(Pediatric) Intracranial Hemorrhage

The pathophysiology of pediatric intracranial hemorrhage is typically related to trauma (including birth trauma), abuse/inflicted injury or intraventricular hemorrhage related to prematurity. Anesthetic management goals for patients with intracranial hemorrhage include avoidance of iatrogenic increases in ICP, pharmacologic and physiologic manipulation of ICP, maintenance of cerebral perfusion pressure and avoidance of secondary injury to ischemic penumbra.

ANESTHETIC CONSIDERATIONS:

- Increased ICP
  - Avoid increased ICP (coughing/straining/gagging) upon securing the airway
    - Consider lidocaine, opioids, NMBAs to decrease cough
  - Maintain CPP (children: 50-60mmHg – not evidence based; adults 60-70mmHg)
  - ICP monitoring if indicated
  - Maintain normocapnia
  - Avoid hypotension, hypoxemia, acidosis
  - Allow for adequate upper extremity/cerebral venous return (avoid venous HTN)
    - HOB >30 degrees if possible
    - If C spine collar ensure not too tight
    - Pneumothorax
  - Avoid hyperthermia, hyperglycemia/hypoglycemia
  - Avoid shivering and treat with NMBAs
  - Caution with NaHCO3/3% saline/mannitol as increased osmolar load and fluid shifting may tear bridging subdural veins and cause SDH; use in refractory intracranial hypertension cases only
  - Caution with IV solutions with excess free H20
- Decreased LOC and aspiration risk → RSI
- Avoid anticoagulation and extension of hemorrhage
- Investigation re: child abuse or inflicted injury
- Investigation re: concomitant injury

ANESTHETIC GOALS:

- Avoid secondary injury to penumbra (hypoxia, hypotension, hyperthermia, hyper/hypoglycemia, seizures)
- Avoid extension of hemorrhage
- Avoid increases in ICP (coughing, bucking, straining)
- Decrease brain metabolic/O2 requirements
- Seizure prophylaxis in high risk patients: pediatric traumatic brain injury, mass lesions, depressed skull fractures

HISTORY

- AMPLE history
- Onset of injury
- Presence of neurologic deficits
- Developmental history, baseline neurological function for age and evaluation of developmental delay
- Pregnancy and delivery history (gestational age, birth weight, apgars, mode of delivery, perinatal complications, postnatal complications)
- Social history

PHYSICAL

- **ABCS – SECURE AIRWAY IF GCS <8**

PEDIATRIC GCS FOR CHILDREN <2YO (ACAD EMERG MED 2005)

EYE OPENING	SCORE
▪ SPONTANEOUS	4
▪ TO VERBAL COMMAND	3
▪ TO PAIN	2
▪ NONE	1
VERBAL RESPONSE	
▪ SMILES, COOS, BABBLES, ORIENTED TO SOUND	5
▪ CRIES, IRRITABLE	4
▪ CRIES IN RESPONSE TO PAIN	3
▪ MOANS IN RESPONSE TO PAIN	2
▪ NO SOUNDS	1
MOTOR RESPONSE	
▪ SPONTANOUES/OBEYS VERBAL COMMANDS	6
▪ WITHDRAWS TO TOUCH	5
▪ WITHDRAWS TO PAIN	4
▪ ABNORMAL FLEXION	3
▪ ABNORMAL EXTENSION	2
▪ NO MOTOR RESPONSE	1

- Focused neurological exam (detail any neurologic deficits – eye deviation, pupil reactivity, posturing, seizures, level of consciousness, symmetric/asymmetric movement of extremities)
- look for any associated/concomitant injuries – fractures, rib fractures, bruises, **retinal hemorrhage**

INVESTIGATIONS

- Type and Screen, Cross Match
- CBC, lytes, BUN, Cr, INR, PTT, glucose
- Sosm

- CT Head if applicable
- Fundoscopy

OPTIMIZATION

- Optimize CPP
  - May require vasopressors/inotropic support
  - Optimize preload
  - Minimize cerebral venous pressure
- Minimize ICP
  - Minimize cerebral venous pressure

ANESTHETIC OPTIONS

- General anesthetic with airway protection

ANESTHETIC SETUP

- Standard CAS monitors
- Temperature probe
- Foley catheter
- Peripheral nerve stimulator
- Glucometer or ABG with glucose measuring capabilities
- Possible intraoperative neurophysiologic monitors – EEG, evoked potentials
- Standard emergency drugs
  - May consider having norpepinephrine, dopamine available
  - May consider having anti hypertensive medications readily available
  - Consider ready availability of mannitol and hypertonic saline
- +/- ICP monitor
- +/- arterial line for invasive BP monitoring and titration of CPP
- +/- central line for vasopressors/inotropes

MANAGEMENT OF ANESTHESIA

- **Induction:** use anesthetics that ↓CMRO2 and ↓CBF (ie not ketamine)
  - **In presence of ↑ICP in TBI conflict between deep RSI & maintenance of CPP**
  - Options = awake direct laryngoscopy, awake fibreoptic intubation, RSI, surgical airway
- **Maintenance:** use TIVA technique if tight brain; no nitrous, consider Sevo (less affect on autoreg)
  - During surgical evacuation of acute subdural or epidural hematoma, BP may drop at time of decompression, requiring aggressive fluid resuscitation
- **Emergence:** controlled, minimize coughing (↑↑ICP) with lidocaine/remifentanil
  - Incremental doses of antihypertensive agents prior to waking prevents rebound hypertension
  - Extubate if patient meets criteria: adequate ventilation, protective airway reflexes, awake

DISPOSITION & MONITORING

- Consider (if indicated):
  - neuro observation unit
  - PICU/NICU

COMPLICATIONS

- **Seizure** – consider anticonvulsant prophylaxis for high risk patients, tx with benzos
- **Impending herniation**: ICP Management Pathway (from Brain Trauma Foundation Guidelines)
  - Temperature control, seizure prophylaxis, HOB up 30 degrees, avoid venous outflow obstruction, sedation +/- paralysis, maintain arterial oxygenation, volume resuscitation to CPP 60 mmHg
  - If ventricular drain in place, CSF drainage should be first line treatment for increase ICP
  - Ventilation can be adjusted to maintain $PaCO_2$ low normal (35 mmHg).
  - For sustained ICP despite above manoeuvres:
    - Increase ventilation to $PaCO_2$ 30-35 mmHg
    - Consider measuring CBF, JVO_2 saturations when doing this
    - If mild hypocapnia unsuccessful at lowering ICP, consider mannitol
      - Limiting factor: serum osmolarity < 320 mOsm/L
      - Maintain euvolemia throughout
    - If above unsuccessful, consider:
      - proven benefits but significant complication rates: barbiturate coma (endpoint EEG burst suppression pattern)
      - unproven beneficial treatments: hyperventilation to $PaCO_2$ < 30 mmHg, decompression craniectomy, hypertensive therapy

PATHOPHYSIOLOGY

TRAUMATIC BRAIN INJURY

- **Injury is the leading cause of death of children and adolescents in most developed countries, and 40% of these fatalities are related to brain injury**
  - 10% of brain injured patients die pre-hospital; 80% are "mild", 10% "moderate", 10% "severe"
  - Mild: GCS 13-15, 3% will deteriorate unexpectedly; CT scan if GCS <15, loss of consciousness >5 min, amnesia, severe headaches, or focal neurologic deficit (attributable to brain)
  - Moderate: GCS 9-12, 10-20% deteriorate—all require admission to high acuity unit with neurologic reassessment for 12-24hrs
  - Severe: GCS 3-8, high risk or significant morbidity/mortality
- **Most common etiology (by age group):**
  - Adolescents: MVAs
  - Children: falls
  - Infants: inflicted injury (shaken baby)
- Overall mortality in children (requiring hospitalization and emergency room visits for TBI) is 4.5% (vs 10.4% in adults)

- **Independent risk factors for poor outcome:**
  - age <4yo
  - low GCS at presentation
  - hypotension
  - hyperglycemia
  - coagulopathy
- **Avoidance of hypotension and hypoxia—secondary brain injury—are the cornerstone to TBI management**
  - One episode of SBP <90mmHg is associated with a 50% ↑ in mortality
  - Phenylephrine does not constrict cerebral blood vessels (Barash 1270); norepinephrine better than dopamine (Miller 2071)
  - Patients with impaired autoregulation, low baseline flows and ↑ICP likely benefit from ↑ CPP—discrimination can be based upon CBF measurement, TCD, Sjvo2, brain tissue Po2
    - CPP target 45 mmHg in children
    - Sjvo2: Normal is 55-75%; <50% for 5 min considered desaturation
    - Pbo2: Normal is ≥ 20mmHg, ≤10mmHg conveys substantial risk hypoxic injury
  - Hypothermia: No overall benefit in multicenter trial; subset <45yr presenting <35°C had an improved outcome (? Study not cooling patients early enough)
  - ICP monitors: insert with neurosurgical consultation
    - If compressed basal cisterns, midline shift, effaced ventricles, contusion or subdural
    - Intended aortic occlusion—potential for dramatic ↑ICP; unless intent for heparinization
    - Consider nature & duration of procedure (long, ++↑↓ pressure/volume)
- **Elevated ICP**
  - Assess CT scan for intracranial hematoma, contusions, and shift of midline
    - A shift of >5mm usually indicates need for surgical decompression
  - It is preferable to keep patients at a PaCO2 of 35mmHg; however in Miller p2071, in discussion with the neurosurgeon, the anesthetist may hyperventilate to:
    - Maintain ICP <20mmHg
    - Prevent or reverse herniation
    - Minimizing retractor pressure
    - Facilitate surgical access
  - Mannitol 20% 1g/kg—if acute herniation over 5min; caution re hemodynamics if hypotensive
  - Furosemide 0.3-0.5mg/kg
  - If intractable ↑ICP, pentobarbital 3-10mg/kg over 0.5-2.5hrs; maintenance 0.5-3mg/kg/hr
  - Monitor for hyperglycemia (harmful) and Na levels (risk of SIADH)
  - ±Phenytoin load 1g IV at rate <50mg/min, then 100mg q8h (post-traumatic epilepsy in 5-15%)

REFERENCES

- UpToDate – approach to pediatric traumatic brain injury
- Miller 7th Edition – neurosurgical anesthesia, neurophysiology chapters
- www.braintrauma.org/guidelines/
- Brain Trauma Foundation Guidelines "Update Notice – CPP" and "Management and Prognosis of Severe Traumatic Brain Injury"