Consequences of Sleep Disordered Breathing from the SHHS, WSCS, NHHS

Andrew G Veale
New Medical Knowledge

- Observe a problem
- Describe the problem
- Look for associations
- Find a test
- Work out a treatment
- Treat more patients (on research money)
- Ask for more money
- Management notice
- Epidemiology - size of the problem
- Show the treatment works
- Determine why the treatment works
- Randomize to prove the treatment works
- Show that it saves money
Lofty
Anatomy
Pathophysiology

- Abnormally narrowed airway
- Increased collapsibility
- Airway collapse – multi-level problem
  - Palate, base of tongue, pharynx, supraglottis or all levels
- Increased effort
- Sympathetic outpouring
- Desaturation
- Arousal
Patient complains of:
- Poor sleep quality
- Always tired
- Impotence
- Headaches
- Enuresis
- GE reflux

Wife complains of:
- Snoring
- Apnoeas
- Mood changes
- Impotence

Doctor should notice:
- Asleep in waiting room
- Difficult Hypertension
- Difficult diabetes
- Congestive cardiac failure
- Obesity
- Known associations (Hypothyroidism, Acromegally, Abnormal facies, Ehlers Danlos Syndr. Etc)
## Epidemiology - Obstructive Sleep Apnea Syndrome

<table>
<thead>
<tr>
<th>Reference, Country</th>
<th>Methods</th>
<th>Subjects, N</th>
<th>Age (yr)</th>
<th>Criteria</th>
<th>Prevalence, %</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lavie (1983), 163</td>
<td>Questionnaire. PSG</td>
<td>1262 (m)</td>
<td>18-67</td>
<td>AI &gt;10, symptomatic</td>
<td>1.0-5.9</td>
</tr>
<tr>
<td>Peter et al. (1985), 164</td>
<td>Questionnaire. PSG</td>
<td>354 (m)</td>
<td>25-55</td>
<td>AI &gt;10, symptomatic</td>
<td>2.3</td>
</tr>
<tr>
<td>Telakivi et al. (1987), 42</td>
<td>Questionnaire. PSG</td>
<td>1939 (m)</td>
<td>30-69</td>
<td>Snoring, EDS, and RDI &gt;10</td>
<td>0.4-1.4</td>
</tr>
<tr>
<td>Gislason et al. (1988), 41</td>
<td>Questionnaire. PSG</td>
<td>3201 (m)</td>
<td>30-69</td>
<td>AI &gt;10, symptomatic</td>
<td>0.7-1.9</td>
</tr>
<tr>
<td>Cirignotta et al. (1989), 156</td>
<td>Questionnaire. PSG</td>
<td>1170 (m)</td>
<td>40-59</td>
<td>AI &gt;10, symptomatic</td>
<td>3.4-5.0</td>
</tr>
<tr>
<td>Telakivi et al. (1987), 42</td>
<td>Questionnaire. PSG</td>
<td>1939 (m)</td>
<td>60-69</td>
<td>AI &gt;10, symptomatic</td>
<td>0.5-1.1</td>
</tr>
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<td>Questionnaire. PSG</td>
<td>354 (m)</td>
<td>30-67</td>
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<td>0.7-1.9</td>
</tr>
<tr>
<td>Gislason et al. (1993), 49</td>
<td>Questionnaire. PSG</td>
<td>555 children</td>
<td>6 mo to 6 yr</td>
<td>Snoring or apnea &amp; ODI4 &gt;3</td>
<td>2.9</td>
</tr>
<tr>
<td>Esnaola et al. (1995), 170</td>
<td>Questionnaire. PSG</td>
<td>1077 (m)</td>
<td>30-70</td>
<td>AHI ≥15</td>
<td>15.3</td>
</tr>
<tr>
<td>Hayon et al. (1997), 171</td>
<td>Telephone (Sleep-EVAL)</td>
<td>2078 (m)</td>
<td>35-64</td>
<td>AHI &gt;10</td>
<td>13.4</td>
</tr>
<tr>
<td>Kripke et al. (1997), 172</td>
<td>Telephone, oximeter, snoring</td>
<td>165 (m)</td>
<td>35-64</td>
<td>AHI &gt;10 and EDS</td>
<td>6.5-9.1</td>
</tr>
<tr>
<td>Bixler et al. (1998), 38</td>
<td>Telephone. PSG (sample)</td>
<td>4364 (m)</td>
<td>20-100</td>
<td>AHI &gt;10 and clinical criteria</td>
<td>All: 3.3</td>
</tr>
</tbody>
</table>
Symptoms, Risk Factors

Source: Random sample of 10,000 NZ adults aged 30-60 yrs, 71% response rate. Data courtesy of Dr Ricci Harris.
4% $O_2$ Desaturations / Hour

- Random sample from electoral roll, 30-60 years
- 169 Maori, 195 non-Maori
- Overnight MESAM4 monitoring at home

Data courtesy of Kara Mihaere
# Hypertension

<table>
<thead>
<tr>
<th>STUDY</th>
<th>Age</th>
<th>N</th>
<th>RDI &lt;1</th>
<th>RDI 1-4.9</th>
<th>RDI 5-14.9</th>
<th>RDI &gt;15</th>
<th>RDI &gt;30</th>
</tr>
</thead>
<tbody>
<tr>
<td>WSCS</td>
<td>30-65</td>
<td>709</td>
<td>1.0</td>
<td>1.2</td>
<td>2.0</td>
<td>2.9</td>
<td></td>
</tr>
<tr>
<td>SHHS</td>
<td>40-97</td>
<td>6132</td>
<td>1.0</td>
<td>1.1</td>
<td>1.2</td>
<td>1.3</td>
<td>1.4</td>
</tr>
<tr>
<td>S Penn</td>
<td>20-100</td>
<td>1741</td>
<td></td>
<td>2.3</td>
<td></td>
<td></td>
<td>6.9</td>
</tr>
<tr>
<td>Vitoria</td>
<td>30-70</td>
<td>552</td>
<td>1.0</td>
<td>2.5</td>
<td>1.3</td>
<td></td>
<td>2.3</td>
</tr>
</tbody>
</table>
Hypertension treatment

Prospective study 420,000 over 10 years

<table>
<thead>
<tr>
<th>Reduction of BP</th>
<th>Reduction in Stroke</th>
<th>Reduction in CAD</th>
</tr>
</thead>
<tbody>
<tr>
<td>5mHg</td>
<td>34%</td>
<td>21%</td>
</tr>
<tr>
<td>7.5mmHg</td>
<td>46%</td>
<td>29%</td>
</tr>
<tr>
<td>10mmHg</td>
<td>56%</td>
<td>37%</td>
</tr>
<tr>
<td>CPAP reduction 5 – 10 mmHg</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>
Figure 100-2 Treatment with therapeutic continuous positive airway pressure (CPAP) decreased systemic blood pressure, whereas use of subtherapeutic CPAP failed to decrease blood pressure. (From Becker HF, Jerrentrup A, Ploch T, et al: Effect of nasal continuous positive airway pressure treatment on blood pressure in patients with obstructive sleep apnea. Circulation 2003;107:68-73.)
Recommendation – When to test

- All new hypertensives
- All hypertensive patients not controlled on 1 drug
- All patients with severe HT
- All patients with loss of nocturnal dip in BP
- All patients with diastolic dysfunction
# Pulmonary Hypertension

<table>
<thead>
<tr>
<th>Study</th>
<th>N</th>
<th>Criteria</th>
<th>Prevalence</th>
</tr>
</thead>
<tbody>
<tr>
<td>Chaouat</td>
<td>220</td>
<td>AHI &gt; 20</td>
<td>17% PAP &gt; 20</td>
</tr>
<tr>
<td>Laks</td>
<td>100</td>
<td>AHI &gt; 20</td>
<td>42% PAP &gt; 20 (20-52)</td>
</tr>
<tr>
<td>Sanner</td>
<td>92</td>
<td>AHI &gt; 10 (10-100)</td>
<td>20% PAP &gt; 20</td>
</tr>
</tbody>
</table>

8 had increased PCWP
all were hypertensive

Pre-capillary factors
- Hypoxia, hypercapnia, Intrathoracic pressure changes, Endothelial damage.

Capillary factors
- Reduction of capillary bed from co-morbidities.

Post-capillary factors
- Increased LVEDP
Atrial Fibrillation

- Cardioverted patients with OSA
  - Untreated: 82% recurrence @ 12 mths
  - CPAP treated: 42% recurrence @ 12 mths
  - Non OSA Pts: 53%

Non OSA Pts: 53%
Sudden death

- 46% OSA patients die between 12mn and 8am
- 21% in people without OSA
- RDI >40 have a 40% greater risk of nocturnal death than RDI 5-39
- 5 to 7% risk in non snorers
- 12% risk in habitual snorers
- 20% risk in OSA OR 3.6
- OR 1.4 in SHHS
Figure 97-4 Importance of monitoring nocturnal oxygen saturation in patients who have sustained a myocardial infarction. Nonsustained ventricular tachycardia (lower panel) and hypoxemia measured by pulse oximetry (upper panel) occurred simultaneously in a patient on the third night after infarction. The patient died on the following day of cardiogenic shock. ECG, electrocardiogram. (From Galatius-Jensen S, Hansen J, Rasmussen V, et al: Nocturnal hypoxemia after myocardial infarction: Association with nocturnal myocardial ischaemia and arrhythmias. Br Heart J 1994; 72:23-30.)
Figure 101-1 Incidence of cardiovascular disease during a 7-year follow-up in middle-aged men otherwise healthy at baseline. Fraction of individuals with incidence of cardiovascular disease, hypertension, coronary artery disease (CAD), and cardiovascular event (stroke, myocardial infarction [MI], or cardiovascular death). Depicted are data from patients without OSA (non OSA) as well as from those incompletely or efficiently treated for their sleep and breathing disorder. (Reprinted from Peker Y, Hedner J, Norum J, et al: Increased incidence of cardiovascular disease in middle-aged men with obstructive sleep apnea: A seven-year follow-up. Am J Respir Crit Care 2002;166:159-165.)
Figure 86-1 Sleep apnea and diabetes mellitus.
Figure 86-2 Intermediate pathways linking sleep apnea, glucose intolerance, and insulin resistance.
Figure 96-2 The mechanisms by which sleep apnea may result in endothelial dysfunction and cerebrovascular and cardiovascular disorders. CBF, coronary/cerebral blood flow; Do2, oxygen delivery; HPT, hypertension; ↑, increase; ↓, decrease. (Adapted from Javaheri S: Heart failure and sleep apnea: Emphasis on practical therapeutic options. Clin Chest Med 2003;24:207-222.)
Figure 6—In a cohort of more than 1600 men, a higher incidence of fatal (top) and non-fatal (bottom) cardiovascular events was observed in the 36% with severe OSA who were noncompliant with CPAP treatment. Reproduced with permission from Reference 140.
Quality of Life

- 80% of OSA patients have EDS & cognitive impairment
- 50% of OSA patients report personality changes
- 4% of a random community sample 18 – 84 – Habitual sleepy drivers
- 34% of OSA patients have had a crash in previous 5 years (24% of controls)

18/06/2009
Driving

- OSA in Driving Simulators
  - Increase off road incidents
  - Slower brake reaction time
  - Increased lateral position deviations

18/06/2009
Driving & the Law

- Falling asleep whilst driving = recklessness
  - Maggie’s Law

- Inform the patient that they should not drive and document this in the record. Inform the patient that this advice will be given to the GP. If the patient continues to drive, despite this advice, then the Licensing Authority MUST be notified.
  - But not all patients with OSA will have a crash
  - If they do crash Low speed bunt or high speed fatal
  - Nod off driving, nod of driving daily, run off road or out of lane, previous crash, risk to others, size of vehicle.

18/06/2009
Quality of Life

- Quality of life
- Health related quality of life
- Functional status

- Generic (SF36)
- Disease specific
  - Functional outcomes of sleep questionnaire (FOSQ)
  - Calgary sleep apnoea quality of life instrument (SAQLI)
  - OSA Patient oriented severity index (OSAPOS1)
- Threshold effect between RDI 1 and 15

18/06/2009