Sleep Apnea is not on the death certificate

No financial disclosures or conflicts of interest

Richard Marcus M.D.
David returns to Italy after a very successful 20 week, 20 city USA tour
Obesity a cause of OSA

- A 10% increase in body weight is associated with a 30% increase in AHI
- Weight loss improves OSA and survival
STOP BANG

S - Snoring
T - Tiredness
O - Observed apnea
P - High blood-pressure (140/95)
B - BMI (>35)
A - Age (>50)
N - Neck circumference (>17in male, >16in female)
G - Gender (male)
Alternative Scoring Models of STOP-Bang Questionnaire Improve Specificity To Detect Undiagnosed Obstructive Sleep Apnea

Frances Chung, M.B.B.S., Yilang Yang, M.D., Russell Brown, M.D., Pu Liao, M.D.
Department of Anesthesia, Toronto Western Hospital, Women's College Hospital, University of Toronto, Toronto, Ontario, Canada

ABSTRACT

Background
Obstructive sleep apnea (OSA) is common among surgical patients. The STOP-Bang questionnaire is a validated screening tool with a high sensitivity. However, its moderate specificity may yield fairly high false positive rate. We hypothesized that the specific combinations of predicting factors in the STOP-Bang questionnaire would improve its specificity.

Methods
After research ethics approval, consented patients were asked to complete the STOP-Bang questionnaire and then underwent sleep studies. The predictive performance of the STOP-Bang alternative scoring models was evaluated. Five hundred sixteen patients with complete data on the STOP-Bang questionnaire and polysomnography were reported.

Results
When the STOP-Bang score was ≥ 3 (any 3 positive items), the sensitivity and specificity for identifying moderate-severe OSA was 87% and 31%, respectively. The specificity for any 2 positive items from the 4 STOP questions plus BMI > 35 kg/m², male gender, or neck circumference > 40 cm for identifying moderate-severe OSA was 65%, 77%, and 79%, respectively. Compared with STOP-Bang score ≥ 3, the predicted probability for severe OSA of the specific combinations of STOP score ≥ 2 + male and STOP score ≥ 2 + BMI increased by 30% and 42%, respectively. For severe OSA, the specific combination of STOP score ≥ 2 + BMI + male demonstrated a specificity of 97% and 80% increase in predicted probability versus any 4 positive items of STOP-Bang questionnaire.

Conclusions
The specific constellations of predictive factors improved the specificity of STOP-Bang questionnaire. For patients with STOP score ≥ 2, male gender, and BMI > 35 kg/m² were more predictive than age ≥ 50 and neck circumference > 40 cm.

Citation
When to suspect OHS
Often misdiagnosed as COPD

OSA and BMI ≥ 30 kg/m² (n=522)

Serum HCO₃⁻ < 27 mEq/L (n=257)
3% with OHS

Serum HCO₃⁻ ≥ 27 mEq/L (n=265)
50% with OHS

Lowest oxygen saturation during sleep
> 60% or AHI < 100 (n=186)
36% with OHS

Lowest oxygen saturation during sleep
< 60% or AHI > 100 (n=79)
76% with OHS

Mokhlesi B et al. Sleep Breath 2007; 11:117
Pathophysiology of OHS

- Obesity (Increased VCO₂)
  - Leptin resistance
  - Increased mechanical load/Relative weak respiratory muscles
  - Blunted ventilatory response
  - Chronic hypercapnia
- Obstructive sleep apnea
  - Upper airway resistance
  - Acute hypercapnia during sleep
  - Increased serum bicarbonate
- Decreased CO₂ response
- Reduced HCO₃⁻ excretion rate
Sleep Duration and Pain

and BMI, depression, HTN, diabetes, CVD, stroke, reaction times, and cognition!

Primary events
- Hypoxemia/hypercapnia
- Reoxygenation/hypocapnia
- Sympathetic overactivity
- DO₂↓ and ↑ CBF
- Nocturnal and diurnal hypertension
- ↓ and ↑ Wall tension
- Alterations in CBF
- ↑ and ↓

Secondary mechanisms
- Sleep apnea & hypopnea
- Negative swings in intrathoracic pressure

Consequences
- Endothelial Dysfunction
- Systemic HTN
- Pulmonary HTN
- Systolic HF
- Diastolic HF
- Arrhythmias
- Atherosclerosis
- CAD
- Stroke

Source: Javaheri, Clinics in Sleep Medicine, December, 2007
Sleep apnea hypopnea

- \( \uparrow \downarrow O_2 \) delivery
  - Organ dysfunction
- Endothelial dysfunction syndrome
  - Vasoconstriction, thrombosis, inflammation
- Hypoxic and hypercapnic pulmonary vasoconstriction
  - \( \uparrow \) Right ventricular afterload
- Sympathetic activation
  - \( \uparrow \) BP/other adverse effects
- Parasympathetic withdrawal
  - \( \uparrow \) Heart rate
- Transmural pressure of left and right ventricles and of pulmonary microvascular bed
  - Changes in right and left ventricular afterload
  - \( \uparrow \) Lung H₂O

Arousal

\( \downarrow Ppl \)
## Prevalence of SDB in Cardiovascular Disease

<table>
<thead>
<tr>
<th>Disorder</th>
<th>Prevalence of OSA</th>
<th>Prevalence of CSA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hypertension</td>
<td>35%</td>
<td>-</td>
</tr>
<tr>
<td>Drug-resistant Hypertension</td>
<td>65-80%</td>
<td>-</td>
</tr>
<tr>
<td>Heart Failure</td>
<td>12-53%</td>
<td>21-37%</td>
</tr>
<tr>
<td>CAD</td>
<td>30%</td>
<td>-</td>
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<tr>
<td>Atrial Fibrillation</td>
<td>49%</td>
<td>-</td>
</tr>
<tr>
<td>Stroke</td>
<td>60%</td>
<td>12%</td>
</tr>
</tbody>
</table>
Prevalence of OSA in patients with type 2 diabetes

This suggests that nearly 15 million of the estimated 22 million diabetic people in the US will have OSA!
The effect of correction of sleep-disordered breathing on BP in untreated hypertension.

Hla KM, Skatrud JB, Finn L, Patta M, Young T.

Department of Medicine, Section of General Internal Medicine, University of Wisconsin Medical School, 2628 Marshall Court, Suite 100, Madison, WI 53705, USA.

Abstract

OBJECTIVES: To compare BP response to 3 weeks of nasal continuous positive airway pressure (CPAP) in hypertensive patients with and without sleep-disordered breathing (SDB).

DESIGN: A controlled, interventional trial of nasal CPAP in patients with and without SDB. Participants and setting: Twenty-four men, aged 30 to 60 years, with mild to moderate untreated hypertension recruited from employee health and primary care clinics.

METHODS: Based on in-laboratory polysomnography, 14 hypertensive patients had SDB, defined by five or more episodes of apnea and hypopnea per hour of sleep (apnea-hypopnea index [AHI], > 5), and 10 had no SDB (AHI, < 5). We performed 24-h ambulatory BP monitoring on all patients at baseline, during CPAP, and after CPAP treatment. In patients with an AHI > or = 5, nasal CPAP was titrated to reduce the AHI to < 5. Patients with an AHI < 5 cm H2O to control for any potential effect of CPAP per se on BP. Both groups received CPAP for 3 weeks.

RESULTS: After adjusting for age and body mass index, the mean nocturnal systolic and diastolic BP changes after CPAP treatment in the SDB group were significantly different from those in the no-SDB group: -7.8 vs +0.3 mm Hg (p = 0.02), and -5.3 vs -0.7 mm Hg (p = 0.03), respectively. There was a similar, although statistically insignificant, difference in the adjusted mean daytime systolic and diastolic BP changes after CPAP treatment between the two groups (-2.7 vs +0.4 mm Hg and -2.3 vs -1.7 mm Hg, respectively).

CONCLUSIONS: Three weeks of nasal CPAP treatment of SDB in hypertensive men caused the lowering of nocturnal systolic and diastolic BP values, suggesting that increased nocturnal BP in persons with hypertension was causally related to the apnea and hypopnea events of SDB.

Comment in

Silent bedpartners: obstructive sleep apnea and hypertension, 6 years later. [Chest. 2002]

PMID: 12377832 [PubMed - indexed for MEDLINE]
OSA a Cause of Systemic Hypertension

Epidemiological studies showing association of OSA with HTN are not the proof of causality. Only well done randomized placebo-controlled trials showing that elimination of OSA improves HTN prove that OSA is a cause of HTN.
Impact of CPAP: Inconsistent Findings!

- Numerous studies have examined the effects of CPAP on glucose metabolism

<table>
<thead>
<tr>
<th># of studies</th>
<th>Positive</th>
<th>Negative</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPAP studies</td>
<td>12</td>
<td>13</td>
</tr>
</tbody>
</table>

Pamidi S, Tasali E. Front Neurol. 2012; 3:126
# CPAP Adherence in RCTs of Glucose Metabolism

<table>
<thead>
<tr>
<th>Author/Year</th>
<th>Randomized treatment duration</th>
<th>Nightly treatment (hours)</th>
</tr>
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<tbody>
<tr>
<td>Coughlin et al. (2007)</td>
<td>6 weeks</td>
<td>CPAP~ 3.9 h, Sham~ 2.6 h</td>
</tr>
<tr>
<td>West et al. (2007)</td>
<td>3 months</td>
<td>CPAP~ 3.3 h, Sham~ 3.5 h</td>
</tr>
<tr>
<td>Lam et al. (2010)</td>
<td>1 week</td>
<td>CPAP~ 6.2 h, Sham~ 4.5 h</td>
</tr>
<tr>
<td>Nguyen et al. (2010)</td>
<td>3 months</td>
<td>CPAP~ 5.1 h, Sham~ 4.9 h</td>
</tr>
<tr>
<td>Sharma et al. (2011)</td>
<td>3 months</td>
<td>CPAP~ 5.1 h, Sham~ 4.8 h</td>
</tr>
<tr>
<td>Kohler et al. (2011)</td>
<td>2 weeks</td>
<td>CPAP~ 6.2 h, Sham~ 6.0 h</td>
</tr>
<tr>
<td>Hoyos et al. (2012)</td>
<td>12 weeks</td>
<td>CPAP~ 3.6 h, Sham~ 2.8 h</td>
</tr>
<tr>
<td>Sivam et al. (2012)</td>
<td>8 weeks</td>
<td>CPAP~ 4.6 h, Sham~ 3.4 h</td>
</tr>
<tr>
<td>Weinstock et al. (2012)</td>
<td>8 weeks</td>
<td>CPAP~ 4.8 h, Sham~ 3.4 h</td>
</tr>
</tbody>
</table>
OSA that becomes more severe in REM
Total AHI 53, NREM AHI 43, REM AHI 80
Obstructive sleep apnea during REM sleep and hypertension. Results of the Wisconsin Sleep Cohort.

Mokhlesi B, Finn LA, Hagen EW, Young T, Hla KM, Van Cauter E, Peppard PE.

Abstract

RATIONALE: Obstructive sleep apnea (OSA) is associated with hypertension.

OBJECTIVES: We aimed to quantify the independent association of OSA during REM sleep with prevalent and incident hypertension.

METHODS: We included adults enrolled in the longitudinal community-based Wisconsin Sleep Cohort Study with at least 30 minutes of REM sleep obtained from overnight laboratory polysomnography. Studies were repeated at 4-year intervals to quantify OSA. Repeated measures logistic regression models were fitted to explore the association between REM sleep OSA and prevalent hypertension in the entire cohort (n = 4,385 sleep studies on 1,451 individuals) and additionally in a subset with ambulatory blood pressure data (n = 1,085 sleep studies on 742 individuals). Conditional logistic regression models were fitted to longitudinally explore the association between REM OSA and development of hypertension. All models controlled for OSA events during non-REM sleep, either by statistical adjustment or by stratification.

MEASUREMENTS AND MAIN RESULTS: Fully adjusted models demonstrated significant dose-relationships between REM apnea-hypopnea index (AHI) and prevalent hypertension. The higher relative odds of prevalent hypertension were most evident with REM AHI greater than or equal to 15. In individuals with non-REM AHI less than or equal to 5, a twofold increase in REM AHI was associated with 24% higher odds of hypertension (odds ratio, 1.24; 95% confidence interval, 1.08-1.41). Longitudinal analysis revealed a significant association between REM AHI categories and the development of hypertension (P trend = 0.017). Non-REM AHI was not a significant predictor of hypertension in any of the models.

CONCLUSIONS: Our findings indicate that REM OSA is cross-sectionally and longitudinally associated with hypertension. This is clinically relevant because treatment of OSA is often limited to the first half of the sleep period leaving most of REM sleep untreated.

KEYWORDS: REM-related sleep apnea; hypertension; obstructive sleep apnea; rapid eye movement; sleep-disordered breathing

Comment in
REM sleep: a nightmare for patients with obstructive sleep apnea? [Am J Respir Crit Care Med. 2014]
CPAP versus oxygen in obstructive sleep apnea.

Gottlieb DJ, Punjabi NM, Mehra R, Patel SR, Guan SF, Babineau DC, Tracy RP, Rueschman M, Blumenthal RS, Lewis FE, Bhatt DL, Redline S

Abstract

BACKGROUND: Obstructive sleep apnea is associated with hypertension, inflammation, and increased cardiovascular risk. Continuous positive airway pressure (CPAP) reduces blood pressure, but adherence is often suboptimal, and the benefit beyond management of conventional risk factors is uncertain. Since intermittent hypoxemia may underlie cardiovascular sequelae of sleep apnea, we evaluated the effects of nocturnal supplemental oxygen and CPAP on markers of cardiovascular risk.

METHODS: We conducted a randomized, controlled trial in which patients with cardiovascular disease or multiple cardiovascular risk factors were recruited from cardiologic practices. Patients were screened for obstructive sleep apnea with the use of the Berlin questionnaire, and home sleep testing was used to establish the diagnosis. Participants with an apnea-hypopnea index of 15 to 50 events per hour were randomly assigned to receive education on sleep hygiene and healthy lifestyle alone (the control group) or, in addition to education, either CPAP or nocturnal supplemental oxygen. Cardiovascular risk was assessed at baseline and after 12 weeks of the study treatment. The primary outcome was 24-hour mean arterial pressure.

RESULTS: Of 318 patients who underwent randomization, 281 (89%) could be evaluated for ambulatory blood pressure at both baseline and follow-up. On average, the 24-hour mean arterial pressure at 12 weeks was lower in the group receiving CPAP than in the control group (-2.4 mm Hg; 95% confidence interval [CI], -4.7 to -0.1; P=0.04) or the group receiving supplemental oxygen (-2.8 mm Hg; 95% CI, -5.1 to -0.5; P=0.02). There was no significant difference in the 24-hour mean arterial pressure between the control group and the group receiving oxygen. A sensitivity analysis performed with the use of multiple imputation approaches to assess the effect of missing data did not change the results of the primary analysis.

CONCLUSIONS: In patients with cardiovascular disease or multiple cardiovascular risk factors, the treatment of obstructive sleep apnea with CPAP, but not nocturnal supplemental oxygen, resulted in a significant reduction in blood pressure. (Funded by the National Heart, Lung, and Blood Institute and others; HeartBEAT ClinicalTrials gov number: NCT01056600.)

Comment in

Commentary on CPAP vs. oxygen for treatment of OSA. [J Clin Sleep Med. 2014]
Obesity: CPAP effects in sleep apnoea-what should be expected? [Nat Rev Endocrinol. 2014]
Cardiovascular morbidity and obstructive sleep apnea. [N Engl J Med. 2014]
Association between sleep apnea, snoring, incident cardiovascular events and all-cause mortality in an adult population: MESA

Joseph Yeboah, Susan Redline, Craig Johnson, Russell Tracy, Pamela Ouyang, Roger S. Blumenthal, Gregory L. Burke, David M. Herrington

- Multi-Ethnic Study of Atherosclerosis (MESA)
- 5338 respondents (sleep questionnaire)
  → 208 Physician diagnosed sleep apnea
  → 1452 Habitual snorers
  → 3678 Neither
- 7.5 year follow-up
- 310 CVD events (MI, stroke, angina, cardiac arrest)
- 189 deaths
Effects of continuous positive airway pressure on blood pressure in patients with resistant hypertension and obstructive sleep apnea: a meta-analysis

Ifukhar, Imran H.; Valentine, Christopher W.; Bittencourt, Lia R.A.; Cohen, Debbie L.; Fedson, Annette C.; Gislason, Thorarinn; Penzel, Thomas; Phillips, Craig L.; Yu-sheng, Lin; Pack, Allan I.; Magalang, Ulysses J.

Abstract

Objective: To systematically analyze the studies that have examined the effect of continuous positive airway pressure (CPAP) on blood pressure (BP) in patients with resistant hypertension and obstructive sleep apnea (OSA).

Methods: Design – meta-analysis of observational studies and randomized controlled trials (RCTs) indexed in PubMed and Ovid (All Journals@Ovid). Participants: individuals with resistant hypertension and OSA; interventions – CPAP treatment.

Results: A total of six studies met the inclusion criteria for preintervention to postintervention analyses. The pooled estimates of mean changes after CPAP treatment for the ambulatory (24-h) SBP and DBP from six studies were 7.21 mmHg (95% confidence interval (CI) -9.04 to 5.38; P < 0.001, I² 55%) and -4.99 mmHg (95% CI -6.01 to -3.96; P < 0.001, I² 31%), respectively. The pooled estimate of the ambulatory SBP and DBP from the four RCTs showed a mean net change of -6.74 mmHg (95% CI -9.98 to -3.49; P < 0.001, I² 61%) and -5.94 mmHg (95% CI -9.40 to -2.47; P < 0.001, I² 76%), respectively, in favor of the CPAP group.

Conclusion: The pooled estimate shows a favorable reduction of BP with CPAP treatment in patients with resistant hypertension and OSA. The effects sizes are larger than those previously reported in patients with OSA without resistant hypertension.

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Treatment of OSA Reduces the Risk of Repeat Revascularization After Percutaneous Coronary Intervention

Xiaofan Wu, MD; Shuzhong Lv, MD; Xiaohong Yu, MD; Linyin Yao, MD; Babak Mokhlesi, MD, FCCP; Yongdong Wei, MD

Original Research: Sleep Disorders | March 2015

BACKGROUND: The impact of OSA treatment with CPAP on percutaneous coronary intervention (PCI) outcomes remains largely unknown.

METHODS: Between 2002 and 2012, we identified 390 patients with OSA who had undergone PCI. OSA was diagnosed through in-laboratory sleep studies and defined by an apnea-hypopnea index ≥ 5 events/h. The cohort was divided into three groups: (1) moderate-severe OSA successfully treated with CPAP (n = 128), (2) untreated moderate-severe OSA (n = 167), and (3) untreated mild OSA (n = 95). Main outcomes included repeat revascularization, major adverse cardiac events (MACEs) (ie, death, nonfatal myocardial infarction, repeat revascularization), and major adverse cardiac or cerebrovascular events (MACCEs). The median follow-up period was 4.8 years (interquartile range, 3.0-7.1).

RESULTS: The untreated moderate-severe OSA group had a higher incidence of repeat revascularization than the treated moderate-severe OSA group (25.1% vs 14.1%, P = .019). There were no differences in mortality (P = .64), MACE (P = .33), and MACCE (P = .76) among the groups. In multivariate analysis adjusted for potential confounders, untreated moderate-severe OSA was associated with increased risk of repeat revascularization (hazard ratio, 2.13; 95% CI, 1.19-3.81; P = .011).

CONCLUSIONS: Untreated moderate-severe OSA was independently associated with a significant increased risk of repeat revascularization after PCI. CPAP treatment reduced this risk.
Sleep Apnea and Non-Fatal Cardiovascular Events

Cumulative incidence of non-fatal CVS events (%)

- Controls
- Snorers
- Mild OSAH
- Severe OSAH
- OSAH with CPAP

Months

Marin et al. Lancet 2005
OSA Patients Die During Sleep

Gami et al, NEJM, 2006
Sleep Apnea and CVD: Conclusions

• Cross-sectional data suggest a significant and independent association between sleep apnea and prevalent CAD

• Untreated sleep apnea is associated with increased odds of developing CAD

• In patients with CAD, untreated sleep apnea is associated with increased death rate

• Observational data suggest that CPAP may reduce CVD events.
The Effect of Sleep Disordered Breathing on the Outcome of Stroke and Transient Ischemic Attack: A Systematic Review

http://dx.doi.org/10.5564/jcsm.3376

Johannes Birkbak, B.Sc. in Medicine; Alice J. Clark, M.Sc.; Naja Hulvæj Rod, Ph.D.

Department of Public Health, University of Copenhagen, Copenhagen, Denmark

ABSTRACT

Study Objectives
The primary objective was to systematically review the literature on how sleep disordered breathing (SDB) affects recurrence and death among stroke or transient ischemic attack (TIA) patients. A secondary objective was to evaluate how treatment of SDB with continuous positive airway pressure (CPAP) affects the risk of recurrence and death in these patients.

Methods
Adults (18+) with a stroke or TIA diagnosis were eligible for inclusion. Case groups consisted of patients with a sleep disorder. The outcomes of interest were all-cause mortality, recurrent vascular events, and case fatality.

Results
Ten articles covering 1,203 stroke and TIA patients were included in the review. The results generally support a dose-response relationship between severity of SDB and risk of recurrent events and all-cause mortality in stroke and TIA patients. Three small-scale articles with substantial risk of bias evaluated the effects of CPAP therapy, and the results are inconclusive. Data on case fatality is too sparse to be conclusive.

Conclusions
Existing studies provide sufficient data to establish obstructive SDB as a negative predictor of all-cause mortality and recurrent vascular events following stroke or TIA. The ability of CPAP treatment to lower the risk of serious adverse outcomes after stroke remains controversial because of substantial risk of bias identified in most of the eligible studies addressing this relation. Additional studies are needed.

Citation
Sleep Apnea and Arrhythmias: SHHS

Mehra et al. AJRCCM. 2006; 170: 910-916
Incidence of AF Based Upon Presence or Absence of OSA in Subjects <65 Years of Age

Average follow-up: 4.7 years

OSA

No OSA

$p = 0.002$

OSA=Obstructive Sleep Apnea

Sleep-disordered breathing in HF

- OSA in CHF:
  - Male sex
  - Older age
  - Postmenopausal state
  - Upper airway anatomical abnormalities
  - Patients may be less obese compared with OSA patients without CHF
  - Patients may be less sleepy compared with OSA patients without CHF
  - OSA is a predictor of mortality in CHF
Effects of OSA on Mortality in CHF Patients

- 164 patients with HF (LVEF < 45%)
- 113 without OSA and 51 with OSA (14 treated and 37 untreated)

Wang et al. JACC 2007; 49: 1625-1631
Diagnosis and Treatment of Sleep Disordered Breathing in Hospitalized Cardiac Patients: A Reduction in 30-Day Hospital Readmission Rates

http://dx.doi.org/10.5664/jcsm.4096
Shilpa R. Kaula, M.D.¹, Brendan T. Keenan, M.S.¹, Lee Goldberg, M.D.², Richard J. Schwab, M.D.¹
¹Center for Sleep and Circadian Neurobiology, University of Pennsylvania, Philadelphia, PA; ²Department of Cardiology, University of Pennsylvania, Philadelphia, PA

ABSTRACT

Background
Sleep disordered breathing (SDB) is associated with significant cardiovascular sequelae and positive airway pressure (PAP) has been shown to improve heart failure and prevent the recurrence of atrial fibrillation in cardiac patients with sleep apnea. Patients who are hospitalized with cardiac conditions frequently have witnessed symptoms of SDB but often do not have a diagnosis of sleep apnea. We implemented a clinical paradigm to perform unattended sleep studies and initiate treatment with PAP in hospitalized cardiac patients with symptoms consistent with SDB. We hypothesized that PAP adherence in cardiac patients with SDB would reduce readmission rates 30 days after discharge.

Methods
108 consecutive cardiac patients hospitalized for heart failure, arrhythmias, and myocardial infarction and who reported symptoms of SDB were evaluated. Patients underwent a type III portable sleep study and those patients diagnosed with sleep apnea were started on PAP. Demographic data, SDB type, PAP adherence, and data regarding 30-day hospital readmission/ED visits were collected.

Results
Of 108 patients, 104 had conclusive diagnostic studies using portable monitoring systems. Seventy-eight percent of patients (81/104) had SDB (AHI ≥ 5 events/h). Eighty percent (66/81) had predominantly obstructive sleep apnea, and 20% (16/81) had predominantly central sleep apnea. None of 19 patients (0%) with adequate PAP adherence, 6 of 20 (30%) with partial PAP use, and 5 of 17 (29%) of patients who did not use PAP were readmitted to the hospital or visited the emergency department (ED) for a cardiac issue within 30 days from discharge (p = 0.025).

Conclusions
Performing diagnostic unattended sleep studies and initiating PAP treatment in hospitalized cardiac patients was feasible and provided important clinical information. Our data indicate that hospital readmission and ED visits 30 days after discharge were significantly lower in patients with cardiac disease and SDB who adhered to PAP treatment than those who were not adherent.
Central Sleep Apnea in HF

- CSA in CHF:
  - Daytime hypocapnea is predictive of CSA in HF
  - Male sex is predictive of CSA in HF
  - Higher NYHA classification is predictive of CSA in HF
  - Atrial fibrillation is predictive of CSA in HF
  - Age predicts presence of CSA in women with HF
  - CSA is a predictor of mortality in CHF
Increasing Prevalence of Diabetes in the US

Annual Number (in Millions) of Persons with Diagnosed Diabetes, 1980-2011

Data from CDC 2012 Fact sheet
LINK:


REFERENCE:

Best Pract Res Clin Endocrinol Metab. 2010 October 1; 24(5): 703–715.

Authors: Sushmita Pamidi, MD, Renee S. Aronsohn, MD, and Esra Tasali, MD
Association of obstructive sleep apnea in rapid eye movement sleep with reduced glycemic control in type 2 diabetes: therapeutic implications.

Grimaldi D', Boccoli G, Touma C, Van Cauter E, Mohseni B.

Objective: Severity of obstructive sleep apnea (OSA) has been associated with poorer glycemic control in type 2 diabetes. It is not known whether obstructive events during rapid eye movement (REM) sleep have a different metabolic impact compared with those during non-REM (NREM) sleep. Treatment of OSA is often limited to the first half of the night, when NREM rather than REM sleep predominates. We aimed to quantify the impact of OSA in REM versus NREM sleep on hemoglobin A1c (HbA1c) in subjects with type 2 diabetes.

Research Design and Methods: All participants underwent polysomnography, and glycemic control was assessed by HbA1c.

Results: Our analytic cohort included 115 subjects (65 women; age, 55.2 ± 9.8 years; BMI, 34.5 ± 7.5 kg/m²). In a multivariate linear regression model, REM apnea-hypopnea index (AHI) was independently associated with increasing levels of HbA1c (P = 0.008). In contrast, NREM AHI was not associated with HbA1c (P = 0.762). The mean adjusted HbA1c increased from 6.3% in subjects in the lowest quartile of REM AHI to 7.3% in subjects in the highest quartile of REM AHI (P = 0.044 for linear trend). Our model predicts that 4 h of continuous positive airway pressure (CPAP) use would leave 60% of REM sleep untreated and would be associated with a decrease in HbA1c by approximately 0.25%. In contrast, 7 h of CPAP use would cover more than 85% of REM sleep and would be associated with a decrease in HbA1c by as much as 1%.

Conclusions: In type 2 diabetes, OSA during REM sleep may influence long-term glycemic control. The metabolic benefits of CPAP therapy may not be achieved with the typical adherence of 4 h per night.

Comment in


Publication Types, MeSH Terms, Substances, Grant Support

LinkOut - more resources

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0 comments

How to join PubMed Commons

Recent Activity

Association of obstructive sleep apnea in rapid eye movement sleep with reduced glycemic control in type 2 diabetes: therapeutic implications. [Diabetes Care. 2014]

Obstructive sleep apnea during REM sleep and hypertension. Results of the Wisconsin Sleep Cohort Study. [Circulation. 2004]

Adjusted mean HbA1c for REM and NREM AHI

Adjusted for age, sex, race, BMI, years of type 2 diabetes, and insulin use
SUMMARY

High prevalence of OSA in:
- Diabetes
- Stroke
- CVD
- HTN
- CHF
- Arrhythmias
- Pulmonary HTN

Now RCTs showing that with CPAP compliance of > 6 hours,
With coverage through Rem sleep,
You can improve all these clinical measures and reduce long term morbidity and mortality