Obstructive Sleep Apnea in View of The Epidemic of Obesity in Children

Sleep Teaching Day
Department of Pediatrics
Golisano Children’s Hospital
Upstate Medical University
April 15, 2016, Syracuse, NY

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Overview

- Pathophysiology of OSAS in children
  - Anatomical factors
  - Functional factors
- The epidemic of obesity
- Treatment
- Case presentations
- Q&A
The Problem

12 yo obese female with loud snoring, poor school performance and always sleepy (AHI 120/hr)
Spectrum of OSAS

- Primary snoring
  - No abnormalities in gas exchange

- Upper airway resistance (UARS)
  - Arousals with no abnormalities in gas exchange

- Mild OSAS
  - Obstructive events, hypoxia, sleepiness

- Moderate OSAS

- Severe OSAS
Prevalence of OSAS

- **Children**
  - 2-4%

- **Adults**
  - 2% of women and 4% of men
  - (SDB 9% women and 24% men)

*Young et al. NEJM 1993*

*Lumeng & Chervin Proc Am Thorac Soc 2008*
Paediatric origins of adult lung diseases • 3

The genesis of adult sleep apnoea in childhood

F McNamara, C E Sullivan

Obstructive sleep apnoea (OSA) and central sleep apnoea have been identified and described in adults, children, and infants.1-5 It is not certain, however, if the adult sleep apnoea syndromes, particularly OSA, originate from childhood or whether paediatric and adult sleep apnoea are separate syndromes. Some investigators have suggested that the pathophysiology, criteria for diagnosis, and the management of paediatric patients with OSA are different from that for adults.6-7 Other investigators have found that risk factors, clinical symptoms, and the consequences of OSA share common features between adults, children and infants.8-11 We propose that the adult sleep apnoea syndrome is related to sleep apnoea in children, and that adult patients with sleep apnoea have been predisposed to developing the possible genesis of adult sleep apnoea during childhood.

The sleep apnoea syndromes
The OSA syndrome in adults was identified more than 30 years ago,12 has been described extensively in adults, and is believed to be caused by collapse of the oropharyngeal airway.1 It is not certain whether adult patients had OSA as infants or children; however, the diagnosis of OSA in adults often occurs several years after the onset of symptoms, sometimes starting during adolescence. Obstructive apnoea is associated with repetitive episodes of hypoxaemia, sleep fragmentation, and cardiovascular and neurobehavioural sequelae (fig 1).13-15 Several risk factors have been identified that predispose an individual to developing OSA including obesity, upper airway muscular abnormalities, craniofacial abnormalities, and the presence of other sleep disorders (table 1).13-15
Childhood OSAS: a continuum, overlap, or distinct disorders?

Arens & Marcus Sleep 2004
### Phenotypes of Childhood OSAS

<table>
<thead>
<tr>
<th></th>
<th>INFANCY</th>
<th>CHILDHOOD</th>
<th>ADOLESCENCE</th>
<th>ADULTHOOD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Prevalence</td>
<td>?</td>
<td>2-4%</td>
<td>?</td>
<td>2 %F, 4%M (9-24%)</td>
</tr>
<tr>
<td>Peak age (yrs)</td>
<td>&lt; 1</td>
<td>2-8</td>
<td>12-18</td>
<td>30-60</td>
</tr>
<tr>
<td>Gender</td>
<td>M &gt; F</td>
<td>M=F</td>
<td>?</td>
<td>M&gt;F</td>
</tr>
<tr>
<td>Weight</td>
<td>Normal</td>
<td>Normal, underweight, or obese</td>
<td>Obese</td>
<td>Obese</td>
</tr>
<tr>
<td>Association</td>
<td>Craniofacial anomalies</td>
<td>T&amp;A Hypertrophy</td>
<td>T&amp;A Hypertrophy</td>
<td>Obesity Functional causes</td>
</tr>
<tr>
<td></td>
<td>Neurological disorders</td>
<td>Obesity Neurological dis.</td>
<td>Obesity Neurological dis.</td>
<td></td>
</tr>
</tbody>
</table>

Arens & Marcus Sleep 2004
Obesity and Excessive Daytime Sleepiness in Prepubertal Children With Obstructive Sleep Apnea
Gozal & Kheirandish-Gozal Pediatrics 2009
Consequences of OSAS

Cognitive dysfunction
- Excessive daytime sleepiness (EDS)
- Altered memory
- Altered executive function
- Behavior abnormalities, short attention span, hyperactivity
- Decreased school performance

Cardiovascular morbidity
- Hypertension
- LV dysfunction
- Accelerated atherosclerosis
- Stroke
- MI
- RV dysfunction
- Autonomic dysfunction

Metabolic dysfunction
- Insulin resistance
- Type 2 diabetes
- Metabolic syndrome
Pathophysiology of OSAS

Schwab RJ. AJRCCM 2003

Pro/Con Editorials

Pro: Sleep Apnea Is an Anatomic Disorder

Is obstructive sleep apnea an anatomic disorder? Of course it is; there is no real debate. Just examine the patient population. Patients with sleep apnea almost invariably are either obese or have abnormal upper airway anatomy (retrognathia, tonsillar hypertrophy, congenital palatal orofacial deformity). In fact, studies have shown that parapharyngeal fat pad volume is greater in obese subjects developing apnea than in nonobese subjects developing apnea (1-4). Moreover, nonobese subjects developing apnea have larger parapharyngeal fat pads than normal subjects (1). However, it is not just the

Con: Sleep Apnea Is Not an Anatomic Disorder

The most fundamental argument for neural events as initiating obstructive sleep apnea is the fact that closure of the pharyngeal airway occurs during sleep. In sleep, there is a reorganization of cortical (e.g., neural) control that includes changes in direct cortical drive to and peripheral reflex control of the muscles of the chest wall and upper airway and ventilation (1). The disease-defining event is state-related, requiring sleep in the presentation of the disease-obstructive sleep apnea-hypopnea syndrome (2, 3). The presence of an anatomic encroachment of the upper airway by itself does not produce an obstruction. Obesity, tonsillar hypertrophy,
Evidence of Functional Factors in Children

- In 10-15% of cases, OSAS continues after adenotonsillectomy*
  
  *Rosen GM. Pediatrics 1994

  Other anatomical causes exist or there is increased AW collapsibility

- Some children with OSAS have normal anatomy

  *Tal A. Chest 2003

  increased AW collapsibility

- Children with OSAS don’t obstruct while awake

  *Guilleminault C. Laryngoscope 2004

  Neuromotor compensation
Collapse occurs when the pressure surrounding the airway (Pcrit) becomes greater than the pressure within the airway.

- Pcrit is a quantitative measure of airway collapsibility.
- Pcrit is the net effect of AW neuromuscular control and structural factors.
- Pcrit is a dynamic measure that becomes more negative with activation of pharyngeal dilators.
$P_{\text{crit}}$ in Children: Controls vs. OSAS


Anatomical Factors Affecting Airway Size
Upper Respiratory Tract Involvement in Children with OSAS

Prospective study of 60 OSAS children between 2-9 years and 300 matched controls

Recruitment from
The Children’s Hospital of Philadelphia between 1999-2005

Polysomnography
MRI of head & neck under sedation

Upper airway structure

NIH HL-62408
Sagittal View: Control vs. OSAS

Control

OSAS

Arens et al. AJRCCM 2001
Adenoid and Tonsils Size

Arens et al. AJRCCM 2001
Skeletal Measurements

- MAND Width (cm)
- MS-CL (cm)
- MAND Vol (cm³)
- MAXL width (cm)
- HP length (cm)
- HP Sag area (cm²)

Comparison of OSA and Controls:

- OSA
- Controls
Soft Tissues

- PTG Axial (cm²)
- Fat pad axial (cm²)
- TNG Sag (cm²)
- SP Sag area (cm²)
- TNG Vol (cm³)
- SP Vol (cm³)

- OSA
- Controls
Correlation between Adenoid & Tonsils Size and OSA Severity (AHI)

Adenoid+Tonsils %Vol diff

AHI

Tonsil+Adenoid Volume %Diff (OSAS to Control)

B

$r = 0.51$
$p = 0.03$
The Epidemic of Obesity
Obesity and Risk for OSAS

The Occurrence of Sleep-Disordered Breathing among Middle-Aged Adults
Wisconsin Sleep Cohort Study

“Male gender and obesity were strongly associated with the presence of SDB”

“An increase BMI in 1SD tripled the prevalence of OSAS”

Young et al. NEJM 1993
Prevalence of Obesity Among Children and Adolescents between 6-19 Years
Obesity and Risk for OSAS in Children

- OSAS in non-obese children 2-4%

- Early studies found that obesity was present in 10% of children with OSAS

- OSAS was reported up to 59% of children with obesity referred for evaluation

- OSAS was noted in 46% of unselected obese children

- More recently, in a large epidemiological study of 400 children, obesity increased the risk for OSAS (OR=4.5)
  - Redline et al. *AJRCCM* 1999
Possible Mechanisms

- Adenotonsillar hypertrophy (↑ somatic growth, inflammation, infection?)
  - decreases AW size

- Excess of other soft tissues around the airway (adipose tissue, fat pads and lateral pharyngeal wall muscles)
  - compresses the airway and increases AW collapsibility

- Restrictive lung disease (low FRC) due to increased visceral fat
  - decreases oxygen reserves
  - decreases AW stiffness (reduced tracheal tethering)

- Decreased ventilatory drive
  - blunted chemo receptor function due to chronic CO2 elevation
  - Hypoventilation

- Short sleep duration
  - Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes
  *Spiegel and Van Cauter J Appl Physiol 2005*
Upper Airway Structure and Body Fat Composition in Obese Children (8-17 years) with OSAS

- **Hypothesis**
  - Our main hypothesis was that the size of lymphoid tissues surrounding the upper airway is larger in the obese OSAS group as compared to obese controls, as found in young non-obese OSAS children.
  
  - Our secondary analysis examined other possible anatomical causes that have been previously noted in obese adults (other upper airway soft tissues and subcutaneous and visceral fat in neck and abdomen).

Arens et al. AJRCCM 2010
METHODS

- Wake MRI was used to determine the size of upper airway structure, and body-fat composition
  - **Airway**
    - Nasopharynx, Oropharynx, Hypopharynx
  - **Lymphoid tissues**
    - Adenoid
    - Palatine tonsils
    - Retropharyngeal nodes
    - Deep cervical lymph nodes
  - Tongue (including the genioglossus and geniohyoid muscles)
  - Soft palate
  - Mandible

- **Body Fat Composition**
  - Neck (subcutaneous & parapharyngeal fat)
  - Abdominal (subcutaneous & visceral fat)
Visceral Fat Vs. Subcutaneous Fat
## Demographics

<table>
<thead>
<tr>
<th></th>
<th>OSAS (n=22)</th>
<th>CON (n=22)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>12.5 ± 2.8</td>
<td>12.3 ± 2.9</td>
<td>NS</td>
</tr>
<tr>
<td>Gender (male/female)</td>
<td>M:14, F:8</td>
<td>M:14, F:8</td>
<td>NS</td>
</tr>
<tr>
<td>Height (cm)</td>
<td>158.5 ± 17.7</td>
<td>158.8 ± 16.3</td>
<td>NS</td>
</tr>
<tr>
<td>Weight (kg)</td>
<td>89.8 ± 35.2</td>
<td>84.6 ± 29.8</td>
<td>NS</td>
</tr>
<tr>
<td>BMI (kg/m²)</td>
<td>34.6 ± 8.3</td>
<td>32.4 ± 6.9</td>
<td>NS</td>
</tr>
<tr>
<td>BMI Z- Score</td>
<td>2.4 ± 0.4</td>
<td>2.3 ± 0.3</td>
<td>NS</td>
</tr>
<tr>
<td>Tanner Stage</td>
<td>3.4 ± 1.7</td>
<td>3.3 ± 1.6</td>
<td>NS</td>
</tr>
<tr>
<td>Ethnicity (AA/Hispanic/Cau/Other)</td>
<td>12/8/1/1</td>
<td>12/7/3/0</td>
<td>NS</td>
</tr>
</tbody>
</table>
## Polysomnography

<table>
<thead>
<tr>
<th></th>
<th>OSAS (n=22)</th>
<th>CON (n=22)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Total Sleep Time (hrs)</strong></td>
<td>6.3 ± 1.5</td>
<td>6.2 ± 0.7</td>
<td>NS</td>
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<tr>
<td><strong>Sleep Efficiency (%)</strong></td>
<td>78.5 ± 16.5</td>
<td>84.3 ± 8.2</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Apnea Index (events/hr)</strong></td>
<td>3.1 ± 5.4</td>
<td>0.2 ± 0.4</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Apnea Hypopnea Index</strong></td>
<td>16.3 ± 12.8</td>
<td>1.1 ± 1.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Baseline SpO(_2) (%)</strong></td>
<td>98.8 ± 1.4</td>
<td>98.5 ± 1.2</td>
<td>NS</td>
</tr>
<tr>
<td><strong>SpO(_2) Nadir (%)</strong></td>
<td>85.2 ± 6.1</td>
<td>94.2 ± 2.9</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td><strong>Baseline ETCO(_2) (mmHg)</strong></td>
<td>38.7 ± 7.7</td>
<td>41.2 ± 2.5</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Peak ETCO(_2) (mmHg)</strong></td>
<td>50.1 ± 4.4</td>
<td>47.6 ± 3.9</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Arousal Index (events/hr)</strong></td>
<td>15.7 ± 12.1</td>
<td>8.0 ± 4.2</td>
<td>&lt;0.05</td>
</tr>
</tbody>
</table>
## Upper Airway Volumetric Analysis (cm$^3$)

<table>
<thead>
<tr>
<th></th>
<th>OSAS (n=22)</th>
<th>CON (n=22)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Airway</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Nasopharynx</td>
<td>8.7 ± 3.5</td>
<td>9.8 ± 2.8</td>
<td>NS</td>
</tr>
<tr>
<td>Oropharynx</td>
<td>3.5 ± 1.7</td>
<td>4.9 ± 2.0</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Hypopharynx</td>
<td>3.2 ± 1.3</td>
<td>3.5 ± 1.5</td>
<td>NS</td>
</tr>
<tr>
<td><strong>Lymphoid Tissues</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Adenoid</td>
<td>10.5 ± 4.4</td>
<td>7.1 ± 3.6</td>
<td>&lt; 0.01</td>
</tr>
<tr>
<td>Tonsils</td>
<td>10.1 ± 3.9</td>
<td>7.8 ± 2.5</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Retropharyngeal Nodes</td>
<td>4.7 ± 2.4</td>
<td>3.1 ± 1.6</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td><strong>Others Tissues</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soft palate</td>
<td>8.6 ± 2.9</td>
<td>8.6 ± 1.8</td>
<td>NS</td>
</tr>
<tr>
<td>Tongue</td>
<td>88.1 ± 17.0</td>
<td>89.7 ± 31.8</td>
<td>NS</td>
</tr>
<tr>
<td>Mandible</td>
<td>63.1 ± 12.7</td>
<td>64.9 ± 18.5</td>
<td>NS</td>
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</tbody>
</table>
### Body Fat Composition (cm³)

<table>
<thead>
<tr>
<th></th>
<th>OSAS (n=22)</th>
<th>CON (n=22)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Head &amp; Neck subcutaneous fat</td>
<td>483.2 ± 170.1</td>
<td>420.1 ± 111.0</td>
<td>NS</td>
</tr>
<tr>
<td>Parapharyngeal fat</td>
<td>10.6 ± 4.7</td>
<td>8.4 ± 2.3</td>
<td>&lt; 0.05</td>
</tr>
<tr>
<td>Abdominal subcutaneous fat</td>
<td>6,939.5 ± 3,115.0</td>
<td>5,916.9 ± 2,289.2</td>
<td>NS</td>
</tr>
<tr>
<td>Abdominal visceral fat</td>
<td>1,434.9 ± 459.6</td>
<td>1,101.4 ± 423.7</td>
<td>&lt; 0.05</td>
</tr>
</tbody>
</table>
Correlation Coefficient:
AHI vs. Airway Lymphoid Tissue Volume

\[ r = 0.42 \]
\[ p < 0.01 \]
Did not show that BMI Z-score was an effect modifier on any anatomic characteristic as it relates to OSAS

In other words:

- Degree of obesity (BMI Z-score) did not seem to affect the size of the upper airway structures or body fat distribution as it relates to OSAS or non-OSAS
CONCLUSIONS

- Upper airway lymphoid hypertrophy is significant in obese children with OSAS.
- The lack of correlation of lymphoid tissue size with obesity suggests that this hypertrophy is caused by other mechanisms.
- Though the parapharyngeal fat-pads and abdominal visceral fat are larger in obese children with OSAS we could not find a direct association with severity of OSAS or with obesity.

Thus, we suggest that the two groups emerging from the present study with similar BMI Z-score, present two distinct phenotypes:

1. Marked visceral adiposity, upper airway lymphoid hypertrophy, and OSAS.
2. Less profound visceral adiposity, smaller upper airway lymphoid tissues, and no evidence of OSAS.

Arens et al. AJRCCM 2010
Treatment for Childhood OSAS

- T&A is recommended first line treatment for pediatric SDB
  - >500,000 annually in US for children < 15 YO

- Meta-analysis shows OSA resolves in 82% of children undergoing T&A
  - Brietzke S et al. OHNS 134(6), 979-984, 2006

- Significant QOL improvement in children whose AHI does not normalize
A Randomized Trial of Adenotonsillectomy for Childhood Sleep Apnea

Carole L. Marcus, M.B., B.Ch., Reneé H. Moore, Ph.D., Carol L. Rosen, M.D., Bruno Giordani, Ph.D., Susan L. Garetz, M.D., H. Gery Taylor, Ph.D., Ron B. Mitchell, M.D., Raouf Amin, M.D., Eliot S. Katz, M.D., Raanan Arens, M.D., Shalini Paruthi, M.D., Hiren Muzumdar, M.D., David Gozal, M.D., Nina Hattiangadi Thomas, Ph.D., Janice Ware, Ph.D., Dean Beebe, Ph.D., Karen Snyder, M.S., Lisa Elden, M.D., Robert C. Sprecher, M.D., Paul Willging, M.D., Dwight Jones, M.D., John P. Bent, M.D., Timothy Hoban, M.D., Ronald D. Chervin, M.D., Susan S. Ellenberg, Ph.D., and Susan Redline, M.D., M.P.H., for the Childhood Adenotonsillectomy Trial (CHAT)

ABSTRACT
CHAT Study

- 464 children (5-9 y.o.) randomized to receive T&A vs. observation (watchful waiting) for 6 months

- PSG, neuropsych, cognitive, behavioral, and health outcomes assessed at baseline and 7 months

- T&A group did not improve in neuropsychological scores (7.1 ± 13.9 vs 5.1 ± 13.4), p =0.16

- However, improvements were noted in behavioral, QOL, and PSG findings among the T&A cohort

4 y.o. boy with snoring

- 1-2 yrs of progressively worsening snoring
  - Mouth breathing
  - Witnessed partial apnea, no obvious apnea
  - Difficulty waking but no daytime lethargy
- Prone to hyperactivity
- 50%ile on height and weight, no comorbidities
- Audible breathing, 3-4+ tonsils, hyponasal voice
- Would you order sleep study?
When to order sleep study in child with suspected OSA

- With complex co-morbidity
  - Obesity
  - Down’s syndrome
  - craniofacial disorders
  - neuromuscular disorders
  - sickle cell disease

- Discordance between history and physical
  - AAO-HNS Clinical Practice Guidelines, 2011
Physical

- septum/concha bullosa
- inferior turbinates
- polyps?
- nasopharynx: adenoid?
Radiologic Work-Up

- Plain films: useful for adenoid
- CT/MRI/sleep endoscopy
  - diagnosis in doubt or surgery contemplated
  - Residual OSAS in complicated cases
    - also study nasopharynx, septum, craniofacial structure
A sleepy teenager
Visit #1: Chief Complaint

- A 15 year old adolescent male with obesity was referred to the Sleep Disorders Center to evaluate for:
  - Difficulty breathing during sleep
  - Decline in school performance (always sleepy)
  - Weight gain
The child’s mother states that the problem began about 2 years ago when she started noticing that he has gained weight.

His progress at school deteriorated secondary to his daytime sleepiness.

His mother states that she always had noticed him snoring mostly after his toddler years.
Sleep schedule

- Sleeps 8-9 hours a night
  - Bed time: around 10 pm
  - Wakes up around 6-6:30 am
- Mother states that he snores and has problems with excessive daytime sleepiness
- She has observed him waking up from his sleep, choking, gasping for air, and at times has observed apneas during his sleep
- He sleeps in his own room
- Nocturnal awakenings: twice to go to the bathroom
- Also states having 2 hour naps during the day after coming back from school
- He states that he does fall asleep during his class at times
Review of systems: Sleep

- The child denies any symptoms of orthopnea or paroxysmal nocturnal dyspnea
- He denies, somniloquy, somnambulism, night terrors, recurrent/frequent nightmares, or nocturnal enuresis
- In addition there are no symptoms consistent with seizures, cataplexy, sleep paralysis or hynagogic hallucinations
- There are no symptoms consistent with restless leg syndrome or periodic limb movements during sleep
Family history

- Mother has diabetes
- Father’s medical history is not known
- Has one older brother and three older sisters who are alive and healthy
Physical Exam

- **Vital signs:**
  BP= 117/68, Pulse 93/min, RR=14 /min, Temp=96.7  SpO2= 100%, Wt= 73.1 kg (**90**th percentile), Ht=163.1 cm, BMI = **27.5** (**96**th percentile)

- **General:** The patient is in no apparent distress

- **Oropharynx:** tonsils 3+, Mallampati score of 2, mild oropharyngeal crowding

- **HEENT:** PERRL, EOMI, nares patent, tympanic membranes normal

- **Skin:** mild acanthosis on the neck

- **Heart:** RRR, S1/S2

- **Lungs:** CTAB

- **Abd:** soft NT, obese

- **Ext:** no c/c/e

- **Neuro:** grossly nonfocal
1st Sleep Study

09/09/2009
SpO2

Obstructive hypopnea
## Sleep Architecture

<table>
<thead>
<tr>
<th>Stages</th>
<th>Time (h:m:s)</th>
<th>% Sleep Time</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wake</td>
<td>14.5</td>
<td>3.9%</td>
<td>1-2%</td>
</tr>
<tr>
<td>Stage 1</td>
<td>15.0</td>
<td>48.4%</td>
<td>45-50%</td>
</tr>
<tr>
<td>Stage 2</td>
<td>89.0</td>
<td>23.1%</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.0</td>
<td>0.0%</td>
<td>15-25%</td>
</tr>
<tr>
<td>REM</td>
<td>94.5</td>
<td>24.5%</td>
<td>20%</td>
</tr>
</tbody>
</table>

### Respiratory Statistics (Normal Values):

- Apnea Hypopnea Index / Respiratory Disturbance Index (<5/hr): 33.2
- Central Apnea Index (<1/hr): 13.1
- Obstructive Apnea Index (<1/hr): 0.9
- Hypopnea Index: 18.1
- Snore Intensity: severe
- Baseline Oxygen Saturation (%): 100
- Minimum Oxygen Saturations (≥92%) (%): <<75
- Baseline ETCO2 (torr): 49
- Max ETCO2 (<53 torr) (torr): 60

### Respiratory Event Durations

<table>
<thead>
<tr>
<th></th>
<th>Apnea</th>
<th>Hypopnea</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>NREM</td>
<td>REM</td>
</tr>
<tr>
<td>Average (seconds)</td>
<td>12.9</td>
<td>14.0</td>
</tr>
</tbody>
</table>
PSG comments

INDICATIONS FOR POLYSOMNOGRAPHY: Kevin is a 15 year old male with a history of snoring, observed apnea during sleep, as well as obesity. A full night polysomnogram was performed to evaluate for sleep disordered breathing. This was reported to be a usual night of sleep.

SLEEP ARCHITECTURE: In terms of Kevin's sleep architecture, this was an abnormal study. The sleep onset latency was normal, the sleep efficiency was normal, but the arousal-awakening index was increased.

RESPIRATORY EVENTS: From a respiratory standpoint, this was an abnormal study. Kevin demonstrated severe snoring with paradoxical breathing. Throughout his study, the baseline oxygen saturation was 100% and the lowest oxygen saturation was <=75%. CO2 was also recorded with a peak of 60 torr. Kevin had a Respiratory Disturbance Index / Apnea Hypopnea Index of 33.2, a Hypopnea Index of 18.1 events per hour and an Obstructive Apnea Index of 0.9 events per hour. These events were both obstructive and central in nature.

RECOMMENDATIONS:
1. Follow up with Dr. Braun-Courville.
2. Refer to ENT to evaluate the need for T&A.
3. Repeat sleep study, after T&A. If T&A not performed, repeat sleep study for titration with noninvasive positive pressure ventilation.
4. Consider referral to RD to help with weight management.
5. Consider evaluation for gastroesophageal reflux.
Follow up visit (#2)

- Patient is s/p T&A
- States that he is feeling much awake during the day and more energetic
- His mother states that he has decreased snoring recently
- Plan to repeat a sleep study in 3 months
2nd Sleep Study (3 months post T&A)

2/19/2010
### Method of Study:

<table>
<thead>
<tr>
<th>EEG (C3/A2, C4/A1)</th>
<th>x</th>
<th>EUG (night/day)</th>
<th>x</th>
<th>BMG (chin)</th>
<th>x</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMG (leg)</td>
<td>x</td>
<td>ERG</td>
<td>x</td>
<td>Nasal-Urinal Thermistor</td>
<td>x</td>
</tr>
<tr>
<td>Respiratory Inductance Plethysmography</td>
<td>x</td>
<td>SpO2 (oximetry)</td>
<td>x</td>
<td>ETCO2</td>
<td>x</td>
</tr>
<tr>
<td>Nasal pressure</td>
<td>x</td>
<td>RIP Sum</td>
<td>x</td>
<td>Oxygen</td>
<td>no</td>
</tr>
</tbody>
</table>

### Sleep Statistics (Normal Values):

<table>
<thead>
<tr>
<th>Time in Bed (h.m.s.)</th>
<th>437.1</th>
<th>COUNT</th>
<th>INDEX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Sleep Time (h.m.s.)</td>
<td>419.0</td>
<td>Awakenings: 18</td>
<td>2.6</td>
</tr>
<tr>
<td>Sleep Latency (&lt;30 min)(h.m.s.):</td>
<td>4:5</td>
<td>Arousals: 139</td>
<td>19.9</td>
</tr>
<tr>
<td>Sleep Efficiency (&gt;85%):</td>
<td>95.9%</td>
<td>REM Latency (&lt;120 min): 7:30</td>
<td></td>
</tr>
<tr>
<td>Lights Off:</td>
<td>09:46:53 PM</td>
<td>Lights On:</td>
<td>05:04:00 AM</td>
</tr>
</tbody>
</table>

### Sleep Architecture

<table>
<thead>
<tr>
<th>Stages</th>
<th>Time (h.m.s)</th>
<th>% Sleep Time</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wake</td>
<td>18:5</td>
<td>15.5%</td>
<td>&lt; 4%</td>
</tr>
<tr>
<td>Stage 1</td>
<td>18:5</td>
<td>4.4%</td>
<td>1-2%</td>
</tr>
<tr>
<td>Stage 2</td>
<td>169.5</td>
<td>40.5%</td>
<td>45-50%</td>
</tr>
<tr>
<td>Stage 3</td>
<td>165:5</td>
<td>38.5%</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.0</td>
<td>0.0%</td>
<td>15-25%</td>
</tr>
<tr>
<td>REM</td>
<td>65:5</td>
<td>15.5%</td>
<td>20%</td>
</tr>
</tbody>
</table>

### Respiratory Statistics (Normal Values):

| Apnea Hypopnea Index / Respiratory Disturbance Index (<5/hr): | 8.4 |
| Central Apnea Index (<1/hr): | 0.7 |
| Hypopnea Index: | 7.8 |
| Baseline Oxygen Saturation (%): | 99 |
| Baseline ETCO2 (torr): | 60-63 |
| Obstructive Apnea Index (<1/hr): | 0.1 |
| Snore Intensity: | mild |
| Minimum Oxygen Satuations (≥92%): | 87 |
| Max ETCO2 (<63torr): | 55 |
### Method of Study:

<table>
<thead>
<tr>
<th>EEG (C3/A2, C4/A1)</th>
<th>x</th>
<th>EUG (night/1sett)</th>
<th>x</th>
<th>BMG (chin)</th>
<th>x</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMG (leg)</td>
<td>x</td>
<td>ERG</td>
<td>x</td>
<td>Nasal-Ural Thermistor</td>
<td>x</td>
</tr>
<tr>
<td>Respiratory Inductance Plethysmography</td>
<td>x</td>
<td>SPO2 (oximetry)</td>
<td>x</td>
<td>ETCU2</td>
<td>x</td>
</tr>
<tr>
<td>Nasal pressure</td>
<td>x</td>
<td>RIP Sum</td>
<td>x</td>
<td>Oxygen</td>
<td>no</td>
</tr>
</tbody>
</table>

### Sleep Statistics (Normal Values):

<table>
<thead>
<tr>
<th>Time in Bed (h.m.s.):</th>
<th>437.1</th>
<th>COUNT</th>
<th>INDEX</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total Sleep Time (h.m.s.):</td>
<td>419.0</td>
<td>18</td>
<td>2.6</td>
</tr>
<tr>
<td>Sleep Latency (&lt;30 min) (h.m.s.):</td>
<td>4.5</td>
<td>139</td>
<td>19.9</td>
</tr>
<tr>
<td>Sleep Efficiency (&gt;=85%):</td>
<td>95.9%</td>
<td>REM Latency (&lt;120 min):</td>
<td>73.0</td>
</tr>
<tr>
<td>Lights Off:</td>
<td>09:46:53 PM</td>
<td>Lights On:</td>
<td>05:04:00 AM</td>
</tr>
</tbody>
</table>

### Sleep Architecture

<table>
<thead>
<tr>
<th>Stages</th>
<th>Time (h.m.s.)</th>
<th>% Sleep Time</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wake</td>
<td>18.5</td>
<td>18.5</td>
<td>&lt; 4%</td>
</tr>
<tr>
<td>Stage 1</td>
<td>18.5</td>
<td>4.4%</td>
<td>1.2%</td>
</tr>
<tr>
<td>Stage 2</td>
<td>169.5</td>
<td>40.5%</td>
<td>45-50%</td>
</tr>
<tr>
<td>Stage 3</td>
<td>185.5</td>
<td>38.5%</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.0</td>
<td>0.0%</td>
<td>15-25%</td>
</tr>
<tr>
<td>REM</td>
<td>65.5</td>
<td>15.5%</td>
<td>20%</td>
</tr>
</tbody>
</table>

### Respiratory Statistics (Normal Values):

<table>
<thead>
<tr>
<th>Apnea Hypopnea Index / Respiratory Disturbance Index (&lt;5/hr):</th>
<th>8.4</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central Apnea Index (&lt;1/hr):</td>
<td>0.7</td>
</tr>
<tr>
<td>Hypopnea Index:</td>
<td>7.8</td>
</tr>
<tr>
<td>Baseline Oxygen Saturation (%):</td>
<td>99</td>
</tr>
<tr>
<td>Baseline ETCO2 (torr):</td>
<td>50-53</td>
</tr>
<tr>
<td>Obstructive Apnea Index (&lt;1/hr):</td>
<td>0.1</td>
</tr>
<tr>
<td>Snore Intensity:</td>
<td>mild</td>
</tr>
<tr>
<td>Minimum Oxygen Saturation (&gt;=92%):</td>
<td>87</td>
</tr>
<tr>
<td>Max ETCO2 (&lt;63 torr):</td>
<td>55</td>
</tr>
</tbody>
</table>

Severe <<75
INDICATIONS FOR POLYSOMNOGRAPHY: Kevin is a 15 year old boy with a history of severe OSA who is s/p adenotonsillectomy in 11/09. He then had a much improved sleep study performed the night of the T&A as part of the obesity study. A full night polysomnogram was performed to evaluate for continued sleep disordered breathing. This was reported to be a usual night of sleep.

SLEEP ARCHITECTURE: In terms of Kevin's sleep architecture, this was an abnormal study. The sleep onset latency was normal and the sleep efficiency was normal, however, the arousal-awakening index was increased. In addition, there was a decrease in REM sleep.

RESPIRATORY EVENTS: From a respiratory standpoint, this was an abnormal study. Kevin demonstrated mild snoring with evidence of paradoxical breathing. Throughout his study, the baseline oxygen saturation was 99% and the lowest oxygen saturation was 87%. CO2 was also recorded with a peak of 55 torr. Kevin had a Respiratory Disturbance Index / Apnea Hypopnea Index of 8.4, a Hypopnea Index of 7.6 events per hour and an Obstructive Apnea Index of 0.1 events per hour. These events were mostly obstructive in nature.

RECOMMENDATIONS:
1. Consider follow up sleep study for CPAP / BiPAP titration
2. Follow up in sleep clinic with Dr. Arens to discuss the results of this study

DIAGNOSIS:
Obstructive Sleep Apnea Syndrome, 780.53-0

S/P Adenotonsillectomy
Obesity
Follow up visit (# 3)

- Currently sleeping 8-9 hours at night
- Feeling much energized and rested in the morning
- School grades have improved to 80s
- Eating well balanced diet and has removed fatty and sugary foods from his diet
- Has lost 10 lbs in 2 months with diet and exercise
  - Patient will undergo a repeat sleep study in a few weeks for CPAP titration
3rd Sleep Study (5 months post T&A)

4/22/10

CPAP titration
CPAP titration
Sleep Statistics (Normal Values):

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Count</th>
<th>Index</th>
</tr>
</thead>
<tbody>
<tr>
<td>Time in Bed (h:m:s)</td>
<td>401.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Sleep Time (h:m:s)</td>
<td>347.0</td>
<td>17</td>
<td>2.9</td>
</tr>
<tr>
<td>Sleep Latency (&lt;30 min) (h:m:s)</td>
<td>2:5</td>
<td>44</td>
<td>7.6</td>
</tr>
<tr>
<td>Sleep Efficiency (&gt;85%)</td>
<td>86.5%</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lights Off:</td>
<td>10:43:33 PM</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Lights On:</td>
<td>05:24:42 AM</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Sleep Architecture

<table>
<thead>
<tr>
<th>Stages</th>
<th>Time (h:m:s)</th>
<th>% Sleep Time</th>
<th>Normal Values</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wake</td>
<td>55.0</td>
<td>&lt; 4%</td>
<td></td>
</tr>
<tr>
<td>Stage 1</td>
<td>17.0</td>
<td>4.9%</td>
<td>1-2%</td>
</tr>
<tr>
<td>Stage 2</td>
<td>112.5</td>
<td>32.4%</td>
<td>45-50%</td>
</tr>
<tr>
<td>Stage 3</td>
<td>142.0</td>
<td>40.9%</td>
<td>5%</td>
</tr>
<tr>
<td>Stage 4</td>
<td>0.0</td>
<td>0.0%</td>
<td>15-25%</td>
</tr>
<tr>
<td>REM</td>
<td>75.5</td>
<td>21.8%</td>
<td>20%</td>
</tr>
</tbody>
</table>

Respiratory Statistics (Normal Values):

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Apnea Index (1/hr)</th>
<th>Snore Intensity</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apnea Hypopnea Index / Respiratory Disturbance Index (&lt;5/hr)</td>
<td>2.8</td>
<td></td>
<td>moderate</td>
</tr>
<tr>
<td>Central Apnea Index (&lt;1/hr)</td>
<td>2.6</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Hypopnea Index</td>
<td>0.2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline Oxygen Saturation (%)</td>
<td>97</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Baseline ETCO2 (torr)</td>
<td>-</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

Respiratory Event Durations

<table>
<thead>
<tr>
<th>Event</th>
<th>Duration (torr)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>-</td>
</tr>
</tbody>
</table>
INDICATIONS FOR POLYSOMNOGRAPHY: Kevin is a 15 year old Male with a history of obesity and sleep fragmentation s/p T&A 11/09. He had CPAP titration performed.

SLEEP ARCHITECTURE: In terms of Kevin's sleep architecture, this was a normal study. The sleep onset latency was normal, the sleep efficiency was normal, and the arousal-awakening index was normal.

RESPIRATORY EVENTS: From a respiratory standpoint, this was a mildly abnormal study. Kevin demonstrated moderate snoring with evidence of paradoxical breathing. The baseline oxygen saturation was 97% and the lowest oxygen saturation was 92%. CO2 was not recorded. Kevin had a Respiratory Disturbance Index/ Apnea-Hypopnea Index of 2.8 events/hour, a Hypopnea Index of 0.2 and an Obstructive Apnea Index of 0 events/hour. These events were mostly central in nature. He was started on a CPAP of 4cm H2O and titrated up to 7cm H2O with improvement in his snoring. He had several brief central events at this pressure with associated arousals but no desaturations.

RECOMMENDATIONS:
1. Start CPAP at 7cm H2O with small Resmed nasal micro mirage mask with CFlex @3 and chin strap
2. Follow up with Dr. Arens to discuss the results of this study

DIAGNOSIS:
Obstructive Sleep Apnea Syndrome, 780.53-0

obesity 278.00
Summary

- OSAS is a common disorder in children and has significant co-morbidities
- Polysomnography provides an important and objective tool to detect and monitor OSAS
- There are multiple causes for OSAS in children including anatomical and functional factors
- The epidemic of obesity impacts on the prevalence of OSAS today
- Treatment needs to be directed toward the underlying cause of the disorder and may include: adenotonsillectomy or other surgical procedures, CPAP, weight management, or all of the above
Acknowledgments

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The Children’s Hospital at Montefiore
- Sin SH
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- Khan UI
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- Lipton ML
- Shifteh K
- Agarwal C
- Coupey S
- John Bent

The Children’s Hospital of Philadelphia
- McDonough JM
- Palmer J
- Dominguez T
- Costarino AT
- Mahboubi S
- Meyer H
- Corbin AM
- Rubin NK
- Zhao H
- Uong E

University of Pennsylvania
- Udupa JK
- Liu J
- Maislin G
- Schwab RJ
- Pack AI

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- Guez A
- Wootton DM
- Persak S
- Xu C

NIH RO1 HL-62408, RO1 HD-53693, RO1 HL-105212
Thank You!