Update On The Management of Traumatic Brain Injury

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Beyond Just Long Island

- Incidence
  - 1.7 million cases of TBI – 52,000 deaths

- Trimodal distribution
  - 0-4, 15-19, 65 and older
  - Mechanism related to age

- Mortality has decreased but Incidence Tripled
Pathophysiology of TBI

**Primary Injury**
- Skull fracture
- Brain contusion
- Intracranial Bleeding
- Parenchymal Injury
- Diffuse axonal injury
- Edema formation

**Major outcome determinants**

**Secondary Injury**
- Hypoxemia*
- Hypotension*
- Hypercarbia/Hypocarbia
- Hyperglycemia
- Seizures
- Vasospasm

**Inflammatory/excitotoxic edema formation**

CPP = MAP - ICP
Radiologic Findings
The Head Has A Body

Management is complicated by associated injuries

Also, systemic effects of TBI:

Cardiopulmonary – neurogenic pulmonary edema and myocardial dysfunction

Metabolic – catecholamine surges leading to hyperglycemia and increased catabolism

Endocrine – pituitary dysfunction, DI, SAIDH

Hematologic – Coagulopathies and DIC
• Severe TBI (GCS 3-8)
• Maintenance of cerebral perfusion
• Prehospital and ICU
• No specific guidelines for intraoperative or anesthetic management
Indications for Surgery in TBI

- Depressed skull fracture (> skull thickness)
- Mass lesion with > 5 mm shift or compression of the basal cisterns
- Acute subdural or extradural hematomas
- Refractory intracranial hypertension
Intra-operative Anesthetic Care

- Correct or maintain conditions that prevent secondary injury.
  - Avoid secondary insults – hypoxemia, etc…
  - Provide adequate anesthesia and analgesia
  - Maintain CPP through ICP and MAP management
Anesthetic Agents

- Thiopental, Propofol, Etomidate
  - Reduce CBF, CBV, CMRO$_2$ and ICP

- Opioids
  - No effect on cerebral hemodynamics (When ventilation is controlled)!

- Inhalational Agents
  - Volatiles dec. CMRO$_2$, but may cause vasodilation (Minimal effect at less than one MAC)
  - Avoid nitrous oxide (inc. CMRO$_2$, vasodilation and inc. ICP)
Hemodynamic Management

- A single episode of hypotension can impact outcome
  - Maintain SBP >90 mmHg
  - CPP > 50, < 70 (no iatrogenic increases > 70)

- Decompression hypotension
  - Usually occurs at the time of dural opening
  - Risk – low GCS, effaced cisterns, B/L dilated pupils
Hemodynamic Management

- Fluid Management - resuscitation to euvolemia and then maintenance
  - Isotonic non-glucose containing solutions
  - NS preferred to LR b/c of sodium content

- Colloid vs. Crystalloid – SAFE Trial
  - Saline vs. albumin for resuscitation – saline wins!
  - Severe TBI – higher mortality and poorer neurologic outcome at 24 months

- Hypertonic saline
  - Less data than for use to treat ICP
  - Works but clinically no difference in outcome
Hemodynamic Management

- Vasopressors – Scant evidence to suggest that there is one perfect choice

- Several small studies comparing specific drugs suggest the following:
  - NE vs. Dopamine equal effects on flow, oxygenation and metabolism, but NE effect is more consistent and DA may lead to higher ICPs
  - Phenylephrine may give the max increase in MAP and CPP without changing ICP
Hyperosmolar Therapy

- Mannitol is still the mainstay
  - Recommended for sign of impending herniation or neurologic deterioration in OR to improve surgical conditions

- Concerns:
  - Hypotension and hypovolemia
  - Expansion of pre-existing intracranial hematoma
Hyperosmolar Therapy

- Hypertonic saline – 3% to 23.4% solutions
  - ~ 5 ml/kg of 3% solution

- Target serum sodium level 145-160 mEq
- Has the advantage of beneficial hemodynamics
  - Increased intravascular volume
  - Increased inotropy
  - In ICU early ICP decreases with more sustained increases in CPP

- Intra-op may be better at improving surgical exposure but no known difference in outcomes
Respiratory Management

- Hypoxemia (PaO2 < 60) has major in effect on secondary injury
  - CBF, CBV and ICP increase linearly with hypoxia
  - For inadequate oxygenation – consider first increasing FiO2, inspiratory time and peak pressures before PEEP (may inc. ICP)
Respiratory Management

- Ventilation and CO2 the double edged sword of ICP control
  - Hypocapnia leads to vasoconstriction and reduction in CBF and CBV (Good)
  - Excessive hypocapnia can lead to cerebral ischemia (Bad)
  - Normocarbia is probably the best goal except in the short to for impending hernia or poor surgical conditions.
Glycemic Control

- Although hyperglycemia clearly associated with worse outcomes in TBI…
  - Unclear whether this is cause or effect
  - Hyperglycemia does lead to secondary injury
  - Intraoperative data is scant, but hyperglycemia common.
  - Frequent monitoring to target 80-180 mg/dl?
Therapeutic Hypothermia

- **Benefits** –
  - Dec. CMRO₂, Dec. Ext. NT release, Attenuates BBB permeability, good for brain protection

- **Perils** –
  - Hypotension, bradycardia, dysrrhythmias, sepsis, pneumonia, coagulopathy

- **BTF guidelines** – Cautious and Optional (Lvl 3)
  - Best evidence if maintained for 48 hours
Coagulopathy and Coagulotherapy

- Coagulopathy in up to 30% of TBI patients
  - Brain Tissue factor release can lead to thrombin formation and factor depletion
  - Risks – GCS < 8, cerebral edema, SAH, shift
    - Hypothermia, acidosis and hypocalcemia
  - rFVIIa – Cochrane review too small to draw conclusion about utility in TBI
  - CRASH-2 – tranexamic acid in trauma – dec. mortality
Anemia and Transfusion

- Anemia is associated with increased in-hospital mortality and poor outcome in TBI
- Little data to support improved outcome with treatment in the setting of TBI
- Liberal transfusions strategies (<10 g/dl trigger) not recommended
Steroid in TBI

CRASH Trial – steroids w/in 8 hours – higher risk death and disability in TBI
Brain Trauma Foundation 2012?
References