Traumatic Brain Injury

Traumatic brain injury is the leading cause of death from blunt trauma and a major cause of morbidity.

ANESTHETIC CONSIDERATIONS:

- Considerations of trauma patient
  - Emergency situation
  - Full stomach/aspiration risk
  - Hemorrhagic shock
  - Hypothermia
  - Coexisting injuries (recognized and unrecognized)
  - Coingestions
- Elevated intracranial pressure – address and monitor ICP, Cushing reflex, herniation
- Secondary brain injury
- Systemic response – neurogenic pulmonary edema, myocardial stunning, DI/SIADH, DIC

ANESTHETIC GOALS:

- Assess severity of TBI
- Prevent secondary brain injury (avoid hypotension, hypoxemia, anemia,hyperthermia, acidosis, hyperglycemia)
- Maintain cerebral perfusion pressure
  - Maintain MAP
  - Reduce and prevent increases in ICP

PRIMARY SURVEY

- ABCDE
- Initial investigations – labs, ABG, CXR

GCS scoring system

Score	Eye Opening	Verbal	Motor
1	None	None	None
2	To pain	Incomprehensible sounds	Extensor posturing (decorticate)
3	To voice	Inappropriate speech (words)	Flexor posturing (decerebrate)
4	Spontaneously	Confused speech	Withdraws from pain
5		Oriented to name	Localizes to pain
6			Follows commands

SECONDARY SURVEY

- AMPLE Hx
- Head-to-toe examination
- Further investigations
- Detailed PMHx

DDX DECREASED LOC (COMA)

- Drugs – iatrogenic, intoxication, withdrawal
- Infection – CNS, systemic
- Metabolic – hypoxemia, hypercapnia, hypo/hyperglycemia, hypothermia, uremia, hepatic failure
- Structural
  - CNS – cerebral contusion, intracranial hemorrhage, seizures/postictal
  - CVS – shock (hypovolemic, obstructive, cardiogenic, distributive)

PHYSICAL

- VS – Cushing reflex (HTN, bradycardia
- CNS – GCS, pupils, neuro deficits
- AW – standard exam, confirm ETT position if already intubated
- CVS – intravascular volume status, associated trauma
- RESP – hypoxemia, pulmonary edema, PTX and associated trauma
- GI – abdominal trauma
- GU – associated trauma

INVESTIGATIONS

Labs

- CBC, lytes, urea, Cr, liver enzymes, glucose, INR, PTT, x-match
- Posm, toxicology screen, +/- BHCG
- ABG, lactate, +/- cardiac enzymes

Imaging

- CXR, EKG
- CT head
  - Midline shift, distortion of ventricles and cisterns, effacement of sulci
  - Hematoma – EDH, SDH, SAH, ICH, IVH
  - Depressed skull #s, intracranial air
- C-spine xrays
  - Normal lateral view not necessarily reassuring (lateral view misses 15-26% of c-spine #s)
- CT/MRI neck
  - CT neck + 3 x-ray views of c-spine has false negative rate of <0.1% for serious c-spine injury
- +/- X-ray pelvis/long bones, FAST

OPTIMIZATION

Optimize cerebral oxygenation and perfusion

- Airway management
  - Adequate systemic oxygenation is essential
    - Maintain SpO2 >90% and PaO2 >60mmHg (ideally >95)
  - Conflicts in airway management with TBI
    - A – possible difficult airway (blood, injury)

- B – hypoxemia, aspiration risk (full stomach)
- C – hypovolemia
- D – ↑ICP, possible skull-base #, uncertain c-spine status, uncooperative/combative

- Ventilation strategy
  - PEEP
    - Isolated head injury – no or low-level PEEP to prevent ↑ICP
    - If ALI (chest trauma, aspiration, etc) use adequate PEEP to correct hypoxemia
  - PaCO2 targets
    - Maintain PaCO2 30-35mmHg routinely
      - Hyperventilation/vasoconstriction can result in ischemia esp w/ low baseline CBF (ie: first 48-72hrs post-TBI)
    - Transient hyperventilation to PaCO2 25-30 in the following settings:
      - Refractory ICP >20mmHg
      - Impending herniation
      - Facilitating surgical access/minimizing retractor pressure
- Circulatory management
  - Avoid hypotension (SBP <90mmHg)
    - Target MAP >70-80mmHg until ICP monitoring instituted and CPP can be targeted directly
    - Target CPP 50-70mmHg
      - Aggressive attempts to maintain CPP >70mmHg with fluids/vasopressors ↑s risk ARDS
    - Discuss BP targets with NSx
  - Fluids to maintain euvolemia
    - Fluid resuscitation until euvolemic then use vasoactive infusions to maintain target MAP
    - Maintain Posm 290-320mOsm/kg (avoid hypoosmolar fluids; RL, dextrose)
    - Consider hypertonic saline (may be optimal?), synthetic colloids, blood products
      - Albumin = worse outcome
  - Correct anemia from blood loss – target Hct >30%

ICP management
- Positioning – HOB 30°, neck midline
- Hyperosmolar therapy
  - 25% Mannitol (0.25-1g/kg q4-6h)
    - Rheologic + osmotic effects
    - Addition of furosemide 0.25-0.5mg/kg iv may ↑duration of mannitol effect on ICP
    - Risks – hypovolemia, hyponatremia, renal failure, rebound edema
  - Hypertonic saline (3% saline 6-8mL/kg or 7.5% saline 4mL/kg at 20-40mL/hr)
    - Osmotic and microcirculatory effects
    - Less electrolyte disturbance and less diuresis compared to mannitol
    - Risks – hypernatremia, metabolic acidosis, pulmonary edema
- Sedation and analgesia
- Post-traumatic seizure prophylaxis (phenytoin preferred)
  - Indicated to ↓incidence of early PTS (w/in 7d, although not associated with worse outcomes), not indicated for prevention of late PTS (beyond 7d)
- Neuromuscular blockade/paralysis
- Barbiturate coma
  - Prophylactic barbiturates to produce burst suppression *not* recommended
    - Hemodynamic instability outweighs beneficial effect on ICP
  - Indicated for controlling ↑ICP refractory to maximum standard medical and surgical Tx
    - Pentobarbital 10mg/kg over 30min, then 5mg/kg q1h x 3, then 1 mg/kg/hr for burst suppression
    - HD stability essential before and during therapy; D/C if unable to maintain MAP
- Temperature management
  - Avoid hyperthermia
  - Hypothermia may be associated with improved neurologic outcomes and survival
    - If normothermic, inducing hypothermia of 33°C does not improve outcome
    - If hypothermic on admission, active rewarming worsens outcome – do not rewarm
- Hyperventilation
  - Prophylactic hyperventilation *not* recommended
  - Hyperventilation may be employed as a *temporizing* measure to reduce elevated ICP
  - Avoid hyperventilation during first 24h after injury, when CBF often critically reduced
  - If hyperventilation employed, monitor cerebral O2 delivery
- Neurosurgical intervention
  - Intraventricular CSF drain insertion, craniotomy and evacuation hematoma, decompressive craniectomy
- Decompressive laparotomy
  - Consider in severe TBI if IAP >20mmHg (eg: 2° to coexisting injuries or vigorous resuscitation)

Identify and treat other life-threatening injuries – prioritize

ANESTHETIC OPTIONS

- **Depends on surgical procedure**
  - Emergency surgery (Neurosurgical vs non-neurosurgical) vs Nonemergency surgery
- **None**
  - Delay 'specialty x' surgical intervention while addressing more life-threatening injuries
    - Most immediately life-threatening injury takes priority
    - For emergent non-neurosurgical procedures, consult NSx intraop for ICP monitoring
  - Delay non-emergent surgery in setting of refractory ↑ICP or very labile ICP
  - Brain death → discuss organ donation
- **Local/Regional**
  - Local infiltration for head pins
  - Local/regional techniques alone may be considered for non-neurosurgical procedures
- **General**
  - Required for emergency Neurosurgery procedures

ANESTHETIC SETUP

Priority is to open cranium

Drugs

- Standard emergency drugs
- Mannitol and/or hypertonic saline
- Phenytoin

Monitors

- Standard CAS monitors including temperature probe + 5 lead EKG
- Arterial line
- +/- CVL (access, vasoactive infusions, aspiration of air if VAE)
- Consider ICP monitoring if:
  - Surgery w/in 48h of severe TBI
  - Severe TBI + abnormal head CT
  - Severe TBI + risk factors (age >40, motor posturing, SBP <90 mmHg)
  - Prolonged surgery (neuro status cannot be monitored under GA)
- Consider advanced neurologic monitoring
  - TCD, jugular bulb oximetry, brain tissue oxygenation monitor

Equipment

- Large bore iv x 2
- Blood products, fluid warmer
- Difficult airway cart

MANAGEMENT OF ANESTHESIA

Induction

- Airway
  - Depending on airway assessment, HD stability, cooperation
    - RSI with MILS
    - Awake fiberoptic
    - Intubation with spontaneous ventilation
    - Surgical airway
  - Avoid nasal intubation if basal skull # suspected
- Thiopental, propofol, etomidate, and midazolam cause cerebral v/c, ↓CMRO2 (→↓ICP), and maintain CO2 responsiveness
  - Any of these agents a reasonable choice as long as hemodynamic stability (MAP) maintained
    - Risk of profound hypoT w/ unrecognized hypovolemia (see Px above)
  - Use of opioid allows lower dose of induction agent → less reduction in MAP
    - High dose/rapid bolus fentanyl or sufentanil may ↑ICP and ↓MAP in severe TBI
- Lidocaine – ↓CMRO2 similar to barbiturates although less effect on MAP (dose 1.5mg/kg)
- Ketamine – may not ↑CMRO2 or CBF when coadministered w/ other induction agents and ventilation controlled
- Succinylcholine – ↑ICP (blunted by induction agents), although less than produced by coughing; not contraindicated as provides most rapid and most optimal intubating conditions
- Importance of deep anesthesia and adequate muscle relaxation prior to airway manipulation and surgical stimulation

Maintenance

- Traumatized brain has lower anesthetic requirements
  - Maintain adequate anesthesia w/o compromising HDs
- All inhaled anesthetics (including N2O) are cerebral vasodilators (↑CBF → ↑ICP), and reduce cerebral autoregulation, CO2 responsiveness and CMRO2
  - Concentrations <1 MAC often consistent with acceptable ICP and surgical conditions
    - Discontinue if ICP uncontrolled or surgical field is 'tight'
  - Avoid N2O in setting of possible intracranial air (missile injury, depressed skull #)
- Maintain muscle relaxation
- Moderate hyperventilation until dura opened as ↑ICP likely more detrimental than short-term hyperventilation
- Maintain HD stability
  - Risk of profound hypoT following craniectomy, when intrinsic stimulus for ↑BP diminishes
  - To avoid postdecompressive hypoT – volume loading in early stages of anesthetic, especially if other injuries and significant blood loss
- Management of 'tight brain' intraop
  - Correct hypercapnia, hypoxemia, HTN and venous obstruction
  - Head up and neutral neck position, increase MAP, administer mannitol, convert to TIVA

Emergence

- Depending on situation
  - Rapid smooth emergence for neuro assessment
  - Transfer to ICU intubated with sedation

DISPOSITION & MONITORING

- ICU for observation and ongoing management
- DVT prophylaxis – intermittent pneumatic compression stockings, heparin should be safe after 48hrs?

COMPLICATIONS

Early complications

- Intracranial HTN, brain herniation, seizures, neurogenic pulmonary edema, myocardial dysfunction, arrhythmias, bradycardia, HTN, coagulopathy/DIC

Late complications

- Seizures, stress ulcers, coma

OBSTETRICS

- Head injury is a leading cause of maternal death 2° to trauma
- CT is investigation of choice in setting of head injury and should be performed within 1hr of hospital admission
- Aggressive management of hypotension and hypoxia to ensure perfusion to brain, other vital organs, and placenta
- Hyperventilation a disadvantage for fetus (↓UBF from ↓maternal C.O. and BP and uteroplacental v/c)
  - Maintain maternal PaCO2 within N range for pregnancy and use lowest possible airway pressures to minimize risk of hypotension and barotrauma
- Mannitol and furosemide can cross placenta; may ↓fetal plasma osmolality and intravascular volume
  - However, concern for fetal effects overridden by acute maternal concerns

PATHOPHYSIOLOGY

Epidemiology
- TBI accounts for 40% of trauma-related deaths and is leading cause of death from blunt trauma
- Hypotension is most important cause of death in TBI
- Predictors of poor outcome in TBI – age >55, poor pupil reactivity, low post-resuscitation GCS, hypoT, hypoxia, unfavorable intracranial Dx on CT, early hyperglycemia >11.1 mmol/L

Primary injury
- Shear forces cause primary damage to neuronal cell bodies and axons, and to the vasculature

Secondary injury
- Only part of the damage to brain during TBI occurs at moment of impact
- Numerous secondary insults compound the initial damage in the ensuing hours and days
  - Hypoxemia and hypotension → ↓O2 delivery → lactic acidosis, free radicals, PGs, excitatory amino acids, lipid peroxidation and cell membrane breakdown, entry of Na/Ca/H2O into cells, leakage of fluid from cells into extracellular space → brain edema, regional and global disturbances in cerebral circulation
  - Metabolic failure, oxidative stress, biochemical events → delayed necrotic/apoptotic cell death
  - Exacerbated by tissue hypoxia/ischemia and inflammatory responses
- The area of brain around the site of direct injury may have ↓ed CBF and defective autoregulation
- Protocols emphasizing early intubation, rapid transport to trauma care facility, prompt resuscitation, early CT scanning, and immediate evacuation of intracranial mass lesions, followed by ICU management (including ICP monitoring) have reduced mortality and morbidity following TBI

Monro-Kellie doctrine
- Vic = Vbr + Vcsf + Vbl
  - Vic ~ 1500cc (brain 85-90%, blood 10%, CSF < 3%)
- N ICP < 10 mmHg
- CPP = MAP – ICP or CVP (whichever greater)
- Brain normally receives 15% of CO
- CBF autoregulation (normotensive, uninjured brain) 60-150 mmHg
  - Autoregulation impaired in TBI
- Flow-metabolism coupling leads to regional differences in CBF

Signs/Sx of ↑ICP
- HA, N/V, changes in vision, neuro deficits, ↓LOC, fixed/dilated pupil(s)
- Cushing reflex (HTN, bradycardia, irregular resps)

Classification of TBI

GCS*	Severity of TBI	Investigations	Management	Outcomes
13-15	Mild	Routine	Avoid hypoxia Fluid resuscitation Manage associated injuries Consider D/C home after several hrs^	3% decompensate unexpectedly (unlikely to deteriorate after 24hrs) At risk for HA, amnesia, emotional lability, sleep disturbance
9-12	Moderate	Early CT head	Intubation for CT head if agitated and requires sedation (risk respiratory depression/aspiration) Stabilize and admit to ICU Serial neuro assessment q2h (GCS, motor, sensory function)# Supportive management as for mild TBI	Low mortality rates Significant long-term morbidity
3-8	Severe	Immediate CT head	CPP and ICP management Early surgical intervention as indicated	High risk of mortality

At time of admission

^ If aSx, alert, awake – may observe for several hrs then safely D/C in care of responsible companion for 24h, with instructions ot return if HA, emesis, LOC, focal neuro deficits

Invasive ICP monitoring indicated if neuro assessment not possible (GA >2h, need for aggressive analgesia, delirium tremens prophylaxis)

Types of injury
- Blunt vs penetrating
- Skull # (depressed, basal, other)
- Contusion
- Epidural hematoma (tear in middle meningeal artery, often associated skull #)
  - Sudden initial LOC followed by lucid interval, then sudden deterioration a few hrs later (hemiparesis, mydriasis, bradycardia reflect uncal herniation and BS compression)
- Subdural hematoma (laceration/tear of cortical bridging veins)
  - Slow expansion with gradual progression of Sx (HA, confusion, drowsiness, eventual lateralizing neuro signs (hemiparesis, hemianopsia, language disturbances)), common in elderly, CSF clear.
  - Tx – burr hole drainage or craniotomy if large clots
- Subarachnoid hemorrhage (cortical vessels or aneurysm rupture)
  - Evolve over time, xanthochromia
- Intracerebral hemorrhage
  - Often acutely increase in size, difficult to Tx, managed conservatively unless size or rate of growth likely to cause herniation
- Intraventricular hemorrhage

General principles
- Early rapid management focused on restoring systemic homeostasis and perfusion-directed care of the injured brain
  - Common goal of Tx of TBI is to maintain cerebral perfusion
- Therapy following severe TBI is directed toward preventing 2° brain injury
- Coexisting injuries
  - C-spine # in 2% of blunt trauma victims and 8-10% of TBI victims with GCS <8
  - Aspiration, pulmonary contusion, ARDS → hypoxemia → worsen 2° brain injury
- Corticosteroids *contraindicated* (worsened outcome with methylprednisolone)

REFERENCES: Anesthesia and Coexisting Disease p.223-225; Miller p.2068-2072, 2295-2299, 2908-2910, Barash p.898-901, 916-917, 1022-1025,1447-1449, Brain Trauma Foundation. Guidelines for the management of severe traumatic brain injury. J Neurotrauma 2007;24:S1-106, Chestnut p.1155-1156, ATLS manual (7th ed)