What IS Sleep Apnea, Anyway?

Barbara Phillips, MD, MSPH, FCCP

Feb 25, 2017
Disclosures

• Leadership position
  • American College of Chest Physicians
  • National Board of Respiratory Care
• Honorarium
  • CHEST Review, CCM International
  • American Thoracic Society
  • Temple Clinic Baylor
  • NIH
• Expert Witness
  • 3 legal cases about commercial drivers and wrongful death
What is hypopnea, anyway?
Moser NJ, Phillips BA, Berry DT, Harbison L.

Abstract
Quantitation of apneas and hypopneas is routinely included in studies of epidemiology, diagnosis, and treatment of sleep-disordered breathing (SDB). The definition of apnea appears clear-cut in the sleep literature. In contrast, the literature contains remarkable variety in both recording techniques and definitions of hypopnea. The purpose of this study was to characterize the variety in the definitions and techniques used to identify hypopnea in clinical sleep laboratories. One hundred surveys were mailed to 100 accredited sleep laboratories. Each laboratory was asked to provide its criteria and equipment used to define hypopnea. Forty-five surveys (45 percent) were returned. No two laboratories used the same definition and measures of hypopnea. We conclude that there is no consensus about either recording techniques or definitions of hypopnea. Thus, epidemiologic studies and reports of interventions on SDB that do not include precise definitions of hypopnea must be interpreted with caution.

PMID: 8308740
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Overview

• Why does it matter?
• Sources of variability
• Do symptoms matter?
• A modest proposal....
Why Does it Matter?

- Undiagnosed disease can cause morbidity, mortality and crash
- Undiagnosed disease can cost money

......but

- Unnecessary OSA treatment causes anguish
- Unnecessary OSA treatment costs money
- Jobs are at stake
- Over-diagnosis affects the credibility of the field
- Over treatment may have adverse outcomes
**Expected healthcare costs (at 2005/6 prices) over 14 years following no treatment or CPAP**

<table>
<thead>
<tr>
<th>Resource</th>
<th>Expected discounted NHS costs over 14 years at 2005/6 prices following:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No treatment (£)</td>
</tr>
<tr>
<td>Clinician visits for OSAHS</td>
<td>0.00 (0)</td>
</tr>
<tr>
<td>Devices</td>
<td>0.00 (0)</td>
</tr>
<tr>
<td>Diagnostic sleep studies</td>
<td>0.00 (0)</td>
</tr>
<tr>
<td>Resources required to manage cardiovascular events</td>
<td>1044.67 (10)</td>
</tr>
<tr>
<td>Resources required to manage strokes</td>
<td>7203.58 (68)</td>
</tr>
<tr>
<td>Resources required to manage RTAs</td>
<td>2396.77 (23)</td>
</tr>
<tr>
<td>Total</td>
<td>10645.02 (100)</td>
</tr>
</tbody>
</table>
### Does CPAP Save Lives?

**Guest JF Thorax 2008**

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Probability after 14 years following:</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>No treatment</td>
</tr>
<tr>
<td><strong>Survival</strong></td>
<td>0.57 (0.49 to 0.65)</td>
</tr>
<tr>
<td><strong>Cardiovascular event</strong></td>
<td>0.35 (0.20 to 0.53)</td>
</tr>
<tr>
<td><strong>Stroke</strong></td>
<td>0.39 (0.23 to 0.60)</td>
</tr>
<tr>
<td><strong>RTA</strong></td>
<td>0.24 (0.21 to 0.28)</td>
</tr>
<tr>
<td><strong>Event-free survival</strong></td>
<td>0.30 (0.13 to 0.46)</td>
</tr>
<tr>
<td><strong>QALYs</strong></td>
<td>7.22 (6.48 to 7.93)</td>
</tr>
</tbody>
</table>
Cost Effectiveness of CPAP...or Not

- Considering QoL, cost of Rx and motor vehicle crash outcomes, CPAP is “economically attractive” (Ayas NT Arch Intern Med 2006)
- The Incremental cost-effectiveness ratio (ICER) of CPAP is much better than use of statins, LVRS and other common therapies. (Alghanim N Lung, 2007)
- “Overall, this study supports the use of CPAP in older people with OSA syndrome and shows that it would be good value for money in the NHS.” (McMillan A Health Tech Assess 2015)
Health Care Utilization Falls on CPAP

Albarrak M Sleep 2005

N=342 men
Why Does it Matter?

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- Undiagnosed disease can cost money

......but

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- Unnecessary OSA treatment costs money
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U.S. DOT Seeks Input on Screening and Treating Commercial Motor Vehicle Drivers and Rail Workers with Obstructive Sleep Apnea

March 8, 2016

WASHINGTON – The U.S. Department of Transportation’s (DOT) Federal Motor Carrier Safety Administration (FMCSA) and Federal Railroad Administration (FRA) today announced that the agencies are seeking public input during the next 90 days on the impacts of screening, evaluating, and treating rail workers and commercial motor vehicle (CMV) drivers for obstructive sleep apnea (OSA). The National Transportation Safety Board recommended that DOT take action to address OSA screening and treatment for transportation workers.
The Drivers’ Perspective

• “The source of the problems the medical community. They saw this sleep apnea condition as a cash cow for them. Instead of practicing good medicine and letting the bottom line take care of itself by creating very satisfied patients, (Some did). The medical community began putting CPAP on just about anyone who walked through the door. They put CPAP machines on people that may have not needed a machine. Then once they put someone on a machine, they did not do appropriate follow-up. And in some cases, individuals were then reported to their company as being non-compliant and were fired.”
Intermittent Hypoxia (IH)
Rosenzweig I Lancet Respir Med 2015

• Short, mild, lower cycle frequency IH is believed to generate adaptive responses in the brain, ie ischemic preconditioning.

• Chronic severe high frequency IH results in disruption of homeostatic mechanisms and inflammation.

• Genetic mechanisms include hypoxia inducible factor-1, vascular endothelial growth factor, erythropoietin, atrial natriuretic peptide, and brain-derived neurotrophic factor.

• Other mechanisms include facilitation of phrenic motor output, chemoreflex activation, vascular remodeling, neo-angiogenesis, productive autophagy, reactive gliosis, various synaptic changes, and modulation of adult hippocampal neurogenesis.
Overview

• Why does it matter?
• Sources of variability
• Do symptoms matter?
• A modest proposal....
A Working Definition of Sleep Apnea (CMS)

- AHI $\geq 15$

*OR*

- AHI $\geq 5$ with
  - Hypertension
  - Stroke
  - Sleepiness
  - Ischemic heart disease
  - Insomnia
  - Mood disorders
Definitions of Respiratory Events

• Apnea: 10 second or more cessation of airflow
  • Respiratory effort absent = central sleep apnea
  • Respiratory effort present=obstructive sleep apnea

• Hypopneas: reduction of airflow and/or effort with associated reduction in oxygen saturation (SaO$_2$)
Definitions

• AHI = Apneas + Hypopneas
  Total Sleep Time, in Hours
• RDI = AHI, more or less (AASM and CMS definitions vary)
• ODI = # of oxygen desaturations/hr of sleep (typically 3 or 4 % falls)
• SDB = Sleep-Disordered Breathing: What you say when you are not sure what you are including. This term may include snoring, RERA’s, oxygen desaturation, central apneas. You MIGHT say this because you don’t know whether or not it’s sleep apnea, because you are not sure what sleep apnea IS.)
What IS Sleep Apnea?
Redline S, AJRCCM 2000

• Sources of variability include
  • Magnitude of flow change
  • Variations in kinds of sensors used (thermocouples, transducers)
  • Differential use of oxygen desats (3 or 4 %)
  • Cutoffs, eg, 5, 10, 15 or more
  • Requirement of symptoms. Or not.
Scoring Apneas and Hypopneas AASM Scoring Manual (Iber C, AASM, 2007)

• Apneas are measured with a thermistor
  – Signal drops by 90% of baseline
  – For at least 10 seconds
  – *Obstructive* if effort persists by RIP; *central* if not; *mixed* if effort resumes before airflow

• Hypopneas are measured with a pressure transducer
  – Signal drops by $\geq 30\%$ of baseline with a $\geq 4\%$ desat OR
  – Signal drops by $\geq 50\%$ of baseline with a $\geq 3\%$ desat or the event is associated with an arousal.
“Chicago Criteria” for Hypopneas

Iber C, AASM Scoring 2007

• ≥ 50% reduction in respiratory amplitude for ≥ 10 sec
• or
• ≥ 20% reduction in respiratory amplitude for ≥ 10 sec associated with ≥ 3% decrease in SpO2  
  or an arousal.
RERA: Respiratory Effort-related Arousal
Guilleminault, 1993

A sequence of breaths characterized by increasing respiratory effort leading to an arousal from sleep which does not meet criteria for an apnea or hypopnea. These events must fulfill both of the following criteria:

1. Pattern of progressively more negative esophageal pressure, terminated by a sudden change in pressure to a less negative level and an arousal
2. The event lasts 10 seconds or longer.

UARS (Upper Airway Resistance Syndrome): $\geq 5$ RERA’s per hour of sleep
Update in AASM Scoring Rules
(Berry RB JCSM 2012)

• Task force met to reach consensus on scoring in light of reality and new technology.
• Statements are almost completely based on consensus; very little data is presented.
• Addressed scoring, sensors, RERA’s, ODI, event duration, CSR and several complicated issues.
• Defined an alternative sensor is used if the recommended sensor fails or the signal is inaccurate.
### Recommended Sensors
(Berry RB JCSM 2012)

<table>
<thead>
<tr>
<th>Respiratory Parameter</th>
<th>Sensor</th>
</tr>
</thead>
</table>
| Airflow (use both oronasal thermal flow sensor and nasal pressure transducer during diagnostic study) | • Oronasal thermal airflow sensor* (to score apnea in diagnostic study)  
• Nasal pressure transducer** (to score hypopnea in diagnostic study)  
• PAP device flow signal (to score apneas and hypopneas in PAP titration study) |
| Respiratory Effort (select one) | • Esophageal manometry  
• Dual thoracoabdominal RIP belts***  
• Dual thoracoabdominal PVDF belts [Acceptable] in adults |
<p>| Oxygen Saturation | Pulse oximetry |</p>
<table>
<thead>
<tr>
<th>Respiratory Event</th>
<th>Sensor</th>
</tr>
</thead>
<tbody>
<tr>
<td>Apnea (select one)</td>
<td>• Nasal pressure transducer*</td>
</tr>
<tr>
<td></td>
<td>• RIPsum**</td>
</tr>
<tr>
<td></td>
<td>• RIPflow**</td>
</tr>
<tr>
<td></td>
<td>• PVDFsum</td>
</tr>
<tr>
<td></td>
<td>[Acceptable] in adults</td>
</tr>
<tr>
<td></td>
<td>• End-tidal PCO₂</td>
</tr>
<tr>
<td></td>
<td>[Acceptable] in children</td>
</tr>
<tr>
<td>Hypopnea (select one)</td>
<td>• Oronasal thermal airflow sensor***</td>
</tr>
<tr>
<td></td>
<td>• RIPsum**</td>
</tr>
<tr>
<td></td>
<td>• RIPflow**</td>
</tr>
<tr>
<td></td>
<td>• Dual thoracoabdominal RIP belts**</td>
</tr>
<tr>
<td></td>
<td>• PVDFsum</td>
</tr>
<tr>
<td></td>
<td>[Acceptable] in adults</td>
</tr>
</tbody>
</table>
Apnea: a drop in the peak signal excursion by ≥ 90% of pre-event baseline using an oronasal thermal sensor (diagnostic study), PAP device flow (titration study), or an alternative apnea sensor, for ≥ 10 seconds.

Hypopnea: peak signal excursions drop by ≥ 30% of pre-event baseline using nasal pressure, PAP device flow (titration study), or an alternative sensor, for ≥ 10 seconds in association with either ≥ 3% arterial oxygen desaturation or an arousal. Scoring a hypopnea as either obstructive or central is optional.
RERA: a sequence of breaths lasting at least 10 seconds with increasing respiratory effort or flattening of the inspiratory portion of the nasal pressure (diagnostic study) or PAP device flow (titration study) waveform with arousal.
Update in AASM Scoring Rules
(Berry RB JCSM 2012)

- RDI = AHI + RERA index
  - (CMS defines RDI as apneas + hypopneas per hour of monitoring).

- ODI = > 3 % arterial desats/hr

- Arousals with 30% reduction in flow can be hypopneas (CMS does not support hypopneas defined with out fall in SaO2)

- AHI can be defined using reduction in flow with an alternative sensor and an arousal...
Different Hypopnea Definitions Affect Prevalence and Severity of OSA (n≈2000)

Campos-Rodriguez F Sleep Med 2016

Hypopnea Definitions Required a Reduction In Airflow And:

- AHI 4%: 4% desat (CMS and AASM 2007)
- AHI 3%: 3% desat ("Chicago")
- AHI 3%a: 3% desat OR an arousal (AASM 2012)
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Different Hypopnea Definitions Affect Prevalence and Severity of OSA (n=2000, women and elderly)
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Different Hypopnea Definitions Affect Prevalence and Severity of OSA ($n \approx 2000$)  
Campos-Rodriguez F Sleep Med 2016

• “Classification of disease severity varied substantially based on criteria used to define hypopneas.”

• Different criteria affected the association between AHI and cardiovascular outcomes.
  — AHI 3%a>30/hr was associated with increased cardiovascular mortality in women but not in older patients
  — AHI 3%a between 15 and 28/hr was not associated with mortality.

• The less stringent (AHI 3%a) criterion for hypopnea has resulted in increased reported prevalence of OSA
  — In this cohort, 82% of women would be eligible for CPAP

• The less stringent (AHI 3%a) criterion for hypopnea likely weakens the association between “OSA” and cardiovascular risk.
Severity Criteria Based on PSG From the American Academy of Sleep Medicine (Sleep, 1999)

- "Mild" sleep apnea is 5-15 events/hr
- "Moderate" sleep apnea is 15-30 events/hr
- "Severe" sleep apnea is over 30 events/hr

("Events" includes apneas, hypopneas, and RERA’s)
### Which Patient Has “Mild” OSA?

<table>
<thead>
<tr>
<th></th>
<th>Patient 1</th>
<th>Patient 2</th>
</tr>
</thead>
<tbody>
<tr>
<td>AHI (events/hr)</td>
<td>40</td>
<td>10</td>
</tr>
<tr>
<td>Apnea duration (secs)</td>
<td>10-22</td>
<td>10-90</td>
</tr>
<tr>
<td>Lowest SaO2 (%)</td>
<td>90</td>
<td>71</td>
</tr>
<tr>
<td>% REM on study</td>
<td>18</td>
<td>0</td>
</tr>
<tr>
<td>Arousals/hr</td>
<td>8</td>
<td>8</td>
</tr>
<tr>
<td>Cardiac arrhythmias</td>
<td>none</td>
<td>v tach</td>
</tr>
</tbody>
</table>
What About “Simple Snoring?”

- Snoring in pregnancy is associated with increased hypertension and growth retardation, controlling for weight, age, smoking (Franklin, Chest, 2000)
- Snoring is associated with cognitive decline (Quesnot, J Am Geriatric Soc, 1999)
- Snoring medical students are more likely to fail exams, controlling for BMI, age, sex (Ficker, Sleep, 1999).
- Snoring is a risk factor for cardiovascular disease in women. (Hu, J Am Coll Cardiol 2000).
- Snoring is a risk for type II diabetes (Al-Delaimy, Am J Epidemiol 2002).
- Snoring women have faster progression of CAD (Leineweber C. Sleep 2004)
What IS Sleep Apnea?
Redline S, AJRCCM 2000

RDI= Respiratory Disturbance Index, D =% desat, A=arousals, H=hypopneas
RDI-2D= apneas and hypopneas based on amplitude Δ+2% desat
RDI-3D, A= Apneas and hypopneas on basis of amplitude Δ+3% desat +/- arousal
RDI-4H=Hypopneas on basis of 4% desat, apneas on amplitude changes alone
RDI-T = Hypopneas and apneas on the basis of amplitude changes alone
What IS Sleep Apnea?
Redline S, AJRCCM 2000

• Correlations strongest for RDIs that require desats
• Prevalence of “sleep apnea” varied from 10.8% to 82.2% for RDI > 15
  • (and basing events on flow/amplitude alone resulted in nearly the whole cohort having “sleep apnea.”)
• RDIs based on desats predicted symptoms much better than those based on amplitude alone or an arousals.
Sleep Heart Health Study: Apneas and Hypopneas

- Decrease in airflow or chest wall movement to an amplitude smaller than approximately 25% (apnea) or 70% (hypopnea) of baseline
- At least 10 seconds
- Associated with oxyhemoglobin desaturation of 4% or greater as compared with baseline
SHHS’s AHI is really an ODI4

- All events (apneas and hypopneas) required a 4% oxygen desaturation to be counted because
- It was not otherwise possible to achieve acceptable inter-rater reliability based on flow rate or arousals.
## Update in AASM Scoring Rules

**Whitney CW Sleep 1998**  
*(ICC= Interclass Correlation)*

<table>
<thead>
<tr>
<th></th>
<th>Scorers</th>
<th></th>
<th></th>
<th></th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>912</td>
<td>914</td>
<td>915</td>
<td>ICC</td>
<td></td>
</tr>
<tr>
<td>AHI-flow (using flow only)</td>
<td>25.9</td>
<td>32.9</td>
<td>27.4</td>
<td>0.74</td>
<td></td>
</tr>
<tr>
<td>AHI-flow and ≥ 3% desaturation</td>
<td>11.35</td>
<td>9.97</td>
<td>10.8</td>
<td><strong>0.97</strong></td>
<td></td>
</tr>
<tr>
<td>AHI-flow and ≥ 4% desaturation</td>
<td>6.1</td>
<td>5.4</td>
<td>5.75</td>
<td><strong>0.99</strong></td>
<td></td>
</tr>
<tr>
<td>AHI-flow and arousal only</td>
<td>5.6</td>
<td>7.2</td>
<td>6.57</td>
<td>0.77</td>
<td></td>
</tr>
<tr>
<td>AHI-flow and ≥ 3% desaturation or arousal</td>
<td>14.1</td>
<td>14.58</td>
<td>14.06</td>
<td><strong>0.95</strong></td>
<td></td>
</tr>
<tr>
<td>AHI-flow and ≥ 4% desaturation or arousal</td>
<td>9.75</td>
<td>10.8</td>
<td>17.3</td>
<td><strong>0.94</strong></td>
<td></td>
</tr>
<tr>
<td>Arousal index</td>
<td>13.5</td>
<td>20.6</td>
<td>17.3</td>
<td>0.54</td>
<td></td>
</tr>
</tbody>
</table>
Inter-Scorer Reliability
(Malhotra A, Kuna S, Magalang U, Sleep 2013)

• Three different reports of interscorer reliability in research studies.
• Highest is for ODI
• Excellent concordance for AHI using 2007 criteria
• Worse concordance for “new” criteria, for N1 vs N3 sleep, for hypopnea, and for central vs obstructive apneas.
• (Push for automated algorithm scoring is beginning).
Overview

- Why does it matter?
- Sources of variability
- Do symptoms matter?
- A modest proposal....
The Cardinal Symptom of OSA is Sleepiness. And it Matters

- Sleepiness predicts CPAP adherence in OSA (Sawyer A Sleep Med Rev 2011)
  - Although this is not a consistent finding (Turnbull CD J Thoracic Dis 2016; Campos-Rodriguez F, Sleep Med 2016)
- Sleepiness may correlate with PSG severity of OSA (Oksenberg A Laryngoscope 2010; Kapur VK Sleep 2005)
- Sleepiness is the symptom/finding most relieved/prevented with CPAP in OSA (McMillan A Health Technol Assess 2015)
- Sleepiness predicts car crash in OSA (Tregear S, JCSM 2009)
- Sleepiness correlates with cognitive dysfunction (Zhou J Sleep Medicine 2016)
- Sleepiness, regardless of the cause, is associated with all-cause mortality in older people (Emppana JP Stroke 2009)
The Cardinal Symptom of OSA is Sleepiness. And it Matters

- But sleepiness is not the only symptom of OSA
- And comorbidities matter, too.
- Our colleagues are a bit like blind men, in this regard.
The Blind Men and the Elephant

"Hey, the elephant is a pillar," said the first man who touched his leg.
"Oh, no! it is like a rope," said the second man who touched the tail.
"Oh, no! it is like a thick branch of a tree," said the third man who touched the trunk of the elephant.
"It is like a big hand fan" said the fourth man who touched the ear of the elephant.
"It is like a huge wall," said the fifth man who touched the belly of the elephant.
"It is like a solid pipe," Said the sixth man who touched the tusk of the elephant.
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John Godfrey Saxe's (1816-1887) version of the famous Indian legend,
The Specialists and the Sleep Apneic

- “Hey, OSA causes resistant hypertension and heart failure,” said the cardiologist.
- “Oh, no! it causes strokes, dementia and headaches,” said the neurologist.
- “Oh, no! it causes ADHD and poor academic performance,” said the pediatrician.
- “It causes car crash,” said the occ med doc.
- “It exacerbates diabetes, PCOS and the metabolic syndrome,” said the endocrinologist.
- “It does all of this, and MORE, including causing fatigue, insomnia, and depression,” said the sleep clinician.
A Working Definition of Sleep Apnea (CMS)

- $\text{AHI} \geq 15$  \hspace{1cm} \text{OR}

- $\text{AHI} \geq 5$ with \textit{SYMPTOMS OR COMORBIDITIES}
  - Hypertension
  - Stroke
  - Sleepiness
  - Ischemic heart disease
  - Insomnia
  - Mood disorders
Overview

- Why does it matter?
- Sources of variability
- Do symptoms matter?
- A modest proposal...
The wide variation in estimates is likely to reflect the definitions used to quantify the OSA...and the different health status of the older populations studied."

McMillan A Health Technol Assess 2015

<table>
<thead>
<tr>
<th>Reference</th>
<th>n</th>
<th>Female (%)</th>
<th>Age (years)</th>
<th>Population</th>
<th>Prevalence of OSA (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carskadon et al., 1981</td>
<td>40</td>
<td>55</td>
<td>62–86</td>
<td>Community</td>
<td>36</td>
</tr>
<tr>
<td>Coleman et al., 1981</td>
<td>83</td>
<td>28</td>
<td>66 ± 5</td>
<td>Sleep clinic</td>
<td>39</td>
</tr>
<tr>
<td>McGinty et al., 1982</td>
<td>26</td>
<td>0</td>
<td>64.4 ± 4.4</td>
<td>Community</td>
<td>39</td>
</tr>
<tr>
<td>Roehrs et al., 1983</td>
<td>97</td>
<td>0</td>
<td>61–81</td>
<td>Sleep clinic</td>
<td>27</td>
</tr>
<tr>
<td>Smallwood et al., 1983</td>
<td>30</td>
<td>20</td>
<td>50–80</td>
<td>Community</td>
<td>37</td>
</tr>
<tr>
<td>Yesavage et al., 1985</td>
<td>41</td>
<td>0</td>
<td>69.5 ± 6.5</td>
<td>Both</td>
<td>73</td>
</tr>
<tr>
<td>Hoch et al., 1986</td>
<td>56</td>
<td>52</td>
<td>69.3 ± 5.4</td>
<td>Community</td>
<td>5</td>
</tr>
<tr>
<td>Knight et al., 1987</td>
<td>27</td>
<td>NG</td>
<td>75.8 ± 5.9</td>
<td>Primary care</td>
<td>37</td>
</tr>
<tr>
<td>Mosko et al., 1988</td>
<td>46</td>
<td>65</td>
<td>68.7 ± 6.7</td>
<td>Community</td>
<td>28</td>
</tr>
<tr>
<td>Ancoli-Israel et al., 1989</td>
<td>233</td>
<td>65</td>
<td>65–101</td>
<td>Nursing home</td>
<td>70</td>
</tr>
<tr>
<td>Hoch and Reynolds 1990</td>
<td>105</td>
<td>53</td>
<td>60–91</td>
<td>Community</td>
<td>26</td>
</tr>
<tr>
<td>Philips et al., 1992</td>
<td>92</td>
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<td>64.2 ± 8.6</td>
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<td>Ancoli-Israel et al., 1995</td>
<td>346</td>
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<td>72.8 ± 6.1</td>
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<td>Bixler et al., 1998</td>
<td>75</td>
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<td>Young et al., 2002</td>
<td>3448</td>
<td>NG</td>
<td>60–99</td>
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<td>54</td>
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<td>Endeshaw et al., 2004</td>
<td>58</td>
<td>76</td>
<td>77.7 ± 6.7</td>
<td>Community</td>
<td>56</td>
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<td>Haas et al., 2005</td>
<td>3643</td>
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<td>70.2 ± 6.9</td>
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<td>Hader et al., 2005</td>
<td>80</td>
<td>50</td>
<td>74.1 ± 6.3</td>
<td>General clinic</td>
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Do Symptoms Matter?


Abstract

BACKGROUND: Mild obstructive sleep apnea (OSA) is a highly prevalent disorder in adults; however, whether mild OSA has significant neurocognitive and cardiovascular complications is uncertain.

OBJECTIVES: The specific goals of this Research Statement are to appraise the evidence regarding whether long-term adverse neurocognitive and cardiovascular outcomes are attributable to mild OSA in adults, evaluate whether or not treatment of mild OSA is effective at preventing or reducing these adverse neurocognitive and cardiovascular outcomes, delineate the key research gaps, and provide direction for future research agendas.

METHODS: Literature searches from multiple reference databases were performed using medical subject headings and text words for OSA in adults as well as by hand searches. Pragmatic systematic reviews of the relevant body of evidence were performed.

RESULTS: Studies were incongruent in their definitions of "mild" OSA. Data were inconsistent regarding the relationship between mild OSA and daytime sleepiness. However, treatment of mild OSA may improve sleepiness in patients who are sleepy at baseline and improve quality of life. There is limited or inconsistent evidence pertaining to the impact of therapy of mild OSA on neurocognition, mood, vehicle accidents, cardiovascular events, stroke, and arrhythmias.

CONCLUSIONS: There is evidence that treatment of mild OSA in individuals who demonstrate subjective sleepiness may be beneficial. Treatment may also improve quality of life. Future research agendas should focus on clarifying the effect of mild OSA and impact of effective treatment on other neurocognitive and cardiovascular endpoints as detailed in the document.
We Don’t Know What Sleep Apnea Is

• There are several reasons for this
  • Definitions and metrics have changed frequently
  • Definitions and metrics have not been consistently validated.
  • Inter scorer reliability is generally poor unless oximetric measures are included

• Because of this, we don’t know
  • What the true prevalence of sleep apnea is
  • What the consequences of sleep apnea are
  • Who needs to be treated
The Emperor Has No Clothes...
...And Oximetry May Be Good Enough
### OSA and Cancer, Adjusted

(Campos-Rodriguez F AJRCCM 2013)

<table>
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<th>TSat&lt;sub&gt;90&lt;/sub&gt;</th>
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T90 Predicts Inverse Relationship for Cardiovascular Morbidity in OHS Better than AHI (Masa JF CHEST 2016)
T<90, NOT AHI, Predicts Death in HF Patients (n=963, Oldenburg O Eur Heart J, 2016)
We Don’t Know What Sleep Apnea Is  
But Here’s What We Do Know

• Measures of SDB that require a metric of oxygenation are more reliable and predictive of symptoms and outcomes.
• Symptoms and comorbidities matter, especially sleepiness.
• Many people can benefit in many ways from treatment, especially with CPAP. But others don’t.
• CPAP is extraordinarily effective, safe and cheap.
My Proposal

• Define OSA based on a combination of oximetric measures (T90, ODI, LoSat) and symptoms/comorbidities that might be expected to improve with treatment. Collect data. Analyze outcomes prospectively.

• Have a low threshold for initiating CPAP treatment, but maybe also for discontinuing it and trying something else.

• Avoid stigmatizing OSA (a basic public health principle for any prevalent, deadly disease).

• Pay more attention to clinical presentation than to squiggly lines.
Early microscope
CPAP as a Therapeutic Trial
(Senn O Chest 2006, n= 33)

- Auto-titrating CPAP, 4-15 cm H₂O, was used as the therapeutic trial
- A successful trial was “yes” to
  - Are you willing to continue CPAP treatment?
  - Was objective CPAP use > 2 hours/night?
- All underwent PSG; sleep apnea was considered an AHI of > 10
- Excluded were those with CHF, OHS, underlying lung disease, prior CPAP Rx, psych or illness, language problems
- Those who were diagnosed with OSA on basis of TT had same outcomes as in-lab diagnosed.