

# Optimization of Patients with Traumatic Brain Injury Before Surgery

LE Hong Chinh. MD

University Medical Center HCMC

# Introduction

## The burden (USA)

- 3.3–5.3 million people affected per year
- Direct cost: \$9.2 billion/year (\$13.1 billion in 2013)
- \$51.2 billion lost to missed work and reduced productivity
- Leading causes: falls, motor-vehicle crashes, assault; males affected ~2× more than females
- Older adults: highest rates of TBI-related hospitalization and death in those  $\geq 75$ ; falls cause 51% of TBI in older adults

# Introduction

## Severity

- Classified by the Glasgow Coma Scale (GCS); severe TBI =  $GCS \leq 8$
- Mortality closely tracks the initial GCS score

## Primary vs. secondary injury

- **Primary injury:** Primary injury occurs at the moment of trauma due to mechanical forces applied to the skull and brain, causing direct neuronal tissue damage.
- **Secondary injury:** begins immediately after the primary injury and evolves over time through neuroinflammation, blood–brain barrier disruption, cerebral edema, increased ICP, ischemia, and cell death; this cascade may be modified to improve outcomes.

# Concept of “high quality” perioperative traumatic brain injury care



# Two scenarios the anesthesiologist will face

## Isolated severe TBI

- Defined by GCS  $\leq 8$
- Highly lethal early: about half of severe TBI patients die within 2 hours of injury

## Severe TBI within polytrauma

- Polytrauma complicates up to 70% of TBI cases
- Polytrauma = AIS  $\geq 3$  injuries in  $\geq 2$  body regions,
- Plus  $\geq 1$  of: SBP  $\leq 90$  mmHg; GCS  $\leq 8$ ; BE  $\leq -6.0$ ; INR  $\geq 1.4$  or PTT  $\geq 40$  s; age  $\geq 70$ .
- 50–60% die within 4 hours (in-scene, ED, or OR); a substantial proportion of survivors die within 24 hours

# Two scenarios the anesthesiologist will face

## **Polytrauma with Severe TBI**

- Limited evidence guides management during emergency extracranial surgery.
- Treatment varies widely among trauma centers.
- Care must balance hemorrhage control with neuroprotection.
- Main priorities: cardiorespiratory optimization, coagulation/transfusion management, prevention of secondary brain injury, and selected use of SMS.

# Hemodynamic Management

## In polytrauma

- Hemorrhage control and massive transfusion are major priorities.
- Permissive hypotension may be used to reduce bleeding, but it is not appropriate when severe TBI is present.

## In polytrauma with TBI

- Hypotension must be avoided to preserve cerebral perfusion and prevent secondary brain injury.
- Recommended targets:
  - SBP  $\geq$  100 mmHg in patients aged 50–69 years
  - SBP  $\geq$  110 mmHg in patients aged 15–49 years or  $>70$  years
  - MAP  $\geq$  80 in severe TBI

# Hemodynamic Management

## Normal saline vs. balanced fluids:

- Isotonic saline is suggested over balanced crystalloids for volume expansion in adult critically ill patients with TBI.
- More hypotonic balanced crystalloids, such as Ringer's lactate or acetate, should probably be avoided.
- Clinical implication: Normal saline is the preferred fluid in TBI to reduce mortality.
- For other acute brain injuries such as SAH or ICH, current evidence is insufficient to prefer normal saline over balanced solutions.

# Hemodynamic Management

## Albumin

- Isotonic saline is suggested over albumin for volume expansion in adult critically ill patients with TBI.
- Clinical implication: Albumin is not preferred for resuscitation or maintenance in brain-injured patients due to limited evidence and possible harm, especially with hypo-osmolar albumin solutions.

## Glucose-containing fluids

- No recommendation.
- Clinical implication: Glucose-containing hypo-osmolar fluids are not advised for resuscitation or maintenance.

# Respiratory Management

- In polytrauma without TBI, prehospital SpO<sub>2</sub> < 94% is associated with higher in-hospital mortality and worse disability.
- In TBI, even a single episode of SpO<sub>2</sub> ≤ 92% increases mortality and disability. Therefore, strict oxygenation control is essential to prevent secondary brain injury.
- However, hyperoxia has been linked to worse outcomes in TBI, possibly due to cerebral/coronary vasoconstriction and oxygen free radical toxicity.
- Current evidence is mixed, but oxygen therapy should aim to avoid both hypoxemia and severe hyperoxia.

# Respiratory Management

- PaCO<sub>2</sub> abnormalities should be avoided in the acute phase of TBI.
- Hypocapnia from hyperventilation can reduce ICP but may cause cerebral vasoconstriction, ischemia, and worse neurological outcomes.
- Hyperventilation may also increase airway pressure, reduce venous return, and worsen hypotension in bleeding trauma patients.
- Hypercapnia can increase cerebral blood volume and exacerbate intracranial hypertension.
- Recommended target in TBI: PaCO<sub>2</sub> 35–40 mmHg. Temporary hypocapnia should be reserved for suspected cerebral herniation while awaiting or during emergency neurosurgery.

# Airway Management in TBI

- Indication for endotracheal intubation and rapid sequence induction: GCS  $\leq$  8.
- Cervical spine injury occurs in about 3.5–6.2% of trauma patients and should always be suspected in TBI.
- Endotracheal intubation and patient repositioning should minimize cervical spine movement.
- Video laryngoscopy with cervical immobilization may reduce upper cervical motion compared with Macintosh laryngoscopy and allows faster intubation than flexible bronchoscopy.
- Awake flexible bronchoscopic intubation minimizes cervical movement but requires expertise and may be difficult in patients with impaired consciousness.

# Airway Management in TBI

- Avoid nasotracheal intubation when skull base fracture is suspected due to risk of intracranial tube passage.

## Tracheostomy in Severe TBI

- Tracheostomy is commonly performed in ICU patients with TBI, often after the first week.
- Early tracheostomy may reduce duration of mechanical ventilation, ICU and hospital length of stay, and ventilator-associated pneumonia.
- BTF guidelines recommend early tracheostomy when expected benefits outweigh the risk of complications.

# Glycemic Control in TBI

- Trauma can cause stress-induced hyperglycemia through increased stress hormones, cytokines, insulin resistance, and altered insulin secretion.
- Stress-induced hyperglycemia is associated with higher morbidity and mortality, but may reflect injury severity rather than direct causation.
- Strict glucose control has not shown clear benefit and may increase harm, including hypoglycemia.
- Tight control to keep glucose <110 mg/dL is generally not required and may be detrimental.
- A moderate strategy aimed at avoiding both hyperglycemia and hypoglycemia appears safer

# Temperature Management in TBI

- Fever is common after severe TBI and may worsen neurological recovery.
- Evidence supports maintaining normothermia in patients with TBI.
- Prophylactic hypothermia has not improved 6-month neurological outcomes and is not recommended by BTF guidelines.
- Hypothermia may reduce refractory intracranial hypertension, but should not be used routinely as a neuroprotective strategy.
- Core body temperature may not accurately reflect brain temperature; direct brain temperature monitoring is more accurate but not routinely available

# Coagulation and Transfusion Management

- In bleeding trauma patients, RBC transfusion is generally recommended Hb is 7–9 g/dL.
- The optimal Hb threshold in polytrauma patients with TBI remains uncertain.
- Most evidence supports a restrictive transfusion strategy; trials show no clear mortality or neurological benefit with liberal transfusion.
- In hemodynamically stable patients, RBC transfusion is recommended when Hb < 7 g/dL.
- In unstable patients or those with cardiovascular disease, transfusion should be individualized.
- Coagulation management should balance hemorrhage control with prevention of secondary brain injury.

# Coagulation and Transfusion Management

## Transfusion Ratios in Massive Bleeding

- Massive transfusion is often required in bleeding polytrauma patients, especially when TBI is present.
- Initial transfusion protocol should use plasma:platelets:RBC = 1:1:1.
- The ratio can later be adjusted based on laboratory values and POC coagulation tests.
- Compared with 1:1:2, the 1:1:1 strategy improves hemostasis and reduces death from exsanguination within 24 hours, although overall mortality is similar.

# Coagulation and Transfusion Management

- Trauma-related coagulopathy increases mortality and may worsen intracranial hematoma progression and neurological outcomes.
- In life-threatening bleeding or emergency neurosurgery:
  - Maintain platelets  $>50,000/\text{mm}^3$
  - Target  $>100,000/\text{mm}^3$  if ongoing bleeding, TBI, or neurosurgery/ICP probe insertion
  - Keep PT and aPTT  $\leq 1.5$  times normal
- Point-of-care tests such as TEG/ROTEM may help guide individualized coagulation therapy.
- TXA should be given within 3 hours in actively bleeding trauma patients; in TBI, may benefit mild–moderate TBI, but has no clear benefit in severe TBI.

# Perioperative Brain Protection

## ICP/CPP Monitoring in Polytrauma with TBI

- Intracranial hypertension and low CPP can worsen secondary brain injury and increase mortality/disability.
- $CPP = MAP - ICP$ ; therefore, both blood pressure and ICP control are critical.
- $ICP > 22$  mmHg is associated with increased mortality.
- In comatose TBI patients with CT signs of intracranial hypertension, invasive ICP monitoring should be considered after life-threatening bleeding is controlled.
- ICP monitoring may help detect and treat intracranial hypertension or low CPP during emergency extracranial surgery.

# Perioperative Brain Protection

## Perioperative ICP/ CPP Management

- Perioperative management in severe TBI focuses on controlling ICP and maintaining adequate CPP.
- Key targets include CPP 60 -70 mmHg and early treatment of intracranial hypertension.
- In suspected cerebral herniation, osmotherapy and temporary hypocapnia may be used while awaiting or during emergency neurosurgery.
- A stepwise approach is recommended: escalate therapy gradually and reserve aggressive interventions for refractory intracranial hypertension.
- Advanced neuromonitoring, such as brain tissue oxygenation, may help personalize therapy in complex cases, but may not be available early and can increase information burden

# Perioperative Brain Protection

## Maintaining CPP by *Reducing ICP*

- Lower ICP to maintain CPP using CSF drainage, if feasible.
- Optimize cerebral venous drainage: head elevation 30° and neutral neck position.
- Prolonged prophylactic hyperventilation with PaCO<sub>2</sub> of 25 mmHg or less is not recommended.
- Use hyperosmolar therapy such as mannitol or hypertonic saline to reduce cerebral edema.
- In stable patients, mannitol 0.25–1 g/kg may be given slowly over 15 minutes to avoid hypovolemia and hypotension.

# Perioperative Brain Protection

## Maintaining CPP by *Reducing ICP*

- Hypertonic saline may improve CPP, but evidence shows no clear superiority over mannitol in mortality or neurological outcomes.
- Monitor serum sodium closely;  $\text{Na}^+ >155$  mmol/L may predict acute kidney injury, and rapid sodium shifts should be avoided.
- Effects of hyperosmolar agents may be altered when the blood–brain barrier is disrupted, as often occurs in TBI.

# Perioperative Brain Protection

## Maintaining CPP by *Increasing Systemic Blood Pressure*

- Increasing MAP should be considered to maintain adequate CPP.
- Blood pressure must be optimized before and during anesthesia, especially in trauma patients with unstable hemodynamics.
- Correct reversible causes of hypotension first: bleeding; anesthetic-induced vasodilation.
- Vasopressors should be considered when needed to maintain MAP and CPP.
- Phenylephrine may increase MAP and CPP more effectively than norepinephrine or dopamine, but optimal vasopressor choice remains uncertain.
- Because cerebral autoregulation is often impaired after TBI, hypotension can directly reduce cerebral blood flow and should be strictly avoided.

# Perioperative Monitoring in TBI Patients

- Standard monitoring should include  $\text{ETCO}_2$ ,  $\text{SpO}_2$ , ECG, invasive arterial blood pressure, urine output, and body temperature.
- In TBI patients undergoing damage control surgery (DCS) or interventional radiology immediately after admission, ICP/ CPP monitoring should be considered if clinically indicated and feasible.
- In patients undergoing emergency neurosurgery, the same standard monitoring is required.
- In TBI patients undergoing urgent extracranial surgery after 24 hours, continue standard monitoring and maintain ICP/ CPP monitoring if already in place.

# Choice of Induction Agents in TBI

- Induction agents should reduce cerebral metabolism and ICP while preserving MAP and CPP.
- Propofol, thiopental, midazolam, and etomidate reduce CMRO<sub>2</sub>, cerebral blood flow, and ICP.
- Propofol and thiopental may cause hypotension and reduce CPP; careful dose titration is required.
- Etomidate preserves MAP and may be preferred in hemodynamically unstable patients, but adrenal suppression remains a concern.
- Ketamine is no longer contraindicated in TBI; current evidence shows no clear harm and suggests possible neuroprotective effects.

# Anesthetic Maintenance in TBI

- No specific maintenance anesthetic has proven superiority in improving TBI outcomes.
- Maintenance can use inhalational or intravenous agents, but must prioritize hemodynamic stability and CPP preservation.
- TIVA with propofol and opioids is useful for cerebral hemodynamic control and neurophysiological monitoring.
- Dexmedetomidine may reduce anesthetic/opioid requirements and postoperative nausea/vomiting, but optimal dosing and comparative effects remain unclear.

# Anesthetic Maintenance in TBI

- Low-dose volatile agents may reduce brain metabolism, but higher concentrations can increase cerebral blood flow and ICP.
- Nitrous oxide should be avoided because it increases cerebral blood flow and ICP.
- If volatile anesthesia is used, MAC < 1 appears appropriate in TBI patients.

# Steroids and Seizure Prophylaxis in Severe TBI

## Steroids in Severe TBI

- Steroids are not recommended to improve outcomes or reduce ICP in TBI.
- High-dose methylprednisolone is associated with increased mortality in severe TBI and is contraindicated.

## Seizure Prophylaxis in Severe TBI

- Prophylactic phenytoin or valproate is not recommended to prevent late post-traumatic seizures.
- Phenytoin is recommended to reduce early post-traumatic seizures, defined as seizures within 7 days after injury, when benefits outweigh treatment-related risks.
- Early post-traumatic seizures have not been clearly associated with worse outcomes.
- Current evidence is insufficient to recommend levetiracetam over phenytoin for preventing early seizures or reducing toxicity.

# Take-Home Messages

- **Goal before surgery:** Prevent secondary brain injury — optimize rapidly and in parallel with resuscitation.
- **Identify the scenario** — isolated severe TBI vs. TBI in polytrauma. In polytrauma with TBI, balance hemorrhage control with neuroprotection; permissive hypotension is not appropriate.
- **Perfusion & oxygenation:** Avoid hypotension (age-based SBP targets, CPP 60–70 mmHg, treat ICP > 22 mmHg); avoid hypoxia and abnormal PaCO<sub>2</sub>; no prophylactic hyperventilation.

# Take-Home Messages

- **Airway:** rapid sequence induction when GCS  $\leq 8$ , with cervical-spine precautions.
- **Coagulation & transfusion:** correct coagulopathy early; massive transfusion 1:1:1; meet platelet/fibrinogen targets; use a restrictive hb threshold.
- **Don't miss the basics:**
  - Normoglycemia and normothermia;
  - Steroids contraindicated;
  - Anesthetic choice matters less than hemodynamic stability and cpp.

**THANK YOU**  
for your attention!

