Adult Traumatic Brain Injury Management Guideline

Policy:

University Hospital is the Level I Trauma Center for the Central New York Region. This region consists of 14 counties and 28 hospitals, as determined by the New York State Department of Health. Upstate Medical University is the definitive care facility for this area. Strong evidence currently exists showing improved patient outcome associated with adherence to evidence based guideline for traumatic brain injury (TBI) management. The purpose of this guideline is to provide a framework to ensure uniform adherence to evidence based Guidelines for the Management of Severe Traumatic Brain Injury.

Definition:

Criteria	Mild TBI	Moderate TBI	Severe TBI
GCS post	13 or greater	9 to 12	8 or less
resuscitation			
Duration of	Less than 30 min	Greater than 30 min	Greater than 30 min
unconsciousness			
Non-Contrast Brain	Normal	Normal or	Normal or
CT findings		Abnormal	Abnormal

Procedure:

A. Emergency room evaluation and management:

Patients with traumatic brain injury sustain primary insult at the time of the accident. Secondary brain injury may occur during pre-hospital and in-hospital assessment and management due mostly to systemic hypotension and hypoxemia.

- a. Initial Emergency Room assessment:
 - i. GCS: All patients will be assessed rapidly as soon as possible and GCS documented using the trauma resuscitation narrator prior to intubation and sedation when possible, at post-resuscitation and as needed in between.
 - ii. Trauma Code activation: Trauma team activation policy will be initiated using the trauma code criteria (CM T-28)
 - iii. Airway: All patients with severe TBI will have a secure and patent airway. If intubation was done in the field, airway patency and tube position/location will be verified as soon as possible.
 - iv. Breathing: Prophylactic or induced hyperventilation to greater than 20 breaths per minute will be avoided in all patients unless with clinical signs of cerebral herniation. If hyperventilation is initiated for treatment of cerebral herniation, the intensity and duration will be guided by serial blood gases, with a target PCO2 between 30-35 mmHg. All patients will have oxygen supplementation. All intubated patients will have an end tidal CO2 monitor.

Respiratory goal: O2 Sat between 96-100%, arterial $PaO2 \ge 100 \text{ mmHg}$, arterial PaCO2 35-40 mmHg (30-35mmHg if with clinical signs of herniation or increased ICP)

- v. Circulation: Hypotension (MAP <70mmHg) will be avoided in all patients and prevented with liberal use of crystalloids with the goal of intravascular euvolemia. (Option: Rapid infusion of 1-2 Liters of normal saline or Ringer's lactate on admission or equivalent to 20 ml/kg fluid). Isotonic saline maintenance fluid may be started at a rate of 1-2 ml/kg/hour, with frequent boluses to avoid and/or correct hypovolemia.Arterial line and central venous pressure monitoring will be instituted as soon as possible. Persistent hypotension refractory to volume resuscitation and/or blood products will be treated with vasopressors. Avoid dextrose-containing solutions. Drug choices include:
 - Norepinephrine 0.01-1 mcg/kg/min if negative for tachycardia
 - Phenylephrine 0.1 10 mcg/kg/min if positive for tachycardia **Hemodynamic goal**: MAP $\ge 70 \text{ mmHg}$ and CVP > 5 mmHg
- vi. Cerebral herniation: Patients with clinical signs of cerebral herniation may be treated briefly by controlled hyperventilation with a target PCO2 of 30-35mmHg. Brain imaging will be facilitated to identify patients who may benefit from emergent neurosurgical intervention. Osmotic therapy will be instituted as well using the following options:
 - Hypertonic saline 23.4% at a dose of 1 ml/kg as IV infusion over 15-20 minutes in patients at risk for **hypervolemia** or with known renal impairment (**central line administration advised**)
 - Hypertonic saline 3% at a dose of 3-4 ml/kg as IV bolus over 10-15 minutes in patients at risk for **hypovolemia** or with known renal impairment, followed by continuous infusion at 0.5-1 ml/kg/hour to keep serum Sodium 145-155.
 - For patients with no or partial response to hypertonic saline within 60 minutes, Mannitol 25% at a dose of 0.25 to 1.0 gram/kg as IV bolus over 10 minutes (caution if hypovolemic or with known renal impairment)
- vii. Spine precaution: After hemodynamic and pulmonary stabilization and treatment of all immediately life-threatening injuries, spine clearance in the trauma patient will be followed .
- viii. Imaging: All patients with TBI and GCS less than 15 will have emergency CT brain without contrast to detect intracranial abnormalities. In patients with mild TBI and GCS = 15 with loss of consciousness and post-traumatic amnesia, CT brain may be done if any one of the following is present:
 - Age > 60 years
 - Drug or alcohol intoxication
 - Headache
 - Vomiting
 - Post-traumatic Seizure
 - Deficit in short term memory

- Physical evidence of trauma above the clavicle
- Focal neurologic deficit
- Known coagulopathy

Patients with moderate to severe TBI with an initial CT Brain within 3 hours of injury will have a repeat CT scan within 12 hours. Subsequent need for follow-up CT brain and other radiologic imaging may be determined by the primary service managing the patient based on patient characteristics.

- ix. Sedation: Initiate sedation after securing airway to achieve RASS 0 to -1 after the initial neurologic exam (deeper sedation may be warranted in patients with intracranial hypertension) Drug choices include:
 - Fentanyl 25-50 mcg/hr
 - Propofol 10-100 mcg/kg/min
 - Dexmedetomidine 0.2-1.5 mcg/kg/hr
 - Midazolam 0.05-0.2 mg/kg/hr
- x. Analgesia: Initiate analgesia after the initial neurologic exam titrated for comfort.

Drug choices include:

- Fentanyl 0.6 3 mcg/kg/hr or prn intermittent boluses
- Morphine 0.05-0.1 mg/kg/hr or prn intermittent boluses
- **B.** Fast Track ICU Admission from ED: Facilitation of patient admission will follow the fast track protocol for trauma patients (CM T-37)
- **C.** Primary Service Identification: Early service identification for each patient is crucial for coordinated and optimal patient management. Every effort will be done to admit the patient to the appropriate service. The emergency physician Attending together with the Trauma service, in consultation with the Neurosurgery and Neurocritical Care service will identify the primary service for each patient admitted with traumatic brain injury.
 - **a.** Trauma service: All patients with MULTISYSTEM TRAUMA requiring intensive care will be admitted to the Trauma/SICU service." After all extracranial life-threatening traumatic injuries have been stabilized, patients with moderate to severe traumatic brain injuries may be transferred to the Neurocritical care service unless Neurosurgery agrees to be the primary service for the patient (such as in patients requiring active neurosurgical intervention). All effort will be done to transfer the patient to the Neuroscience Critical Care Unit (9F). Trauma will keep all multisystem traumas. Trauma will round on the patient daily until there is no need for the trauma surgeon's care.
 - **b.** Neurosurgery service: All patients with isolated traumatic brain injuries (or nonlifethreatening, stable extracranial injuries) will be admitted to the Neurosurgery service once cleared by Trauma Service.
 - **c.** Neurocritical Care Service: All patients with isolated traumatic brain injuries (or non-lifethreatening, stable extracranial injuries) will be admitted to the Neurocritical Care service once cleared by Trauma Service.
- **D.** Intensive Care Management
 - a. Monitoring

- i. Blood Pressure: Continuous arterial blood pressure monitoring will be done for all severe TBI and selected moderate TBI patients as soon as possible. Care of patients with arterial line will follow the hospital protocol (CM A-03).
- ii. Oxygenation: Pulse oximetry monitoring will be done for all patients continuously.
- iii. Cardiac: Telemetry monitoring will be done for all patients admitted to the intensive care unit.
- iv. Ventilation: End Tidal Carbon Dioxide (ET CO2) monitoring will be done for intubated patients continuously. Mechanical ventilation setting will be adjusted to achieve the following goals:
 - Tidal volume 6-8 ml/kg
 - PEEP 5-12 mmHg
 - FiO2 30-50%
 - Plateau pressure < 30 mmHg
- v. Intracranial pressure: Intracranial pressure (ICP) monitoring, Licox or Jugular venous brain oxygen monitoring will be done in all salvageable patients with the following conditions:
 - Severe TBI (GCS ≤ 8 after resuscitation) with abnormal CT brain (hematomas, contusions, swelling, herniation, or compressed basal cisterns)
 - Severe TBI (GCS ≤ 8 after resuscitation) with normal initial CT brain and two of the following features on admission:
 - a. Age over 40 years
 - b. Unilateral or bilateral motor posturing
 - c. Systolic Blood Pressure < 90 mmHg
 - Moderate to severe TBI (GCS 9-12) with abnormal CT brain and evidence of beginning/impending hydrocephalus, large intraventricular hemorrhage with occlusion of third and fourth ventricle, or significant mass effect, at the discretion of the Trauma/Neurosurgery/Neurocritical Care Attending.
 - All other TBI patients at high risk for developing intracranial hypertension at the discretion of the Neurosurgery and/or Neurocritical Care Attending.

Ventricular pressure measurement using external ventricular drain (EVD) is the preferred method for ICP monitoring. Maintenance and care of EVD will follow the hospital policy (<u>CM I-01</u>). Intraparenchymal ICP monitors may be used instead of EVD at the discretion of the Attending Neurosurgeon or Neurointensivist. Subarachnoid, subdural or epidural ICP monitors are less accurate and not recommended.

vi. Brain oxygen monitoring: Selected patients at high risk for secondary cerebral ischemia or who have evidence of ongoing cerebral insult may benefit from global (Jugular Venous Saturation) or regional (Brain Tissue Oxygen) brain oxygen monitoring to complement the standard ICP monitoring. Insertion of these monitors will be done at the discretion of the Trauma/Neurosurgery/Neurocritical Care Attending. Other noninvasive devices using the near-infrared spectroscopy (NIRS) technology may be employed if available.

- vii. Other monitoring devices:
 - EEG monitoring: Patients with clinical seizure or who are suspected to have subclinical status epilepticus will be monitored continuously with EEG. Patients receiving metabolic suppression therapy for management of refractory intracranial hypertension will also have continuous EEG monitoring to guide therapy.
 - Microdialysis: Selected patients at high risk for secondary cerebral ischemia or who have evidence of ongoing cerebral insult may benefit from neurochemical monitoring of the injured brain to detect early markers of tissue ischemia and metabolic crises using cerebral microdialysis. Insertion of these monitors will be done at the discretion of the Trauma/Neurosurgery/Neurocritical Care Attending.

E. Imaging: Serial imaging using CT or MRI will be done as clinically indicated. Patient transport will be minimized unless the anticipated result of imaging has a high probability of potentially and significantly altering the current direction of care. **F.** ICP management and goal: The goal of ICP management is to prevent development of intractable intracranial hypertension. In patients with ICP monitoring, the goal is ICP < 20mmHg. Lower goal may be necessary in patients with clinical symptoms not attributable to systemic/metabolic abnormalities and ICP between 14 to 20mmHg. A stepwise approach for management of intracranial hypertension (ICP > 20mmHg for > 5 minutes) is outlined below:

viii. General management

- Ensure adequate systemic oxygenation (PaO2>100, O2Sat 96-100%). If hypoxic, adjust ventilator to achieve optimal oxygenation goals.
- Ensure adequate pulmonary ventilation (PCO2 35-40mmHg). If hypercapneic, adjust ventilator to achieve optimal PCO2 range.
- Ensure euvolemia with liberal crystalloid resuscitation (Urine output 0.5-1 cc/kg/hour, CVP > 5 mmHg). Increase maintenance fluid to achieve goal
- Ensure adequate circulation with MAP goal > 70 mmHg. In patients with ICP monitor, use CPP goal of 60-75 mmHg instead of the MAP goal. Start vasopressor to achieve goal, once euvolemic status has been achieved
- Ensure optimal patient position: Maintain head of bed (HOB) elevation to at least 30 degrees from horizontal plane.
- Ensure normothermia (Temperature goal < 38 degrees C). Use cooling blanket, acetaminophen PO/PR 650 mg Q4-6 hours. Consider using temperature modulation with surface cooling to maintain temperature between 36.0 37.0 degrees C (CM H-11) in patients with refractory hyperthermia (defined as persistent temperature elevation to >38 degrees C for longer than 24 hours despite aggressive management). Shivering during temperature

modulation may be managed using the hospital protocol (PROC CM H-11C)

- Ensure adequate sedation and analgesia using the hospital Sedation protocol
- ix. Specific management
 - CSF drainage: Drain 2-5 ml CSF and lower EVD setting (e.g. from 10 cm to 5 cm above external auditory meatus). Repeat draining 2-5 ml CSF every 10-15 minutes until ICP < 20 mmHg.
 - Hyperosmolar therapy: If no EVD or if no response to CSF drainage, institute hyperosmolar therapy using the following every 4 to 6 hours PRN for ICP > 20 over 5 minutes:
 - a. Hypertonic saline 23.4% at a dose of 0.5-1 ml/kg as IV infusion over 15-20 minutes in patients at risk for hypervolemia or with known renal impairment or high osmolar gap.
 - b. Hypertonic saline 3% at a dose of 3ml/kg as IV bolus over 10-15 minutes in patients at risk for hypovolemia or with known renal impairment or high osmolar gap, followed by infusion of 0.5 to 2 cc/kg/hr adjusted to keep serum Na between 145-155 (Duration of infusion longer than 48 hours not advised. Taper slowly to avoid rebound cerebral edema.) BMP will be ordered every 4-6 hours to follow serum sodium level
 - c. If no response within 60 minutes to either a or b, Mannitol 25% at a dose of 0.25-1.0 gram/kg as IV infusion over 10 minutes in euvolemic patients without known renal impairment. Basic Metabolic Panel and Serum Osmolality will be ordered every 4-6 hours to determine the osmolar gap (Actual Serum Osmolality Calculated Osmolarity). Discontinue mannitol if osmolar gap > 10 (Option: Change in osmolar gap > 10 from baseline)

The use of hyperosmolar therapy in patients without ICP monitoring should be limited to those with clinical signs of transtentorial herniation or progressive neurologic deterioration not attributable to exracranial causes.

- Metabolic suppression: If no/poor response to CSF drainage or hyperosmolar therapy, initiate metabolic suppression therapy using any of the following:
 - a. Pharmacologic metabolic suppression with Pentobarbital 10 mg/kg given as 100-250 mg IV bolus every 10-15 minutes until EEG burst suppression and 50-400 mg/hr to maintain EEG burst suppression of about 1-4 burst per minute. Repeat bolus may be necessary in patients not achieving burst suppression especially if the ICP elevation persist. If burst suppression after loading has been achieved and ICP still elevated, discontinue infusion and consider

other treatment. If ICP is controlled with bolus administration, maintain burst suppression for at least 24 hours followed by weaning (reduction of infusion by 0.5 mg/kg/hr every 6 hours until off). If ICP rises during weaning, resume infusion at prior dose and retry weaning after 24-48 hours of no ICP elevation.

- b. Induced hypothermia using Arctic Sun with target temperature of 33 degrees C using the hypothermia protocol (CM H-11) Shivering during temperature modulation may be managed using the hospital protocol (PROC CM H-11C). Induced hypothermia is typically maintained for 24 hours or longer until ICP is controlled and risk of further cerebral edema and mass effect has abated before rewarming over 24-36 hours. Rewarming should be stopped and/or patient placed back on induced hypothermia if during rewarming, the patient develops rebound intracranial hypertension.
- Controlled hyperventilation may be done in refractory cases by increasing the minute ventilation to achieve arterial PaCO2 of 30-35 mmHg. End Tidal CO2 poorly correlates with actual arterial PaCO2 and may not be used as the only monitoring parameter during controlled hyperventilation. Brain tissue oxygenation monitoring keeping the brain tissue oxygen > 20 mmHg, or jugular venous oxygen saturation >50% is recommended for prolonged controlled hyperventilation. Hyperventilation duration longer than 24 hours is not recommended.
- x. Repeat CT brain should be done to determine the presence of a surgical brain lesion. Prompt neurosurgical referral/consultation will be initiated in salvageable patients for definitive management of surgical intracranial pathology.
- G. Hemodynamic management and goal
 - xi. CVP: All patients should be assessed frequently for volume status. In patients with central venous access (Central Line or PICC Line), central venous pressure (CVP) monitoring may be done with goal of CVP > 5 mmHg (Option: Urine output > 0.5 ml/kg/hour over 4-6 hours in absence of cerebral salt wasting or diabetes insipidus). Aggressive fluid resuscitation may be done using isotonic or hypertonic non-glucose containing crystalloids to achieve goal.
 - xii. MAP: In patients without ICP monitoring, MAP should be maintained between 70-90 mmHg. After adequate fluid resuscitation, vasopressor therapy may be initiated to achieve MAP goal.
 - xiii. CPP: In patients with ICP monitoring, instead of MAP, use cerebral perfusion pressure (CPP) with goal of 60-75mmHg. After adequate fluid resuscitation, vasopressor therapy may be initiated to achieve CPP goal.
 - xiv. Cerebral Oxygenation: In patients with brain tissue oxygen monitor in the peri-injury region, brain tissue oxygen saturation (PBtO2) should be

maintained with goal PBtO2 > 20-25 mmHg. Patients with jugular venous oxygen saturation (SjvO2) monitoring should be maintained between 55-75%, Strategies to improve PBtO2 and/or SjvO2 include:

- Increasing systemic oxygen by increasing FiO2
- Optimizing systemic oxygen delivery by keeping Hemoglobin > 10 g/dL
- Optimizing CPP by
 - a. Increasing MAP
 - b. Decreasing ICP

• Reducing metabolic demand using metabolic suppression therapy **H.** Sedation: Patients with TBI need to be examined for clinical deterioration. However, agitation and pain can exacerbate secondary brain injury and may limit efficacy of overall treatment strategy if not adequately controlled. As such, short-acting sedatives and analgesics are the preferred agents. The following drugs may be used:

> Dexmedetomidine 0.2- 1.5 mcg/kg/hr IV infusion Propofol 10-100 mcg/kg/min IV infusion Midazolam 0.05-0.2 mg/kg/hr IV infusion

I. Agitation/delerium/dysautonomia and motor restlessness are common complications post traumatic brain injury: The period of post traumatic amnesia is a subtype of delirium characterized by aggression, disinhibition, akathisia, disinhibition, and emotional liability that can be adversely affected and exacerbated by narcotics and benzodiazepine medications. Enviornmental factors adding to agitation and affecting sleep wake cycle should be minimized, and modifying these should be the first line of treatment. Turn off unnecessary alarms and maintain a low stimulation room. Minimize physical restraints. The following drugs may be used:

i. Propranolol start 40-60mg per day in divided doses BID-QID(preferred to metoprolol for agitation and dysautonomia in TBI as has better CNS penetration. Also prophylactic for headaches, decreases restlessness and disinhibition)
ii. Seroquel start 12.5mg BID to 25mg BID depending on age and comorbidities
iii. Valproic Acid start 250mg BID and titrate up (longer onset of action but mood stabilizing and decreases motor restlessness. Also prophylactic for headaches)

J. Analgesia: Patients with severe TBI experience significant pain and discomfort from initial injury and subsequent interventions. Uncontrolled pain can contributes significantly to agitation and increased requirement for sedation complicating the overall care. If a patient is not verbal, be aware that pain is one of many reasons for agitation, and there should be assessment for other causes. Although pain medication may make the clinical evaluation for deterioration difficult, this may be mitigated by using ultra-short acting analgesics. The following drugs may be used:

i. Fentanyl 0.6-3 mcg/kg/hr IV infusion (2 mcg/kg Loading Dose)

ii. Morphine 0.05-0.5 mg/kg/hr IV infusion (0.1 mg/kg Loading Dose)

iii Assure specific type of pain being treated is adequately addressed and documented i.e. Headaches should be treated with agents that do not significantly affect cognition such as atypical antidepressants or Propranolol or Depakote.

K. Nutrition: Patients with moderate to severe TBI have increased metabolic demand and needs to have adequate nutritional intake as soon as possible. Enteral feeding will be initiated within 24 hours or until no further anticipated surgical intervention is planned. Small bowel tube feeding is preferred than gastric tube feeding. If enteral feeding cannot be started within 72 hours, parenteral nutrition will be started. The goal is to achieve 100% of full caloric replacement by day 3 post-injury. Feeding formulation should contain at least 15% of total calories from protein and should provide 120-150% replacement of resting metabolism expenditure (reduce by 25% if on metabolic suppression therapy).

L. Prophylaxis

i. Seizure prophylaxis: Patients with abnormal CT have increased risk of seizure and may be given antiepileptic drugs (AED) for seizure prophylaxis for up to 7 days. Patients with GCS > 12 or normal CT brain, or patients with no epileptiform discharge on EEG may not need AED. Levetiracetam 750 mg BID PO or IV will be started for up to 7 days as indicated. Patients developing adverse effect (profound lethargy or delirium) to levetiracetam may be switched to lacosamide 100 mg BID PO/IV or phenytoin 100 mg TID PO/IV (if no fever or thrombocytopenia). Anti seizure prophylaxis will be discontinued after 7 days from trauma unless the patient develops post-traumatic seizure, or at the discretion of the Attending Physician.

ii. DVT prophylaxis: All patients will be screened using the hospital protocol (CM D-07) to include a caprini score and recommended therapy. Holding prophylaxis should be a decision that the attending neurosurgeon or trauma surgeon makes.

iii. GI prophylaxis: All patients will receive GI prophylaxis to reduce stress ulceration per hospital protocol (CM S-23)

iv. Infection prophylaxis: Periprocedural antibiotics for intubation will be administered to reduce the incidence of pneumonia. Early tracheostomy (within 7 days from injury) in patients with low likelihood of being extubated by day 7 may be performed to reduce mechanical ventilation days and pneumonia. All patients will be evaluated daily for early extubation in qualified patients. Routine ventricular catheter exchange is not recommended to reduce infection.

M. Potentially harmful intervention

i.Steroids: Not recommended for improving outcome and reducing intracranial pressure. High dose steroid is associated with increased mortality in moderate to severe TBI and is contraindicated.

ii. Prophylactic hyperventilation: Prophylactic hyperventilation is not recommended and should be avoided in first 24 hours after injury. If used for control of ICP, SjvO2 or PBtO2 measurements are recommended to monitor oxygen delivery.

iii. Prophylactic barbiturate coma: Prophylactic use of high dose barbiturates to induce coma lowers blood pressure without improving outcome. However, it may be used in refractory intracranial hypertension.

iv. Prophylactic induced hypothermia: Not associated with reduction in mortality in unselected patients. Subgroup analysis suggest benefit if maintained for more than 48 hours.

v. Antiseizure prophylaxis beyond 7 days is not recommended N. ICU care: Rehabilitation Physician consultation and facilitation of transfer to a rehabilitation facility with expertise to manage patients with brain injury will be done in coordination with social work department and case management. Rehabilitation Physician consultation can assist with patient, family, and staff brain injury education, agitation control, delirium control, dysautonomia control, headache control, post concussion symptom management and early coma or brain injury assessment and recovery planning.

O. All patients will have physical and occupational therapy evaluation as soon as lifethreatening injuries have been stabilized. Speech therapy evaluations should be considered in any patients with moderate to severe brain injuries for cognitive and/or swallow evaluations; any patients with trachs will also benefit from assistance for communication or evaluation of swallow function. Even vented patients can engage in bedside therapies, should have regular range of motion on all stable limbs, be challenged with focused stimulation programs, and sit up as able. Regular skin checks and repositioning should be done by nursing.

P.Quality Process Development and Review: The brain trauma committee will participate in and report quality improvement initiatives. Outcomes that will be measured include Length of Stay, Variances, Cost per case, Patient Satisfaction, Patient Outcomes (Severity of Illness, Morbidity, Mortality, Incidents, Readmissions, infection rates), and Staff Satisfaction

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