Common issues in the Pediatric PARU- Perspectives of a Pediatric Anesthesiologist

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Anesthesia may be conceptually considered as consisting of separate phases of induction, maintenance and emergence.

Emergence is commonly welcomed as the end of the vigil, characterized by the elimination of confounding and potentially deleterious physiological and pharmacological influences---an anesthetic “Miller time”.

Is routine emergence from anesthesia routine---and uncomplicated?

It seems reasonable to conclude that better use of even our current monitoring capacity affords us the opportunity to minimize risk from emergence effects, which, unwitnessed and unappreciated, might lead to patient harm.

Philip W Lebowitz, MD
Recovery from Anesthesia - Overview

- Residual effects of anesthetic agents
  - Respiratory issues
  - Circulatory issues
- Pain
- PONV
- Emergence delirium
- Malignant Hyperthermia
- Cardiac Arrest
- Slow awakening
Residual ventilatory depressant effects of anesthetics

- Depressed hypoxic ventilatory responses
- Depressed hypercarbic ventilatory responses
- Depressed airway reflexes-
  - MAC awake = .5 MAC
  - OSA
  - Laryngospasm
- Return of sympathetic reflexes- “Hemodynamic vulnerability”
  - Shivering
  - Hypertension
  - Tachycardia
  - Increased O2 consumption
- Impaired circulatory reflex responses?
- Impaired thermoregulatory function
Common respiratory problems

- Upper airway obstruction
  - Residual anesthesia
  - Timing of extubation - awake or deep?
    - MAC awake
    - Laryngospasm-post-obstructive pulmonary edema
    - OSA
- Positioning
- Available equipment
- Airway management skills
## Pathophysiologic approach to hypoxia

<table>
<thead>
<tr>
<th>Condition</th>
<th>PaCO2</th>
<th>A-aDO2</th>
<th>100% O2</th>
</tr>
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<tbody>
<tr>
<td>Hypoventilation</td>
<td>Inc</td>
<td>N1</td>
<td>Inc</td>
</tr>
<tr>
<td>Low FIO2</td>
<td>N1</td>
<td>N1</td>
<td>Inc</td>
</tr>
<tr>
<td>Shunt (V/Q = 0)</td>
<td>N1</td>
<td>Inc</td>
<td>Nr</td>
</tr>
<tr>
<td>V/Q mismatch</td>
<td>N1</td>
<td>Inc</td>
<td>Inc</td>
</tr>
<tr>
<td>Diffusion Barrier</td>
<td>N1</td>
<td>Inc</td>
<td>Inc</td>
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Causes of hypoxemia- intubated patient

- **DOPE**
  - Displaced endotracheal tube- mainstem or out
  - Obstructed endotracheal tube
  - Pneumothorax
  - Esophageal intubation
Causes of hypoxia in extubated patients

- Airway obstruction
- Hypoventilation
  - Pain and splinting
  - Excessive sedation-residual anesthesia
- Inadequate FiO2
- Secretions
- Pulmonary edema- ?post obstructive
- Pulmonary embolism
- Pneumonia- Aspiration?
- Pneumothorax
- Bronchospasm
- Atelectasis
- Chronic lung disease
- Low cardiac output
Postoperative Hypoxia
Treatment of hypoxia

- Assure adequate gas exchange
  - Supplemental Oxygen
  - Intubate if necessary
  - Consider PEEP in intubated patients
  - Consider CPAP mask in OSA patients
- Patient positioning
- Further evaluation as indicated
Opioid and Sedative Synergism

“Supplemental oxygen... merely postpones the patient’s insidious progress from bradypnea to apnea.”

“...What it does do is mask that natural opiate-induced progression from bradypnea to apnea, by failing to allow the patient to become hypoxemic, which would otherwise cause a pulse oximeter alarm, thereby alerting clinicians to the respiratory danger......

“...breathing is the only thing that counts.”
ALVEOLAR GAS EQUATION

- $\text{PAO}_2 = \text{FiO}_2 \times (\text{PB} - \text{PH}_2\text{O}) - \text{PaCO}_2/R$
- With a PaCO2 of 100 breathing room air
  - $\text{PAO}_2 = 0.21 \times 740 - (100/0.8) = 30$
- With a PaCO2 of 100 breathing 30 % blow by
  - $\text{PAO}_2 = 0.30 \times 740 - (100/0.8) = 97$
- When in doubt get a blood gas!!!!
Major factors contributing to emergence hemodynamics

- **Pre-op medical conditions**
  - CV disease- CAD, CHF, valvular ht disease
  - HTN
  - Pulmonary disease
  - Other end organ disease

- **Surgical issues**
  - Nature of procedure
  - Location and length of incision
  - Duration of procedure
  - Surgery and extubation needs – neuro

- **Anesthetic technique**
Factors causing impaired hemodynamics

- Alterations in blood pressure
- Hypoxemia/Hypercarbia
- Altered temperature states
- Intervascular volume changes (hyper or hypovolemia)
- Residual drug effects and reactions
- Influence of surgical procedure
Hemodynamics 101  The Immature Heart and Anesthesia -
Baum et al Anesthesiology 87:1528-1548, 1997

Stroke volume

Cardiac output

Blood pressure*

Heart rate*

Systemic vascular resistance

- Myocardial contractility
- Preload
- Afterload

Fig 2. Hemodynamic relations.

*Measurable. Box indicates variables manipulated by therapy.
Signs Of Failing Circulation

- Poor perfusion - capillary refill
- Weak pulses
- End Organ effects
  - Mentation
  - Urine output
  - ECG changes
- Failing pulse oximeter
- Fall in end-tidal CO2
- ABG - lactic acidosis, base deficit
- Low SVO2 - < 50% (venous blood gas analysis)
- Blood pressure - caution!
Hemodynamic Response to Shock - don’t chase the blood pressure. \[ BP = CO \times SVR \]
Alpha adrenergic agonists

- Clonidine
- Dexmedetomidine - PRECEDEX
  - Selective alpha2-adrenoceptor agonist
  - Receptor affinity eight times that of the prototype alpha agonist clonidine
- Hypnotic and anxiolytic properties are attributed to the binding of alpha 2 adrenoreceptors within the locus ceruleus
- Analgesic properties of the drug stem from stimulation of alpha 2 adrenoreceptors in the brain, spinal cord, and peripheral sites
inhibits norepinephrine release
inhibits noradrenergic activity
Dexmedetomidine

- Decreases norepinephrine levels
- Reduces brain noradrenergic activity
- Produces sedation
- Inhibits sympathetic activity
- Decreases blood pressure and heart rate (opposite effect with loading)
- Reduces the need for morphine
Dexmedetomidine

- Occasional bradycardia or hypotension usually in the face of hypovolemic shock
- Only case report in pediatrics: post-op AV canal developed bradycardia
- More caution if < 6 mo
- Occasional failure when used as monotherapy
Dexmedetomidine

- Distribution t1/2 ~ 6 min
- Elimination t1/2 ~ 2 hrs
- Load 1 mcg/kg over 10 min
- CI at 0.2-0.7 mcg/kg/hr
- Need higher dose 0.3 up to 1.5 mcg/kg/hr in peds
- Not recommended for administration > 24 hrs...multiple case reports document giving longer...no data on pharmacokinetics
- Metabolized in liver phase 1 and 2, eliminated by kidneys
Strategies for Post-op Pain Control

“Avoid intense single modality therapy in treating acute pain”
"Gate Theory"
Mechanisms of spinal analgesia
Analgesic Options

- Narcotics
- Non-narcotic analgesics
- Local anesthetic infiltration
- Peripheral nerve blocks
- Major neuraxial conduction blocks
- Epidural and spinal opiates
- Other drug to reduce anesthetic requirements
  - Alpha agonists - precedex
  - Low dose ketamine
PONV

- 20-30% of patients undergoing GA have PONV
- Leading cause of unexpected hospital admission after planned ambulatory surgery
PONV risk factors

- **Patient Specific**
  - Female gender
  - Non-smoking status
  - History of PONV

- **Anesthesia specific**
  - Post op Opioids
  - Volatile anesthetics (TIVA better)
  - Nitrous Oxide
Cortex
- benzodiazepines
- cannabinoids

Visceral Afferents
- metoclopramide (high dose)
- serotonin antagonists

Chemoreceptor Trigger Zone
- butyrophenones
- metoclopramide
- phenothiazines
- serotonin antagonists

Vomiting Center
- anticholinergics
- antihistamines
PONV prevention/treatment options

- Premedicate and reduce anxiety
- Prehydrate/superhydrate
- Modify anesthetic (regional, propofol, opioid sparing analgesics)
- Combination antiemetics (prophylactically and intraoperatively)
PONV treatment

- Scopolamine patch (anticholinergic side effects)
- Antihistamines (diphenhydramine)
- Bemzamides (metochlopramide)
- Buterophynone (droperidol-black box warning!!!)
- Phenothiazines (promethazine)
- Serotonin antagonists (ondansetron, granisteron, dolasteron)
- Steroids- dexamethasone
- Substance P receptor antagonists (NK1-neurokinin)-Aprepitant
- Chinese acupuncture point P6- ReliefBand
What is Malignant Hyperthermia?

Malignant hyperthermia is an inherited disorder of skeletal muscle triggered in susceptibles (human or animal) in most instances by inhalation agents, and/or succinylcholine resulting in hypermetabolism, skeletal muscle damage, hyperthermia and death if untreated.
Signs of Malignant Hyperthermia

- **Specific**
  - Increased CO2 Production - end tidal CO2 - early sign
  - Muscle Rigidity – trunk or limb
  - Rhabdomyolysis - myoglobinuria - Cola colored urine
  - Marked Temperature Elevation (>38.8 ° or an increase of 1-2 degrees every 5 minutes)
  - Increased resting CPK

- **Non Specific**
  - Tachycardia
  - Hypertension
  - Tachypnea
  - Skin mottling
  - Acidosis (Resp/Metabolic)
  - Hyperkalemia
  - Fever - late sign
  - DIC
What to do if you suspect an MH crisis?
Get help, Get Dantrolene

- Notify surgeon or anesthesiologist
- Discontinue volatile anesthetics and succinylcholine
- Hyperventilate with 100% oxygen
- Halt procedure as soon as possible
- Dantrolene 2.5 mg/kg- max 30 mg/kg
- Bicarbonate
- Cool the patient
- Dysrhythmias
- Hyperkalemia
- Frequent ABG’s and other appropriate lab work (electrolytes –potassium, CPK > 20,000 U/L in less than 24 hrs, urine for myoglobin)
Mnemonic for the treatment of MH

- **Some**: STOP all triggering agents go to 100% O2
- **Hot**: HYPERVERTILATE
- **Dude**: DANTROLENE: 2.5 mg/kg immediately
- **Better**: BICARBONATE, sodium, 1 mEq/kg to start
- **Give**: GLUCOSE, 0.5 g/kg; INSULIN, 0.15 U/kg
- **Iced**: IV fluids, cooling blanket
- **Fluids**: Fluid output: FUROSEMIDE, mannitol prn
- **FAST**: TACHYCARDIA: be prepared to treat V-tach.
- For consultation to help with patient management, call the **MH Hotline**: 1-800-MH-HYPER (1-800-644-9737) or 1-315-464-7079 if outside the U.S.
- Report patients who have had acute MH episodes to the **North American MH Registry of MHAUS**: 1-412-692-5464 by means of a confidential AMRA report. The patient can call this number to add their name to the Registry database.
Case 1

- 4 yo male, uneventful ASD repair using sevoflurane, isoflurane, and non-depolarizing muscle relaxant- pancuronium
- Went to PARU post-op and was extubated when met criteria
- 20 minutes later EKG developed broad QRS complex bradycardia>>>then V-Fib arrest
Treatment

- Re-intubated, CPR, defibrillated, labs drawn
  - K = > 9 mEQ/L, BD -4.3, CPK 613 K 48 hrs later, Tmax 37.7 C
- Rx - hyperkalemia
  - HCO3
  - CaCL2
  - Epinephrine
  - GIK
- Muscle biopsy positive for Duchene's Muscular Dystrophy
MHAUS Reports 3 Unique Cases of Hyperkalemic Cardiac Arrest

- **Case 2**
  - 7 y.o., 30 kg asymptomatic female developed laryngospasm at the end of GA for T & A with Sevoflurane anesthesia without muscle relaxant
  - Succinylcholine 6 mg was administered...and V-fib ensued
Hyperkalemic Cardiac Arrest

- Seen in patients with occult or undiagnosed myopathy (Duchennes-symptoms of muscle weakness can be delayed until 6-8 yrs of age) or assymptomatic “dystrophinopathy”
- Can occur wit inhalation anesthetics or succinylcholine
- MH or “hyperkalemic syndrome”?.... different pathogenesis- leaky muscle membranes?
Sudden/Unexpected Cardiac Arrest in Young Patients—take home points

- Sudden cardiac arrest in healthy children in the OR or PARU in the absence of airway compromise, hypovolemia, or known cardiac abnormalities—think hyperkalemia!
  - Presume **hyperkalemia** and initiate treatment
    - Hyperventilate, bicarbonate, Calcium Chloride, glucose and insulin, epinephrine, defibrillation if necessary
- Usually secondary to **occult myopathy**
- Measure CK, Myoglobin, ABG’s, until normalized
- Consider Dantrolene
- Resuscitation may be difficult and prolonged
The patient who is slow to awaken

- Sufficient ventilation? - residual anesthetic agent hyper or hypocarbia?
- Hypoxia?
- Acid base
- Low cardiac output (diminished drug metabolism and elimination)
- Hypothermia?
- Medications (clonidine, lithium - lower anesthetic requirements) - iatrogenic? - caution with reversal agents
- Premedication with prolonged CNS effects? (anticholinergic, BZD, butyrophenone?)
- Synergism?
- Residual paralysis
- Advanced age?
- Glucose - diabetic
- Metabolic - electrolytes
- Intercurrent CNS event - Stroke?
THE END