OSA and Hypertension – Scope of the Problem

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Outline

- Does OSA cause daytime HTN, and if so, by what mechanism
- How to diagnose OSA in patients with HTN, and vice versa
- Does increased BP in OSA affect target organs
- Does treatment of OSA decrease BP
- Which antihypertensive should be used to treat HTN in OSA patients
OSA is an independent risk for hypertension and effective CPAP therapy can reduce the blood pressure.
Obstructive Sleep Apnoea
Intermittent hypoxia, sleep fragmentation, increased respiratory efforts

Intermittent Hypoxia

Reduction or cessation of flow

Increase in respiratory effort

Cardiovascular activation

Arousal
Clinical OSA: Just the Tip of the Iceberg

Diagnosed cases

Undiagnosed cases – millions at risk
Obesity and ageing; lack of awareness; stigma

‘Subclinical’ OSA

Clinical OSA
Prevalence of OSA


- 9–50% of males and 4–17% of females reported snoring

- Clinically significant OSA 3-7% for adult males and 2-5% for adult females

- 75% of severe OSA remains undiagnosed
Snoring and breathing pauses during sleep in the Malaysian population  
Kamil et al Respirology 2007

N= 1611 adults 30-70 years old, cross sectional population based survey

Definition:
Snoring- self report with confirmation from bed partner
Habitual snoring – snores 3 or more nights a week
Loud habitual snoring – ‘louder than talking’
Excessive daytime sleepiness (EDS) – ESS>11
OSAS- loud snoring, breathing pauses (with or without without choking or gasping) and EDS

<table>
<thead>
<tr>
<th></th>
<th>Male (n = 853)</th>
<th>Female (n = 758)</th>
<th>All respondents (n = 1611)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Non-snoring</td>
<td>33.9</td>
<td>38.9</td>
<td>36.3</td>
</tr>
<tr>
<td>Snoring*</td>
<td>66.1</td>
<td>61.1</td>
<td>63.7</td>
</tr>
<tr>
<td>Habitual snoring**</td>
<td>51.5</td>
<td>42.6</td>
<td>47.3</td>
</tr>
<tr>
<td>Loud habitual snoring**</td>
<td>28.3</td>
<td>17.9</td>
<td>23.4</td>
</tr>
<tr>
<td>Sleeping pauses**</td>
<td>19.2</td>
<td>10.7</td>
<td>15.2</td>
</tr>
<tr>
<td>Excessive daytime sleepiness*</td>
<td>17.1</td>
<td>12.3</td>
<td>14.8</td>
</tr>
<tr>
<td>Clinically suspected OSAS*</td>
<td><strong>8.8</strong></td>
<td><strong>5.1</strong></td>
<td>7.1</td>
</tr>
</tbody>
</table>

Univariate analysis for comparison between genders. *P<0.05, **P<0.001.
Sleep apnoea prevalence in CV diseases

- Hypertension
- CHD
- Heart failure
- Stroke
- Refractory hypertension

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Courtesy of Patrick Levy, ERS2012
Sleep Apnoea → Cardiovascular Risk

Several Ongoing Epidemiological Studies

1. Sleep Heart Health Study

   Community dwelling middle-aged men and women across the United States

2. Wisconsin Sleep Cohort Study

   Population based sample of working men and women from Wisconsin
The Sleep Heart Health Study

- **Design:**
  - Prospective cohort study
  - Multi-center
  - Assessment of sleep on to ongoing cohort studies of cardiovascular and respiratory disease
  - Wide geographic and ethnic representation of middle-aged and older US adults

Quan et al: The Sleep Heart Health Study: Design, Methods, Rationale
Sleep 1997; 20(12) 1077-1085
The SHHS: Field Sites

Baseline N = 6441

Minneapolis (1085)
Framingham (1000)
South Dakota (201)
Pittsburgh (398)
New York (760)
Hagerstown (1184)
Sacramento (501)
Phoenix (201)
Tucson (911)
Oklahoma (200)
Oklahoma (200)

Outcome and Covariate Data

- Prevalent disease
  - Cardiovascular disease, myocardial infarction, stroke, angina, PTCA, congestive heart failure, hypertension, diabetes, asthma and COPD

- Demographic
  - Age, race, education, marital status, occupation, etc.

- Health Habits
  - Smoking, alcohol use, caffeine, etc.

Quan et al: The Sleep Heart Health Study: Design, Methods, Rationale
Sleep 1997; 20(12) 1077-1085
Outcome and Covariate Data

- Anthropometrics (BMI, waist girth, hip circumferences)
- Medication use (prescription and non-prescription)
- Electrocardiogram
- Subset of the cohort
  - Echocardiogram
  - Holter data
  - Head MRI scans
  - Physical activity
  - Serum (fasting, non-fasting, post glucose challenge)
  - Spirometry

Quan et al: The Sleep Heart Health Study: Design, Methods, Rationale
Sleep 1997; 20(12) 1077-1085
SHHS: Longitudinal Design

Baseline PSG (N = 6441)  Follow-up PSG (N = 3291)

Continuous Outcome Surveillance

Outcome Surveillance

Censoring Date
April 1, 2006

End of Follow-up

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### Adjusted odds ratio for prevalent hypertension

<table>
<thead>
<tr>
<th>AHI (events/hr)</th>
<th>Odds ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>&lt; 1.5</td>
<td>1.00</td>
<td>Reference</td>
</tr>
<tr>
<td>1.5 – 4.9</td>
<td>1.07</td>
<td>0.91 – 1.26</td>
</tr>
<tr>
<td>5.0 – 14.9</td>
<td>1.20</td>
<td>1.01 – 1.42</td>
</tr>
<tr>
<td>15.0 – 29.9</td>
<td>1.25</td>
<td>1.00 – 1.56</td>
</tr>
<tr>
<td>≥ 30</td>
<td>1.37</td>
<td>1.03 – 1.83</td>
</tr>
</tbody>
</table>

Adjusted for age, sex, ethnicity, BMI, neck, waist/hip ratio, alcohol use, and smoking.

Sleep apnea and Incident Hypertension

Prospective Study of Sleep-disordered Breathing and Hypertension
The Sleep Heart Health Study

George T. O’Connor¹, Brian Caffo², Anne B. Newman³, Stuart F. Quan⁴,⁵, David M. Rapoport⁶, Susan Redline⁷, Helaine E. Resnick⁸, Jonathan Samet², and Eyal Shahar⁹

¹Pulmonary Center, Boston University School of Medicine, Boston, Massachusetts; ²Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, Maryland; ³University of Pittsburgh, Pittsburgh, Pennsylvania; ⁴Arizona Respiratory Center, University of Arizona, Tucson, Arizona; ⁵Division of Sleep Medicine, Harvard Medical School, Boston, Massachusetts; ⁶Department of Medicine, New York University School of Medicine, New York, New York; ⁷Department of Medicine, Case Western Reserve University, Cleveland, Ohio; ⁸American Association of Homes and Services for the Aging, Washington, D.C.; and ⁹Division of Epidemiology and Biostatistics, Mel and Enid Zuckerman College of Public Health, University of Arizona, Tucson, Arizona

### TABLE 4. ADJUSTED ODDS RATIOS FOR INCIDENT HYPERTENSION AMONG SLEEP HEART HEALTH STUDY SUBJECTS WHO WERE NORMOTENSIVE AT BASELINE, ACCORDING TO BASELINE APNEA-HYPOPNEA INDEX, STRATIFIED BY BODY MASS INDEX

<table>
<thead>
<tr>
<th>Baseline AHI</th>
<th>BMI ≤27.3</th>
<th>OR (95% CI)</th>
<th>BMI &gt;27.3</th>
<th>OR (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–4.9</td>
<td>887</td>
<td>—</td>
<td>541</td>
<td>—</td>
</tr>
<tr>
<td>5–14.9</td>
<td>213</td>
<td>0.89 (0.59–1.34)</td>
<td>378</td>
<td>0.92 (0.67–1.27)</td>
</tr>
<tr>
<td>15–29.9</td>
<td>58</td>
<td>0.93 (0.46–1.90)</td>
<td>164</td>
<td>1.13 (0.76–1.68)</td>
</tr>
<tr>
<td>≥30</td>
<td>21</td>
<td>2.71 (1.24–5.93)</td>
<td>65</td>
<td>1.18 (0.64–2.19)</td>
</tr>
</tbody>
</table>

Incident Hypertension in SDB
n=1889

Marin J. JAMA 2012; 307:2169
OSA and refractory hypertension

% controlled HT

599 HT - Relationship between OSA and HT severity

80% of OSA patients among refractory HT

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- Does treatment of OSA decrease BP
- Which antihypertensive should be used to treat HTN in OSA patients
Mechanisms of Hypertension in OSA

Foster GE et al. Exp Physiol 2007;92;51-65
OSA Causes Increased Sympathetic Tone

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Basic mechanisms linking OSA and cardiovascular diseases

OSA is associated with the development of various cardiovascular diseases and this association now represents a significant public health burden. The identification of the mechanisms underlying the cardiovascular disease processes is of major importance and, despite significant effort being made, the process remains incompletely understood. Clinical studies using OSA populations have often been limited by inadequate control for frequent comorbid conditions, in particular obesity, pre-existing cardiovascular disease or medications. Moreover, OSA is a complex and heterogeneous disease and differences in morphology and length of the nocturnal airflow cessations and associated oxygen desaturations, which are not captured by the traditional OSA severity marker, as well as differences in duration of the disease prior to diagnosis are major contributors for the large variability observed between studies results, including differences in response to short-term continuous positive airway pressure (CPAP) therapy. Cell culture and animal models have been developed to overcome some of these hurdles. These models allow single components of the disease to be studied; precisely controlling the triggering events in both severity and duration, and providing genetic homogeneous populations. Thus, in vitro and animal studies have greatly contributed to our current knowledge.

OSA comprises various pathophysiological triggers, but beside sleep fragmentation, intrathoracic pressure swings and recurrent hypercapnia, it is the OSA unique form of hypoxia in particular, with repetitive short cycles of desaturation followed by rapid re-oxygenation, termed intermittent hypoxia, that plays a pivotal role in the cardiovascular disease process. The pathogenesis is probably multifactorial and our current concept involves sympathetic nervous system overactivity, systemic inflammation and oxidative stress leading to endothelial dysfunction and, possibly, metabolic dysfunction which are the most important pathways.

However, it should be noted that some beneficial effects of intermittent hypoxia have been evidenced in both animal models and OSA with regard to long-term facilitation and hypoxic ventilatory response. Exposure to acute intermittent hypoxia or repeated carotid sinus nerve stimulation causes a persistent increase in respiratory activity; whether this is beneficial in OSA remains unknown. It has been suggested that alteration in long-term facilitation and hypoxic ventilatory response in reply to intermittent hypoxia exposure could lead to breathing stability and reduction in apnoea/hypopnoea index, although this is still conflicting. There are also potential beneficial effects with regard to cardiovascular consequences that have been suggested in animals exposed to intermittent hypoxia. This might also be the case in OSA with respect to vascular adaptations and possibly lower mortality.

![Diagram of mechanisms linking OSA, intermittent hypoxia, and cardiovascular diseases](https://example.com/diagram.png)
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- Does treatment of OSA decrease BP
- Which antihypertensive should be use to treat HTN in OSA patients
## Evaluation

### Classification of Blood Pressure (BP)*

<table>
<thead>
<tr>
<th>CATEGORY</th>
<th>SBP mmHg</th>
<th>DBP mmHg</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normal</td>
<td>&lt;120</td>
<td>and &lt;80</td>
</tr>
<tr>
<td>Prehypertension</td>
<td>120-139</td>
<td>or 80-89</td>
</tr>
<tr>
<td>Hypertension, stage 1</td>
<td>140-159</td>
<td>or 90-99</td>
</tr>
<tr>
<td>Hypertension, stage 2</td>
<td>≥ 160</td>
<td>≥ 100</td>
</tr>
</tbody>
</table>

Key: SBP = systolic blood pressure  
     DBP = diastolic blood pressure

### Diagnostic Workup of Hypertension

- Assess risk factors and comorbidities.  
- Reveal identifiable causes of hypertension.  
- Assess presence of target organ damage.  
- Conduct history and physical examination.  
- Obtain laboratory tests; urinalysis, blood glucose, hematocrit and lipid panel, serum potassium, creatinine, and calcium. Optional: urinary albumin/creatinine ratio.  
- Obtain electrocardiogram.

### Assess for Major Cardiovascular Disease (CVD) Risk Factors

- Hypertension  
- Obesity  
- Dyslipidemia  
- Diabetes mellitus  
- Cigarette smoking  
- Physical inactivity  
- Microalbuminuria, estimated glomerular filtration rate <60 mL/min  
- Age (>55 for men, >65 for women)  
- Family history of premature CVD  
- (men age <55, women age <65)

### Assess for Identifiable Causes of Hypertension

**Sleep apnea** is an identifiable cause of hypertension – NIH, JNC7 (2003)

- Drug induced/related  
- Chronic kidney disease  
- Primary aldosteronism  
- Renovascular disease  
- Cushing’s syndrome or steroid therapy  
- Pheochromocytoma  
- Coarctation of aorta  
- Thyroid/parathyroid disease

## Treatment

### Principles of Hypertension Treatment

- Treat to BP <140/90 mmHg or BP <130/80 mmHg in patients with diabetes or chronic kidney disease.  
- Majority of patients will require two medications to reach goal.

### Algorithm for Treatment of Hypertension

#### Lifestyle Modifications

- Not at Goal Blood Pressure (<140/90 mmHg)  
  (<130/80 mmHg for patients with diabetes or chronic kidney disease)

#### Initial Drug Choices

**Without Compelling Indications**

- Stage 1 Hypertension (SBP 140-159 or DBP 90-99 mmHg)
  - Thiazide-type diuretics for most. May consider ACEI, ARB, BB, CCB, or combination.

**With Compelling Indications**

- Stage 2 Hypertension (SBP ≥ 160 or DBP ≥ 100 mmHg)
  - 2-drug combination for most (usually thiazide-type diuretic and ACEI, or ARB, or BB, or CCB).
  - Drug(s) for the compelling indications  
    - See Compelling Indications for Individual Drug Classes  
    - Other antihypertensive drugs (diuretics, ACEI, ARB, BB CCB) as needed.

**Not at Goal Blood Pressure**

- Optimize dosages or add additional drugs until goal blood pressure is achieved. Consider consultation with hypertension specialist.

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**U.S. DEPARTMENT OF HEALTH AND HUMAN SERVICES**  
National Institutes of Health  
National Heart, Lung, and Blood Institute
Table 2. Secondary causes of hypertension

- Sleep apnoea
- Drug-induced or drug-related
- Chronic kidney disease
- Primary aldosteronism
- Renovascular disease
- Chronic steroid therapy and Cushing syndrome
- Phaeochromocytoma
- Acromegaly
- Thyroid or parathyroid disease
- Coarctation of the aorta
- Takayasu Arteritis
OSAHS...A or B, and C
Obstructive Sleep Apnoea Hypopnoea Syndrome

- A – Excessive Daytime Sleepiness
- B – At least two of the following items
  - Nocturnal gasping or breathing arrests
  - Frequent awakenings
  - Unrefreshing sleep
  - Daytime fatigue
  - Loss of concentration
- C - > 5 obstructive breathing episodes per hour during sleep

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ERS/ESH Task Force Report 2013

Pre-test probability of OSA

High

- Elevated or high normal conventional BP (SBP ≥130 or DBP ≥85 mmHg)
  - ABPM and PSG according to guidelines

- Normal conventional BP (SBP <130 and DBP <85 mmHg)
  - PSG according to guidelines
    - Nondipper
      - ABPM
    - Dipper
      - Clinical follow-up

Low

- Elevated or high normal conventional BP (SBP ≥130 or DBP ≥85 mmHg)

- Normal conventional BP (SBP <130 and DBP <85 mmHg)

If OSA +

- ABPM (if not performed previously)

- Adequate treatment

- Follow-up PSG + ABPM
Outline

- Does OSA cause daytime HTN, and if so, by what mechanism
- What is the best method to measure BP in OSA patients
- Does increased BP in OSA affect target organs
- Does treatment of OSA decrease BP
- Which antihypertensive should be used to treat HTN in OSA patients
Epidemiology and Prevention

Prospective Study of Obstructive Sleep Apnea and Incident Coronary Heart Disease and Heart Failure
The Sleep Heart Health Study

Daniel J. Gottlieb, MD, MPH; Gayane Yenokyan, MD, PhD; Anne B. Newman, MD, MPH; George T. O’Connor, MD, MSc; Naresh M. Punjabi, MD, PhD; Stuart F. Quan, MD; Susan Redline, MD, MPH; Helaine E. Resnick, PhD, MPH; Elisa K. Tong, MD, MA; Marie Diener-West, PhD; Eyal Shahar, MD, MPH
### Sleep Apnea: Incident CVD

#### Men: Age < 70 years

<table>
<thead>
<tr>
<th>Outcome</th>
<th>Predictor Variable</th>
<th>Hazard Ratio</th>
<th>95% CI</th>
</tr>
</thead>
<tbody>
<tr>
<td>Incident CVD</td>
<td>AHI &lt; 5 vs. AHI &gt; 30</td>
<td>1.68</td>
<td>1.02 – 2.76</td>
</tr>
<tr>
<td></td>
<td>AHI continuous (per 10 unit increase)</td>
<td>1.10</td>
<td>1.00 – 1.21</td>
</tr>
</tbody>
</table>

Gottlieb et al. Circulation 2010; 122: 325-360
Prospective Study of OSA and Incident CHD and HF
Sleep Heart and Health Study

OSA have a 58% higher adjusted risk of incident HF

Cardiovascular mortality

Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study

<table>
<thead>
<tr>
<th></th>
<th>Healthy men (n=264)</th>
<th>Simple snorers (n=377)</th>
<th>Untreated mild-moderate OSAH (n=403)</th>
<th>Untreated severe OSAH (n=235)</th>
<th>OSAH treated with CPAP (n=372)</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Non-fatal cardiovascular events</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of events</td>
<td>12</td>
<td>22</td>
<td>36</td>
<td>50</td>
<td>24</td>
</tr>
<tr>
<td>Events per 100 person years</td>
<td>0.45</td>
<td>0.58</td>
<td>0.89</td>
<td>2.13*</td>
<td>0.64</td>
</tr>
<tr>
<td><strong>Cardiovascular death</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Number of events</td>
<td>8</td>
<td>13</td>
<td>22</td>
<td>25</td>
<td>13</td>
</tr>
<tr>
<td>Events per 100 person years</td>
<td>0.3</td>
<td>0.34</td>
<td>0.55</td>
<td>1.06†</td>
<td>0.35</td>
</tr>
</tbody>
</table>

OSAH = obstructive sleep apnoea-hypopnoea syndrome; CPAP = continuous positive airway pressure. *p < 0.0001 versus healthy men; †p = 0.0012.

Table 2: Incidence of cardiovascular events during the 10-year follow-up in healthy men, snorers, and patients untreated and treated for OSAH.
Sleep Apnea and Fatal Cardiovascular Events

Marin et al. Lancet 2005
Effects of CPAP Therapy on CVD

<table>
<thead>
<tr>
<th>Fully adjusted odds ratio (95% CI)</th>
<th>p</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age, years</td>
<td>1.09 (1.04–1.12)</td>
</tr>
<tr>
<td>Diagnostic group</td>
<td></td>
</tr>
<tr>
<td>Snoring</td>
<td>1.03 (0.31–1.84)</td>
</tr>
<tr>
<td>Untreated mild-moderate OSAH</td>
<td>1.15 (0.34–2.69)</td>
</tr>
<tr>
<td>Untreated severe OSAH</td>
<td>2.87 (1.17–7.51)</td>
</tr>
<tr>
<td>CPAP</td>
<td>1.05 (0.39–2.21)</td>
</tr>
<tr>
<td>Cardiovascular disease</td>
<td>2.54 (1.31–4.99)</td>
</tr>
</tbody>
</table>

Marin et al. Lancet 2005
Sleep Apnea and CVD

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

- Untreated sleep apnea is associated with increased odds of developing CAD and CHF.

- In patients with CAD, untreated sleep apnea is associated with increased death rate.
Does OSA cause daytime HTN, and if so, by what mechanism

What is the best method to measure BP in OSA patients

Does increased BP in OSA affect target organs

Does treatment of OSA decrease BP

Which antihypertensive should be use to treat HTN in OSA patients
Randomized Clinical Trial of CPAP on BP

- Double-blind randomized trial (118 men)
- Sleep Apnea: ODI > 10 events/hr + Epworth score > 9
- Sub-therapeutic vs. therapeutic CPAP
- 24-hr ambulatory blood pressure (baseline and 4-wks post Rx)
- Mean BP: –2.5 mmHg (CPAP) vs. +0.8 mmHg (sham)

Before CPAP intervention

After CPAP intervention
CPAP Lowers BP in SDB
9 Weeks

Becker HF. Circulation; 2003; 107:68
Effect Of CPAP On Incident Hypertension Or CV Events In SDB Without EDS (n=725)

Barbé F. JAMA 2012; 307:2161
CPAP and Blood Pressure Meta-Analysis
Haentjens P, Arch Intern Med 2007

CPAP reduces BP but the effect is rather small 1.69mmHg in 24hr mean BP 0.89mmHg per 10 point increase in AHI at entry 1.39mmHg for each 1 hour increase in nightly CPAP use

<table>
<thead>
<tr>
<th>Study</th>
<th>Weighted Mean Difference (95% CI) in 24-Hour MBP (in mmHg)</th>
<th>Weight, %</th>
<th>Weighted Mean Difference (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Engleman et al.19 1996</td>
<td>-2.58 (7.26 to 2.11)</td>
<td>3.9</td>
<td></td>
</tr>
<tr>
<td>Dimson et al.20 1998</td>
<td>-1.30 (3.36 to -0.31)</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>Faccenda et al.21 2001</td>
<td>-1.00 (-2.60 to 0.60)</td>
<td>15.4</td>
<td></td>
</tr>
<tr>
<td>Garabedian et al.22 2001</td>
<td>-1.67 (-6.09 to 2.70)</td>
<td>4.3</td>
<td></td>
</tr>
<tr>
<td>Johnson et al.13 2002</td>
<td>-1.33 (-6.30 to 3.53)</td>
<td>2.4</td>
<td></td>
</tr>
<tr>
<td>Barnes et al.24 2002</td>
<td>-4.43 (-5.49 to 4.62)</td>
<td>3.4</td>
<td></td>
</tr>
<tr>
<td>Becker et al.25 2003</td>
<td>-10.50 (-18.50 to -2.40)</td>
<td>1.5</td>
<td></td>
</tr>
<tr>
<td>Bents et al.26 2002</td>
<td>-1.37 (-0.97 to 0.99)</td>
<td>4.3</td>
<td></td>
</tr>
<tr>
<td>Robinson et al.27 2006</td>
<td>-0.74 (-4.40 to 2.90)</td>
<td>5.8</td>
<td></td>
</tr>
<tr>
<td>Norman et al.28 2006</td>
<td>-7.00 (-11.66 to 2.34)</td>
<td>3.9</td>
<td></td>
</tr>
<tr>
<td>Arias et al.29 2006</td>
<td>-0.45 (-7.82 to 6.94)</td>
<td>1.7</td>
<td></td>
</tr>
<tr>
<td>Campos-Rodriguez et al.30 2006</td>
<td>-0.80 (-2.70 to 4.30)</td>
<td>13.3</td>
<td></td>
</tr>
<tr>
<td>Overall (95% CI)</td>
<td>-1.69 (-2.69 to -0.69)</td>
<td>100.0</td>
<td></td>
</tr>
</tbody>
</table>

Data in Heterogeneity: $I^2 = 40.5\%$, $Q = 17.4\%$
Significant correlation between the improvement in nighttime blood BP and the duration of CPAP use.
Factors affecting BP reduction by CPAP therapy in OSA patients

- Presence or absence of daytime sleepiness
- OSA severity
- Blood pressure levels before treatment
- Resistant hypertension
- Patients’ age and sex
- Duration of treatment
- CPAP proper titration
- Patients’ compliance with CPAP treatment

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Antihypertensives for OSA patients

- In patients with untreated OSA and hypertension, BP lowering drugs effectively reduced daytime BP, without clear differences between different classes of medications (Zou et al. Sleep Med 2010)

- Normalisation of BP during daytime can be easily achieved, but CPAP treatment is necessary to eliminate BP fluctuations at night
Additive effects of CPAP and Valsartan

a) SBP mmHg

b) DBP mmHg

CPAP
Valsartan
Valsartan + CPAP

Time h

09:00 11:00 13:00 15:00 17:00 19:00 21:00 23:00 01:00 03:00 05:00 07:00 09:00

Pepin AJRCCM 2010
Take Home Message

- Obstructive sleep apnea is an independent risk factor for the development of systemic hypertension.

- Hypertension associated with obstructive sleep apnea may be relatively resistant to drug therapy.

- In hypertensive patients with obstructive sleep apnea, specific therapy of obstructive sleep apnea may reduce blood pressure.
Quantity and Quality of Sleep are Paramount for Human Health
Sleep 2014

20th - 22nd March 2014
Cititel Hotel
Kuala Lumpur

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