Guidelines for the Critical Care Management of Severe Head Injury

2008

University Mississippi Medical Center
Section of Neurotrauma
Department of Neurosurgery
Introduction

A. Background

1. Trauma is the leading cause of death and disability among Americans less than 45 years of age, and is the fourth leading cause of death overall. The most common cause of death within this group is brain injury.

2. Severe brain injury is defined as head trauma resulting in an admission Glasgow Coma Scale score of 3-8.

3. Traumatic brain injury (TBI) results in an alteration of cerebral physiology which may be amenable to interventions directed at limiting the injury cascade and, therefore, secondary injury.

4. Neurotrauma care will be based on evidence based medicine guidelines and established institutional protocols. These guidelines include:
   1) Guidelines for Pre-hospital Management of TBI – Brain Trauma Foundation (2007)
   2) Management and Prognosis of Severe Brain Injury – Brain Trauma Foundation (2007)
   4) Surgical Management of TBI – Brain Trauma Foundation (2006)

(All above guidelines have been reviewed and approved by the Section of Neurotrauma and Critical Care and the Executive Committee and Board of Directors of the American Association Neurological Surgeons and Congress Neurological Surgeons)

B. Purpose

1. The purpose of this document is to provide standardized guidelines for the critical care management of the head injured patient at University Mississippi Medical Center.

2. These guidelines are based on all guidelines listed above but most particularly on the Management and Prognosis of Severe Traumatic Brain Injury. They have been modified and expanded to address issues specific to University Mississippi Medical Center and also reflect approaches to certain issues not addressed in the guidelines.
3. These guidelines do not replace the physician’s judgment in individual cases, but may be considered reasonable and current approaches to the management of the critically ill adult head injury patient.

4. While this document does not address specific guidelines for the management of pediatric head injury patients, many of the same principles are applicable.

5. These guidelines are intended to foster a coordinated, cooperative environment among the multidisciplinary team caring for head injured patients, which includes, but is not limited to, Emergency Medicine, Neurosurgery, Trauma Surgery, and Critical Care Medicine and Nursing.
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I. Initial Resuscitation of Blood Pressure and Oxygen

A. Background
In head injured patients, both hypotension (defined as a systolic BP < 90 mmHg) and hypoxia (defined as apnea, cyanosis, or a PaO₂ < 60 mmHg) are associated with worsened clinical outcome. This occurs presumably because hypotension and hypoxia cause secondary injury in vulnerable brain tissue. While these insults may occur at any point in the clinical course of a patient with head injury, they often occur in the pre-hospital setting or during Emergency Department (ED) resuscitation.

Patients with severe TBI may mask hypovolemic hypotension because of the Cushing's response to intracranial hypertension. As such, patients may benefit from central venous monitoring during the period of acute fluid resuscitation to adequately assess their intravascular volume; a PA catheter may be inserted in select cases. A gradual increase in blood pressure associated with a gradual decrease in pulse (even if both are within normal limits) should suggest the development or progression in intracranial hypertension.

B. Goals of Therapy
1. To avoid hypotension and hypoxia in patients with severe head injury.
2. To urgently treat hypotension and hypoxia, thus minimizing exposure of vulnerable brain tissue to these secondary insults.

C. Guidelines
1. Hemodynamic resuscitation should begin in the ED with the placement of two large-bore (14 or 16 G) IV’s when possible. A groin line may be placed in lieu of a peripheral IV.
2. A subclavian central line should be placed for volume-status assessment. The groin line may be used to place a long central venous pressure line as long as two large-bore IV’s are available for fluid resuscitation. Internal jugular lines are not practical in the ED setting since most patients will be wearing cervical collars.
3. Foley catheter should be inserted during the initial resuscitation.
4. Systemic blood pressure should be recorded every five minutes during the initial resuscitation (ED) via automated sphygmomanometer. An arterial blood-pressure catheter (radial) should be placed as soon as possible to allow for continuous blood pressure readings.
5. Volume resuscitation with 0.9%NS or blood (when appropriate) is the first intervention.
6. Strict avoidance of hypotonic (0.45% and 0.225% NaCl) and D5-containing solutions should be observed during acute resuscitation and as routine maintenance fluids in the intensive care unit (ICU).
7. At a minimum, systolic BP should be maintained above 90 mmHg. Ideally, mean arterial pressure (MAP) will be maintained above 80 mmHg, since a systolic blood pressure of 90 mmHg may be inadequate in the setting of elevated ICP.

8. Systemic hypertension generally should not be treated in the acute setting of TBI, since this may reflect the body’s natural response to intracranial hypertension.

9. Antihypertensive medications may be administered if the systolic blood pressure is greater than 220 mm Hg. Beta-blockers are the drug of choice in the absence of bradycardia. Calcium channel blockers or nitrates may be used as well, though the latter may theoretically increase ICP by causing cerebral vasodilation. Hydralazine should be avoided since it is thought to uncouple cerebral blood flow from metabolism.

10. Pressors may be necessary in addition to volume resuscitation, especially in the setting of acute spinal cord injury (SCI). Neo-Synephrine, Dopamine or Epinephrine are the pressors of choice. Neo-Synephrine may induce bradycardia in SCI and is generally reserved for use in TBI. The use of pressors in the acute setting should be agreed upon by ED, Trauma, Anesthesia and Neurosurgery attendings as applicable.

11. Early intubation may be necessary to avoid hypoxemia in patients with severe head injury. While there is theoretical concern about pulmonary oxygen toxicity in patients receiving an FiO2 > 0.6, concerns of systemic and cerebral hypoxia take precedence.

12. Treatment with supplemental oxygen will be initiated in the Field and continued after arrival to ED. 100% O2 will be given prior to intubation and continued during the initial post-intubation period. FiO2 will be adjusted according to the post-intubation arterial blood gas (ABG).

13. Rapid-sequence induction (RSI) should be carried out using Etomidate (0.1-0.3 mg/kg) and succinyl choline or Rocuronium as the agents of choice. Succinylcholine may theoretically increase ICP by depolarizing skeletal muscle, though patient outcome is unlikely affected by its use. Rocuronium, on the other hand is longer acting than succinylcholine – a concern if intubation is not readily accomplished. Furthermore, it prevents the neurological examination of patients for a longer time than succinylcholine. Rocuronium may be reversed after 20-30 minutes with Neostigmine (20-70 mcg/kg) and glycopyrrolate (0.6 mcg/kg).

14. Ventilation rate should be controlled to maintain adequate oxygenation. Because of the significant effects of ventilation on cerebral blood flow, hyperventilation during the acute resuscitation should be reserved only for patients with evidence of acute brain herniation. Hyperventilation can also decrease venous return, cardiac output, and blood pressure, thereby increasing the incidence of secondary brain injury.
15. Initial serological studies must include the following
   a. Post-resuscitation Arterial Blood Gas
   b. Chem-7 (Lytes, BUN, Creatinine and Glucose)
   c. CBC with platelets
   d. Coagulation panel (PT/PTT)
   e. Serum Osmolarity
   f. Type and Screen (cross # of units as necessary)
   g. Urine toxicology
II. Intracranial Pressure (ICP) Monitoring

A. Background
ICP is frequently elevated in patients with severe head injury. Mechanisms causing elevated ICP include cerebral edema, intracranial hematoma formation, and hydrocephalus. While there is no prospective randomized controlled trial showing that treatment of elevated ICP improves outcome from severe head injury, there is a large body of evidence suggesting that monitoring of ICP and its subsequent treatment do, in fact, impact head injury outcome in important ways. Normal ICP is from 10-15 mmHg; elevated ICP begins at 20-25 mmHg.

B. Goals of Therapy
1. To detect elevated ICP in order to allow surgical and medical management to lower ICP and maintain cerebral perfusion pressure (CPP).
2. To allow for drainage of CSF (when available) as a means for treating elevated ICP.

C. Guidelines
1. Indications for ICP Monitoring
   a. ICP monitoring is appropriate for all patients with severe head injury (GCS 3-8) with an abnormal admission head CT scan following hemodynamic resuscitation.
   b. ICP monitoring is appropriate for all patients with severe head injury (GCS 3-8) with a normal head CT scan if two or more of the following are present:
      i. age > 40
      ii. unilateral or bilateral motor posturing
      iii. one or more episodes of hypotension (i.e. systolic blood pressure < 90 mmHg)
   Note: Placement of an ICP monitor should not obscure efforts of cardiopulmonary resuscitation in the setting of hemodynamic instability.
   c. ICP monitoring may also be considered in patients with head injury who are undergoing non-neurosurgical operative procedures early in their hospital course, during which time neurologic examination will be unavailable.
2. Technology for ICP Monitoring
   a. Placement of a ventricular catheter is the preferred method for ICP monitoring as it allows CSF drainage for the treatment of elevated ICP.
   b. When placement of a ventricular catheter with microsensor is not deemed appropriate (i.e. slit ventricles, concerns of coagulopathy or platelet dysfunction, minimal findings on CT Scan), then use of a parenchymal monitor is preferred over other methods.
c. All efforts should be made to normalize PT/PTT and platelets (>100k) before insertion. Activated factor VII is appropriate for emergent situation in the setting of coagulopathy.

3. Threshold for Treatment of ICP
   a. ICP treatment should be initiated at an upper threshold of 20 mmHg. Limits should be coordinated with the CPP.
   b. ICP treatment should be taken in context of clinical examination and CPP data.

4. Methods of CSF Drainage
   a. Two basic techniques of CSF drainage exist: intermittent and continuous.
   b. Intermittent drainage for elevated ICP is the preferred method. When intermittent drainage is used, the opening and closing pressures and volume of CSF drained should be recorded, as this may give an indication of intracranial compliance.
   c. Continuous CSF drainage at a specified pressure-height is an alternative, with the recognition that this method may interfere with continuous monitoring of ICP and may increase the incidence of post-traumatic hydrocephalus.

5. Infection Control
   a. Although there is some evidence to suggest that they are not necessary, empiric antibiotics will be used for prophylaxis against infection here at UMC during ICP monitoring due to our historical low rate of infection with them. The antibiotic chosen should include gram positive coverage and should have good CNS penetrance. The current protocol will consist of one dose of antibiotic (Ancef if no contraindication) at the time of insertion and one dose within 4 hours of insertion.
   b. Great attention will be paid to sterile placement and maintenance of ICP monitors as conditions during placement and instrumentation are the greatest risks for infection. Sterile gloves, gown, and mask should be worn during insertion along with preprocedureal hand-washing.
   c. CSF will be sent for analysis from ventricular catheters as needed for infection surveillance and diagnosis.
      i. CSF will be sent as needed to rule out infection (e.g. after fever spikes, etc.).
      ii. After initial placement, CSF catheters and ports will be sterilized with betadine and/or alcohol prior to obtaining CSF.
   d. Antibiotic coverage will be initiated for treatment of suspected or confirmed ICP monitor infection. Gram positive organisms are most likely, but specific antibiotic coverage will be tailored to bacteriologic isolates and their sensitivities.
III. Metabolic and Electrophysiological Monitoring

A. Background

Despite advances in understanding and treatment of severe head injury, significant morbidity and mortality remain. Monitors aimed at determining the metabolic and functional status of the brain are indispensable for state-of-the-art evaluation of post-traumatic cerebral pathophysiology.

Current tools for the evaluation of cerebral metabolic status include jugular venous saturation monitors, cerebral blood flow monitors, brain tissue oxygen monitors, and microdialysis catheters. Current electrophysiological monitors include electroencephalography (EEG), motor evoked potentials (MEP), somatosensory evoked potentials (SSEP), and brainstem auditory evoked response (BAER). Current imaging technologies also include CT perfusion scans (CTP), diffusion weighted imaging (DWI), diffusion-perfusion MRI, and magnetic resonance spectroscopy (MRS).

B. Goals of Therapy

To comprehensively monitor cerebral metabolic and functional endpoints to allow the physician to determine the physiological, functional, and metabolic status of the brain to allow for targeted therapy for each patient.

C. Guidelines

a. Based on physician discretion, patients with severe TBI requiring an ICP monitor may have one, or more, brain tissue monitors and microdialysis catheters to assess the brain tissue oxygen levels and cerebral metabolism. (See also Chapter on Brain Oxygen Monitoring)

b. Based on physician discretion, patients with ICP monitors may have jugular bulb catheters placed to help determine mixed venous cerebral oxygen extraction, and metabolic state.

c. Based on physician discretion, some patients may also have parenchymal CBF monitors placed as part of the metabolic monitoring array.

d. Based on physician discretion, patients with severe TBI may have 24-hour electrophysiological (EEG) monitoring started as close to admission to the ICU as possible. MEP, SSEP, and BAER may also be evaluated at regular intervals. Electrophysiological monitoring and assessment will continue until it is deemed no longer necessary by the Neurosurgery attending in consultation with the neurocritical care team.

e. Based on physician discretion, patients will have CT perfusion scans and MR perfusion/diffusion scans as needed for quantitative and qualitative determination of cerebral blood flow and perfusion.

f. Based on physician discretion, MR spectroscopy may be obtained as needed to determine the patient’s cerebral metabolic picture as a function of neuroanatomical geography.
g. There is good documentation both in the literature and in our institution of significant risks associated with transport of patients out of the ICU particularly for long diagnostic procedures. Therefore MRI scanning particularly with long multi-sequence studies should be reserved for patients who are stable from the standpoint of intracranial hypertension, hemodynamic stability and the ability to be on portable ventilation support. In the early stages of TBI the amount of information gained by MRI scanning has little effect on treatment protocols. The performance of early MRI scanning simply to “see what things look like” should be discouraged.
IV. Cerebral Perfusion Pressure (CPP)

A. Background
Cerebral perfusion pressure (CPP) = MAP – ICP. Cerebral ischemia may be the most important secondary event affecting outcome after severe traumatic brain injury. Cerebral perfusion pressure therapy is designed to prevent secondary ischemic insults to vulnerable traumatized brain tissue. While the optimal CPP may vary from individual to individual, evidence from studies using transcranial Doppler ultrasound (TCD) and from the Traumatic Coma Data Bank suggest that a threshold CPP of at least 70 mmHg is an appropriate goal. More recent data suggests that a CPP of > 55-70 mmHg is preferred, though this target may vary according to the patient’s individual autoregulatory set point.

B. Goals of Therapy
1. To avoid secondary cerebral ischemia of traumatized brain by ensuring adequate cerebral perfusion.
2. To maintain euvolemia or slight hypervolemia, in order to ensure adequate cerebral and systemic organ perfusion.

C. Guidelines
1. CPP Management
   a. A CPP of > 55-70 mmHg will be maintained using volume and pressors as necessary.
   b. Threshold CPP will be tailored to individual patients using cerebral monitoring tools such as ICP, TCD, jugular venous oxygen saturation (SJVO₂), and PbRO₂.
2. Hemodynamic Monitoring
   a. Insufficient evidence exists currently to recommend one method of hemodynamic monitoring in the head injured patient over another. Therefore, we will recommend that all patients undergoing ICP monitoring receive a central venous catheter (CVP) preferably via a subclavian route.
   b. Patients will also receive an arterial catheter, preferably at the radial site. By convention, the arterial pressure transducer will be placed at the level of the right atrium.
   c. In order to minimize the effect of head-of-bed (HOB) elevation on CPP, HOB should be raised no more than 20°. This strategy also addresses the observation that cerebral blood flow drops as the HOB elevation exceeds 30° – even in the setting of a constant CPP.
   d. Pulmonary artery catheters will be used at the discretion of the treating physician, particularly in patients who cannot be managed via other methods (CVP line, clinical evaluation, cardiac echo).
3. Pressor and Volume Therapy
   a. CVP will be maintained at 5-10 mmHg, which is slightly hypervolemic. If a PA catheter is used, then PCWP will be maintained at 8-12 mmHg. Because these pressure measurements may be altered by changes in intrathoracic pressure which accompany PEEP, ARDS/lung injury, and increased intraabdominal pressure, attention must be made to correlate intravascular pressures with other measures of volume status such as urine output and heart rate in order to ensure euvolemia or slight hypervolemia.
   b. When pressors are used for CPP management, attention will be made to ensure that the patient is adequately fluid resuscitated before instituting a pressor infusion. Initial pressor of choice is phenylephrine (5-400 mcg/min), with dopamine (5-20 mcg/kg/min) an alternative choice. Except in patients with associated spinal cord injury in whom Dopamine will be the pressor agent of choice. Norepinephrine is considered a second-tier choice and if considered, great attention must be paid to volume status in order to avoid precipitating systemic metabolic acidosis.

4. Anti-hypertensive Medications (AHM)
   a. AHM should be used with caution in patients with severe TBI. In most cases elevated BP is a reflection of the Cushing response and is a sign of existing or impending elevated ICP.
   b. Before initiating Anti-hypertensive (BP > 190 systolic) therapy the patient’s sedation/paralysis status should be accessed. In addition the general systemic issues (i.e. pulmonary status, fever, hemodynamic parameters) should be carefully addressed.
   c. Patients with a history of baseline hypertension should be treated appropriately
   d. If treatment of systemic hypertension is based on suspected disautoregulation this should be proved by SVJO₂ and/or PbO₂ values.
V. Mechanical Ventilation

A. Background
The principle purpose of mechanical ventilation in severe head injury is to ensure adequate systemic and cerebral oxygenation. Airway protection from endotracheal intubation is also important to avoid upper airway obstruction and aspiration. In the past, aggressive hyperventilation has been a cornerstone of head injury management because of the ability to decrease ICP by decreasing cerebral blood volume via hypocapnic cerebral vasoconstriction. Recent evidence has demonstrated, however, that aggressive prophylactic hyperventilation actually worsens outcome after severe head trauma. The presumed mechanism for this is exacerbation of cerebral ischemia from the hypocapnic vasoconstriction. Although some difference of opinion exists, hyperventilation is best suited for a short-term strategy for lowering ICP until other measures can be instituted and should be avoided as a long-term intervention.

B. Goals of Therapy
1. To ensure adequate oxygenation, ventilation, and airway protection.
2. To aid in short-term control of elevated ICP while avoiding secondary brain injury from hypocapnic vasoconstriction-induced cerebral ischemia.

C. Guidelines
1. Modes and Methods of Mechanical Ventilation
   a. The initial goal in all patients will be normoventilation, recognizing that pH defines ventilatory status, as driven by the medulla. In patients with normal lungs, a normal ABG is 7.40/40/100. A CO₂ of 30-35 in these patients represents mild hyperventilation. In patients with metabolic acidosis or alkalosis or baseline respiratory alkalosis, there is insufficient current knowledge in the absence of CBF monitoring to know how CBF is affected. Thus, a normal pH will be the goal of ventilation.
   b. Initial ventilator settings will involve volume-controlled ventilation (AC or SIMV) with PEEP of 5. Patients who self hyperventilate (neurogenic hyperventilation) will be placed on volume control mode with high minute ventilation that matches the patient own minute ventilation then slowly wean minute ventilation slowly over 12-14 hours to acceptable level (10-12 L/min).
   c. A goal P_aO₂ of 100 mmHg will be maintained.
   e. PEEP of ≤ 10 cm H₂O will be tolerated without concern for exacerbation of ICP. On a patient by patient basis, the effect of PEEP on ICP will be considered.
   f. Please see section III on initial resuscitation for reference to Rapid-sequence induction (RSI).
   g. Because ventilation is a primary ICP-management modality, all changes in ventilation parameters in patients with ICP monitors must be cleared by Neurosurgery.
h. Periprocedural antibiotics (2 doses of Ancef) should be considered prior to or as soon thereafter endotracheal intubation.

2. Hyperventilation
   a. Prophylactic hyperventilation will not be instituted
   b. Hyperventilation may be used as an acute strategy to lower ICP in patients with evidence of acute brain herniation while the patient is being transported to CT, OR, or other interventions such as ventriculostomy placement are being instituted.
   c. Hyperventilation using manual bagging may be more immediately effective than just changing ventilator settings.
   d. After hyperventilation has been used, it will be withdrawn slowly in a stepwise fashion over 2-4 hours to avoid rebound in ICP.

3. Tracheostomy
   a. Controversy exists regarding the timing of tracheostomy after severe head injury.
   b. In general, tracheostomy will be considered when a patient has been intubated for 14 days and extubation is not deemed to be imminent.
   c. Early tracheostomy (prior to 14 days) may be considered in patients with the likely need for long-term mechanical ventilation or airway protection provided ICP has been stable for prior 24hrs.
   d. Although early hyperventilation has been shown to be associated with worse outcomes in severe TBI due to decreased cerebral blood flow. When primary tier therapy has failed controlled hyperventilation has been used as secondary tier therapy. If this therapy is used patients should be at least 48hrs post injury and be monitored by SVJO₂ and PbO₂ monitors. Any significant change in oxygen extraction or O₂ delivery is a contradiction to this therapy.
VI. Sedation and Analgesia

A. Background
Sedation and analgesia in patients with severe head injury are important aspects of patient care that influence patient comfort, ability to tolerate mechanical ventilation and critical care procedures, and intracranial pressure (ICP) management. The use of sedatives can improve ICP control by preventing agitation and reducing the cerebral metabolic rate of oxygen consumption (CMRO2), but may obscure the neurologic examination. Protocols for the use of sedatives in head injured patients vary widely, but most published series use significant doses of various agents including opiates, benzodiazepines, and propofol. Many published protocols have also used prophylactic neuromuscular blockade. While the value of detailed serial neurologic examination in the patient with severe head injury (GCS 3-8) is debated, a sedation protocol that maximizes patient comfort, ICP control, and allows accurate neurologic examination is the ideal.

B. Goals of Therapy
1. To ensure patient comfort after severe head injury in the setting of critical care interventions.
2. To serve as an adjunct for ICP management and perhaps to decrease secondary brain injury by decreasing cerebral oxygen utilization.
3. To avoid interfering with clinical neurologic assessment as feasible.

C. Guidelines
1. Analgesia and Sedation are considered separate issues which must be addressed individually in each head injured patient.
2. They should be tailored to patient needs. Both will be kept at full dose in case of continued ICP elevation.
3. Analgesia
   a. Opiates infusion are considered the method of choice
      1) Morphine Sulfate 2-10mg/h
      2) Fentanyl 100-200mcg/h is a second choice
4. Sedation in ICP Control
   a. Ramsey Scale will be used for agitation assessment.
   b. Sedation will be used initially in all severe TBI patients in conjunction with other ICP control measures.
   c. In paralyzed patients, the sedative of choice will be Ativan 2-10mg/h or Midazolam 2-10mg/h (although accumulation is a potential problem in Midazolam).
   d. In non-paralyzed patients, either Ativan 1-2mg/h or alternatively Propofol 20-50mg/kg/min will be used and titrated to effect. Either drug will be stopped in AM at least 30min before morning rounds for neurologic examination
   e. In patients receiving sedation, ongoing need for analgesia will be assessed and managed accordingly.
5. Neuromuscular Blockade
   a. Neuromuscular blockade should be restricted to elevated ICP not controlled with sedation alone, or for severe pulmonary disease such as ARDS.
   b. Paralytics use should be stopped as soon as possible. If the indication persists for more than 3 days, a drug holiday for 14 h or more is advocated to minimize the occurrence of critical care myopathy and prolonged severe muscle weakness.
   c. Cisatracurium is the preferred neuromuscular blocker of choice since it is metabolized by Hoffman’s Hydrolysis and the concern of prolonged paralysis is less than with amino steroid based NMB. Vecuronium is a good second choice.
   e. At least 1-2 twitches will be kept at all times while NMB are in use.
   f. Sedative infusions will continue in all patients receiving neuromuscular blockade.
VII. Mannitol and Hypertonic Saline therapy for ICP Control

A. Background
Mannitol is effective for treatment of elevated ICP after severe head injury. It is thought that an intact blood-brain barrier is necessary for maximal effectiveness of mannitol. Effects within minutes are observed due to rheologic effects on blood volume. Osmotic effects are seen within 15-30 minutes. Mannitol may be more effective when administered as intermittent boluses, rather than continuous infusion. Recent data also suggests that hypertonic saline solutions (3% or 7.5% NaCl) effectively reduce ICP. These solutions can be used as a primary treatment for increased ICP or as an adjunct to mannitol. Hypertonic saline (HTS) may also be effective as a salvage treatment in patients whom mannitol therapy has failed.

B. Goals of Therapy
1. To treat acutely elevated ICP or diminished CPP, while avoiding hypovolemia.
2. To treat clinical signs of cerebral herniation prior to ICP monitoring or if ICP does not reflect focal tissue shifts (e.g. focal temporal lobe pathology).

C. Guidelines
1. Mannitol dosing – 0.25-1.0 gm/kg as needed consideration may be given to regular interval dosing (Q6 hrs), but continuous infusions will, in general be avoided.
2. Serum osmolarity should be kept below 340 mOsm, esp. if renal failure is a concern.
3. A suggested regimen is to check serum osmolarity 1 hour after prior mannitol dose.
4. Fluid replacement, usually with NS, will be undertaken to maintain appropriate volume status, usually euvolemia, as indicated in Section V.
5. Hypertonic saline solutions (3% or 7.5% NaCl) may be used at the discretion of the treating physician.
6. Attempts should be made to keep serum sodium values at the high normal or slightly above normal range with the use of appropriate saline solutions. The maneuver should not be performed at the risk of hypovolemia.
VIII. Barbiturates

A. Background
The use of high-dose barbiturate therapy (“pentobarb coma”) for the control of intractable intracranial hypertension is controversial. While barbiturates do lower ICP by decreasing cerebral metabolism and altering vascular tone, significant data regarding improved outcome is lacking. Additionally, high-dose barbiturates have significant systemic complications, most notably hemodynamic compromise and increased infection risk. Therefore, barbiturate therapy in severe head injury is a second-tier therapy, usually reserved for potentially salvageable patients with refractory intracranial hypertension.

B. Goals of Therapy
To treat refractory intracranial hypertension, while avoiding systemic cardiovascular complications which may diminish CPP.

C. Guidelines
1. Barbiturate therapy may be considered in patients with persistently elevated ICP, especially if CPP remains diminished, despite maximal medical and surgical treatment, which usually includes CSF drainage, mannitol therapy, sedation +/- paralysis, and mild hyperventilation. Consideration can be given to earlier institution of barbiturates in individual situations.

2. Barbiturate therapy consists of pentobarbital with a loading and maintenance dose
   a. Loading dose: 10mg/kg over 30 minutes or 5 mg/kg Qhr x 3
   b. Maintenance dose: Initially 1 mg/kg/hr, adjusted Qhr based on ICP and EEG

3. Monitoring with EEG for burst-suppression is mandatory.
   a. Initial interburst interval = 10-15 seconds, modify based on ICP control.
   b. Pentobarbital loading should not be delayed for EEG placement.

4. Pulmonary-artery (PA) catheter placement may be useful.
   a. Pressor and inotropic support are usually needed for patients undergoing barbiturate therapy.
   b. Pentobarbital loading should not be delayed for PA catheter placement.

5. Weaning of pentobarbital infusion, initiate after 24 hours of acceptable ICP control.
   a. Decrease pentobarbital dose by 50% each day.
   b. Discontinue pentobarbital 48 hours after wean initiated, if tolerated.
   c. In individual situations, more rapid weaning of pentobarbital infusion may be considered, as tolerated.
IX. **Glucocorticoids**

A. **Background**

Glucocorticoids have been used in the past for treatment of brain edema in a variety of conditions, including head trauma, stroke, brain tumor, and cerebral abscess. Although currently used for vasogenic edema in tumor and abscess, they have been shown as ineffective in lowering ICP or improving outcome in patients with head trauma and stroke.

B. **Goals of Therapy**

None.

C. **Guidelines**

1. Glucocorticoids will not be used for the treatment of head trauma.
2. Glucocorticoid treatment for other indications may be provided in patients with head trauma. These indications include:
   a. Other conditions requiring corticosteroids (e.g. asthma, prior outpatient corticosteroid use).
   b. In patients with head trauma who are receiving glucocorticoids for other indications, insulin infusion will be used to maintain serum glucose from 100-200.
   c. In patients with head trauma who are receiving glucocorticoids for other indications, non-depolarizing neuromuscular blocking agents will be avoided if at all possible, because of concerns of persistent paralysis from muscle damage induced by the combination of the two agents.
   d. Low dose corticosteroids may be used at a dose of 25mgs of cortisone acetate if hypothalamic/pituitary dysfunction is suspected. A decreased serum cortisol level is confirmatory.
X. Brain Oxygen Monitoring

A. Background

Although ICP and CPP directed therapy has dominated modern severe TBI treatment, more sophisticated technologies are being used to determine cerebral oxygen and metabolic activity. Many of these methods are still experimental and are beyond the scope of these guidelines. But two existing well documented and relatively simple technologies and their treatment thresholds will be discussed. Jugular venous saturation monitoring (SjO₂) has been assessed in a number of studies beginning in the early 1990’s. The SjO₂ probe is placed in the region of the jugular bulb with a retrograde IJ stick. Levels of saturation are measured with a digital monitor. Brain tissue oxygen monitoring (PbrO₂) has been more frequently used technology since the late 1990’s. The probe is a fine parenchymal device into a selected area of the brain parenchymal and connected to a digital continuos monitor.

B. Goals

a. The detection (canary in the coal mine) and prevention of secondary brain injury following severe TBI
b. The detection of hyperemic brain syndrome and progressive cerebral infarction (High SjO₂)

C. Guidelines

a. Jugular venous saturation <50% is a treatment threshold.
b. Brain tissue oxygen tension <15mm of Hg is a treatment threshold.
c. Significant changes in SjO₂ values and PbrO₂ values should be accompanied by a through evaluation of the patient from both a neurologic and systemic stand point.
d. Current evidence suggests that episodes of desaturation SjO₂ < 50-55% are associated with worse outcomes. Low values of PbrO₂ (<10-15mmHg) and the extent of their duration (>30 minutes) are associated with high rates of mortality.
XI. Hypothermia

A. Background
Hyperthermia, even of 1-2° C, worsens brain injury after experimental trauma. Hyperthermia is thought to worsen secondary brain injury after stroke, intracranial hemorrhage, and trauma in patients as well. Hypothermia has been considered as a neuroprotective strategy in all of these diseases. Presumably, hypothermia (usually to 32-33° C) decreases cerebral oxygen utilization and acts as a neuroprotectant. Despite reports from small series of improved outcome after induced hypothermia, larger clinical trials have not consistently shown benefit. Also, there are concerns about systemic effects of hypothermia including coagulopathy, increased infection risks, and cardiac arrhythmias. At present, a strategy of avoiding hyperthermia is essential, with the role of induced hypothermia being less certain.

B. Goals of Therapy
1. To avoid hyperthermia-induced secondary brain injury.
2. To consider induced hypothermia as a 2nd tier therapy for refractory elevated ICP.

C. Guidelines
1. Goal temperature (measured intravascularly or rectally) will be 35°C/95°F.
2. For T > 37.5° C, antipyretics such as acetaminophen will be initiated.
3. Mechanical measures such as cooling blankets, fans, and/or body suits will be used to keep T < 37.5° C.
4. Appropriate measures will be taken to identify and treat infectious sources.
5. Around the clock antipyretics will be considered for patients with recurrent fever spikes.
6. Patients with shivering from fever or hypothermia measures will be treated as needed with agents, per Sedation and Analgesia Guidelines.
7. More aggressive hypothermia to 32-33° C using nasogastric lavage and other measures may be considered in cases of refractory elevated ICP as secondary tier therapy.
XII. Integrated Approach to ICP and CPP Management

Maintain neutral head position, head of bed at 25°, loose ETT taping
Maintain adequate intravascular volume
Analgesia and paralytics as needed; maintain T < 37.5°; seizure prophylaxis
Research studies of new monitoring tools or therapies per protocol

- Insert ICP Monitor
- Maintain CPP > 60 mmHg
- ICP > 20-25 and/or CPP < 60?
  - Yes
    - Ventricular Drainage (if available).
    - Sedation per protocol.
    - Maintain pH 7.38-7.42 and pCO₂ 38-42.
  - No
- ICP > 20-25 and/or CPP < 60?
  - Yes
    - Hyperventilation to p₅CO₂ 30-35 mmHg
  - No
    - Consider Repeating CT Scan
    - Hypothemia
    - Decompressive Craniectomy
    - High-dose Barbiturate Therapy
    - Hyperventilation to p₅CO₂ < 30 mmHg;
      (monitoring SJVO₂, AVDO₂, and/or CBF recommended)
- Carefully Withdraw ICP Treatment

Mannitol *
(0.25-1.0 g/kg IV bolus)

May Repeat Mannitol if Serum Osmolarity < 340 mOsm/L
& Pt Euvolemic

* and or Hypertonic saline
XIII. **Anti-Seizure Prophylaxis**

A. **Background**
Posttraumatic seizures may occur early, within 7 days of injury, or late, after 7 days. Seizures may worsen ICP control and worsen secondary brain injury, especially if status epilepticus occurs. Prospective, randomized trials have shown that prophylactic anticonvulsants may prevent the occurrence of early, but not late posttraumatic seizures.

B. **Goals of Therapy**
To prevent seizures in patients with head trauma

C. **Guidelines**
1. After head injury, phenytoin (IV load 17mg/kg and initial maintenance, 100mg IV TID or QID) will be instituted.
2. Keppra may be used as an alternative in certain situations.
3. In general, prophylactic anticonvulsants will be discontinued after seven days
4. For patients with early posttraumatic seizures after institution of anticonvulsants, in general, anticonvulsants will be continued beyond the 7 day period.
5. Patients who have undergone decompressive craniectomy or are still on paralytic agents will remain on anticonvulsants.
6. Patients who have frequent seizures, status epilepticus, or are not on anticonvulsants and are paralyzed and sedated, will undergo continuous EEG monitoring.
XIV. General Critical Care Issues

A. Background
Patients with severe head injury are critically ill and therefore have multiple issues related to general critical care management. These include, but are not limited to, nutritional support, prevention of deep venous thrombosis and gastrointestinal ulcers, and routine issues of IV fluid and blood product management.

B. Goals of Therapy
1. To prevent complications of critical illnesses that may effect severe head injury patients.
2. To ensure adequate nutrition in critically ill head injury patients.

C. Guidelines
1. Nutritional Support (please see nutritional guidelines)
   i. Enteral feeding will commence as early as feasible, preferably within 24 hours post-trauma. 140% of resting metabolism for most patients; 100% of resting metabolism for patients receiving neuromuscular blockade.
   ii. For patients who cannot receive enteral feeding, TPN will be provided.
2. DVT Prophylaxis (See Chapter)
3. Ulcer Prophylaxis
   i. An H$_2$-blocker or proton pump inhibitor will be initiated at time of ICU admission.
   ii. Sucralfate or omeprazole are alternatives.
4. IV Fluids
   i. All infusions will be mixed in 0.9% NS.
   ii. An even or slightly positive (~500-1000 cc) daily fluid balance will be maintained.
   iii. Hyponatremia will be aggressively avoided and treated.
      a. For Na < 135, 3% NaCl administration will be considered.
      b. Severe fluid restriction will not be undertaken, as hypovolemia is to be avoided in patients with severe head injury.
5. Hematology
   i. DIC may occur in the setting of severe head injury.
   ii. In the absence of bleeding, CBC/plts, PT/PTT will be checked daily.
   iii. Platelet count will be maintained above 100,000 at the discretion of the Neurosurgery Attending.
   iv. RBC transfusion may be considered for hematocrit < 30%, even in the absence of DIC.
XV. Deep Venous Thrombosis Prophylaxis

A. Background
It is well known that in the setting of traumatic brain injury most patients develop a hypercoagulable state. Hence risk for deep venous thrombosis (DVT) and pulmonary emboli (PE) is increased. Historically, with out any treatment, risk of DVT may be as high as 20-25% in this population. With the use of graduated compression stockings (GCS) and intermittent pneumatic compressors (IPC) the risk drops to approximately 8%-10% (17). Norwood et al found in 150 TBI patients in an observational study using intermittent pneumatic compression (IPC) devices and graduated compression stockings (GCS) and LMWH (enoxaparin 30mg bid) within 24 hours of admission or surgery only a 2% incidence of DVT. However there was a 4% risk of further intra-cranial hemorrhage requiring further cranial surgery (18).

These patients have experienced some degree of intra-cranial hemorrhage usually in the form of contusions, SDH, EDH, or traumatic subarachnoid hemorrhage and are at greater risk for further intracranial hemorrhage particularly in the face of anticoagulation. Many of these patients will also have an external ventricular drain placed within the first 24 hours and the drain may malfunction and need to be change unpredictably with regard to time.

B. Goals of Therapy:
Prevention of DVT and PE is the goal without increasing the risk of intracranial bleeding significantly. If DVT does occur and risk of full anticoagulation is still unacceptable prevention of fatal or severe pulmonary emboli by placement of a temporary inferior vena cava filter is in order.

C. Guidelines:
1. All patients with TBI should be started on GCS and IPC on admission if possible. IPC’s are preferable over plexipulses.
2. All TBI patients should be started on low dose unfractionated heparin (LDUH) 5000units SQ q 12 hours at 48 hours following admission. If an external ventricular drain (EVD) has been placed the LDUH should be started 48 hours after the EVD has been placed.
3. If there in any evidence of new intracranial hemorrhage (ICH) the LDUH will be discontinued, a PTT should be checked and Protamine given if appropriate.
4. Surveillance lower extremity Doppler ultrasound should be ordered between 48 and 72 hours of admission and repeated every 5-7 days during the patients stay in the Neuro-Critical Care Unit (NCCU).
5. If a lower extremity DVT is identified and the patient is not yet a candidate for full anticoagulation a retrievable inferior vena cava filter should be placed and anticoagulation started at the highest level acceptable short of full anticoagulation. If small calf DVT are found it may be acceptable to follow these with daily lower extremity doppler exams rather than placing a filter if the window to be able to advance to anticoagulation is short.

6. Twenty four hours after the EVD has been removed the level of LDUH can be raised to 5,000 units’ SQ q 8 hours. If this is well tolerated for 48 hours the level of anticoagulation can be raised to LMWH (enoxaparin 30mg SQ Bid) if there is no contraindication.

7. Using Heparin carries the risk of developing heparin induced thrombocytopenia. Hence, any patient receiving heparin should have their platelet count monitored at least every other day.

8. If remarkable upper extremity swelling occurs upper extremity doppler studies should be performed to see if an upper extremity DVT has developed. If the upper extremity DVT is large and proximal and the patient can not be fully anticoagulated consideration should be given to placing a superior vena cava filter. Whatever level of anticoagulation can be tolerated up to full anticoagulation should be implemented with or without the filter.

9. If the presence of a DVT is associated with high fever and a leukocytosis, septic thrombophlebitis should be considered and blood cultures should be obtained daily and repeated until the infection has cleared. Vascular surgery consultation may be indicated for surgical drainage as well as appropriate antibiotics.

10. Duration of DVT prophylaxis should extend until discharge from the hospital.
XIX. **Timing of Non-Neurosurgical Operative Procedures**

A. **Background**

Patients with severe head injury often have other injuries which require operative intervention. Strategies designed to treat non-neurologic injuries may be at odds with the guidelines for management of severe head injury as hypovolemia or hypotension and hypoxia are to be avoided. Issues related to non-neurosurgical operative procedures may exacerbate secondary brain injury if attention is not taken to avoid hypotension and aggressive volume resuscitation, especially with hypotonic fluids.

B. **Goals of Therapy**

To allow safe and successful treatment non-neurologic injuries, while avoiding secondary brain injury.

C. **Guidelines**

1. In general, neurologic management issues will take precedence in patients with severe head injury and multi-system trauma.
2. If a non-neurologic injury is immediately life-threatening, urgent medical and surgical intervention will be undertaken as appropriate, with attention to maintaining a minimum systolic BP > 90 mmHg (MAP > 90 mmHg strongly recommended) with volume and pressors, and P_{a}O_{2} > 100 mmHg. ICP monitoring will be considered during the non-neurosurgical operative intervention with the threshold CPP of 60 mmHg being preferred.
3. For non-life threatening non-neurologic injuries requiring operative management, timing of surgery will be at the discretion of the Neurosurgery Attending, with input from the appropriate surgical service(s).
4. Discussion will be undertaken between the surgical, neurosurgical, and anesthesia services prior to operative intervention in order in ensure accurate communication regarding goals of intraoperative neurologic management.
XVII. Decompressive Craniectomy

A. Background
The use of decompressive craniectomy in the management of intracranial hypertension has never been subjected to a prospective randomized control study. However, several recent articles have shown a statistical significant improvement in outcome in both case controlled studies and comparison studies with the Traumatic Coma Data Bank Statistics for patients undergoing decompressive craniectomy with established or impending uncontrollable intracranial hypertension.

B. Goals of Therapy
To treat refractory or impending intracranial hypertension while avoiding the potential side effects and complications of other secondary tier therapies.

C. Guidelines
1. “Early” Decompressive Craniectomy
   - Associated with surgery for intracranial mass lesions (See Surgical TBI Guidelines)
   - If possible ICP monitor inserted prior to or simultaneously with craniotomy
   - Procedure
     a) Trauma Skin Flap
     b) Bone flap at least 15cm$^2$ in diameter with subtemporal decompression
     c) Mass lesion removed
     d) Decision concerning duraplasty and non-replacement of flap made on the basis of:
        1) ICP values when available
        2) Appearance of brain
     e) Duraplasty is mandatory and natural tissues are used for duraplasty (ex. Pericranium, Temporallis Fascia, Fascia Lata)
     f) Flap frozen or inserted in abdominal pouch
   - Procedure in generally unilateral hemispheric decompression

2. “Late” Decompressive Craniectomy
   - Within 72hrs of injury
   - Patients selected have failed standard primary tier therapy (See Algorithm)
   - ICP’s >30 for greater than 20 minutes despite maximum primary tier therapy
   - Most procedures hemispheric on worst side of lesion
   - Will also combine with parenchymal resection when indicated (ex. Temporal lobectomy)
- In cases of diffuse swelling without hemispheric predominance bifrontal procedure is done with division of sagittal sinus and falx ample duraplasty will be performed
- Decompressive Craniectomy more than 72hrs post injury usually reserved for patients with associated vascular insult (ex. large posttraumatic stroke or expanding mass lesion)
XVIII. References


