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

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Excerpts from preface to Recovery from Anesthesia

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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
Why infants are at increased risk for perioperative Hypoxemia

- Immature centers for respiratory control and irregular breathing
- Hypoxia (paradox) depresses ventilatory drive
- Trace anesthetics abolish hypoxic ventilatory responses
- Small FRC, high O2 demand
- Anesthesia reduces FRC -> more atelectasis
- Infants are prone to UAO
- Fetal hgb - high affinity, low unloading ability
- Higher incidence in general
Non-invasive assessment of respiratory function - physical exam

- audible breath sounds - wheezing?
  - Inspiratory - extrathoracic
  - Expiratory - intrathoracic
- respiratory rate
- sternal / chest wall retractions
- use of accessory muscles
- nasal flaring
- grunting
- paradoxical respiration
- color
Rapid Cardiopulmonary Assessment: Classification of Physiologic Status

*Respiratory distress:* Increased work of breathing

*Respiratory failure:* Inadequate oxygenation or ventilation
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></th>
<th>PaCO2</th>
<th>A-aDO2</th>
<th>100% O2</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Hypoventilation</strong></td>
<td>Inc</td>
<td>Nl</td>
<td>Inc</td>
</tr>
<tr>
<td><strong>Low FIO2</strong></td>
<td>Nl</td>
<td>Nl</td>
<td>Inc</td>
</tr>
<tr>
<td><strong>Shunt (V/Q = 0)</strong></td>
<td>Nl</td>
<td>Inc</td>
<td>Nr</td>
</tr>
<tr>
<td><strong>V/Q mismatch</strong></td>
<td>Nl</td>
<td>Inc</td>
<td>Inc</td>
</tr>
<tr>
<td><strong>Diffusion Barrier</strong></td>
<td>Nl</td>
<td>Inc</td>
<td>Inc</td>
</tr>
</tbody>
</table>
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 = examine your patient
  - Call for help
  - Patient positioning
  - JAW THRUST better than chin lift
  - Consider CPAP mask in OSA patients
  - r/o laryngospasm - consider sux or propofol
  - Intubate if necessary
  - Consider PEEP in intubated patients
  - Further evaluation as indicated
Pulse Oximetry pitfalls

- Hypoxia can occur in the absence of cyanosis (SpO2 ~85%)
- Place probe properly - signal artifact
- Not a monitor of perfusion or adequacy of cardiac output!
- Motion artifact
- Must examine patient if on supplemental O2!!!!!!!
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

- $PAO_2 = FiO_2 \times (PB-PH_2O) - PaCO_2/R$
- With a $PaCO_2$ of 100 breathing room air
  - $PAO_2 = 0.21 \times 740 - (100/0.8) = 30$
- With a $PaCO_2$ of 100 breathing 30% blow by
  - $PAO_2 = 0.30 \times 740 - (100/0.8) = 97$
- When in doubt get a blood gas!!!
Acoustic Respiratory rate monitors

- Detect changes in respiratory pattern or rate
- Linked to pulse ox and disable PCA
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

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 $BP = CO \times SVR$
Emergence Delirium

- A dissociated state of consciousness in which the child is inconsolable, irritable, uncompromising or uncooperative, typically thrashing, crying, moaning or incoherent.

- Paranoid ideation

- Do not recognize familiar objects or people - parents freak out

- Self limiting 5-15 min

- No long term sequellae
ED causative factors

- Rapid emergence
- Intrinsic characteristics of the anesthetic
- Postoperative pain
- Surgery type
- Age
- Preoperative anxiety
- Child temperament
- Adjunct medication: ketamine, atropine, barbiturates, benzos
ED treatment

- Opioids
  - Intranasal fentanyl 2 mcg/kg
  - IV fentanyl 1 mcg/kg
- Propofol 0.5 mg/kg iv
- Midazolam .02 mg/kg iv
- Flumazenil .01 mg/kg iv
- Alpha agonist clonidine/ precedex
- R/O hypoxia, hypercarbia, hypotension, hypoglycemia, increased ICP
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
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
- 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 pediatrics
- 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
Pain Assessment - Overview

- Rule out treatable causes of agitation
  - Hypoxia
  - Hypercarbia
  - Cerebral hypoperfusion
  - Bladder distention
- Identify cause
- Monitor appropriately
Pain Assessment - overview

- Decision points
  - Agent(s) to use
  - Route of administration
  - Mode of administration
- Titrate >> Observe >> Monitor response with formalized pain scale
- Allow adequate time in transition from iv to PO pain meds
- Careful if accidental overdose...
Perioperative Management of Children with Obstructive Sleep Apnea

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Obstructive sleep apnea syndrome (OSA) affects 1%–3% of children. Children with OSA can present for all types of surgical and diagnostic procedures requiring anesthesia, with adenotonsillectomy being the most common surgical treatment for OSA in the pediatric age group. Thus, it is imperative that the anesthesiologist be familiar with the potential anesthetic complications and immediate postoperative problems associated with OSA. The significant implications that the presence of OSA imposes on perioperative care have been recognized by national medical professional societies. The American Academy of Pediatrics published a clinical practice guideline for pediatric OSA in 2002, and cited an increased risk of anesthetic complications, though specific anesthetic issues were not addressed. In 2006, the American Society of Anesthesiologists published a practice guideline for perioperative management of patients with OSA that noted the pediatric-related risk factor of obesity, and the increased perioperative risk associated with adenotonsillectomy in children younger than 3 yr. However, management of OSA in children younger than 1 yr-of-age was excluded from the guideline, as were other issues related specifically to the pediatric patient. Hence, many questions remain regarding the perioperative care of the child with OSA.

In this review, we examine the literature on pediatric OSA, discuss its pathophysiology, current treatment options, and recognized approaches to perioperative management of these young and potentially high-risk patients.

(Anesth Analg 2009;109:60-75)
Figure 3. Algorithm for risk assessment and disposition planning

Table 4. A Severity Ranking System Based on Polysomnography-gold standard

<table>
<thead>
<tr>
<th>Apnea-hypopnea index</th>
<th>Oxygen saturation nadir</th>
</tr>
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<tbody>
<tr>
<td>Normal</td>
<td>0–1</td>
</tr>
<tr>
<td>Mild OSA</td>
<td>2–4</td>
</tr>
<tr>
<td>Moderate OSA</td>
<td>5–9</td>
</tr>
<tr>
<td>Severe OSA</td>
<td>&gt;10</td>
</tr>
</tbody>
</table>

Peak ETCO₂ values and percent of time spent with ETCO₂ >50 mm Hg should also be considered when assessing severity.

OSA = obstructive sleep apnea syndrome.

<table>
<thead>
<tr>
<th>Oximetry Score</th>
<th>Comment</th>
<th>Criteria</th>
<th>Recommendation</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>No. of Drops in SaO₂ &lt;90%</td>
<td>No. of Drops in SaO₂ &lt;85%</td>
</tr>
<tr>
<td>1</td>
<td>Normal study/inconclusive for OSA</td>
<td>&lt;3</td>
<td>0</td>
</tr>
<tr>
<td>2</td>
<td>OSA, mild</td>
<td>≥3</td>
<td>≤3</td>
</tr>
<tr>
<td>3</td>
<td>OSA, moderate</td>
<td>≥3</td>
<td>&gt;3</td>
</tr>
<tr>
<td>4</td>
<td>OSA, severe</td>
<td>≥3</td>
<td>&gt;3</td>
</tr>
</tbody>
</table>
Table 8. Clinical Features that Predict Respiratory Compromise After Adenotonsillectomy and, in Some Cases, Persistent Obstructive Sleep Apnea

<table>
<thead>
<tr>
<th>Clinical Feature</th>
</tr>
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<tbody>
<tr>
<td>Severe obstructive sleep apnea on polysomnography</td>
</tr>
<tr>
<td>History of prematurity, especially with respiratory disease</td>
</tr>
<tr>
<td>Age &lt;3 yr</td>
</tr>
<tr>
<td>Morbid obesity</td>
</tr>
<tr>
<td>Nasal problems (deviated septum, enlarged turbinates)</td>
</tr>
<tr>
<td>Mallampati score 3 or 4</td>
</tr>
<tr>
<td>Neuromuscular disorders/disordered pharyngeal tone</td>
</tr>
<tr>
<td>Genetic or chromosomal disorders</td>
</tr>
<tr>
<td>Craniofacial disorders</td>
</tr>
<tr>
<td>Enlarged lingual tonsils</td>
</tr>
<tr>
<td>Upper respiratory infection within 4 wk of surgery</td>
</tr>
<tr>
<td>Cor pulmonale</td>
</tr>
<tr>
<td>Systemic hypertension</td>
</tr>
<tr>
<td>Marked obstruction on inhalational induction</td>
</tr>
<tr>
<td>Disordered breathing in the postanesthesia care unit</td>
</tr>
<tr>
<td>Difficulty breathing during sleep</td>
</tr>
<tr>
<td>Growth impairment due to chronic obstructed breathing</td>
</tr>
</tbody>
</table>


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

Cortex
- benzodiazepines
- cannabinoids

Visceral Afferents
- metoclopramide (high dose)
- 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)- FDA black box < 7 yo
- 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
Masqueraders

- Osteogenesis imperfecta
- Myotonic dystrophy
- Neuroleptic Malignant Malignant Syndrome
- Sepsis
- Brain injury - hypoxic, encephalitis, meningitis
- Thyrotoxicosis
- Pheochromocytoma
- Drugs - Anticholinergic syndrome, Cocaine, Aspirin toxicity
- Heatstroke
- Iatrogenic overheating
- Hypoventilation (equipment malfunction)
- CO2 absorption during laparoscopy
- Faulty temperature probe
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** HYPERVENTILATE
- **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.
MHAUS Reports 3 Unique Cases of Hyperkalemic Cardiac Arrest

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 bradycaardia>>>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 he 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 (Duchenne-symptoms of muscle weakness can be delayed until 6-8 yrs of age) or asymptomatic “dystrophinopathy”
- Can occur with 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 hypo carbia?
- 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