Perioperative Upper Airway Considerations in Pediatric Obstructive Sleep Apnea

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Conflict of Interest

• None to declare

OBJECTIVES
At the end of this session audience members will be able to:

• Understand how OSA related upper airway structure, function and related pathophysiology impact anesthetic management in children.

• List key limitations of published pediatric OSA associated management guidelines.

• Appreciate a role for anti-inflammatory agents to modulate perioperative respiratory adverse events (PRAEs) in children with OSA.
**Obstructive Sleep Disordered Breathing (SDB)**

- A syndrome of upper airway dysfunction during sleep characterized by snoring and/or increased respiratory effort that result from increased upper airway resistance and pharyngeal collapsibility

![SBD Spectrum]

SDB Spectrum

- Normal Primary Snoring
- Upper A/W Resistance Syndrome (UARS)
- Obstructive Hypoventilation (OH)
- Obstructive Sleep Apnea (OSA)

**Pediatric OSA Prototype = Adenotonsillar hyperplasia**
Pediatric OSAS & Societal Impact

- Common
- 1-5% of children; ↑ with obesity
- ↑ Surgical prevalence
- M>F & age phenotypes
- Secondary & associated morbidity
- ↓ socioeconomic status
- Expensive
- ↓ School and job performance
- ↑ healthcare utilization
- Altered CVS health trajectory
- Shorter life span
- Treatment & health trajectory

Jennun F et al. Thorax 2013

Pediatric mortality after adenotonsillectomy

<table>
<thead>
<tr>
<th>Source</th>
<th>Years</th>
<th>15-30 Day Death Rate (per 10,000)</th>
</tr>
</thead>
<tbody>
<tr>
<td>US (Brown K Anesth Analg 2014)</td>
<td>1970s</td>
<td>0.3-0.6</td>
</tr>
<tr>
<td>US out-patient (Shay S Laryngoscope 2015)</td>
<td>2010</td>
<td>0.6</td>
</tr>
</tbody>
</table>


The Elephant in the Room: Lethal Apnea at Home after AT

Death or Neurologic Injury After Tonsillectomy in Children with a Focus on Obstructive Sleep Apnea: Houston, We Have a Problem!

Charles J. Gott, MD*, Karen L. Pannier, PhD, t and Karen B. Durieux, MD, MPH*

Mortality and Major Morbidity After Tonsillectomy: Etiologic Factors and Strategies for Prevention

Jelle L. Goldhagen Anesthesia- and opioids-related malpractice claims following tonsillectomy in USA: LexisNexis claims database 1984-2012

Pediatric Anesth 2014
OSA associated with increased respiratory complications & dose-response evident.

Adenotonsillectomy Complications: A Meta-analysis

Pediatrics 2015

Impacts of Disease Severity on Postoperative Complications in Children With Sleep-Disordered Breathing

End-Organ Dysfunction

OSA Syndrome

Oxidative Stress

↑ Pco₂

Inflammatory Cascade

Autonomic Dysfunction

Frequent Micro-arousals

Respiratory Morbidity

(Asthma & URTI)

Gutierrez MJ. Pediatri Pulmonl 2013


Tan HL Nat Sci Sleep 2013

Polysomnography (PSG) is diagnostic “Gold Standard”

“DIAGNOSTIC” SEVERITY is based on the following:

- PSG data-Apnea-hypopnea index

<table>
<thead>
<tr>
<th>Severity of OSA</th>
<th>Adult AHI</th>
<th>Pediatric AHI</th>
</tr>
</thead>
<tbody>
<tr>
<td>None</td>
<td>0-5</td>
<td>0</td>
</tr>
<tr>
<td>Mild OSA</td>
<td>6-20</td>
<td>1-5</td>
</tr>
<tr>
<td>Moderate OSA</td>
<td>21-40</td>
<td>6-10</td>
</tr>
<tr>
<td>Severe OSA</td>
<td>&gt; 40</td>
<td>&gt; 10</td>
</tr>
</tbody>
</table>

- Limited access and expensive
Pediatric management guidelines are confusing...

PSG Indications
“Everyone”—Prescriptive—Not really necessary
AHl Diagnostic Threshold for “Severe” OSA—are Risk for PRAE?
Yes—No—What does “severe” mean?—Not acknowledged
PSG Alternatives Acceptable?
Yes—No—Not acknowledged

OSAS diagnosis is moving out of the sleep lab

Questionnaires
• Symptoms not diagnostic
• Physical findings “unreliable”

Other options
• Single-channel recordings
• Oximetry + airflow or EOG
• Home-based sleep studies
• PSG and polygraphy
• Biological Markers (De Luca Canto G Sleep Med Rev 2015)
  • Blood
  • Urinary—most promising
  • Salivary
  • Exhaled condensate

“Typical” tonsillectomy disposition planning

Definition “Severe” OSA leading to ↑ risk of PRAE: NO CONSENSUS
Unknown how PRAE risk modulated by associated pathophysiology, age, comorbidities, skill of providers (and opioids)
Intensive care unit admission criteria: NO CONSENSUS
Monitor according to local practice
Does risk for PRAE vary by procedure?
OSA age-related airway phenotypes

Pediatric OSA Endotypes

<table>
<thead>
<tr>
<th>SYMPTOM</th>
<th>INFANT (0-2 yrs)</th>
<th>CHILD (2-8 yrs)</th>
<th>PRE-teen/Teen (9-21 yrs)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lymphoid hyperplasia (adenoids +/- tonsils)</td>
<td>+/-</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Soft tissue</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Edema</td>
<td>+/-</td>
<td>++</td>
<td>+++</td>
</tr>
<tr>
<td>&quot;Genetic&quot; (e.g. Hunter’s, Prader-Willi, Beckwith-Wiedemann)</td>
<td>++</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Craniofacial Syndromes</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Uvula &amp; Vatersa (e.g. Craniosynostosis &amp; P Kryder)</td>
<td>+++</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td>Foramen Magnum (e.g. Arnold-Chiari)</td>
<td>++</td>
<td>++</td>
<td>+/-</td>
</tr>
<tr>
<td>Neuromuscular (e.g. C palsy &amp; Trisomy 13)</td>
<td>+</td>
<td>+++</td>
<td>+</td>
</tr>
<tr>
<td>Prematurity (&lt; 32 wks)</td>
<td>+++</td>
<td>+</td>
<td>-</td>
</tr>
<tr>
<td>Inflammatory (e.g. Asthma &amp; Sickle Cell Dis.)</td>
<td>+/-</td>
<td>+++</td>
<td>++</td>
</tr>
</tbody>
</table>

Factors contributing to airway patency and collapse in pediatric OSA

- Promote A/W Collapse
  - Negative pressure on inspiration
  - Fat deposition
  - Small mandible
- Promote A/W Patency
  - Genioglossus contraction
  - Lung volume (longitudinal tracheal traction)

Relationship between air flow & resistance

\[ Q = \Delta P/R \]

- Normal
  - Infant: 2 mm, ↓ 50%, ↑ 16X
  - Adult: 8 mm, 6 mm, ↓ 25%, ↑ 3X

\[ R = \frac{\Delta P}{Q} \cdot \frac{1}{R} \]
Pharyngeal wall tension, tracheal traction & abdominal pannus

Does dysfunctional neuro-motor control of the upper airway have a role?

- Day vs night time obstruction
- ↑ EMG genioglossus activity
- Not all children with anatomical obstruction have OSA
- Variable OSA cure rate following adenotonsillectomy

Pathophysiology of pediatric OSA:
Structural lab model

\[
\text{Cross-sectional area of a collapsible tube (A)} \propto \frac{1}{P_{\text{trans}}}
\]

Non-OSA = \( P_{\text{close}} < 0 \, \text{cm H}_2\text{O} \)

OSA = \( P_{\text{close}} > 0 \, \text{cm H}_2\text{O} \)
### OSA and upper airway neuromotor control

#### Upper Airway Findings

<table>
<thead>
<tr>
<th>Receptor (Location)</th>
<th>OSA Phenotypes</th>
</tr>
</thead>
<tbody>
<tr>
<td>Collapsibility (genioglossus)</td>
<td>Infant (0-2 yrs)</td>
</tr>
<tr>
<td>Ventilatory drive</td>
<td>O2 (Peripheral)</td>
</tr>
<tr>
<td>Arousalibility</td>
<td>O2 (Peripheral)</td>
</tr>
</tbody>
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**O2 administration improves ventilatory control instability**

Arens R & Marcus C. *Sleep* 2004

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### Upper airway collapsibility: Anesthetic agents & opioids

<table>
<thead>
<tr>
<th>Generic Drug Name</th>
<th>Airway Collapse</th>
<th>Mechanism of Action</th>
</tr>
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<tbody>
<tr>
<td>Midazolam</td>
<td>+</td>
<td>CNS GABA&lt;sub&gt;A&lt;/sub&gt;, α dose</td>
</tr>
<tr>
<td>Sevoflurane</td>
<td>+++</td>
<td>CNS GABA&lt;sub&gt;A&lt;/sub&gt;, α dose</td>
</tr>
<tr>
<td>Desflurane</td>
<td>+++</td>
<td>CNS GABA&lt;sub&gt;A&lt;/sub&gt;</td>
</tr>
<tr>
<td>Propofol</td>
<td>++</td>
<td>CNS GABA&lt;sub&gt;A&lt;/sub&gt;/NMDA, α dose</td>
</tr>
<tr>
<td>Dexmedetomidine</td>
<td>+/-</td>
<td>CNS α&lt;sub&gt;2&lt;/sub&gt; adrenergic agonist</td>
</tr>
<tr>
<td>Ketamine</td>
<td>+/-</td>
<td>NMDA receptor antagonist; ↑ EMG genioglossus (rats)</td>
</tr>
</tbody>
</table>

GABA<sub>A</sub> receptors-stimulation leads to myo-relaxation

Anesthetic agents enhance GABA<sub>A</sub> receptor activity

Campagna JA NEJM May 2003

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### GABA<sub>A</sub> receptor activity augmented by anesthetic agents in presence of IL-1

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### Prevention of PRAEs in OSA patients: Is there a role for steroids or NSAIDs?
Perioperative steroid administration improving adenotonsillectomy outcomes

A Randomized Trial of Adenotonsillectomy for Childhood Sleep Apnea

Caulfield HM, Clin Otolaryngol 2012

Future of pediatric tonsillectomy and perioperative outcomes

Oxidative stress & sensitivity to opiates in pediatric OSAS

Laboratory Investigations

Recurrent Hypertension Postoperative Increases Subsequent Restlessness

Anesthesiology 2016

Anesthetic Respiratory Considerations

Muscle relaxants, reversal agents & the upper airway

Impaired Upper Airway Integrity by Residual Neuromuscular Blocking Agents on Postoperative Respiratory Outcomes

A Prospective Study

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Summary: Perioperative Upper Airway Considerations in Children with OSA

• PATIENT
  • Prone to impaired airway neuro-motor function due to drugs
  • Spectrum of comorbidities secondary to OSA or “age-specific”

• PREOPERATIVE
  • Risk Stratification for PRAE
    • Age < 3 yrs & “significant” comorbidities
    • “Severe” OSA by HS/PSG or Overnight Pulse Oximetry
    • “Prescriptive PSG” needed?
    • “Innovativeness” of surgery & anesthesia
  • Need for postoperative opioids

• POSITION
  • Head up “Tracheal Tethering”, avoid being supine

Summary continued

• PROCEDURE
  • Goals: prepare for “challenging” airway & PRAEs
  • No one “best” anesthetic technique
  • “Pharyngeal sparing” approach
  • Emerge awake and consider nasopharyngeal airway

• POSTOPERATIVE
  • Monitoring
    • Continuous SpO2 preferably on room air and asleep
    • Appropriate duration unknown (2-6 hrs)
    • Significance of minor/major PRAEs during recovery unknown
      (Weingarten G-Anesth-Analg 2015)
  • Analgesia
    • Multimodal and avoid opioid infusions or “around-the-clock”
    • Optimal approach unknown (Buckton P-J Pediatr Anesth 2011)
  • Parent Preparation
    • 2019 AAOHN Tonsillectomy Guideline

Thank you!

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