Preanesthetic examination of the child
Red flags

Clinical Red Flags

Francis Veyckemans
BAPA-SKA Refresher course
Leuven 2020
We only find what we look for.
We only look for what we know.
The child with

- Sleep-disordered breathing
- Bronchopulmonary dysplasia
- A mediastinal mass
Range of Sleep Disordered Breathing

SNORING

- Normal Breathing
- Occasional Snoring
- Regular Snoring
- Upper Airway Resistance Syndrome

NO DISEASE

OBSTRUCTIVE SLEEP APNEA

- Mild Sleep Apnea
- Moderate Sleep Apnea
- Severe Sleep Apnea

DISEASE
Sleep-disordered breathing

- **Simple snoring**: 20% of children microarousals during sleep
- **Increased upper airway resistance**: some behavioural problems
- **Hypopnea**: partial airway obstruction
- **Obstructive apnea**: 1-3% episodes of complete airway obstruction
Obstructive sleep apnea syndrome

3 types of OSAS:
1: hypertrophy of tonsils / adenoids
2: obesity with moderate tonsils / adenoids
3: orofacial pathology
   - midfacial hypoplasia (achondroplasia ...)
   - macroglossia, small pharynx
   - hypotonia (T 21, Prader Willi, polyhandicap)
   - micrognathia
Normal  Snoring  OSAS
Signs & symptoms of OSAS

* During sleep:
  - snoring
  - respiratory arrests
  - restlessness, nightmares
  - neck hyperextension

* Adenoid facies (open mouth)

* Diurnal somnolence

* Mood disorders

* Increased risk in African American ethnicity
Risks of OSAS

- Mood disorders
- Learning difficulties
- Risk of chronic cor pulmonale

- More respiratory complications: obstruction, desaturation, laryngospasm
- Decreased response to $\uparrow CO_2$
- Increased sensitivity to opiates ($\downarrow$ n of $\mu$ opiates in the forebrain)
Polysomnography: gold standard

Obstructive apnea/hypopnea index per hour of sleep

> 1.5 = pathologic
1.5 - 5 = mild OSAS
> 5 = moderate OSAS
> 10 = severe OSAS

80% during REM sleep (> in adults: non-REM sleep)
Sleep oximetry: McGill Oximetry score

- at least 3 clusters of nadir SpO$_2$

- if SpO$_2$ > 92%: OK

- if SpO$_2$ < 92%: specialist’s opinion polysomnography?
Outside the ENT context

Clinical questionnaires to be used as screening tools:
- Spruyt-Gozal: presence of OSAS?
- STBUR: increased risk for respiratory complications?

Can be completed before the visit
But parents’ reliability?
Spruyt-Gozal questionnaire

_Chest_ 2012; 142: 1508-15

In the last 6 months,

1. Have you had to help your sleeping child to start breathing again?
2. Does your child stop breathing while asleep?
3. Does your child have difficulty breathing while sleeping?
4. Has your child’s breathing while asleep been a subject of concern for you?
5. How noisy is his/her snoring?
6. How often, does your child snore?

Responses to these questions are a score from 0 to 4 according to the frequency of the event

0 if “never”
1 if “rarely” (1 night per week)
2 if “occasionally” (2 nights per week)
3 if “frequently” (3 to 4 nights per week)
4 if “almost always” (more than 4 nights per week)

except for question 5, assessing snoring:

0: just perceptible or light snoring
1: moderate snoring
2: heavy snoring
3: very heavy snoring
4: extremely heavy snoring
Spruyt Gozal score

Score: where Q = question

\[ A = \frac{Q_1 + Q_2}{2} \]

\[ B = \frac{A + Q_3}{2} \]

\[ C = \frac{B + Q_4}{2} \]

\[ D = \frac{C + Q_5}{2} \]

total score = \[ \frac{D + Q_6}{2} \]

If score > 2.75 = at least moderate SAOS

(= apnea/hypopnea index ≥ 5/h)

Sensitivity 92% specificity 81%
STBUR Questionnaire

While sleeping does your child ...

1. snore > half of the time?
2. snore loudly?
3. have trouble breathing or struggle to breathe?
4. have you ever seen your child stop breathing while sleeping?
5. does your child wake up feeling not well rested in the morning?

*Pediatr Anesth* 2013; 23: 510-6
2016; 26: 759-66
STBUR Questionnaire

While sleeping does your child ...

1. snore > half of the time?
2. snore loudly?
3. have trouble breathing or struggle to breathe?
4. have you ever seen your child stop breathing while sleeping?
5. does your child wake up feeling not well rested in the morning?

If 3 ≥ + answers

2 × risk of respiratory complications

Pediatr Anesth 2013; 23: 510-6
2016; 26: 759-66
Drug-induced sleep endoscopy

Upper airway examination
- under deep sedation /GA
- spontaneous ventilation
- no CPAP, no upper airway maneuvers
to evaluate site(s) of upper airway collapse
in children with sleep-disordered breathing

and small tonsils

or with an at risk morphology
  that persists after adenotonsillectomy
Drug-induced sleep endoscopy (2)

In adults:
TCI propofol starting target 1.5 – 3 µg/ml
BIS 50-70

In children?
- inhalation induction,
- venous access, stop sevo,
- bolus propofol 1-2 mg/kg, EtSevo 0%
If severe: preoperative stabilisation

Nasal cpap
Nasal High flow air/O₂
Nasopharyngeal airway
Anesthetic management

1) Identify patients at preanesthetic evaluation
easy if ENT procedure
ask for snoring, ! if African origin
look at the tonsils (Mallampati)

2) No T&A as outpatient if
(probably) severe SAOS
< 3 years
+ comorbidity /syndrome
Anesthetic management (2)

3) Induction, awakening and PACU:
   - risk of upper airway obstruction
     think nasopharyngeal airway

4) If severe:
   - decrease opiate dose by 50%
     but ...
   - NSAIDs
   - even dexmedetomidine as some effect

5) Plan postoperative monitoring
Opioid Sensitivity in Children with and without Obstructive Sleep Apnea

Michael C. Montana, M.D., Ph.D., Lindsay Juriga, B.S., Anshuman Sharma, M.D., Evan D. Kharasch, M.D., Ph.D.

ANESTHESIOLOGY 2019; 130:936–45

<table>
<thead>
<tr>
<th></th>
<th>Remifentanil (ng/ml)</th>
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<tr>
<td></td>
<td><strong>Total</strong> (n = 30)</td>
<td><strong>Non-OSA</strong> (n = 15)</td>
<td><strong>OSA</strong> (n = 15)</td>
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<tr>
<td><strong>Age</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Mean ± SD</td>
<td>11 ± 2</td>
<td>11 ± 2</td>
<td>11 ± 2</td>
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<tr>
<td>Range</td>
<td>8–14</td>
<td>8–14</td>
<td>8–13</td>
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<tr>
<td><strong>Weight (kg)</strong></td>
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<tr>
<td>Mean ± SD</td>
<td>57.2 ± 24.9</td>
<td>48.3 ± 22.4</td>
<td>66.1 ± 24.8</td>
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<tr>
<td>Range</td>
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<td>24.9–113.9</td>
<td>29.2–119.4</td>
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<td><strong>Sex</strong></td>
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<tr>
<td>Female</td>
<td>10 (33.3%)</td>
<td>4 (26.7%)</td>
<td>6 (40.0%)</td>
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<tr>
<td>Male</td>
<td>20 (66.7%)</td>
<td>11 (73.3%)</td>
<td>9 (60.0%)</td>
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<tr>
<td><strong>Race</strong></td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>Caucasian</td>
<td>23 (76.7%)</td>
<td>12 (80.0%)</td>
<td>11 (73.3%)</td>
</tr>
<tr>
<td>Black</td>
<td>6 (20.0%)</td>
<td>3 (20.0%)</td>
<td>3 (20.0%)</td>
</tr>
<tr>
<td>Native American</td>
<td>1 (6.7%)</td>
<td>0 (0%)</td>
<td>1 (6.7%)</td>
</tr>
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</table>

A

B
Bronchopulmonary dysplasia
Bronchopulmonary dysplasia

50 years ago: sequellae of RDS caused by hyaline membrane disease + aggressive ventilation and $O_2$ toxicity

Nowadays:
chronic lung disease resulting from injury to the developing lung and pulmonary vasculature in very premature infants
Bronchopulmonary dysplasia

50 years ago: sequellae of RDS caused by hyaline membrane disease + aggressive ventilation and O$_2$ toxicity

Nowadays:
Chronic lung disease resulting from injury to the developing lung and pulmonary vasculature in very premature infants

* < 32 weeks gestational age
= before alveolization of lung
* alveolar growth is guided by vascular growth
Bronchopulmonary dysplasia

Premature birth

Postnatal factors
- mechanical ventil
- hyperO₂
- inflammation
- infection

↓ Vascular endothelial growth factor

↓ angiogenesis
↓ alveolisation
↑ risk of pulmonary HyperT

Prenatal factors
- genetics (♂)
- chorioamnionitis
- intraut growth delay
- maternal smoking
- drugs
Bronchopulmonary dysplasia

**Classic**
- reduced alveolar surface
- hyperinflation/atelectasis
- interstitial fibrosis and epithelial lesions (metaplasia, hyperplasia)
- bronchial muscles hyperplasia
- hypertensive vascular lesions

**New**
- ↓ n alveolae (large)
- homogenous lesions
- minimal epithelial lesions
- no or mild muscle anomalies
- abnormal pulmonary vessels
Bronchopulmonary dysplasia

Large spectrum of disease

Official definition:

- birth ≤ 32 weeks gestational age
- need for > 21% O₂ during at least 28 days before 36 weeks PCA or discharge home
- evaluation at 36 weeks PCA

• Mild: if on room air
• Moderate: if < 30% O₂
• Severe: if > 30% O₂ or CPAP, high flow
Bronchopulmonary dysplasia: the obvious

- Chronic lung disease: hyperreactivity
- Sometimes tracheo- or bronchomalacia
- Diuretics, bronchodilators, steroids
- Difficult venous access

Ventilation goals under anesthesia:
- low Vt, permissive hypercapnia
- long expiratory time (> auto-PEEP)
Bronchopulmonary dysplasia:

Check the cardiac status:
- any shunt? ASD, VSD, PDA
- pulmonary hypertension?
- pulmonary vein stenosis?
- cardiomyopathy?
Any shunt?

Left to right shunt (ASD)
- increases pulmonary blood flow
- contributes to the development of pulmonary hypertension

But
- can act as a pop-off valve
  in case of acute pulmonary hypertension
- check pressure gradient and flow
  😞 if low-velocity flow
Pulmonary hypertension

- in 15-25% of cases of BPD
- may develop even if cardiac echo normal at discharge
- regular cardiac echo for screening
- 😞 if poor weight gain
  increasing need for $O_2$
  acute degradation in case of viral infection
- even mild hypoxemia can increase PAP
Pulmonary veins stenosis
Pulmonary veins stenosis

- in 25% of cases with pulmonary hypertension
- acquired intraluminal stenosis
  (following increase flow, e.g. L \(\Rightarrow\) R shunt)
- may develop late
- high mortality: pulmonary hypertension
  acute RV failure
Bronchopulmonary dysplasia

Can be a hidden cardiac infant
Mediastinal mass?
Anterior mediastinal mass

Major anesthetic risk because risk of

- **Difficult/ impossible ventilation after intubation** because of tracheal/bronchial stenosis/compression

- **Hemodynamic collapse due to compression of heart or great vessels** pericardial effusion
Effects of general anesthesia

- \( \downarrow \) pulmonary volume
  = loss of stenting effect of inflated lung
- muscular relaxation (intercostal, bronchial)
  = \( \downarrow \) resistance to compression
- paralysis = \( \downarrow \) transpleural gradient
  = \( \downarrow \) diameter of intrathoracic airways
- intubation
  = loss of expiratory glottic brake
Predictive signs for complications

• Mass is asymptomatic in 40%!

• Except for orthopnea:
  - coughing when supine
  - no correlation with severity of tracheal compression (☠️ if < 30% free surface)
Anterior mediastinal mass

Anesthesia asked for

- Diagnosis: adenectomy (ask for X-Ray)
  - bone marrow aspiration/biopsy
  - imaging (CT)
- Insertion of a CV line for chemotherapy
- Ovarian/testicular sampling before chemotherapy

† different risk/benefit ratios options
Clinical examination

- palpebral edema?
- large external jugular veins?
- coughing when going supine?
- orthopnea?
- dyspnea at rest?
- auscultation!
- look at the chest-XRay and CT
Clinical examination
CT scan
Lymphoma!

- Some tumors double in size every 48 h!

uyên Get the most recent imagery possible
Principles of management

- Maintain spontaneous ventilation
- Induction in semi-sitting position if orthopnea, dyspnea
- Provide CPAP or PEEP
- Be ready to change quickly patient’s position (ventral, sternal lift)
  Look at RX & CT before starting
- If superior vena cava syndrome:
  Use a vein in the lower limb
In textbooks

- Rigid bronchoscope ready?

- If compression of great vessels: cardiac bypass ready?
Reduce the tumor size before anesthesia?

« steroids or radiotherapy before biopsy could make the precise diagnosis difficult to establish »

- in fact: 5 days of corticotherapy do not interfere with diagnosis

Anaesthesia 2008; 63: 837-47

☞ collegial decision with oncologists

but some tumors resist to steroids!
Awakening

- as dangerous as induction!
- because
  - residual effects of anesthetics/sedatives
  - local edema
  - cough
- beware of dexamethasone: risk of [tumor lysis syndrome](#) and hyperkalemic cardiac arrest (look at preoperative uric acid level)
BAPA Antwerp April 25th

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Challenging the Unknowns

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