiding bolus administration of midazolam and fentanyl during ICP crisis is suggested due to risks of cerebral hypoperfusion[24]. Furthermore, there is concern for a paradoxical response from bolus doses of versed and fentanyl which may actually increase ICP in this population. By comparing the intracranial hypertension (ICH) pressure-time exposure before and after drug administration, authors noted an overall increase in ICH burden following drug administration[49].

Evidence Against

Hypothermia

- The Cool Kids Trial was a randomized controlled trial comparing hypothermia versus normothermia in pediatric patients with severe TBI. The study was terminated early due to futility after an interim data analysis cited that hypothermia for 48 hours with slow rewarming does not reduce mortality or improve global functional outcomes[2].
- A recent randomized controlled study out of New Zealand reported that early therapeutic hypothermia continued for 72 hours with slow rewarming did not improve outcomes and recommended that hypothermia not be used outside of clinical trial[7].
- A meta-analysis of the efficacy and safety of hypothermia in pediatric TBI found no benefit to this therapy and reported that it may increase the risk of mortality and arrhythmia in this population. Authors further stated that there is no evidence that therapeutic hypothermia improves prognosis of children[51].
- Another meta-analysis of potential benefits of therapeutic hypothermia in pediatric TBI found a slightly increased risk of mortality and cardiac arrhythmias compared with the normothermic group[28].

Practice Recommendations & Principles of Clinical Management

- **TBI Definitions:**
  - **Mild TBI:** GCS 13-15
  - **Moderate TBI:** GCS 9-12
  - **Severe**: <=8
- The primary goal in treating severe TBI is to avoid secondary injury, specifically hypotension and hypoxia, as both are associated with poor outcomes.
- Assume every patient who suffered a severe TBI also has elevated ICP. Tier I is the foundation of TBI management and is applicable to patients in the early stages of care including pre-trauma center care & ED as well as the operating room and inpatient settings.
- TBI management is based on a tiered approach designed to be followed in a stepwise manner. If ICP and CPP are not well-controlled with the initial level of care, the next tier of therapy should be instituted. As the patient improves, de-escalation of care progresses in the reverse order.

Tier I Therapy

Positioning

- Goal: Optimize cerebral blood flow
- Maintain head in midline position.
- Elevate head of bed 30 degrees when hemodynamically stable.
- Reverse Trendelenburg may be used with spine precautions.
- Apply an appropriate well-fitted collar that is intended for long term use.
- The collar should not be too tight which may occlude venous return.

Respiratory

- Goals: Avoid hypoxia
- Intubate for uncontrolled airway or GCS <9
  - Preoxygenate prior to intubation.
  - Intubate using Rapid Sequence Intubation according to the DCMC RSI Guideline.
    a. Unless CPR in progress or impending.
- Optimize oxygenation/ventilation.
- Initially provide 100% FiO2 to maintain PaO2 as high as possible.
- Wean oxygen to maintain PaO2 > 80mmHg and SaO2 >94%
- PaCO2 35-40mmHg - trend end tidal with blood gases.
- Mode of ventilation chosen at discretion of provider.
- Use lowest appropriate physiological PEEP.
- Ensure adequate oxygenation while suctioning; increase to 100% FiO2 during suctioning.

Cardiovascular

- Maintain appropriate age-specific blood pressure
- Fluid resuscitation with NS, and/or blood products as indicated
  - 60cc/kg total fluids as needed to maintain blood pressure
  - Total volume should include 3% NaCL boluses
- Vasopressor support as appropriate.

Fluids & Electrolytes

- Goal: Normovolemia
  - Keep total fluids at maintenance
    a. adjust maintenance fluids accordingly.
- Goal: Normoglycemia
  - Initiate Insulin as indicated per the PICU Non-Ketotic Hyperglycemia Protocol.

- Administer D10W 5ml/kg for blood sugar < 60mg/dL.

- Goal: Avoid hyponatremia which may lead to cerebral edema
  - Maintain serum Na > 140mg/dL
  - NS or 3% NaCl as needed to achieve this goal
  - 3% NaCl 2-5mg/kg over 60 minutes or less
    a. adjust rate based on the clinical situation to include IV push in urgent situations.
  - Monitor serum Na q4-6 hrs

Hematology

- Goals: Minimize risk of further bleeding
- Avoid hypotension
  - Correct coagulation abnormalities with vitamin K and blood products.
  - Goal:
    a. INR < 1.4
    b. Platelets >100,000
    c. Hemoglobin >= 7 with higher goals as clinically indicated[44]

Temperature

- Goal: Normothermia
  - Maintain temperature less than or equal to 37 degrees with the following:
  - Antipyretics
  - Apply cooling blanket per normothermia protocol.
  - Monitor for shivering which increases metabolic demand.
  - Shivering can be controlled with vecuronium in intubated patients sedated to RASS -4 or -5.

Sedation and Analgesia

- Continuous analgesic and sedative infusions may be used per routine ICU care
- Continuous Analgesics: Fentanyl, Morphine, Hydromorphone
- Continuous Sedatives: Dexmedetomidine, Midazolam
- Avoid bolus doses of sedatives and analgesics to treat acute elevations in ICP
- Sedation holiday – may be needed to evaluate neurological status at discretion of team.

Neuromuscular Blockade

- Neuromuscular blockade may be added to intubated patients sedated to RASS -4 or -5
- Vecuronium initial bolus dose 0.1mg/kg; if effective start continuous infusion - 0.05-0.1mg/kg/hr.
- Initiate continuous EEG monitoring with continuous infusion.
- Start scheduled ocular lubricant (Lacri-lube®) therapy.

Ventriculostomy Placement

- Placement of both external ventricular drain (EVD) and intracranial pressure (ICP) monitor is recommended for patients with severe TBI.
- Change EVD dressing q7 days, or when visibly soiled, loose, or when chlorhexidine impregnated patch is more than 50% saturated, per nursing policy. ICP monitors may or may not have a dressing.
- Administer Cefazolin 33 mg/kg IV push prior to monitor placement and x 1 post-operatively
- Transduce/record ICP and CPP q1 hour
- Maintain CPP > 40mmHg for children less than 1 year old
- Maintain CPP > 50mmHg for children 1-12 years
- Clamp EVD and open to drainage if ICP >20 sustained for 10-20 minutes unless otherwise instructed by the neurosurgery team.

Hyperosmolar therapy

- Administer 3% NaCl 2-5mL/kg infused over 60 minutes (or less per provider discretion) q4hr prn ICP > 20mmHg and as needed to keep serum Na+ at desired range.

- Consider a 3% NaCl continuous infusion (0.1-1ml/kg/hr, central line preferred for prolonged administration) and/or adding sodium to feeds.
- Check serum sodium q 4hrs.
- Hold for serum sodium greater than 155-160mg/dL

Tier II Therapy

Initiate for ICH refractory to previous measures:

- Repeat imaging to rule out surgical lesions and evaluate ventricle size
- Decompressive craniectomy
- Moderate Hypothermia (32-34℃)
- Hyperventilation(28-24mmHg) (Consider advanced neuromonitoring at this time)
- Higher levels of hyperosmolar therapy
  - **Sodium Chloride 23.4% (HTS 23.4%)**
    a. Consider administration of HTS 23.4% for patients who meet criteria for ICP >20 mmHg that are refractory to 3% NaCl bolus x 2 doses, and have received routine analgesia and sedation or who are fluid overloaded or at risk for fluid overload.
    b. Dose of HTS 23.4% is 0.5 mL/kg, Max 30 mL, given over 10 minutes.
    c. Sodium Chloride 23.4% is contraindicated in patients with Serum Sodium >155 mmol/L.
  - **Mannitol 25%**
    a. Consider administration of Mannitol 25% (0.25- 0.5 grams/kg/dose) q 4hr prn ICP > 20mmHg refractory to above measures. Monitor closely for hypotension.
    b. Infuse over 20-30 minutes; the rate may be adjusted based on the clinical situation to include IV push in urgent situations.
    c. Check serum osmolarity q 4hrs.
    d. Consider holding Mannitol when serum osmolarity is >320 mOsm
- Barbiturate Therapy- Pentobarbital
  - For intermittent ICH, bolus with 2-5 mg/kg every 4-6 hours as needed.
  - For persistent ICH, bolus with 2-5mg/kg and begin continuous infusion at 1 mg/kg/hour.
  - Continuous EEG monitoring with intermittent or continuous pentobarbital therapy.
  - Adjust infusion to achieve burst suppression.
  - Stop midazolam, vecuronium, and anticonvulsant medications while in burst suppression.
  - Barbiturate therapy should be used with caution in limited time frames.
- Advanced Neuro-monitoring (ie: PRx, CBF, TCD)
  - Case specific and Neurosurgery Attending must be consulted for placement and guidance.

Anti-Seizure Medications

- Seizure prophylaxis is indicated:
  - Patients with severe TBI (GCS <= 8) - this applies to patients treated at either Tier I or Tier II.
  - Children <3 years with moderate to severe TBI
  - Suspected Abusive Head Trauma with intracranial hemorrhage, cerebral contusion, or infarction
- After starting seizure prophylaxis
  - Begin EEG and continue for a minimum of 24 hours.
  - Consult neurology for EEG interpretation and further recommendations.
- Keppra or Fosphenytoin are both considered appropriate for loading and maintenance
  Dosing:
  - Keppra 30mg/kg IV followed by 15mg/kg IV/PO every 12 hours (max dose 3g).
  - Fosphenytoin 20mg PE/kg IV followed by 2mg PE/kg IV every 8 hours (max dose 1.5g).
- See updated EEG guideline
- For patients with TBI AND witnessed seizure activity or EEG with seizures:

- Consider STAT dose of Ativan 0.1mg/kg (max 4 mg IV) for cessation of seizure. May repeat x 1.

Discontinuation of Anti-Seizure Medications (ASM)

- Improved neuro exam and EEG of minimum of 24 hours without evidence of seizures.
  - May discontinue ASM after 7 days without repeat EEG
- Witnessed seizures, evidence of seizures on EEG or continued poor neuro exam
  - Neurology recommendations for time frame for ASM
  - ASM likely to continue for 3 months with neurology following

Consults and Referrals

Neurology Consult

- For any patient meeting criteria for EEG monitoring:
  - Patients with severe TBI (GCS <=8)
  - Moderate TBI with AMS
  - Children <3 year with moderate-severe TBI.
  - Suspected Abusive Head Trauma with space occupying lesions
- For patients with TBI and witnessed seizure activity.
- Further Consults as needed specific to patient needs

Audiology Consult

- For any patient with moderate or severe TBI
- For any patient with multiple/severe skull fractures

ENT consult

- For any patient with temporal bone fracture

Follow-Ups

Neurology

- Patients with any clinical or subclinical seizure activity following head injury
- Patient put on ASM that will continue past discharge date
- Any patient deemed appropriate for follow up per neurology discretion

ENT

- Per ENT discretion with/without hearing screen

High Risk TBI/Concussion

- Follow up in multidisciplinary TBI clinic 2 weeks post discharge

Outcome Measures

TBI Management

- All patients with Severe TBI that do not progress to Tier II therapies will have appropriate CPP maintained.
- All patients with Severe TBI will have an ICP placed.

Seizure Management

- All patients with severe TBI, moderate TBI with AMS, and children <3 year with moderate-severe TBI will have EEG monitoring initiated within 6 hours of admission to PICU.
- All patients with a severe TBI (GCS <=8) will have EEG monitoring sustained for a minimum of 24 hours.

Related Policies

Management of 23.4% Hypertonic Saline for Intracranial Pressure Reduction	01/2021
Administering Intravenous Hypertonic Saline Pedi	06/2020
External Ventricular, Lumbar, and Subdural Drains - NICU	07/2020
Seizures – Managing the Patient at Risk- Acute	07/2015

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Signatures

The signatures below indicate support for the attached guideline, protocol and/or algorithm. The intent is not to be prescriptive but to provide a cohesive, standardized, and evidence-based (when available) approach to patient care. The physician must consider each patient and family's circumstance to make the ultimate judgment regarding best care.

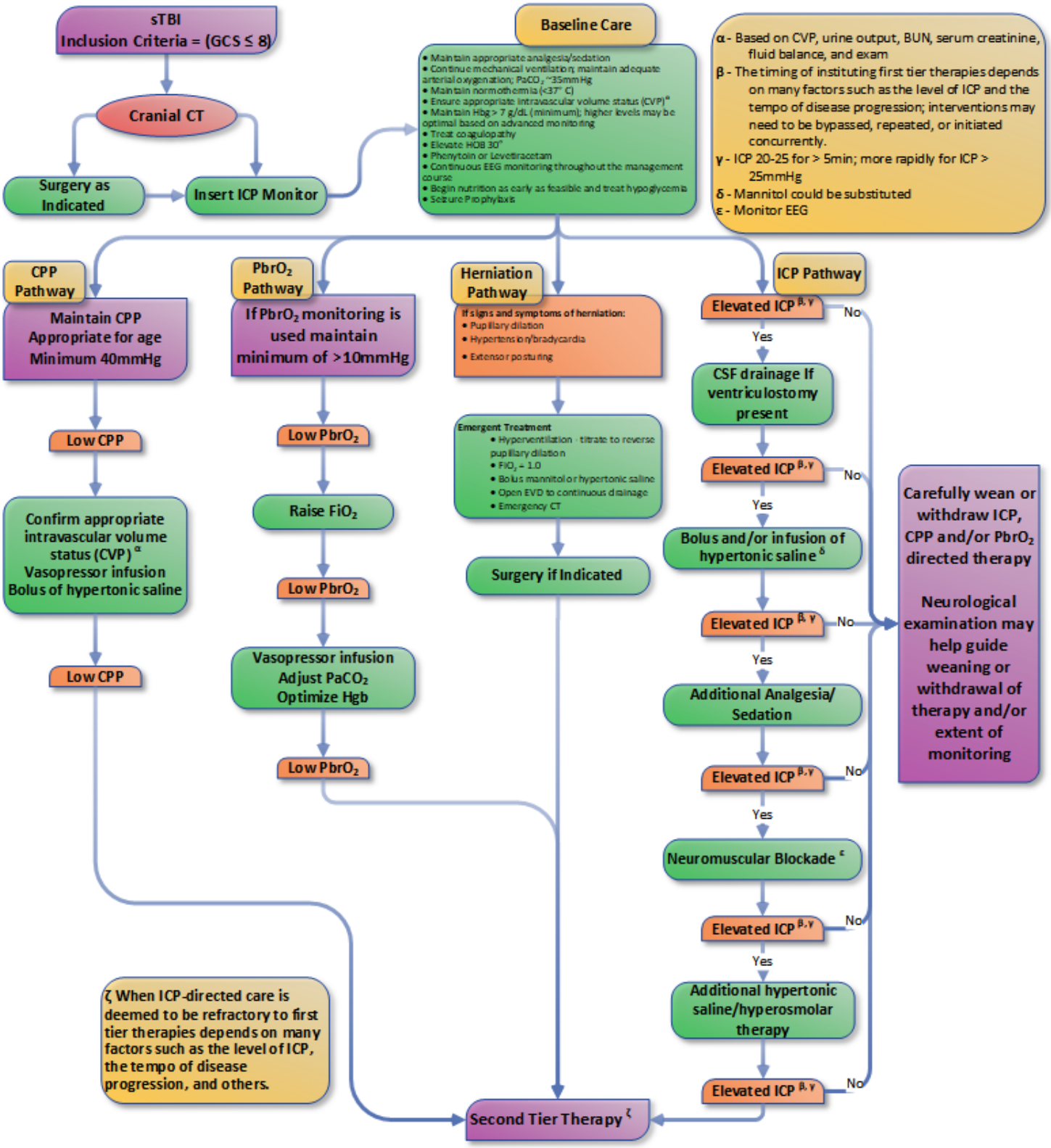
Approved by Trauma Council: 12November2021

By: _____ **12Nov2021**
Dr. Jessica Naiditch, Trauma Council Chair Date

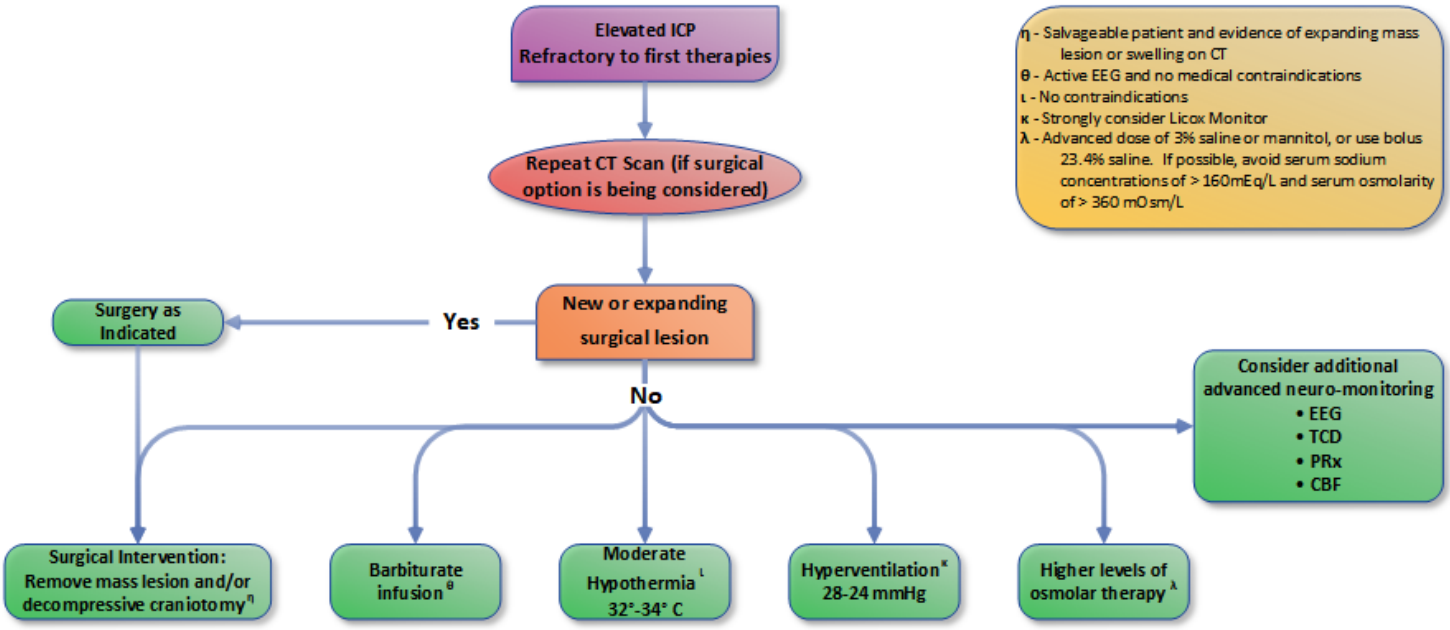
By: _____ **12Nov2021**
Dr. Nilda Garcia, Surgeon in Chief Date

Origination Date: October 2011
Revision Date: May 2013, May 2017, February 2021, November 2021

Traumatic Brain injury (TBI)
Tier I



Traumatic Brain injury (TBI)
Tier II



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