Obstructive Sleep Apnea
Diagnosis and Management

TEXAS ACADEMY OF FAMILY PRACTICE
NOVEMBER 10, 2018
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Speaker Disclosure

- Dr. Brown has disclosed that he has no actual or potential conflict of interest in relation to this topic.
Learning Objectives

By the end of this activity, the participant will be better able to:

1. Identify common symptoms and risk factors for obstructive sleep apnea (OSA).
2. Describe how to screen and diagnose OSA.
3. Explain the different treatment options for OSA.
4. Identify the different co-morbidities that are associated with OSA.
“The stupid-lazy child who frequently suffers from headaches at school, breathes through his mouth instead of his nose, snores and is restless at night, and wakes up with a dry mouth in the morning, is well worthy of the solicitous attention of the school medical officer.”

“Chronic enlargement of the tonsillar tissues is an affection of great importance, and may influence in an extraordinary way the mental and bodily development of children.”

“At night the child’s sleep is greatly disturbed; the respirations are loud and snorting, and there are sometimes prolonged pauses, followed by deep, noisy inspirations.”

The Discovery of Sleep Apnea

- Sleep apnea was discovered independently by Gastaut, et al. in France, and Jung and Kuhlo in Germany. These findings were reported in 1965. These findings were widely ignored in America.
- The frequently cited study of Burwell and colleagues (1956) used Joe from the Pickwick Club but erred badly in that they evaluated their somnolent obese patients only during waking and attributed the cause of the somnolence to hypercapnia.
- There is no evidence that hypercapnia causes true somnolence but is associated with impaired cerebral function. However, the term Pickwickian became an instant success and probably stimulated more interested in research.
- 1972, Christian Guilleminault joined Stanford. He had extensive experience with the European studies of sleep apnea. Until he arrived, Stanford did not routinely monitor respiratory and cardiac functioning. They became routine in 1972.
A clinical discipline can only be said to exist if it represents an organized body of knowledge, and if this body of knowledge can be effectively taught. (Dement)

Birthday of Sleep Medicine

Three Known Sleep Disorders
• Sleep Apnea
• Restless Legs Syndrome
• Narcolepsy
Obstructive Sleep Apnea

1972 – First CME Conference on Sleep
1972 – Tracheostomy only effective treatment
1981 – Sullivan uses CPAP to treat OSA
1981 – Fujita describes UPPP for OSA
1991 – Johns develops Epworth Sleepiness Scale
2008 – Young finds high mortality risk with OSA
2010 – Redline, OSA associated with increased stroke in men

SDB and Mortality: The Wisconsin Sleep Cohort—Young et al., SLEEP, Vol. 31, No. 8, 2008
Mallampati class IV is not useful in ruling in patients with severe OSA, and Mallampati class I is not useful in ruling out OSA in the sleep clinic population. Hukins, J Clin Sleep Med. 2010 Dec 15; 6(6): 545–549.
MRI UPPER AIRWAY

- Normal
- Apnea
Change in Upper Airway During Sleep

- Loss of wake stimulus
- Muscles relax and cross-sectional area is reduced
- Change in AP and lateral dimensions
- AP due primarily to posterior movement of soft palate
- Lateral changes due to thickening of the lateral pharyngeal walls

Schwab, RJ et al. Upper Airway Imaging in Obstructive Sleep Apnea, in Lee-Chiong et al. Sleep Medicine, 2002.

FIGURE 11. Axial retropalatal MRI of a normal subject during wakefulness and sleep. Airway cross-sectional area is reduced during sleep in the lateral and anterior-posterior dimensions. State-dependent narrowing in the lateral dimension is related to thickening of the lateral pharyngeal walls.
Primary Snoring

During snoring, air flow is partially blocked.
OBSTRUCTIVE APNEA

During sleep apnea, air flow is completely blocked.

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• African Americans, Hispanic men, and Pacific Islanders have more OSA than White Europeans.
• Asians have more severe apnea with equal or lower BMI’s.

<table>
<thead>
<tr>
<th>Disorder</th>
<th>ICD-10-CM code</th>
</tr>
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<tbody>
<tr>
<td><strong>Obstructive sleep apnea disorders</strong></td>
<td></td>
</tr>
<tr>
<td>Obstructive sleep apnea, adult</td>
<td>G47.33</td>
</tr>
<tr>
<td>Obstructive sleep apnea, pediatric</td>
<td>G47.33</td>
</tr>
<tr>
<td><strong>Central sleep apnea syndromes</strong></td>
<td></td>
</tr>
<tr>
<td>Central sleep apnea with Cheyne-Stokes Breathing</td>
<td>RO6.3</td>
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<tr>
<td>Central apnea due to a medical Disorder without Cheyne-Stokes breathing</td>
<td>G47.37</td>
</tr>
<tr>
<td>Central sleep apnea due to high altitude periodic breathing</td>
<td>G47.32</td>
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<tr>
<td>Central sleep apnea due to a medication or Substance</td>
<td>G47.39</td>
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<td>Primary central sleep apnea</td>
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<tr>
<td>Primary central sleep apnea of infancy</td>
<td>P28.3</td>
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<tr>
<td>Primary central sleep apnea of prematurity</td>
<td>P28.4</td>
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<td>Treatment-emergent central sleep apnea</td>
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<tr>
<td><strong>Sleep related hypoventilation disorders</strong></td>
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<tr>
<td>✓ Obesity hypoventilation syndrome</td>
<td>E66.2</td>
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<td>✓ Congenital central alveolar hypoventilation syndrome</td>
<td>G47.35</td>
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<tr>
<td>✓ Late-Onset central hypoventilation with hypothalamic Dysfunction</td>
<td>G47.36</td>
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<tr>
<td>✓ Idiopathic central alveolar hypoventilation</td>
<td>G47.34</td>
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<tr>
<td>✓ Sleep related hypoventilation due to a medication of Substance</td>
<td>G47.36</td>
</tr>
<tr>
<td>✓ Sleep related hypoventilation due to a medical disorder</td>
<td>G47.36</td>
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<tr>
<td><strong>Sleep related hypoxemia disorder</strong></td>
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<tr>
<td>✓ Sleep related hypoxemia</td>
<td>G47.36</td>
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<td><strong>Isolated symptoms and normal variants</strong></td>
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<tr>
<td>✓ Snoring</td>
<td>R06.83</td>
</tr>
<tr>
<td>✓ Catathrenia</td>
<td></td>
</tr>
</tbody>
</table>

ICSD, International Classification of Sleep Disorders
PSG of Obstructive Apnea

- No airflow with continued effort
- Must be at least 10 seconds
- No desaturation requirement
- No arousal requirement
Obstructive Sleep Apnea
Hypopneas

- Reduced airflow at nose and mouth
- Associated with a 4% desaturation
- Non-Medicare can be 3% desaturation or an arousal
Central Apnea

- No airflow and no effort
- 10 seconds for adult, 2 breaths for children
- No desaturation or arousal requirement
Complex Apnea (Treatment Emergent Apnea)

Clear Obstructive apneas on Diagnostic Study

Central Sleep Apneas appear with any CPAP pressure
Severe Sleep Apnea

55 YO WM BMI = 44.1
ESS: 18

Rx: Insulin Glargine
Insulin Aspart
Furosemide

AHI = 115.2
Supine AHI = 122.6
Min O2 = 50%
Positional/REM Sleep Apnea

![Graph showing positional REM sleep apnea metrics](image)
### TABLE 2

**STOP-BANG test for predicting probability of obstructive sleep apnea**

At least three positive responses are consistent with a high pretest probability of obstructive sleep apnea:

- **Snore:** Have you been told that you snore?
- **Tired:** Are you often tired during the day?
- **Obstruction:** Do you know if you stop breathing or has anyone witnessed you stop breathing while you are asleep?
- **Pressure:** Do you have high blood pressure or are you on medication to control high blood pressure?
- **Body mass index:** Is your body mass index > 35 kg/m²?
- **Age:** Are you age 50 or older?
- **Neck:** Do you have a neck circumference > 17 inches (men) or > 16 inches (women)?
- **Gender:** Are you male?

Based on the Stop-Bang, how many questions did you answer yes?

1. None
2. One
3. Two
4. Three or More
Prevalence of OSA

• Severity usually defined by apnea-hypopneas index
• Defining OSA as AHI ≥ 5 events/hr. Wisconsin Cohort study find a prevalence of 24% in men and 9% in women 30 – 60 years old
• Prevalence of OSA with daytime sleepiness is 3 – 7% in adult men and 2 – 5% in adult women
• Prevalence studies are about the same world – wide

Epidemiological aspects of obstructive sleep apnea
Prevalence Increasing with Obesity

- Related to obesity – as global obesity epidemic spreads, prevalence will go up.
- Recent re-examination of the Wisconsin data adjusted for current levels of obesity, showed a marked increase.
- 34% of men and 17.4% of women have an AHI ≥ 5.
- EDS 14% of men and 5% of women.

Symptoms

- Snoring
- Pauses in breathing (Unless Hypopneas)
- Gasping and choking
- Breakthrough Snores
- Frequent urination
- Excessive daytime sleepiness
- Difficulty falling or staying asleep
- Excessive sweating
- GERD
- Decreased Libido
- Depression/Irritability

Spicuzza L. Et al. Ther Adv Chronic Dis. 2015, Vol. 6(5) 273–285
Common Physical Examination Findings in Obstructive Sleep Apnea

- Obesity (body mass index $\geq 30$ kg/m$^2$)
- Neck circumference $> 17$ inches in men, $> 16$ inches in women
- Deviated nasal septum
- Narrow mandible
- Narrow maxilla
- Dental overbite and retrognathia
- High and narrow hard palate
- Elongated and low-lying uvula
- Prominent tonsillar pillars
- Large tongue
Retrognathia

Risk Factors

- Risk increases with increased body weight and neck circumference
  - Most significant risk factor is being overweight
  - 10% increase in weight is associated with a 6-fold increase in risk of AHI> 15
  - 10% weight reduction can lower risk by 26%
  - Neck Circumference > 17 in in men and >16 in women
- Women have marked increase in apnea after menopause
- Down Syndrome (57 – 94% have at least mild apnea)
- Familial factors
- Smoking and alcohol near bedtime
- PCOS, Hypothyroidism, Pregnancy
What Percentage of your patients diagnosed with sleep apnea are on CPAP?

1. < 25%
2. 26 – 50%
3. 51 – 75%
4. 76 – 100%
What percentage of your apnea patients use CPAP regularly?

1. < 25%
2. 26 – 50%
3. 51 – 75%
4. 76 – 100%
What Percentage of your adult apnea patients have had Upper-airway surgery?

1. < 25%
2. 26 – 50%
3. 51 – 75%
4. 76 – 100%
Sleep Apnea Co-Morbidities

- Drug-Resistant Hypertension: 83%
- Obesity: 77%
- Pacemakers: 59%
- Congestive Heart Failure: 76%
- Atrial Fibrillation: 49%
- Diabetes: 48%
- All Hypertension*: 37%
- Coronary Artery Disease*: 30%

*Male subjects only

References:
- Logan et al., J Hypertens 2001
- O’Keeffe & Patterson, Obes Surg 2004
- Garrigue et al., Circulation 2007
- Oldenburg et al., Eur J Heart Fail 2007
- Gami et al., Circulation 2004
- Einhorn et al., Endocr Pract 2007
- Sjostrom et al., Thorax 2002
- Schafer et al., Cardiology 1999
AHI > 30 had 3-fold increase for all cause mortality
• There was a 4 – 5 fold increase if take out CPAP users
• Strong cardiovascular link if remove CPAP users.
• Indication that CPAP lowers blood pressure.

n = 1522

Wisconsin Sleep Cohort Study 18 year Follow-Up

OSA and Stroke Risk

- 8 year prospective study in men and women
- n = 5422 with 2462 men and 2960 women
- Men with moderately severe OSA had an almost 3-fold increase in ischemic stroke
- This was not seen in women
- Risk of stroke increased 6% for every increment of AHI from 5 – 25
- Age and AHI were the only significant risk factors after adjusting for confounds
- Reductions in cerebral tissue hemoglobin saturation levels occur with apneas and that the severity of tissue deoxygenation correlates with length of the respiratory disturbance and degree of related desaturation
- Marked surges in systemic blood pressure occur with each apneic and hypopneic event, followed by abrupt drops in systemic blood pressure
- Parallel large fluctuations in cerebral blood flow velocity
- Atrial fibrillation is estimated to increase the risk of stroke by twofold or more and moderate to severe OSA increases the risk of atrial fibrillation by fourfold

Symptoms of OSA in Children

<table>
<thead>
<tr>
<th>SLEEP</th>
<th>WAKEFULNESS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snoring</td>
<td>Poor school performance</td>
</tr>
<tr>
<td>Witnessed apnea</td>
<td>Aggressive behavior</td>
</tr>
<tr>
<td>Choking noises</td>
<td>Hyperactivity</td>
</tr>
<tr>
<td>Increased work of breathing</td>
<td>Attention deficit disorder</td>
</tr>
<tr>
<td>Paradoxical breathing</td>
<td>Excessive daytime sleepiness</td>
</tr>
<tr>
<td>Enuresis</td>
<td>Morning headaches</td>
</tr>
<tr>
<td>Mouth breathing</td>
<td>Age-inappropriate napping</td>
</tr>
<tr>
<td>Restless sleep</td>
<td>Difficult to arouse from sleep</td>
</tr>
<tr>
<td>Diaphoresis</td>
<td>Depression</td>
</tr>
<tr>
<td>Hyperextended neck</td>
<td></td>
</tr>
<tr>
<td>Frequent awakenings</td>
<td></td>
</tr>
<tr>
<td>Dry mouth</td>
<td></td>
</tr>
</tbody>
</table>

Physical Examination in OSAS

General
- Sleepiness
- Obesity
- Failure to thrive

Head
- Swollen mucus membranes
- Deviated septum
- Adenoidal facies
  - Infraorbital darkening
  - Elongated midface
  - Mouth-breathing
- Tonsillar hypertrophy
- High arched palate
- Overjet
- Posterior buccal cross-bite
- Crowded oropharynx
- Macroglossia
- Glossoptosis
- Midfacial hypoplasia
- Micrognathia/retrognathia
- Increased neck circumference

## OSA in Adults vs. Children

<table>
<thead>
<tr>
<th>Symptom</th>
<th>Adult</th>
<th>Child</th>
</tr>
</thead>
<tbody>
<tr>
<td>Snoring</td>
<td>Alternating with pauses</td>
<td>Continuous</td>
</tr>
<tr>
<td>Daytime Sleepiness</td>
<td>Main Symptom</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Obesity</td>
<td>Very Common</td>
<td>Infrequent</td>
</tr>
<tr>
<td>Mouth Breathing</td>
<td>Usually not</td>
<td>Common</td>
</tr>
<tr>
<td>Male Preponderance</td>
<td>Yes (8-10:1)</td>
<td>No (1:1) prepubertal</td>
</tr>
<tr>
<td>Enlarged Tonsils</td>
<td>Uncommon</td>
<td>Most Common</td>
</tr>
<tr>
<td>Surgery as treatment</td>
<td>Select cases, Minority, UPPP</td>
<td>Most Cases, T&amp;A</td>
</tr>
<tr>
<td>CPAP</td>
<td>Most Common</td>
<td>Select Cases, Minority</td>
</tr>
</tbody>
</table>
Down Syndrome

- OSAS – 54 – 100%
- Physical Factors
  - Small Midface and Cranium
  - Narrow Nasopharynx
  - Large Tongue
  - Muscular Hypotonia
  - Obesity
  - Small Larynx
- Congenital heart disease exists in up to 56% of DS patients
- DS patients with OSA tend to have worse cognitive outcomes than those without
- Between 50% and 75% of patients with OSA and DS will have clinically significant residual disease post-Adenotonsillectomy
- Roughly two-thirds of patients continued to use CPAP after the 12-month trial ended

Craniofacial Abnormalities

- Mandibular Hypoplasia
- Pierre-Robin (85% of Infants)
- Maxillary Hypoplasia
- Treacher-Collins (95%)
- Crouzon Syndrome (50%)

Patented Devices

Figure 3. One of many devices invented to stop snoring.
Treatment Strategies

- Position training
- Medication:
  - Protriptyline
  - SSRI’s
  - Modafinil
  - Leukotriene antagonist
- Weight Loss
- Oral Appliance
- EPAP
- Oral Pressure Therapy
- Nasal PAP
- Surgical Intervention
- Upper-Airway stimulation
NASAL CPAP

- CPAP
- Cflex/EPR
- BiPAP
- Autoset
- Adaptive Servo-Ventilation (ASV)
CPAP Pressure and Upper Airway

FIGURE 14. Axial retropalatal MRI of a normal subject (same subject as in Figure 13) with CPAP ranging from 0 to 15 cm H$_2$O. Progressive increases in CPAP result in airway enlargement and thinning of the lateral pharyngeal walls. The distance between the lateral pharyngeal fat pads does not change with incremental increases in CPAP.

Schwab, RJ et al. Upper Airway Imaging in Obstructive Sleep Apnea, in Lee-Chiong et al. Sleep Medicine, 2002.
Split Night Sleep Study

38 YO WM, Snoring, Observed pauses in breathing, Daytime Sleepiness, Epworth = 19
BMI = 40.6 Kg/m²

Pre-CPAP
AHI = 88 episodes/hr., Low O₂ = 81%

Post-CPAP
AHI = 21 episodes/hr., Low O₂ = 80%
CPAP Pressure of 11cm H₂O
AHI = 0/episodes/hr., Low O₂ = 90%
CPAP Pressure of 10 cm H₂O
AHI = 0 episodes/hr., Low O₂ = 92%
Weight Loss and OSA over 1 Year

- Subjects who lost 10 kg or more had a reduction in AHI of 11.3 events/hr.
- After 1 year, in the stable group, the AHI actually increased
- Shows that apnea can worsen with time

A Randomized Study on the Effect of Weight Loss on Obstructive Sleep Apnea Among Obese Patients With Type 2 Diabetes: The Sleep AHEAD Study Arch Intern Med. 2009 September 28; 169(17): 1619–1626
Bariatric Surgery and AHI

Bariatric Surgery

- Surgical weight loss resulted in reductions of AHI in nearly all patients.
- The majority of patients showed residual apnea.
- Reliance on weight loss as a “cure” may lead to inappropriate cessation of CPAP.
- Subjects who thought their snoring was resolved were at highest risk of discontinuing CPAP.
- CPAP Pressure Changed from 11.5 to 8.4 cm H$_2$O
• OA therapy has proved effective over the past 10 years in treating patients with OSA, by reducing the apnea and hypopnea index (AHI), improving oxygen saturation during sleep, reducing snoring
• In all studies, CPAP showed better results than OAs in bringing the AHI <10
• several clinical studies that compared OA with UPPP and demonstrated the superiority of OA with 78% reduction in OSA in OA
• AHI decreased from 31 ep/hr. to 14 ep/hr. n = 2724

• **Expiratory Positive Airway Pressure**
• **One Way Valve** – causes pressure on expiration that opens the upper airway
• The high expiratory resistance results in positive pressure throughout exhalation, which splints open the upper airway, making it more resistant to collapse on subsequent inspiration.
• The nasal EPAP device significantly reduced the AHI and improved subjective daytime sleepiness compared to the sham treatment in patients with mild to severe OSA with excellent adherence.
Upper-Airway Stimulation

- Neurostimulator implanted in chest
- Delivers electrical stimulation to the hypoglossal nerve
- Synchronized with ventilation by sensing lead

Upper-Airway Stimulation

Figure 1—Multilevel upper airway improvement with stimulation during drug-induced sedation endoscopy. The outlined areas demonstrate the increase in cross-sectional area of both the retropalatal and retroglossal portions of the upper airway with hypoglossal nerve stimulation.

Figure 5—Titration of upper airway stimulation therapy during polysomnography (PSG). PSG snapshot showing an approximately 6-min respiratory window. The left side of the figure shows periodic airflow limitation, fluctuating respiratory effort, and associated oxygen desaturations consistent with obstructive sleep apnea. Device activation is illustrated by the vertical arrow. After the device synchronizes with ventilatory effort, immediate improvement in control of breathing and oxyhemoglobin is observed.

Upper-Airway Stimulation

- n = 23 in each group
- Therapy withdrawal group had the device turned off for 5 days before PSG

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Baseline</th>
<th>12 months</th>
<th>p value</th>
</tr>
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<tbody>
<tr>
<td>AHI</td>
<td>32</td>
<td>15.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>ESS</td>
<td>11.6</td>
<td>7.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>FOSQ</td>
<td>14.3</td>
<td>17.3</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>% TST&lt; 90%</td>
<td>8.7</td>
<td>5.9</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

Median 11.0 6.0 –4.0
Interquartile range 8.0 to 15.0 4.0 to 10.0 –8.0 to –1.0
Percentage of sleep time with oxygen saturation <90%
8.7±10.2 5.9±12.4 –2.5±11.1 0.01
Median 5.4 0.9 –2.2
Interquartile range 2.1 to 10.9 0.2 to 5.2 –6.6 to –0.3
Pillar Implants

- Stiffens the soft palate, decreasing its flutter and stabilizing the retro-palatal airway. This is accomplished in a nondestructive way by the placement of small woven inserts into the soft palate, under local anesthetic.
- After refusal or failure of CPAP; First-line therapy for interested patients who meet criteria
  - Mild to moderate OSA
  - AHI doesn’t significantly improve for most patients
  - Procedure alone may not adequately treat a patient’s OSA