Title of Session

Presenter name
The Current State in OSA Diagnosis and Treatment

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Conflicts of Interest

- Symposium Honorarium from ResMed, Respironics, Fisher-Paykel
- Research medical devices from ResMed, Respironics, Fisher-Paykel
Outline

- OSA in brief
- OSA Diagnosis
- OSA treatment
OSA in brief
Obstructive sleep apnea

“Repetitive upper airway obstruction during sleep resulting in intermittent hypoxia and sleep fragmentation caused by arousals”
Flow proportional to difference between upstream pressure and $P_{crit}$
Effect of sleep on upper airway collapsibility

**Figure 1.** In the Starling resistor model, the collapsible segment of the tube is bound by an upstream and downstream segment with corresponding upstream and downstream pressures (Pus and Pds) and resistances (upstream resistance pressure and downstream resistance; data not shown). See text for further explanation (adapted in part from Gossard et al.). \( V_{\text{max}} = (P_{\text{us}} - P_{\text{crit}}) / R_{\text{us}} \)
DISEASE STATUS | UPPER AIRWAY
---|---
Apnea | 5
Hypopnea | 0
Snoring | -5
Normal | -10

Mechanical Loads

Compensatory Neuromuscular Responses

$P_{CRIT}$ (cmH$_2$O)
Diagnosis

A. The presence of one or more of the following:

- The patient complains of sleepiness, nonrestorative sleep, fatigue, or insomnia symptoms.
- The patient wakes with breathing holding, gasping, or choking.
- The bed partners or other observer reports habitual snoring, breathing interruptions, or both during the patient’s sleep.
- The patient has been diagnosed with hypertension, a mood disorder, cognitive dysfunction, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, or type 2 diabetes mellitus.

B. Polysomnography (PSG) or OCST demonstrates

- Five of more predominantly obstructive respiratory events (obstructive and mixed apneas, hypopneas, or respiratory effort related arousals (RERA)) per hour of sleep during a PSG or per hour of monitoring (OCST).

C. Polysomnography (PSG) or OCST demonstrates

- Fifteen or more predominantly obstructive respiratory events (apneas, hypopneas, or RERAs) per hour of sleep during a PSG or per hour of monitoring (OCST).

International Classification of Sleep Disorders (ICSD)-3 2014
OSA diagnosis
Types of sleep test

• 1. Type 1: full attended polysomnography (≥ 7 channels) in a laboratory setting
• 2. Type 2: full unattended polysomnography (≥ 7 channels)
• 3. Type 3: limited channel devices (usually using 4–7 channels)
• 4. Type 4: 1 or 2 channels usually using oximetry as 1 of the parameters

1. We recommend that clinical tools, questionnaires and prediction algorithms not be used to diagnose OSA in adults, in the absence of polysomnography or home sleep apnea testing. (STRONG)

Screening tool

- Berlin Questionnaire
- Sleep Apnea Clinical Score (SACS)
- Epworth Sleepiness Scale Score
- STOP
- STOP-Bang
<p>| | |</p>
<table>
<thead>
<tr>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>1. Snoring</strong></td>
<td></td>
</tr>
<tr>
<td>Do you snore loudly (louder than talking or loud enough to be heard through closed doors)?</td>
<td>Yes/No</td>
</tr>
<tr>
<td><strong>2. Tired</strong></td>
<td></td>
</tr>
<tr>
<td>Do you often feel tired, fatigued, or sleepy during daytime?</td>
<td>Yes/No</td>
</tr>
<tr>
<td><strong>3. Observed apnea</strong></td>
<td></td>
</tr>
<tr>
<td>Has anyone observed you stop breathing during your sleep?</td>
<td>Yes/No</td>
</tr>
<tr>
<td><strong>4. Blood pressure</strong></td>
<td></td>
</tr>
<tr>
<td>Do you have or are you treated for high blood pressure?</td>
<td>Yes/No</td>
</tr>
<tr>
<td><strong>5. BMI more than 35 kg/m²?</strong></td>
<td></td>
</tr>
<tr>
<td><strong>6. Age</strong></td>
<td></td>
</tr>
<tr>
<td>Age over 50 yr old?</td>
<td>Yes/No</td>
</tr>
<tr>
<td><strong>7. Neck circumference</strong></td>
<td></td>
</tr>
<tr>
<td>Neck circumference greater than 40 cm?</td>
<td>Yes/No</td>
</tr>
<tr>
<td><strong>8. Gender</strong></td>
<td></td>
</tr>
<tr>
<td>Gender male?</td>
<td>Yes/No</td>
</tr>
</tbody>
</table>

**High risk of OSA**: answering yes to three or more items

**Low risk of OSA**: answering yes to fewer than three items
• Seventeen studies including 9,206 patients met criteria for the systematic review
• In the sleep clinic population, the sensitivity was 90%, 94% and 96% to detect any OSA (AHI >5), moderate-to-severe OSA (AHI >15), and severe OSA (AHI>30) respectively
• The corresponding NPV was 46%, 75% and 90%
• **Specificity was 49%, 30%, and 25%**

- STOP-Bang may not be good predictor for young and lack of hypertension population
- A prospective cross-sectional study was performed among young doctors <40 years old with HSAT

<table>
<thead>
<tr>
<th>Table 4</th>
<th>The predictors for obstructive sleep apnea</th>
</tr>
</thead>
<tbody>
<tr>
<td>Predictor of OSA (AHI ≥5/h)</td>
<td>Odds ratio</td>
</tr>
<tr>
<td>Snoring</td>
<td>34.5</td>
</tr>
<tr>
<td>Male gender</td>
<td>18.8</td>
</tr>
<tr>
<td>Perception of inadequate sleep</td>
<td>7.4</td>
</tr>
</tbody>
</table>

2. We recommend that polysomnography, or home sleep apnea testing with a technically adequate device, be used for the diagnosis of OSA in uncomplicated adult patients presenting with signs and symptoms that indicate an increased risk of moderate to severe OSA. (STRONG)

Uncomplicated patients

1. Not suspecting non-obstructive sleep-disordered breathing (e.g., central sleep apnea, hypoventilation and sleep related hypoxemia) such as significant cardiopulmonary disease, potential respiratory muscle weakness due to neuromuscular conditions, history of stroke and chronic opiate medication use
Uncomplicated patients

- 2. Not suspecting other sleep disorders e.g. central hypersomnolence, parasomnias, sleep related movement disorders or interfere with accuracy of HSAT (e.g., severe insomnia)
Uncomplicated patients

3. Environmental or personal factors that preclude the adequate acquisition and interpretation of data from HSAT
Increased risk of moderate to severe

- Indicated by the presence of excessive daytime sleepiness and at least two of the following three criteria: habitual loud snoring, witnessed apnea or gasping or choking, or diagnosed hypertension

A technically adequate HSAT device incorporates a minimum of the following sensors: nasal pressure, chest and abdominal respiratory inductance plethysmography, and oximetry, or else PAT with oximetry and actigraphy.

A technically adequate diagnostic test includes a minimum of 4 hours of technically adequate oximetry and flow data obtained during a recording attempt that encompasses the habitual sleep period.
Buy Home Sleep Test

Sale!

$12,000.00
$8,900.00

Home Sleep Test (HST) is a sleeping test service at your own home for maximum comfortable. Alternatively, you can choose to be tested at the hospitals where we provide the service, contact us to find more details.

Add to cart

Portable Monitoring (Sleep Test Level 3)
$5,500.00
$3,900.00

Buy Home Sleep Test
$12,000.00
$8,900.00
Peripheral Arterial Tone
PAT signal

Sympathetic activation

Digital vasoconstriction

PAT Amplitude

PAT attenuation

$\alpha$- receptors

Sympathetic activation
Baseline PAT attenuates with transition from stage II to REM
An HSAT should not be used for general screening of asymptomatic clinical populations

Diagnosis, assessment of treatment efficacy, and treatment decisions must not be based solely on automatically scored HSAT data

The raw data from the HSAT device must be reviewed and interpreted by a physician who is either board certified in sleep medicine or overseen by a board certified sleep medicine physician

3. We recommend that if a single home sleep apnea test is negative, inconclusive, or technically inadequate, polysomnography be performed for the diagnosis of OSA. (STRONG)

4. We recommend that polysomnography, rather than home sleep apnea testing, be used for the diagnosis of OSA in patients with significant cardiorespiratory disease, potential respiratory muscle weakness due to neuromuscular condition, awake hypoventilation or suspicion of sleep related hypoventilation, chronic opioid medication use, history of stroke or severe insomnia. (STRONG)
5. We suggest that, if clinically appropriate, a split-night diagnostic protocol, rather than a full-night diagnostic protocol for polysomnography be used for the diagnosis of OSA. (WEAK)

6. We suggest that when the initial polysomnogram is negative and clinical suspicion for OSA remains, a second polysomnogram be considered for the diagnosis of OSA. (WEAK)

1A. Hypopnea
   a. The peak signal excursions drop by ≥30%
   b. The duration of the ≥30% drop in signal excursion is ≥10 seconds
   c. There is a ≥3% oxygen desaturation or the event is associated with an arousal

1B. Hypopnea
   a. The peak signal excursions drop by ≥30%
   b. The duration of the ≥30% drop in signal excursion is ≥10 seconds
   c. There is a ≥4% oxygen desaturation

Calibration Model for Apnea-Hypopnea Indices: Impact of Alternative Criteria for Hypopneas

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Moderate to severe OSA 22%→45%
Severe OSA 8%→18%
Normal 48%→17%

Figure 1—Distribution of obstructive sleep apnea (OSA) severity using different hypopnea criteria to determine apnea-hypopnea index (AHI). OSA classification: normal AHI < 5; mild 5 ≤ AHI < 15; moderate ≤ AHI < 30; severe ≥ 30.

Are the ICSD-3 criteria for sleep apnoea syndrome too inclusive?

Consistent with the findings of Heinzer and colleagues, the ICSD-3 criteria identified a high prevalence of obstructive sleep apnoea syndrome even though we used a higher AHI cut-off and more conservative hypopnoea scoring criteria. A number of cardiovascular and metabolic disorders included in the ICSD-3 criteria might not be causally related to obstructive sleep apnoea. For many of these disorders, evidence that they improve with treatment of obstructive sleep apnoea is either weak or completely absent. We agree with Heinzer and colleagues that symptoms should only be considered when they cannot be attributed to other factors and that the ICSD-3 criteria need revision.
OSA treatment
OSA

Narrow/crowded/collapsible upper-airway

Low arousal threshold

High loop gain

Required breathing level

Ventilatory disturbance

Poor muscle responsiveness

EMG_DTA

EMG_Law

Epiglottic pressure

Airflow

EEG
Continuous positive airway pressure (CPAP)
Pressurized airflow is generated with fan-driven or turbine systems, adjustable by varying valve diameter or turbine speed.
Nowsaday CPAP
Nowaday CPAP mask
1. We recommend that clinicians use PAP, compared to no therapy, to treat OSA in adults with excessive sleepiness. (STRONG)

2. We suggest that clinicians use PAP, compared to no therapy, to treat OSA in adults with impaired sleep-related quality of life. (CONDITIONAL)

3. We suggest that clinicians use PAP, compared to no therapy, to treat OSA in adults with comorbid hypertension. (CONDITIONAL)

4. We recommend that PAP therapy be initiated using either APAP at home or in-laboratory PAP titration in adults with OSA and no significant comorbidities. (STRONG)

5. We recommend that clinicians use either CPAP or APAP for ongoing treatment of OSA in adults. (STRONG)

6. We suggest that clinicians use CPAP or APAP over BPAP in the routine treatment of OSA in adults. (CONDITIONAL)
7. We recommend that educational interventions be given with initiation of PAP therapy in adults with OSA. (STRONG)

8. We suggest that behavioral and/or troubleshooting interventions be given during the initial period of PAP therapy in adults with OSA. (CONDITIONAL)

9. We suggest that clinicians use telemonitoring-guided interventions during the initial period of PAP therapy in adults with OSA. (CONDITIONAL)

Mandibular advancement device (MAD)
Mandibular advancement device (MAD)

- Adjustable-MAD is recommended in CPAP/BPAP intolerance or the patients who preferred other treatment options
- May be used in combination with CPAP in CPAP pressure intolerance

Mandibular advancement device (MAD)

- Overall a-MAD is less effective in reducing overall RDI compared to CPAP
- However, in mild OSA, a-MAD may be as effective as CPAP

Remotely Controlled Mandibular Positioner (RCMP)
Remotely Controlled Mandibular Positioner (RCMP)

- Effective target protrusive position (ETPP) as measured by the use of RCMP was significantly associated with success of MAD therapy.
- RCMP might be a promising instrument for predicting MAD treatment outcome and targeting the degree of mandibular advancement needed.

Uvulopalatopharyngoplasty (UPPP)
In patients having difficulty with other treatments, surgical procedures for the nose and throat can be a beneficial alternative.

Surgical therapy can also be effective when used as an adjunct to improve tolerance and success with CPAP or an oral appliance.
Drug-induced sleep endoscopy (DISE)

- DISE is an additional method to reveal obstruction sites that have not been detected in awake patients.
- DISE demonstrated the importance of identifying multilevel obstruction, especially in relation to retrolingual and laryngeal collapse in OSAS.

Drug-induced sleep endoscopy (DISE)

- Greater sedative depth increased upper airway collapsibility under DISE assessment
- DISE under Bispectral Index (BIS)-guided propofol infusion, and especially a level of 65–75, offers an objective and reproducible method to evaluate upper airway collapsibility

Maxillomandibular advancement surgery (MMA)
Table 2. Rates of Surgical Success or Cure by Preoperative AHI Severity

<table>
<thead>
<tr>
<th>Surgical Successa</th>
<th>Preoperative AHI Cohort, Events/h</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>&lt;30 (n = 61)</td>
</tr>
<tr>
<td>AHI cure, No. (%)</td>
<td>34 (55.7)b</td>
</tr>
<tr>
<td>AHI Success-10, No. (%)</td>
<td>47 (77.0)b</td>
</tr>
<tr>
<td>AHI Success-15, No. (%)</td>
<td>51 (83.6)c</td>
</tr>
<tr>
<td>AHI Success-20, No. (%)</td>
<td>51 (83.6)d</td>
</tr>
</tbody>
</table>

- Preoperative AHI of fewer than 60 events/h was the factor most strongly associated with the highest incidence of surgical cure.

- Patients with high residual RDI and AHI scores (despite prior treatments by means of uvulopalatopharyngoplasty, partial glossectomy, and/or nasal surgery) are highly likely to benefit from management of OSA by means of MMA.
Positional therapy
Data from Asians revealed prevalence of positional OSA to be 67% in which almost of these patients (47%), RDI was normalized during non-supine position.

Prior studies demonstrated equal efficacy compared to CPAP in mild positional OSA who demonstrated AHI<5 in non-supine position.

Long term compliance monitoring is a major problem.

However; recent study up to 1 year follow up demonstrated mean usage of 7.3 ± 0.9 h/night and 69 ±26% of the nights

Furthermore, 75% of the patients reported a better sleep quality since the start of SPT treatment

Weight reduction

- Weight reduction in recommended in **ALL** OSA patients who are overweight or obese
- However, current evidence demonstrated that in moderate to severe OSA, weight reduction should not be the sole treatment for OSA
- Scarce data was observed with resolution of OSA in mild group with weight reduction

Bariatric surgery is superior to conventional therapy in terms on weight loss.

There was also a trend towards greater reduction in AHI.

Hypoglossal nerve stimulation

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Baseline</th>
<th>12 Months</th>
<th>Change</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Primary outcomes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>AHI score†</td>
<td>32.0±11.8</td>
<td>15.3±16.1</td>
<td>−16.4±16.7</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Median</td>
<td>29.3</td>
<td>9.0</td>
<td>−17.3</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>23.7 to 38.6</td>
<td>4.2 to 22.5</td>
<td>−26.4 to −9.3</td>
<td></td>
</tr>
<tr>
<td>ODI score‡</td>
<td>28.9±12.0</td>
<td>13.9±15.7</td>
<td>−14.6±15.8</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Median</td>
<td>25.4</td>
<td>7.4</td>
<td>−15.7</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>19.5 to 36.6</td>
<td>3.5 to 20.5</td>
<td>−24.0 to −8.6</td>
<td></td>
</tr>
<tr>
<td><strong>Secondary outcomes</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>FOSQ score§</td>
<td>14.3±3.2</td>
<td>17.3±2.9</td>
<td>2.9±3.1</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Median</td>
<td>14.6</td>
<td>18.2</td>
<td>2.4</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>12.1 to 17.1</td>
<td>16.2 to 19.5</td>
<td>0.7 to 4.7</td>
<td></td>
</tr>
<tr>
<td>Epworth Sleepiness Scale score¶</td>
<td>11.6±5.0</td>
<td>7.0±4.2</td>
<td>−4.7±5.0</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Median</td>
<td>11.0</td>
<td>6.0</td>
<td>−4.0</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>8.0 to 15.0</td>
<td>4.0 to 10.0</td>
<td>−8.0 to −1.0</td>
<td></td>
</tr>
<tr>
<td>Percentage of sleep time with oxygen saturation &lt;90%</td>
<td>8.7±10.2</td>
<td>5.9±12.4</td>
<td>−2.5±11.1</td>
<td>0.01</td>
</tr>
<tr>
<td>Median</td>
<td>5.4</td>
<td>0.9</td>
<td>−2.2</td>
<td></td>
</tr>
<tr>
<td>Interquartile range</td>
<td>2.1 to 10.9</td>
<td>0.2 to 5.2</td>
<td>−6.6 to −0.3</td>
<td></td>
</tr>
</tbody>
</table>

Upper airway stimulation maintained a sustained benefit on patient-reported outcomes (ESS, FOSQ, snoring) at 48 months in select patients with moderate to severe OSA.

However, prior studies utilized rigorous inclusion criteria including moderate to severe OSA (AHI, 20-65), failure of CPAP therapy, body mass index (BMI) <32 kg/m2, and absence of complete circumferential palatal collapse on DISE.

Muscle training

- A systematic review and meta-analysis that includes data from nine studies involving a total of 120 adult patients showed that oropharyngeal training reduces the AHI by approximately 50% and increases nadir oxygen saturation by > 2.5%

Some pre-clinical works identified the main cause of sleep-related hypotonia of the pharyngeal muscles to be the central reduction of norepinephrine from wakefulness to sleep.

Inhibitory effect of acetylcholine through muscarinic receptors was found to be responsible for the REM-related hypotonia.

Taranto-Montemurro L, et al. The Combination of Atomoxetine and Oxybutynin Greatly Reduces Obstructive Sleep Apnea Severity: A Randomized, Placebo-Controlled, Double-Blind Crossover Trial. Am J Respir Crit Care Med. 2018
A trial investigated for the first time the efficacy of the combination of a noradrenergic (atomoxetine) and an antimuscarinic (oxybutynin) on OSA severity and on the responsiveness to esophageal pressure swings of the genioglossus muscle versus placebo.

Taranto-Montemurro L, et al. The Combination of Atomoxetine and Oxybutynin Greatly Reduces Obstructive Sleep Apnea Severity: A Randomized, Placebo-Controlled, Double-Blind Crossover Trial. Am J Respir Crit Care Med. 2018
20 people completed a randomized, placebo-controlled, double-blind, crossover trial

comparing one night of atomoxetine 80mg plus oxybutynin 5mg (ato-oxy) to placebo administered prior to sleep

Taranto-Montemurro L, et al. The Combination of Atomoxetine and Oxybutynin Greatly Reduces Obstructive Sleep Apnea Severity: A Randomized, Placebo-Controlled, Double-Blind Crossover Trial. Am J Respir Crit Care Med. 2018
Ato-oxy lowered AHI by 63% [34-86%], from 28.5 [10.9-51.6] events/h to 7.5 [2.4-18.6] events/h (p<0.001)
Excessively large ventilatory response to very small changes in CO2

This scenario leads to hypocapnia and subsequent reductions in respiratory drive, which can perpetuate recurrent upper airway collapse

Ventilatory Response to CO$_2$

<table>
<thead>
<tr>
<th>Alveolar CO$_2$ mmHg</th>
<th>Pulmonary Ventilation L/min</th>
</tr>
</thead>
<tbody>
<tr>
<td>20</td>
<td>10</td>
</tr>
<tr>
<td>30</td>
<td>20</td>
</tr>
<tr>
<td>40</td>
<td>30</td>
</tr>
<tr>
<td>50</td>
<td>40</td>
</tr>
<tr>
<td>10</td>
<td>50</td>
</tr>
</tbody>
</table>

PAO$_2$ = 37
PAO$_2$ = 47
PAO$_2$ = 110
Oxygen supplementation
Carbonic anhydrase inhibitor

<table>
<thead>
<tr>
<th>Variable</th>
<th>β</th>
<th>SEM</th>
<th>Odds ratio</th>
<th>p-value</th>
<th>Interpretation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Constant</td>
<td>-1.97</td>
<td>1.02</td>
<td></td>
<td>0.01</td>
<td></td>
</tr>
<tr>
<td>Loop gain</td>
<td>15.41</td>
<td>7.40</td>
<td>3.7</td>
<td>0.038</td>
<td>Higher loop gain → success</td>
</tr>
<tr>
<td>V\text{passive}</td>
<td>5.27</td>
<td>3.71</td>
<td>4.8</td>
<td>0.15</td>
<td>Reduced collapsibility → success</td>
</tr>
<tr>
<td>Compensation</td>
<td>15.09</td>
<td>6.62</td>
<td>45.5</td>
<td>0.023</td>
<td>Greater compensation → success</td>
</tr>
<tr>
<td>V\text{passive}\times\text{compensation}</td>
<td>-58.53</td>
<td>29.97</td>
<td>0.11</td>
<td>0.036</td>
<td>Poor collapsibility and poor compensation → failure</td>
</tr>
<tr>
<td>Loop gain\times\text{compensation}</td>
<td>-80.34</td>
<td>34.16</td>
<td>0.17</td>
<td>0.019</td>
<td>Low loop gain and poor compensation → failure</td>
</tr>
<tr>
<td>Arousal threshold\times\text{compensation}</td>
<td>-86.43</td>
<td>29.53</td>
<td>0.012</td>
<td>0.003</td>
<td>Low arousal threshold and higher compensation → success</td>
</tr>
</tbody>
</table>

- Hypnotic sleep promotion agents to increase the respiratory arousal threshold (experimental)
- Zopiclone increased the respiratory arousal threshold versus placebo without impairing genioglossus muscle activity or its responsiveness

Conclusion

- Polysomnography is still a standard diagnostic tool for OSA
- However; in uncomplicated cases, HSAT may be considered
- CPAP is currently a standard treatment for OSA
- Personalized OSA treatment focusing on anatomical therapies, muscle function therapies, loop gain therapies, and arousal threshold therapies are new targeted therapies for OSA
THANK YOU FOR YOUR ATTENTION

narichac@hotmail.com
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