Cardiovascular Disease and Sleep Apnea

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Purpose

Describe the types and prevalence of sleep apnea and its meaning in patients at risk for or who already have cardiovascular disease
• Recognizing patients with cardiovascular disease who have coexisting sleep apnea
• Understanding the mechanisms by which sleep apnea may contribute to the progression of the cardiovascular condition
• Review positive airway pressure treatment strategies
Main Questions

• Does sleep apnea initiate the development of cardiovascular disease?
• Does sleep apnea accelerate heart disease progression?
• Does treatment of sleep apnea cause fewer cardiovascular events and reduce mortality?
The Letter Conundrum

- **SDB** – sleep disordered breathing
- **OSA** – obstructive sleep apnea
- **CSA** - central sleep apnea
- **CSR** – Cheyne Stokes respiration
- **AHI** – apnea hypopnea index
- **CAI** – central apnea index
- **PAP** – positive airway pressure
- **CPAP** – continuous positive airway pressure
Sleep disordered breathing and cardiovascular disease

- Highly prevalent in patients with CV disease
- OSA affects at least 15 million adult Americans
- CAD, stroke, atrial fibrillation
- CSA occurs mainly in patients with heart failure
Evidence-based Research Limitations

- Close association between obesity and OSA obscures the differentiation
- Multiple comorbidities (CV disease, metabolic syndrome and diabetes)
- Randomization of sleep apnea patients to treatment or no treatment; need to treat patients with severe daytime somnolence
Topics of Interest

- Definitions – obstructive, central, mixed, complex
- Diagnosis
- Pathophysiology
- Hypertension
- Heart Failure
- Stroke
- Arrhythmias
- Myocardial Ischemia and Infarction
- Pulmonary Arterial Hypertension (PAH)
- End Stage Renal Disease (ESRD)
- CPAP Treatment
Definitions

- **Obstructive Apnea** – repetitive cessations of ventilation during sleep caused by collapse of the pharyngeal airway
Definitions

- **Obstructive Hypopnea** – repetitive interruptions of ventilation during sleep caused by incomplete collapse of the pharyngeal airway.
Definitions

- Central Sleep Apnea – repetitive cessation of ventilation during sleep resulting from loss of ventilatory drive
Definitions

- Normal Breathing
Definitions

Normal Breathing
- Airway is open
- Air flows freely to lungs

Obstructive Sleep Apnea
- Airway collapses
- Blocked air flow to lungs
Definition – OSA syndrome

• Apnea or hypopnea – 10 seconds in length
• Hypopneas - majority
• Sleep Heart Health Study > 6000 adults with hypopneas associated with ≥4% decrease in O₂ saturation were independently associated with CV disease¹

Definition & Prevalence OSA syndrome

- AHI > 5 with symptoms of excessive daytime sleepiness
- AHI = number of apneas and hypopneas per hour of sleep
- 1 in 5 adults - mild OSA (AHI ≥ 5)
- 1 in 15 adults - moderate to severe OSA (AHI ≥ 15)
- Significant progression of OSA over time \(^1,^2\)
  (Wisconsin Sleep Cohort study and Cleveland Family Study)

Prevalence of CSA

- CAI ≥ 2.5 - 0% (20-44), 1.7% (45-64) and 12% (>65)\(^1,2\)
- CAI ≥ 1 - 9% (40 to 97) in men in SHHS\(^3\)
- Higher in those with diabetes, HF, LV dysfunction and stroke
- Fewer women with heart failure have CSA
- CAI ≥ 15 - 40% of men with heart failure\(^4\)

Pathophysiology of OSA

- Small pharyngeal airway
  - ▲ Airflow resistance during wake
  - ▲ Intrapharyngeal negative pressure during inspiration when awake
  - via mechanoreceptors in larynx
  - ▲ Activity of number of pharyngeal dilator muscles
  - Maintain airway patency
Pathophysiology of OSA

- Small pharyngeal airway
  - ↑ Airflow resistance during wake
  - ↑ Intrapharyngeal negative pressure during inspiration when awake via mechanoreceptors in larynx
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Pathophysiology of OSA

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Pathophysiology of OSA

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Maintain airway patency
Pathophysiology of OSA

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- ↑ Airflow resistance during wake
- ↑ Intrapharyngeal negative pressure during inspiration when awake
  via mechanoreceptors in larynx
- ↑ Activity of number of pharyngeal dilator muscles
- Maintain airway patency
- Pharyngeal narrowing & intermittent collapse
- ↓O₂ and ↑CO₂
- Stimulate ventilatory effort
- Arousals
- Sleep deprivation
Pathophysiology of OSA

Deficient pharyngeal anatomy
Pathophysiology of OSA

Deficient pharyngeal anatomy

Ventilatory control system instability
  Cycling respiratory output to ventilatory pump muscles & upper airway dilatation muscles
  Apneas and hypopneas
Pathophysiology of OSA

Deficient pharyngeal anatomy

Ventilatory control system instability
- Cycling respiratory output to ventilatory pump muscles & upper airway dilatation muscles
- Apneas and hypopneas

Variable surface tension of pharyngeal airway
Pathophysiology of OSA

- Deficient pharyngeal anatomy
- Variable surface tension of pharyngeal airway
- Asynchronous timing of activation of upper airway versus pump muscles
- Ventilatory control system instability
  - Cycling respiratory output to ventilatory pump muscles & upper airway dilatation muscles
  - Apneas and hypopneas

Apneas and hypopneas
Pathophysiology of OSA

Deficient pharyngeal anatomy

Variable surface tension of pharyngeal airway

Arousal threshold

Asynchronous timing of activation of upper airway versus pump muscles

Ventilatory control system instability

Cycling respiratory output to ventilatory pump muscles & upper airway dilatation muscles

Apneas and hypopneas

Variable surface tension of pharyngeal airway
Pathophysiology of OSA

Deficient pharyngeal anatomy

Variable upper airway dilator muscle control

Variable surface tension of pharyngeal airway

Ventilatory control system instability
  Cycling respiratory output to ventilatory pump muscles & upper airway dilatation muscles
  Apneas and hypopneas

Asynchronous timing of activation of upper airway versus pump muscles

↑ Arousal threshold
Pathophysiology of OSA

Deficient pharyngeal anatomy:
- Loss of lung volume
  - Longitudinal traction
  - More collapsible
- Variable surface tension of pharyngeal airway

Ventilatory control system instability:
- Cycling respiratory output to ventilatory pump muscles & upper airway dilatation muscles
  - Apneas and hypopneas

Variable upper airway dilator muscle control

Asynchronous timing of activation of upper airway versus pump muscles

Arousal threshold

Variable surface tension of pharyngeal airway
Mechanism of disease and CV Risk

Cardiovascular variability

↑HR and ↑BP variability during wake

Cardiovascular Disease Risk
Mechanism of disease and CV Risk

Cardiovascular variability
↑HR and ↑BP variability during wake

Endothelial dysfunction
(Systemic inflammation, sympathetic activation, pressor surges, oxidative stress)

Cardiovascular Disease Risk
Mechanism of disease and CV Risk

Cardiovascular variability

Endothelial dysfunction
(Systemic inflammation, sympathetic activation, pressor surges, oxidative stress)

Insulin resistance/metabolic dysregulation
Glucose intolerance
Leptin decrease

Cardiovascular Disease Risk

HR and BP variability during wake
Mechanism of disease and CV Risk

Cardiovascular variability
↑HR and ↑BP variability during wake

Endothelial dysfunction
(Systemic inflammation, sympathetic activation, pressor surges, oxidative stress)

Insulin resistance/metabolic dysregulation
Glucose intolerance
↓Leptin

Thrombosis
Platelet activation
↑fibrinogen

Cardiovascular Disease Risk
Mechanism of disease and CV Risk

Cardiovascular Disease Risk

- Intrathoracic pressure changes
  - Mueller maneuver
- Thrombosis
  - Platelet activation
- Fibrinogen
- Endothelial dysfunction
  - (Systemic inflammation, sympathetic activation, pressor surges, oxidative stress)
- Insulin resistance/metabolic dysregulation
  - Glucose intolerance
  - Leptin
- Cardiovascular variability
  - HR and BP variability during wake
Mechanism of disease and CV Risk

Cardiovascular Disease Risk

- **Endothelial dysfunction**
  - Systemic inflammation, sympathetic activation, pressor surges, oxidative stress

- **Insulin resistance/metabolic dysregulation**
  - Glucose intolerance
  - Leptin

- **Oxidative Stress**
  - Thiobarbituric acid reactive substances, isopropanes, oxidized LDL

- **Intrathoracic pressure changes**
  - Mueller maneuver

- **Cardiovascular variability**
  - HR and BP variability during wake

- **Thrombosis**
  - Platelet activation
  - Fibrinogen
Mechanism of disease and CV Risk

Inflammation / Hypoxemia
(Pentraxin3, IL-6, CRP, NO, NFκB, TNFα, adhesion molecules, serum amyloid A, leukocyte activation)

Cardiovascular variability
↑HR and ↑BP variability during wake

Endothelial dysfunction
(Systemic inflammation, sympathetic activation, pressor surges, oxidative stress)

Insulin resistance/metabolic dysregulation
Glucose intolerance ↓Leptin

Thrombosis
Platelet activation ↑fibrinogen

Oxidative Stress
(Thiobarbituric acid reactive substances, isopropanes, oxidized LDL)

Intrathoracic pressure changes
Mueller maneuver
Mechanism of disease and CV Risk

Cardiovascular Disease Risk

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Endothelial dysfunction
(Systemic inflammation, sympathetic activation, pressor surges, oxidative stress)

Insulin resistance/metabolic dysregulation
Glucose intolerance
↓ Leptin

Thrombosis
Platelet activation
↑ fibrinogen

Oxidative Stress
↑ (thiobarbituric acid reactive substances, isopropanes, oxidized LDL)

Inflammation/Hypoxemia
↑ (Pentraxin3, IL-6, CRP, NO NFκB, TNFα, adhesion molecules, serum amyloid A, leukocyte activation)

Vasoactive substances
recurrent hypoxemic stress
↑ endothelin

Intrathoracic pressure changes
Mueller maneuver

Leptin
Mechanism of disease and CV Risk

- **Sympathetic Activation**
  - HR during wake

- **Vasoactive substances**
  - Recurrent hypoxemic stress
  - Endothelin

- **Inflammation / Hypoxemia**
  - (Pentraxin3, IL-6, CRP, NO, NFκB, TNFα, adhesion molecules, serum amyloid A, leukocyte activation)

- **Cardiovascular variability**
  - HR and BP variability during wake

- **Endothelial dysfunction**
  - (Systemic inflammation, sympathetic activation, pressor surges, oxidative stress)

- **Insulin resistance / metabolic dysregulation**
  - Glucose intolerance
  - Leptin

- **Thrombosis**
  - Platelet activation
  - Fibrinogen

- **Oxidative Stress**
  - (thiobarbituric acid reactive substances, isopropanes, oxidized LDL)

- **Cardiovascular Disease Risk**

- **Intrathoracic pressure changes**
  - Mueller maneuver
Mechanism of disease and CV Risk

Occluded pharynx (Mueller Maneuver)
Mechanism of disease and CV Risk

Occluded pharynx (Mueller Maneuver)

↓

Negative intrathoracic pressure

↓
Mechanism of disease and CV Risk

Occluded pharynx (Mueller Maneuver)

↓

Negative intrathoracic pressure

↓

↑ Transmural gradients across atria, ventricles & aorta

↓
Mechanism of disease and CV Risk

Occluded pharynx (Mueller Maneuver)

↓

Negative intrathoracic pressure

↓

↑ Transmural gradients across atria, ventricles & aorta

↓

△ Ventricular function

△ Autonomic & hemodynamic instability

↑ Myocardial oxygen demand

↓
Mechanism of disease and CV Risk

- Occluded pharynx (Mueller Maneuver)
- \( \rightarrow \) Negative intrathoracic pressure
- \( \rightarrow \) Transmural gradients across atria, ventricles & aorta
- \( \rightarrow \) Ventricular function
- \( \rightarrow \) Autonomic & hemodynamic instability
- \( \rightarrow \) Myocardial oxygen demand
- \( \rightarrow \) Preload
- \( \rightarrow \) Afterload
- \( \rightarrow \) Atrial size
- \( \rightarrow \) Wall stress
- \( \rightarrow \) Stroke volume

Impaired diastolic function
Thoracic aortic dilation / dissection
Ventricular hypertrophy / remodeling
Mechanism of disease and CV Risk

From the Journal of the American College of Cardiology
Hypertension

• 50% of OSA patients are hypertensive
• 30% of hypertensive patients have OSA
• OSA may predominantly raise systolic BP\(^1\)
• Linear/causal relationship between 24-hour BP and AHI\(^2,3\)

\(^1\)Haas DC et al. Age-dependent association between sleep disordered breathing and hypertension: importance in discriminating between systolic/diastolic hypertension and isolated systolic hypertension in the Sleep Heart Health Study. *Circulation* 2005.
Hypertension

• Wisconsin Sleep Cohort study\(^1\), Sleep Heart Health Study\(^2\), Nurses Health Study\(^3\), Outcomes of Sleep Disorders in Older Men Study\(^4\)

• OSA is an independent risk factor for essential HTN

• Nocturnal BP ‘nondipping’ is associated with coexisting OSA\(^5\), cerebral white matter disease\(^6\) and all-cause mortality\(^7\)

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Hypertension

- CPAP therapy decreases Mean Arterial Pressure (MAP) by 2-5 mmHg\textsuperscript{1}
- Better antihypertensive response if daytime sleepiness\textsuperscript{2,3,4} and more severe disease\textsuperscript{5}
- CPAP markedly reduced BP and sympathetic production in sleep\textsuperscript{6}
- CPAP reduces arterial stiffness\textsuperscript{7}
- Larger prospective studies needed

\textsuperscript{1}Giles TL et al. CPAP for obstructive sleep apnoea in adults. \textit{Cochrane Database Syst Rev} 2006.
\textsuperscript{2}Robinson GV et al. CPAP does not reduce blood pressure in nonsleepy hypertensive OSA patients. \textit{Eur Respir J} 2006.
\textsuperscript{3}Faccenda JF et al. Randomized placebo-controlled trial of CPAP on BP in the sleep apnea-hypopnea syndrome. \textit{Am J Respir Crit Care Med} 2001.
Progression of HTN in OSA

- Intermittent Hypoxia
- Chemoreceptor Stimulation (6-OH dopamine)
- Sympathetic Activation
- Renin-angiotension-aldosterone System (RAAS) (aldosteronism)
Heart Failure

- SDB is present in 75% of patients with symptomatic or decompensated HF\(^1,\)\(^2\)
- 40-50% prevalence of all HF patients\(^3,\)\(^4\)
- Prevalence of 69% in HFNEF (40%/29%)\(^5\)
- Men (obesity) > women (older age)
- HF patients with SBD are older and have higher BMI, BNP, PCWP and lower PaCO\(_2\)

Heart Failure

- OSA and CSA are prevalent
- OSA may not manifest with sleepiness
- CSA/CSR – independent predictor of mortality
- HTN - most common risk factor for ventricular hypertrophy and failure
- Nocturnal O2 desaturation – predicts impaired ventricular relaxation during diastole
- LVH may be more closely linked to HTN during sleep

Progression of HF in OSA

Increased sympathetic outflow

Cytokines, catecholamines, endothelin - Ventricular hypertrophy

Negative intrathoracic pressure - increases preload and afterload

Inducing hypoxia and increases in RV afterload

Increasing risk of MI – increase myocardial demand in recurrent hypoxia
Progression of HF in CSA

- Higher resting sympathetic drive
- Pulmonary congestion
- Increased chemoreflexes
- Increased cardiac filling pressures with LV dysfunction
- Greater cardiac electrical instability
- Prolonged circulation time
Heart Failure

• OSA is associated with altered cardiac structure and function\(^1,^2,^3,^4\) and they may be reversible with CPAP

• 3 months of CPAP – improved abnormal diastolic filling and function\(^2\)

• CPAP – improved LVEF and quality of life\(^5,^6\)

• CPAP - decreases myocardial irritability and risk of arrhythmia\(^7\)

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\(^1\) Otto ME et al. Comparison of structural & functional changes in obese otherwise healthy adults w/ vs w/o OSA. *Am J Cardiol* 2007.


\(^3\) Shivalkar B et al. OSA syndrome: more insights on structural and functional cardiac alterations, and the effects of treatment with CPAP. *J Am Coll Cardiol* 2006.


Heart Failure

• Effect of CPAP on CSA in patients with HF is not well understood
• CANPAP study did not improve survival\textsuperscript{1}
• Cardiac resynchronization – CSA/CSR patients\textsuperscript{2,3,4}
• Not clear if treatment of OSA or CSA with CPAP will reduce incidence of HF or improve survival

\textsuperscript{3}Gabor JY et al. Improvement in Cheyne-Stokes Respiration following cardiac resynchronization therapy. \textit{Eur Respir J} 2005.
\textsuperscript{4}Luthje L et al. Cardiac resynchronization therapy and atrial overdrive pacing for the treatment of central sleep apnea. \textit{Eur J Heart Fail} 2009.
Stroke

• High prevalence 40-70% (OSA and CSA)\textsuperscript{1}
• Biased and study only stroke survivors and rehab patients
• SDB is a risk factor for stroke\textsuperscript{2} and stroke is a risk factor for SDB\textsuperscript{3}
• Severe SDB is an independent risk factor for stroke in men\textsuperscript{4}

\textsuperscript{1}Chan W et al. Sleep apnea in patients with TIA and minor stroke: opportunity for risk reduction of recurrent stroke. \textit{Stroke} 2010.
\textsuperscript{3}Dyken ME. Obstructive sleep apnea and stroke. \textit{Chest} 2009.
\textsuperscript{4}Redline S et al. Obstructive sleep apnea hypopnea and incident stroke: The Sleep Heart Health Study. \textit{Am J Respir Crit Care Med} 2010.
Stroke

• Increased rate of stroke or death in patients with OSA over 4 years\(^1\)
• 10 year f/u data of patients with stroke – increased risk of death with OSA (HR 1.76; 95% CI: 1.05 to 2.95; \(P=0.03\)) but not with CSA (HR 1.07; 95% CI: 0.65 to 1.76; \(P=0.80\))\(^2\)

Stroke in OSA

- Impaired endothelial function
- BP fluctuations
- Prothrombotic and proinflammatory states
- Altered cerebral autoregulation
- Accelerated atherogenesis
- Reductions in cerebral blood flow / hypoxia

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Stroke

- CPAP normalizes cerebral blood flow after 4 to 6 weeks\(^1\)
- Hypoxic events - poor post stroke recovery
- Improved long-term survival in CPAP compliant post stroke patients\(^2\)
- Low compliance
- Unclear if treatment of SDB reduces risk of stroke

\(^1\)Foster GE. Effects of CPAP on cerebral vascular response to hypoxia in patients with OSA. *Am J Respir Crit Care Med* 2007.
Arrhythmia

- Noc arrhythmias - 50% of SDB patients
- Increase with AHI and hypoxia
- Most common – Afib, nonsustained VT, sinus arrest, 2nd degree AVB, PVC’s (66%)
- 17 fold increased odds of an arrhythmia (Afib and nonsustained VT) after SDB
- Afib - CSA
- Complex ventricular ectopy – OSA

Arrhythmia

- Prevalence of undiagnosed OSA in patients with pacemakers is 59% and SDB present in 68% with AVB\(^1\)
- Postop Afib – more likely in OSA patients\(^2\)
- 50% Afib patients at cardioversion likely to have OSA\(^3\)

Arrhythmia in OSA

Cardiac vagal activation

Pressor surges

Sympathetic activation to the periphery

Hypoxemia

Transmural pressure changes

Systemic inflammation
Arrhythmia

• Risk of arrhythmia in OSA decreases with CPAP therapy\(^1\)
• Lower recurrence of Afib after elective cardioversion in OSA patients treated with CPAP (82% vs 42%) after 1 year\(^2\)
• 58% decrease in PVC’s with CPAP in patients with OSA & systolic dysfunction\(^3\)

Atrial overdrive pacing for OSA

• Initial report showed 50% reduction of OSA\(^1\)
• No effect on OSA severity or nocturnal oxygen desaturation\(^2,3,4\)

Cardiovascular Disease and SDB

- Prevalence of SDB in CAD – 2x increase\(^1\,^2\)
- Risk of CAD – primarily in men < 70\(^3\)
- OSA patients have subclinical CAD (coronary artery calcification)\(^4\)
- OSA with no CVD have microcirculation oxidant production and endothelial dysfunction\(^5\)
- Untreated severe OSA (men) at 10 yrs - increased fatal and nonfatal CV events\(^6\)

\(^2\) Shafer H et al. Obstructive sleep apnea as a risk marker in CAD. *Cardiology* 1999.
\(^3\) Gottlieb DJ et al. Prospective study of OSA and incident CHD and heart failure: the SHHS. *Circulation* 2010.
\(^5\) Patt BT et al. Endothelial dysfunction in the microcirculation of patients with OSA. *Am J Respir Crit Care Med* 2010.
Cardiovascular Disease and SDB

• ST depressions occur in 1/3 of patients with severe OSA\textsuperscript{1}
• Nocturnal angina and ST depressions are diminished with CPAP\textsuperscript{2,3}
• Patients with SDB and CAD – 5 yr f/u composite endpoint (death, MI, CVA) - men 28 vs 16%, women 20 vs 14%\textsuperscript{4}

\textsuperscript{1}Hanly P et al. ST - segment depression during sleep in OSA. \textit{Am J Cardiol} 1993.
\textsuperscript{2}Franklin KA et al. Sleep apnoea and nocturnal angina. \textit{Lancet} 1995.
\textsuperscript{3}Peled N et al. Nocturnal ischemic events in patients with OSA syndrome and ischemic heart disease: effects of CPAP treatment. \textit{Am J Coll Cardiol} 1999.
\textsuperscript{4}Mooe T et al. Sleep disordered breathing and coronary artery disease: long-term prognosis. \textit{Am J Respir Crit Care Med} 2001.
Cardiovascular Disease and SDB

• Sudden cardiac death – no OSA (6 to 11am), OSA (10pm to 6am)¹
• Decreased occurrence of CV death, ACS, HF, revascularization – if treat OSA²
• Men with severe OSA – marked increase in fatal and nonfatal CV events (much less if treated with CPAP)³

CAD and OSA after PCI

• OSA associated with restenosis and remodeling\(^1\)
• Increase incidence of revascularizations and cardiac mortality\(^2\)
• Less increase in LVEF and wall motion\(^3\)
• Treatment of OSA after PCI – reduced cardiac deaths\(^4\)

\(^3\)Nakashima H et al. OSA inhibits the recovery of LV function in patients with acute myocardial infarction. *Eur Heart J* 2006.
\(^4\)Cassar A et al. Treatment of OSA is associated with decreased cardiac death after PCI. *J Am Coll Cardiol* 2007.
Cardiovascular Disease in OSA

- Increased BP
- Hypoxemia and acidosis
- Intrathoracic and cardiac transmural pressure changes
- Systemic inflammation
- Endothelial dysfunction and vascular remodeling
- Sympathetic vasoconstriction
Cardiovascular Disease and SDB

• CPAP improves early atherosclerosis\(^1\) and microvascular disease and endothelial dysfunction\(^2\)

• No randomized control trials for treatment of OSA and risk of CAD, MI or sudden death

• **RICCADSA** study – CPAP on composite endpoint in CAD & OSA patients over 3 years

• **HeartBEAT** study – CPAP or low flow O\(_2\) will change cardiac biomarkers

• **SAVE** study – CPAP to treat OSA to prevent CVD

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\(^1\) Drager LF et al. Effects of CPAP on early signs of atherosclerosis in OSA. *Am J Respir Crit Care Med* 2007.

Pulmonary Arterial Hypertension (PAH)

• Prevalence of PAH in OSA – 17 to 42%
• Pulmonary hypertensive OSA patients - increased pulmonary vascular reactivity to hypoxia¹
• ? OSA causes sustained daytime PAH
• Pulmonary artery pressures decrease after treatment with CPAP²,³

End Stage Renal Disease (ESRD)

- Prevalence of OSA in ESRD is 40 to 60% (often mixed sleep apnea)
- OSA - contribute to ESRD by chronic HTN
- OSA may increase risk of CV complications
- Prevalence of OSA in ESRD is the same before and after HD¹
- Nocturnal peritoneal dialysis attenuate sleep apnea²

ESRD in OSA

Uremia – destabilized ventilatory control & upper airway edema

Hypoxemia

Sympathetic nerve discharge at kidney

Glomerular hyperfiltration

Chronic Hypertension

Systemic Inflammation

↑ Cystatin C
Treatment of SDB

• **OSA**
  - Weight loss
  - Positional sleep
  - Oral appliance
  - Surgery – tracheostomy, UPPP
  - Positive airway pressure

• **CSA**
  - Optimize HF treatment (ACE-I, diuresis, β blocker)
  - Cardiac resynchronization therapy (CRT)
  - CPAP, BiPAP, Adaptive pressure support servoventilation
  - Supplemental O2
  - Acetazolamide
Summary

• A persuasive body of data supporting a causal association between SDB and cardiovascular disease

• Randomized control trials are underway to investigate whether
  – SDB accelerates heart disease progression
  – treatment results in fewer cardiovascular events
  – treatment reduces mortality
Contact Information

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