Advances in Trauma and Acute Management of Traumatic Brain Injury

TBI: A Multidisciplinary Approach to Rehabilitation Across the Continuum of Care
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Chris Zacko M.S., M.D., FAANS
Assistant Professor of Neurosurgery
Director of Neurotrauma and Neurocritical Care
Penn State Hershey Medical Center

Disclosures

• No financial disclosures

Overview/Goals of Discussion

• Based upon the Brain Trauma Foundation’s (BTF) 3rd Edition of the Guidelines for the Management of Severe Traumatic Brain Injury (STBI)
• Review Systemic and General Critical Care
• Review CNS-specific Neurocritical Care and ICP control management options
• Emerging concepts
The 3rd Edition of the Guidelines for Severe TBI’s

Changes in the 3rd edition include:

- **Four (4) new topics** include i) a meta-analysis of Prophylactic Hypothermia, ii) Brain Oxygen Monitoring and Treatment, iii) Infection prophylaxis, iv) DVT prophylaxis

- **Two (2) expanded topics** are i) Hyperosmolar Therapy (expanded from Mannitol), and ii) Anesthetics, Analgesics, and Sedatives (formerly Barbiturates)

- A conversion from level of recommendation nomenclature to the standardized EBM terms “Level 1, Level 2, and Level 3”. To do so, the BTF established and integrated the assistance of the “Evidence-Based Practice Center” @ Oregon Health Sciences University to include literature searches through April 2006

Topics Covered in the 3rd Edition

- Infection Prophylaxis - New
- DVT Prophylaxis - New
- Prophylactic Hypothermia - New
- Brain Oxygen Monitoring and Treatment - New
- Hyperosmolar Therapies - Newly expanded
- Anesthetics Analgesics and Sedatives - Newly expanded
- Indications for Intracranial Pressure Monitoring
- Intracranial Pressure Monitoring Technology
- Intracranial Pressure Treatment Threshold
- Cerebral Perfusion Pressure
- Hyperventilation
- Blood pressure and oxygenation
- Nutrition
- Anti-seizure Prophylaxis
- Steroids

Limitations of the BTF Guidelines

- The Guidelines do NOT include chapters on ICP Management in the Elderly or Decompressive Cranectomy due to a lack of a literature base
- Only reviewed human literature
- Doesn’t necessarily consider specific patient factors

- There are also published guidelines at [www.braintrauma.org](http://www.braintrauma.org) covering Prehospital Management, Surgical Management, Penetrating Injuries, and Prognosis of Severe TBI’s
Do The Guidelines Make A Difference?

- There has been a progressive and significant reduction in mortality in severe TBI patients 50% to <25% in the last 30 years
- Adherence to a protocol based on the BTF guidelines can result in a significant decrease in hospital days and charges for TBI patients who live > 48 hours (The Journal of Trauma, Injury, Infection and Critical Care Volume 56(3), March 2004, pp 492-500)
- ICU stay was reduced by 1.8 days (p = 0.021) and total hospital stay was reduced by 5.4 days (p < 0.001). The charge reduction (calculated in 1997 dollars) per patient for the length of stay decrease was $6,577 in 1995–96 and $8,266 in 1997–2000 (p = 0.002)
- Compliance with BTF ICP monitoring Guidelines improved mortality to 32.7% from 53.9% (Talving et al 2013)

Outcomes And Following The Guidelines

<table>
<thead>
<tr>
<th>Group</th>
<th>GOS=1 Died</th>
<th>GOS=2&amp;3 Severe Disability</th>
<th>GOS=4&amp;5 Good Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Pre-TBI Guidelines</td>
<td>16 (43%)</td>
<td>11 (50%)</td>
<td>10 (27%)</td>
</tr>
<tr>
<td>Post-TBI Guidelines</td>
<td>9 (16%)</td>
<td>8 (14%)</td>
<td>39 (70%)</td>
</tr>
</tbody>
</table>
Goals At-a-Glance

1. Oxygen Saturation >93%, Avoid <90%
2. PaO\textsubscript{2} 95-100mmHg (once outside initial 24 hours of normobaric hyperoxygenation). Avoid <60 mmHg
3. PaCO\textsubscript{2} 35-40
4. MAP >70mmHg (>90mmHg until CPP can be measured)
5. SBP >95mmHg. Avoid any episodes <95mmHg
6. CVP 5-8mmHg.
7. Temperature: 35-37°C. Avoid >38.5 °C
8. Labs: HCT ≥ 30, platelets ≥ 100,000, INR ≤ 1.2, Na 135-145

General and Systemic Critical Care

1. Early Intubation, Adequate Ventilation And Oxygenation:
   a. Goals: Oxygenate to PaO\textsubscript{2}>95; avoid PaO\textsubscript{2} <60 or O2 sat <90%
   b. Mild hyperventilation to a PaCO\textsubscript{2} no lower than 35, especially in the first 48-72 hours of admission when CBF is critically reduced
      • No prophylactic hyperventilation, although it can be used as a temporizing measure when clinical signs of herniation are present
      • Hyperventilation to lower then 35 should be guided by PbtO\textsubscript{2} or jugular bulb parameters

General Critical Care

2. Initiate Comprehensive Monitoring And Resuscitation:
   • Severe TBI’s should have ICP monitor’s, central lines and arterial lines - Avoid SBP ≤ 95
   • Multimodality monitoring at discretion of individual
3. Proper Positioning: Head of Bed 30° with head straight (avoid jugular venous outflow obstruction)
4. Control Body Temperature: aggressively treat fevers >38°C
5. Adequate Sedation/Analgesia: Fentanyl, Versed and Propofol
Anti-seizure Prophylaxis

Level 2:
1. Anticonvulsants are indicated to **decrease the incidence of early PTS**. However, early PTS is not associated with worse outcomes.

2. Prophylactic use of phenytoin or valproate is not recommended for preventing late posttraumatic seizures (PTS) < 7 days OK.

Who Gets AED?

1. All patients meeting specific indications after TBI should receive prophylactic AED for 7 days after TBI. This has been shown to decrease incidence of early post-traumatic seizures (no benefit on outcome or late seizures).

2. These indications are:
   a) GCS <10
   b) Cortical contusion
   c) Depressed skull fracture
   d) SDH
   e) EDH
   f) ICH
   g) Penetrating head wound
   h) Seizure within 24 hours of TBI

   - **Original study done with Dilantin**
   - **Many practitioners using Levetiracetam due to more favorable side effect profile**

Steroids

Level 1:
1. The use of steroids is **not recommended** for improving outcome or reducing intracranial pressure (ICP)s.

2. In patients with moderate or severe traumatic brain injury (TBI), high dose methylprednisolone is associated with **increased mortality** and is contraindicated.

   → Steroids
DVT Prophylaxis - NEW

Level 3:
1. Graduated compression stockings or intermittent pneumatic compression (IPC) stockings are recommended, unless lower extremity injuries prevent their use. Use should be continued until patients are ambulatory.
2. Low molecular weight heparin (LMWH) or low dose unfractionated heparin should be used in combination with mechanical prophylaxis. Due to concerns of increased risk of intracranial bleeding, treatment with LMWH or low-dose unfractionated heparin should be avoided early after trauma and in the period preceding and immediately after craniotomy.
3. There is insufficient evidence to support recommendations regarding the preferred agent, dose, or timing of pharmacologic prophylaxis for deep vein thrombosis.

Infection Prophylaxis - New

Level 2:
1. Perioperative antibiotics for intubation should be administered to reduce the incidence of pneumonia. However, it does not change length of stay or mortality.
   - Sirvent, et al., statistically significant decrease in the incidence of pneumonia in the treated group (23% vs. 64%, P = 0.036), but no difference in mortality. (Sirvent JM, Torres A, Mustafa E, et al.: Protective effect of intravenously administered cefuroxime against nosocomial pneumonia in patients with structural coma. Am J Respir Crit Care Med 155:1729-1734, 1997)
2. Early tracheostomy should be performed to reduce mechanical ventilation days. However, it does not alter mortality or the rate of nosocomial pneumonia.

Level 3:
1. Routine ventricular catheter exchange or prophylactic antibiotic use for ventricular catheter placement is not recommended to reduce infection.
2. Early extubation in qualified patients can be done without increased risk of pneumonia.
Nutrition

Level 2:
• Replace 140% of resting metabolism expenditure in non-paralyzed patients and 100% of resting metabolism expenditure in paralyzed patients using enteral or parenteral formulas containing at least 15% of calories as protein with goals being met by the 7th day after injury
• Nutritional replacement should begin no later than 72 hours after injury

Level 3:
• The method of feeding should be a jejunal feeding tube or gastrojejunostomy

Glycemic Control

• Treatment threshold is ≥150 to maintain blood glucose level <180
• Avoid <100 and >180
• Target is 140-180
Goals of Neurocritical Care

• Resuscitation to ensure adequate tissue metabolism
• Prevent secondary insults
• Attenuate secondary injury

Prognostic Value of Secondary Insults

• Cerebral ischemia
• Intracranial hypertension
• Systemic hypotension
• Hypoxia
• Fever
• Hypocapnia
• Hypoglycemia

All independent risk factors for worsened survival


Mechanisms of Secondary Injury

• Hypoxia-Ischemia
• Edema
• Excitotoxicity (via Glutamate)
• Calcium Dysregulation
• Cytoskeletal Proteolysis and Secondary Axotomy
• Neuronal Depolarization
• Disturbance of Ionic Homeostasis
• Metabolic and Mitochondrial dysfunction
• Lipid Peroxidation
• Alterations in vascular permeability (disruption of autoregulation and BBB breakdown)
• Oxidative Stress/Free radical and nitric oxide production
• Intracranial Hypertension
• Secondary Hemorrhage
• Neuroinflammation
• Apoptotic and Necrotic Cell Death
Determinants Of Head Injury Mortality

6 Powerful Predictors of Outcome:
1) Age (>40yo)
2) Admission GCS
3) Admission motor score
4) SBP <90mmHg, (even a single episode doubles mortality)
5) Pupil status
6) Intracranial pathology (BTF): SDH worse than EDH as an example

Management Strategies for TBI

- ICP-directed (traditional and most widespread)
- CPP-directed (Rosner)
- Volume-directed (Lund)
- Autoregulation-directed/Optimal CPP
- \( \text{PbO}_2 \) directed therapy
- Compliance-directed Therapy (ICP Wave)

Classification Of TBI For Targeted Therapies
**Overview Of ICP Control Options**

- Initial therapeutic measures
  - Intubation with controlled ventilation, optimize positioning, controlled ventilation, mild to moderate sedation
  - CSF drainage
  - Hyperosmolar Therapy
  - Mannitol
  - Hypertonic Saline
  - Surgical Decompression
- Modulation of metabolic demand
  - Heavy anesthesia, sedation, analgesia
  - Neuromuscular Paralysis
  - Hypothermia (therapeutic, not prophylactic)
- Barbiturate Coma

**Goals At-a-Glance**

1. CEREBRAL PHYSIOLOGY:
   - CPP 50-70mmHg
   - ICP <20mmHg with treatment threshold >20mmHg
2. Licox: Treatment threshold is when <15
   - Assessment of cerebral oxygenation is especially important in the first 48 hours when CBF can be reduced by as much as 50%.
3. SjvO2: Normal is 55-70% with treatment threshold <50%
   - <55% indicates inadequate oxygen delivery or excessive demand
   - <50% Hypoxemia
   - <40% Ischemia
   - >75% indicates hyperemia or inability to extract oxygen

**CSF Drainage**

1. Treatment should begin when ICP>20mmHg (Level 2)
   a) Prophylactic antibiotics are NOT necessary for placement of an EVD.
   b) Routine flushing and catheter exchanges are NOT recommended as they increase infection risk
Indications For ICP Monitoring

Level 2: GCS ≤ 8 after resuscitation and an abnormal*** HCT (in salvageable patients)

***An abnormal HCT is one that reveals hematomas, contusions, swelling, herniation, or compressed basal cisterns

Level 3: GCS ≤ 8 after resuscitation and a normal HCT if 2 or more of the following are present upon admission:
   a. Age >40yo
   b. Unilateral or bilateral motor posturing
   c. SBP < 90 mmHg

Compliance with BTF ICP monitoring Guidelines improved mortality to 32.7% from 53.9% (Talving et al 2013)

CPP Recommendations

LEVEL 2:
- Aggressive attempts to maintain cerebral perfusion pressure (CPP)
- Above 70 mm Hg with fluids and pressors should be avoided because of the risk of adult respiratory distress syndrome (ARDS)

LEVEL 3:
- CPP < 50 mm Hg should be avoided
- The CPP value to target lies within the range of 50-70 mm Hg. Patients with intact pressure autoregulation tolerate higher CPP values
- Ancillary monitoring of cerebral blood flow, cerebral oxygenation, cerebral oxygen extraction or lactate production, and cerebral metabolism can facilitate CPP management

Hyperventilation Recommendations

Level 2:
- Prophylactic hyperventilation (PaCO₂ <25 mm Hg) is not recommended

Level 3:
- Hyperventilation is recommended as a temporizing measure for the reduction of elevated intracranial pressure (ICP)
- Hyperventilation to a PaCO₂ below 29 mm Hg should be avoided during the first 24 hours after injury, when cerebral blood flow (CBF) is often critically reduced
- If hyperventilation is used, jugular venous oxygen saturation (SjO₂) or brain tissue O₂ partial pressure (PbtO₂) measurements are recommended to help determine if hyperventilation is causing ischemia

Compliance with BTF ICP monitoring Guidelines improved mortality to 32.7% from 53.9% (Talving et al 2013)
Hyperosmolar Therapy - Newly Expanded

1. Mannitol remains effective for control of elevated ICP (Level 2)
   a) Bolus administration (0.25-1.0 g/kg intravenously) is preferred
   b) Avoid arterial hypotension (SBP <90 mmHg). Consider crystalloid "chaser" or pressor after administration
   c) Be cautious with the presence of hypotension, sepsis, nephrotoxic drugs, or pre-existing renal disease → check serum osmolality (hold if >320)
2. Restrict mannitol use prior to ICP monitoring to patients with signs of lateralization, transtentorial herniation, or progressive neurological deterioration not attributable to extracranial causes (Level III)
3. Hypertonic saline (HTS) is gaining popularity as an option. Some reserve it for elevated ICP refractory to mannitol
   a) Exclude hyponatremia before use to avoid Central Pontine Myelinolysis

The Pediatric Head Injury Guidelines currently recommend continuous infusion of 3% HTS for ICP control (Level III recommendation)

Hyperosmolar Therapies

- In patients with a subdural or temporal lobe hematoma undergoing surgical evacuation, an early preoperative single, high dose of mannitol (1.2-1.4 g/kg) is recommended, provided the patient is not hypotensive.
- In patients with absent motor response, bilateral pupillary widening and severe diffuse brain swelling on CT scan, an early initial high dose of mannitol (1.2-1.4 g/kg) is recommended

Hypertonic Saline

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Hypertonic Saline</th>
<th>Mannitol</th>
</tr>
</thead>
<tbody>
<tr>
<td>Volume</td>
<td>Volume expander</td>
<td>Initially expands plasma volume... but causes diuresis and possible hypotension</td>
</tr>
<tr>
<td>Serum osmolality</td>
<td>Increased</td>
<td>Increased</td>
</tr>
<tr>
<td>Crossing BBB</td>
<td>Less likely</td>
<td>More likely</td>
</tr>
<tr>
<td>Impact on ICP</td>
<td>Similar reduction</td>
<td>Similar reduction</td>
</tr>
<tr>
<td>Impact on CPP</td>
<td>Maintains CPP</td>
<td>May reduce CPP due to hypotension/diuresis</td>
</tr>
<tr>
<td>Immunomodulation</td>
<td>May decrease inflammation</td>
<td>No significant effect</td>
</tr>
</tbody>
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10/22/2014
Decompressive Craniectomy

1. When the above measures do not control ICP, consider wide decompressive decompression
   a) Unilateral frontotemporoparietal craniectomy with duraplasty from a pterional approach. Bifrontal also an option
   b) Must decompress the temporal fossa
   c) Dural opening at the base
   d) Bifrontal craniectomy if it's a diffuse injury

2. After decompression, ICP treatment threshold is likely lower (>15mmHg and not 20mmHg)


Modulation Of Metabolic Demand

- Heavy anesthesia, sedation, analgesia
- Neuromuscular Paralysis
- Hypothermia (therapeutic, not prophylactic)
- Barbiturate Coma

Heavy Sedation and Neuromuscular Paralysis

1. PARALYSIS: Vecuronium
   a) Paralyze to a train of four (TOF) of 2/4 as deeper neuromuscular paralysis is associated with increased risk of pulmonary complications due to paralysis of pulmonary ciliary motility.

2. ANALGESIA/SEDATION: Fentanyl/Versed

*** Despite the fact that sedation and neuromuscular paralysis have not been shown to improve outcome they remain a common management strategy. That said their use is being revisited, particularly paralysis. However, we continue to implement them as no alternative has been established.
   - Propofol is not recommended for long-term use.
   - CAVEAT: Benzodiazepines have been shown in animals to negatively influence cognitive outcomes
Anesthetics, Analgesia, And Sedatives

Level 2:
• Propofol is recommended for the control of ICP, but not for improvement in mortality or 6 month outcome. However, high-dose propofol can produce significant morbidity (propofol infusion syndrome, hypotension, immunosuppression)

**** Caution must be taken when using doses greater than 5mg/kg/hr or when usage of any dose exceeds 48 hours in critically ill adults.

Prophylactic Hypothermia

Level 3:
a. Induced prophylactic hypothermia is not associated with a significant decrease in mortality when compared to normothermic controls in a meta-analysis of 15 clinical trials
b. However, in a sub-group analysis, maintenance of target temperatures for more than 48 hours is significantly associated with decreased mortality
c. Induced prophylactic hypothermia is associated with a significant increase in the percentage of patients with better Glasgow Outcome Scale (GOS) scores when compared to scores for normothermic controls

Therapeutic Hypothermia

1) When control of ICP is intractable to the above measures, cooling to 34°C (+/- 1 degree) is recommended
a) Maintain target temperature for at least 48 hours (level III data in prophylactic hypothermia analysis).
b) Rewarm slowly when hypothermia no longer necessary (0.2-0.5 degrees/hour)
2) No Level I or II data supporting prophylactic use of hypothermia
3) During rewarming a patient will likely require increased fluids and possibly pressors (watch for hyperkalemia)
Barbiturate Coma

Level 2:
1. Prophylactic administration of barbiturates to induce burst suppression EEG is NOT recommended.
2. High-dose barbiturate administration is recommended to control elevated ICP refractory to maximum standard medical and surgical treatment. Hemodynamic stability is essential before and during barbiturate therapy.
3. Prophylactic administration of high dose barbiturates is contraindicated for intracranial pressure (ICP) control in patients with diffuse injuries and is associated with significant hypotension.

REVIEW

1. Oxygen Saturation >93%. Avoid <90%
2. PaO₂ 95-100mmHg (once outside initial 24 hours of normobaric hyperoxygenation). Avoid <60 mmHg
3. PaCO₂ ~35-40 Avoid prophylactic hyperventilation
4. MAP >70mmHg (>90mmHg until CPP can be measured)
5. SBP >95mmHg. Avoid any episodes <95mmHg
6. CVP 5-8mmHg.
7. Temperature: 35-37°C. Avoid >38.5 °C
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Review

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Don’t List

- HypXension
- Prophylactic Hyperventilation
- Steroids
- Anti-seizure Prophylaxis Beyond a Week
EMERGING CONCEPTS
Classification Of TBI For Targeted Therapies

Management Strategies for TBI
• ICP-directed (traditional and most widespread)
• CPP-directed (Rosner)
• Volume-directed (Lund)
• Autoregulation-directed/Optimal CPP
• PbO2 directed therapy
• Compliance-Directed Therapy (ICP Wave)

Multi-modality Monitoring
• Data derived from different monitoring modalities can provide a sophisticated biochemical profile of regional and global brain environments
• Real time measurement of multiple indices of cerebral physiology may ultimately allow for more dynamic assessments of cerebral health and allow the development of individualized and goal-directed cerebral therapies (including predictive non-linear modeling of multiple dynamic physiologic parameters)
Multi-modality Monitoring

<table>
<thead>
<tr>
<th>INVASIVE</th>
<th>NON-INVASIVE</th>
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<tbody>
<tr>
<td>ICP</td>
<td>Continuous EEG</td>
</tr>
<tr>
<td>Licox (regional)</td>
<td>Bispectral Index Monitoring (BIS)</td>
</tr>
<tr>
<td>Jugular Bulb Oxygenation (global)</td>
<td>Evoked Potentials</td>
</tr>
<tr>
<td>Microdialysis (regional)</td>
<td>Transcranial Doppler (TCD)</td>
</tr>
<tr>
<td>Cortical Spreading Depression</td>
<td>Near Infrared Spectroscopy (NIRS)</td>
</tr>
<tr>
<td>PRx, POx, ICP Wave</td>
<td>EEG</td>
</tr>
<tr>
<td>Cerebral Blood Flow (regional)</td>
<td>(Hemex vs. C-Flow)</td>
</tr>
<tr>
<td>Targeted Temperature Management (hypothermia)</td>
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</tbody>
</table>
Brain Oxygen Monitoring and Thresholds

Level 3:
- Jugular venous saturation (<50%) or brain tissue oxygen tension (<15mm Hg) are treatment thresholds
- Jugular venous saturation or brain tissue oxygen monitoring measure cerebral oxygenation

Cortical Spreading Depression

- **DEFINITION:** A depolarization wave in cerebral gray matter that propagates across the brain at slow velocity, 2-5mm/min
- CSD has been described since 1940's
- Until recently, their detection and significance has been difficult to ascertain
- Linked to mechanisms of migraine, stroke, SAH, TBI
- May explain the patient who won’t wake up, but when given time does (when all else has been constant)

Cortical Spreading Depression

- **INDUCED:** A deliberate perturbation of the brain (experimentally via electrical or mechanical stimulation)
- CSD silences spontaneous and evoked synaptic activity for 5-15 minutes
- Returns to normal function occurs spontaneously
Cortical Spreading Depression

- Dramatic failure in ionic homeostasis,
- Efflux of excitatory amino acids from neurons,
- Increased energy metabolism
- Changes in CBF

Microdialysis

Measures indices of the local tissue’s metabolic environment:
- Lactate
- Pyruvate
- Glucose
- Glycerol

Higher Lactate/Pyruvate ratios are indicative of anaerobic metabolism
Cerebral Blood Flow

- A newer technology using a proprietary technology in the clinical application of either thermal diffusion or lasers to measure perfusion
- Only measures very specific and local region of blood flow
- Potential applications, validation, and clinical uses still being investigated

Non-Invasive Monitoring

Continuous EEG

- The summation of excitatory and inhibitory postsynaptic potentials produced by the cortex
- Allows both spatial and temporal information simultaneously
- Most commonly used to detect subclinical non-convulsive status (NCS)
- Also used to verify burst suppression in barbiturate coma
- Can be used to alert for changes in CBF (when it falls below 30 mL/100g/min)
### Bispectral Index Monitoring (BIS)

- A simplified array that can be used to represent electrographic activity and monitor depth of anesthesia
- May provide a continuous value indicating level of consciousness – thus guiding sedative use and possibly changes in neurological function (ischemia? Aid prognosis predictions?)
- A 4 electrode strip placed on forehead and uses a complex mathematical algorithm to provide a weighted summation of multiple EEG parameters
- Interfrequency phase relationships are quantified providing a single 0-100 index value
  - <70 Low likelihood of awareness
  - <60 Extremely low likelihood of awareness
  - <40 for surgical hypnosis

### Evoked Potentials (EP’s)

- EP’s allow detection of the cerebral response to an external stimulus (auditory, visual, tactile, or motor action):
  - BAER’s (brainstem auditory…)
  - SSEP’s (somatosensory…)
  - VEP’s (visual…)
  - MEP’s (motor…)
- Predominately used to diagnose deficits, detect subclinical deficits, or localize pathology
- Whereas EEG provides acute information, EP’s delineate more specific pathways that have been shown to provide useful information as to the level of impairment and prognosis
- BUT there have been attempts to use these for prognostic data, but routine use not commonplace

### Transcranial Doppler Ultrasonography

- Measures velocity and used primarily in the ICU to evaluate CBF (and detect vasospasm) and can be done bedside
- Other uses: detect emboli, detect ischemia, collateral flow assessment (can detect anterograde vs. retrograde flow)
- Some advocate using the pulsatility index (PI) to indirectly estimate ICP
  - PI the systolic flow-diastolic flow/mean velocity
- Used in conjunction with S_o2 to aid interpretation as it can reveal hyperemia
- Disadvantages: Operator dependent, patient anatomy can alter effectiveness
Near-Infrared Spectroscopy (NIRS)

- Involves a light-emitting diode with 1 or 2 detectors that measures the ratio of oxyhemoglobin to total hemoglobin (similar to pulse oximetry)
- Depending on the amount of light absorbed and returned, oxygenation can be estimated intracranially
- Still being validated, but advantages include continuous data, minimal maintenance, and produces a single index
- Major limitation is that it gives a ratio of oxygenation without indicating whether it is because of inadequate perfusion or excessive metabolic use

Endocrine Function Evaluation

(NOT IN GUIDELINES) Due to the high incidence of pituitary and hypothalamic injuries that accompany TBI’s, particularly when skull base fractures are present, one should strongly consider screening endocrine function either by day 7 after injury or with unexplained hemodynamic instability

<table>
<thead>
<tr>
<th>ACUTE</th>
<th>SUBACUTE/CHRONIC</th>
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<tbody>
<tr>
<td>ACTH</td>
<td>GH</td>
</tr>
<tr>
<td>ADH</td>
<td>ACTH Stim</td>
</tr>
<tr>
<td>Cortisol (+/- stim)</td>
<td></td>
</tr>
<tr>
<td>Prolactin</td>
<td></td>
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<tr>
<td>T3, T4, TSH</td>
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Goal Directed Fluid Management

- New technologies have become readily available to for hemodynamic assessment and volume assessment. Examples include:
  - Arterial pulse waveform analyses (PiCCO® and Vigileo®)
  - Impedance Cardiography (ICG)
  - Cheetah NICOM®
  - Ultrasound and IVF compressibility
- Use of goal-directed therapy and limiting crystalloid infusion is an area of active research