Sleep Apnea and Congestive Heart Failure, Diagnosis and Management

Rami Kahwash, MD
Presentation Outline

- Overview of sleep disordered-breathing (SDB)
- Pathophysiology of the cardiovascular consequences of OSA and CSA
- Treatment of OSA - Focus on the cardiovascular impact of treatment
- Treatment of CSA
Case History

♥ 61 y/o commercial driver presents to the clinic with:
  ♥ Excessive daytime sleepiness
  ♥ Snoring and pauses in breathing reported by the wife

♥ Difficult to concentrate on the job!
Sleep History

♥ Seven and half hours of allowed sleep per night
♥ Tired in the morning
♥ Awakens 3-4 times at night to “use rest room”
♥ Persistent loud snoring
♥ Leg jerks and kicks- restless sleep
Case History

♥ MHX:
♥ Hypertension
♥ Hypercholesterolemia

♥ ROS:
♥ 35 lbs weight gain/past two year

♥ SoHx:
♥ 40 Pack/year
NEXT STEP

POLYSYMNOGHRAPHY
Obstructive Apnea: partial or complete cessation of upper airway despite continued efforts to breath

Blood oxygen levels reduce to ≤ 4% of baseline value
Central sleep apnea: cessation of upper airway flow without respiratory effort.
Sleep Medicine Nomenclatures

♥ Apnea: complete cessation of airflow > 10 seconds
♥ Hypoapnea: decrease in airflow > 30 %, longer than 10 sec, associated with decrease in O2 sat > 4 % or arousal
♥ AHI: number of events per hour
  ♥ 5 - 15 Mild
  ♥ 15 - 30 moderate
  ♥ > 30 severe
# Prevalence of Obstructive Sleep Apnea

The Wisconsin Sleep Cohort, NEJM 1993

The Occurrence of Sleep-Disordered Breathing among random 602 Middle-Aged Adults.

<table>
<thead>
<tr>
<th>Age (yr)</th>
<th>Women</th>
<th></th>
<th></th>
<th>Men</th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>APNEA–HYPOPNEA SCORE</td>
<td></td>
<td></td>
<td>APNEA–HYPOPNEA SCORE</td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>≧5</td>
<td>≧10</td>
<td>≧15</td>
<td>≧5</td>
<td>≧10</td>
<td>≧15</td>
</tr>
<tr>
<td>30–39</td>
<td>6.5 (1.4–11)</td>
<td>4.9 (0.6–9.8)</td>
<td>4.4 (1.1–7.3)</td>
<td>17 (9.6–25)</td>
<td>12 (5.4–19)</td>
<td>6.2 (1.9–10)</td>
</tr>
<tr>
<td>40–49</td>
<td>8.7 (4.2–13)</td>
<td>4.9 (1.7–8.1)</td>
<td>3.7 (1.0–6.5)</td>
<td>25 (18–32)</td>
<td>18 (11–24)</td>
<td>11 (6.7–16)</td>
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<tr>
<td>50–60</td>
<td>16 (5.2–26)</td>
<td>5.9 (0.0–12)</td>
<td>4.0 (0.0–10)</td>
<td>31 (21–40)</td>
<td>14 (7.5–20)</td>
<td>9.1 (5.1–13)</td>
</tr>
<tr>
<td>30–60*</td>
<td>9.0 (5.6–12)</td>
<td>5.0 (2.4–7.8)</td>
<td>4.0 (1.5–6.6)</td>
<td>24 (19–28)</td>
<td>15 (12–19)</td>
<td>9.1 (6.4–11)</td>
</tr>
</tbody>
</table>

*Values are adjusted to the age distribution of the survey population.
Prevalence in Middle Aged Adults

<table>
<thead>
<tr>
<th>Condition</th>
<th>% Men</th>
<th>% Women</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI ≥ 5</td>
<td>24</td>
<td>9</td>
</tr>
<tr>
<td>AHI ≥ 5 + daytime somnolence</td>
<td>4</td>
<td>2</td>
</tr>
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</table>

AHI = Apnea Hypopnea Index

Young; NEJM 1993
Prevalence of OSA in Stable Outpatients with Heart Failure

<table>
<thead>
<tr>
<th>Study</th>
<th>Year</th>
<th>Prevalence</th>
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<tbody>
<tr>
<td>Oldenburg 2007</td>
<td></td>
<td>36%</td>
</tr>
<tr>
<td>Schulz 2007</td>
<td></td>
<td>43%</td>
</tr>
<tr>
<td>Macdonald 2008</td>
<td></td>
<td>30%</td>
</tr>
<tr>
<td>Paulino 2009</td>
<td></td>
<td>57%</td>
</tr>
<tr>
<td>Khayat 2009</td>
<td></td>
<td>57%</td>
</tr>
<tr>
<td>Bitter 2009</td>
<td></td>
<td>48%</td>
</tr>
<tr>
<td>7</td>
<td></td>
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</tbody>
</table>
Physical Examination in OSA

- Malampati Class (I-IV)
- **Obesity and thick neck**
  - > 17 inch males
  - > 16 inch females
- **Craniofacial anatomy**
  - Inferiorly positioned hyoid bone
  - Mandibular insufficiency
  - Increased mid-facial height
- **Nasal obstruction**
Mallampati Class: Independent Predictor of the Presence and Severity of OSA

Nuckton et al, Sleep 2006

<table>
<thead>
<tr>
<th>Variable</th>
<th>Odds Ratio (95% CI)</th>
<th>p value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mallampati score, (I-IV)</td>
<td>2.5 (1.2, 5.0)</td>
<td>.01</td>
</tr>
<tr>
<td>(per 1-point increase)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Neck circumference,</td>
<td>1.9 (1.0, 3.5)</td>
<td>.04</td>
</tr>
<tr>
<td>(per 2.5-cm increase)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Witnessed apnea</td>
<td>1.9 (1.2, 3.1)</td>
<td>&lt; .01</td>
</tr>
<tr>
<td>Hypertension</td>
<td>4.9 (1.2, 20)</td>
<td>.03</td>
</tr>
</tbody>
</table>

Mallampati class
Macroglossia

Tongue ridging

Lateral Narrowing

SCHELLENBERG et al
AJRCCM 2000
Overjet

Nasion

Retrognathia

Gnathion

SCHELLENBERG et al
AJRCCM 2000
Cardiovascular Consequences - Pathophysiology

Figure 2—Proposed pathophysiologic interactions between OSA, cardiovascular risk mechanisms and overt cardiovascular disease. In OSA, disordered breathing events result in acute stressors (left) which, over time, may promote intermediary mechanisms (blue circle) that could contribute to clinical cardiovascular disease (green circle). Interactions with other risk factors (orange circle) may dictate individual phenotypic outcomes or the cardiovascular response to OSA treatment.

OSA AND Hypertension

♥ 40% of patients with OSA have hypertension
♥ 50% of patients with hypertension have OSA
♥ OSA patients are more likely to be nocturnal “non-dippers”
♥ Treatment of OSA reduces blood pressure
OSA-Induced Hypertension-Animal Model

OSA and CHF-the Sleep Heart Health


n=6,424
SBD and Heart Failure in Clinical Practice

Congestive heart failure

Diastolic dysfunction

Coronary disease or hypertension

Systolic dysfunction

OSA

CSA
Treatment of OSA

- Weight loss
- Positive airway pressure
- Surgery
- Oral appliances
- Oxygen
Effects of CPAP

♥ Respiratory Effects:

♥ Elimination of upper airway obstruction
♥ Unloading of respiratory muscles
♥ Improved work of breathing
♥ Improved gas exchange and elimination of desaturation
Causative Role of Severe Untreated OSA in Cardiovascular Events
mean F/u 10 YR
(Marin et al, Lancet 365:1046, 2005)
CPAP Improves LVEF in Patients with CHF

CPAP group had a significant absolute increase of 8.8±1.6 percent in the left ventricular ejection fraction (P<0.001)

Kaneko et al, NEJM 348:1233, 2003
CSA : Treatment

- Management of CHF
- Supplemental Oxygen
- Acetazolamide
- Theophylline
- Pacemaker
- Heart Transplantation
CANPAP

♥ 258 patients age 18-79 with heart failure, ejection fraction < 40%, and central sleep apnea despite optimal medical therapy

♥ Randomized. Mean age 63 years. Baseline ejection fraction 24.5%

<table>
<thead>
<tr>
<th>Nocturnal CPAP†</th>
<th>No CPAP</th>
</tr>
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<tbody>
<tr>
<td>Titrated as tolerated to 10 cmH₂O</td>
<td>n=130</td>
</tr>
<tr>
<td>n=128</td>
<td></td>
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</tbody>
</table>

Endpoints (mean follow-up 2 years):
♥ Primary: Death or heart transplantation
♥ Secondary: Apnea hypopnea index, quality of life

† CPAP was used an average of 4 hours per day during the trial

CANPAP

Primary Endpoint: Death or hospitalization

\[ p = 0.54 \]

Average sleep time was 304 minutes in the CPAP group and 308 minutes in the control group. Apnea hypopnea index at baseline was 40 apneas/hour.

There was no difference in the frequency of death or hospitalization between groups or in the cumulative number of hospitalizations (\( p = 0.83 \)).
SDB and Cardiovascular Diseases- Conclusions

♥ OSA has a causative relation with hypertension
♥ OSA is an independent risk factor for:
  ♥ Heart failure
  ♥ CAD
  ♥ Stroke
  ♥ Cardiac arrhythmia
♥ OSA causes worse outcomes in stroke, CAD, and Afib. Main therapy is CPAP.
♥ CSA coexist with heart failure and aggravate its control. Main therapy is optimizing HF control
Thank You