MCOEM Spring Chapter Meeting
April 5, 2014

“Sleep Apnea- An Overview with Emphasis on Cardiovascular Correlations”
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History of Present Illness

57 year old man with h/o HTN, CVA, PVD who presents with chief complaint of poor sleep and snoring for many years. He also complained of feeling very fatigued during the day. His ESS was 20/24. He frequently awakens with a choking sensation.
Case Presentation (2)

Medications
Atenolol, Lisinopril, Lipitor

Social History: Patient delivers baked goods (drives a straight truck)

Physical Exam
BP = 140/80
BMI = 35 kg/m2, Neck size = 18 in
ENT: posterior pharyngeal crowding
Case Presentation (3)

Clinical Course

The patient underwent in-lab PSG that revealed severe OSA (AHI=30). In-lab CPAP titration showed that a setting of 7 cwp was effective.

Diagnoses

1. Obstructive Sleep Apnea
2. Hypertension
Sleep Apnea and Cardiovascular Disease

MCOEM Spring Update
April 5, 2014

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Sleep Apnea and Cardiovascular Disease

- Sleep Apnea Background
  - Definition of Sleep Apnea
  - Clinical Findings
  - Upper Airway Physiology
  - Epidemiology of OSA
- OSA and Daytime / Nighttime BP Disturbance
- OSA – Risk factor for CV Disease (HTN / CAD / Cardiac Arrhythmia)
Obstructive Sleep Apnea: Definition

• Functional **obstruction** of the upper airway during sleep.

• Repetitive **breathing pauses** (APNEA) and **disturbed sleep** (AROUSALS).
Obstructive Sleep Apnea: Clinical Findings

- Sleepiness
- Severe Snoring
- Obesity
Obstructive Sleep Apnea: Consequences of Sleepiness

- Impaired judgment
- Reduced concentration
- Delayed reaction time
- Microsleeps
Obstructive Sleep Apnea: Motor Vehicle Accidents

The Association between Sleep Apnea and the Risk of Traffic Accidents (Teran-Santos J et al., NEJM 1999; 340: 847-851)

• **Design**: Case control study of the relation of sleep apnea and the risk of traffic accidents

• **Subjects**: 102 drivers who received emergency treatment

• **Conclusion**: Strong association between sleep apnea and the risk of traffic accidents.
Upper Airway Obstruction During Sleep

AIRWAY LOADING
Nasal Obstruction / Airway Narrowing

Negative Inspiratory Pressure

Activity of Dilator Muscles

WAKE
Increased Activity
Airway Patency Maintained

SLEEP
No Increased Activity
Airway Patency Lost
Normal Upper Airway

During normal sleep, air flows freely past the structures in the throat.
Obstructive Apnea
CPAP Therapy
Obstructive Sleep Apnea: Epidemiology

- Male predominance (4:1)
- Peak occurrence between ages 40 and 50 yrs.
- Incidence of Obstructive Sleep Apnea Syndrome (AHI>15 and sleepiness) in the adult population between 1% and 5%.
- Incidence of Obstructive Sleep Apnea Syndrome (AHI>15 and sleepiness) in the adult male population of 5% to 9%.
- African Americans (elderly) when compared to Caucasians have twice the relative risk of severe Obstructive Sleep Apnea
- Up to 40% of patients with Essential Hypertension have undiagnosed / untreated Obstructive Sleep Apnea
Arterial Blood Pressure Changes During Obstructive Sleep Apnea

Chest
Abdomen
Blood Pressure
Cardiac Output
Obstructive Sleep Apnea
Three Physiological Study Areas

• The Mueller Maneuver

• Breath-Hold / Diving Refex

• Sleep State Change
Mueller Maneuver

Mechanical Effects of OSA on Circulation

• Voluntary inspiratory efforts against a closed glottis

• Effects include
  – Large decrease in intrathoracic pressure
  – Decreased in LV stroke volume (increased afterload)
  – Increase in RV stroke volume
  – Blood pools in the pulmonary circulation
Breath-Hold / Diving Reflex

• Hypoxic stimulus coupled with an inability to move air

• Effects include
  – Increase in sympathetic nerve activity to peripheral vasculature – vasoconstriction
  – Increase in parasympathetic activity to the heart – bradycardia
  – Acts to maintain blood flow to the heart / brain and decrease metabolic demands on the heart
Sleep State Change

• At apnea termination, patients may progress from NREM or REM sleep to transient wakefulness.

• Transition to wakefulness is associated with relative tachycardia and increased blood pressure.
Obstructive Sleep Apnea and Increased Sympathetic Activity

Surges of muscle sympathetic nerve activity during obstructive apnea are linked to hypoxemia

Leuenber U et al.

Methods: Studied muscle sympathetic nerve activity (MSNA; peroneal microneurograph) and BP during awake regular breathing and during sleep. Also studied the effects of 100% O2 on MSNA and BP

Subjects: OSA (n=12) and Age-matched controls (n=15)
Obstructive Sleep Apnea and Increased Sympathetic Activity
Obstructive Sleep Apnea and Increased Sympathetic Activity

Results

• In awake regularly breathing patients with OSA (n=12) resting MSNA was higher than in age-matched controls (n=15)

• Apneas during sleep associated with surges in MSNA followed by transient rises in BP when breathing resumed

• In contrast to room air apneas – hyperoxic apneas of similar duration were associated with attenuated MSNA responses
Obstructive Sleep Apnea and Increased Sympathetic Activity

Conclusions

• BP oscillations that occur with apnea during sleep may in part be mediated by intermittent surges of sympathetic activity resulting in vasoconstriction.

• Because they are blunted by O2 administration – they appear to be linked to intermittent arterial hypoxemia and stimulation of arterial chemoreceptors.
Obstructive Sleep Apnea and Daytime Hypertension

Obstructive sleep apnea as a cause of systemic hypertension. Evidence from a canine model.
*J Clin Invest* 1997;99:106-9
Obstructive Sleep Apnea and Daytime Hypertension

Mean Nighttime BP

Mean Daytime BP
Obstructive Sleep Apnea and Daytime Hypertension

Results

• Canine model of OSA produces a nighttime BP disturbance just like that in humans

• Also generates an awake daytime mean arterial BP rise to 18% above baseline

• Daytime HTN resolves within one month of cessation of the OSA
The Sleep Heart Health Study

Association of sleep-disordered breathing, sleep apnea, and hypertension in a large community-based study

Nieto et al.
JAMA 2000;283:1829-1836

Purpose: To assess the association between SDB and hypertension in a population-based sample

Subjects: 6,132 SHHS participants (age ≥ 40 years, 52.8% female)
Patient Cohorts and Study Sample

• Subjects recruited from participants in ongoing cohort studies (CV / Resp Disease)

• Inclusion / Exclusion Criteria
  – >= 40 years of age
  – No history of treatment of sleep apnea with CPAP
  – No tracheostomy
  – No current home O2 therapy
Baseline Examination

- A self-administered sleep habits questionnaire

- Home visit:
  - Brief health interview
  - Medication history
  - Blood pressure measurement
  - Anthropometric measurements
  - Full unattended PSG (6,841 home studies – 6% failure rate)
Study Variables

• Hypertension
  – BP > 140/90 and/or
  – current treatment with Anti-HTN Meds

• Apnea-Hypopnea Index

• Arousal Index

• % of Total Sleep Time with oxygen saturation below 90%

• Anthropometric Variables
Results

• Mean systolic and diastolic blood pressure and the prevalence of HTN increased with increasing AHI.

• Some of this association was explained by BMI and other anthropometric variables (neck circumference and waist/hip ratio).

• After adjusting for all these variables there was a significantly elevated odds of HTN in more severe OSA (OR=1.4)
  AHI ≥ 30
  % time below a 90% O2 saturation ≥ 12%
Conclusion

These findings from the largest cross-sectional study to date show that OSA is associated with systemic hypertension, particularly middle-age individuals.
Nasal CPAP

Effects of CPAP Versus Supplemental Oxygen on 24-Hour Ambulatory Blood Pressure
Norman, Daniel et al.
_Hypertension_ 2006;47(5):840-845

Study Design
A randomized double-blind, placebo controlled study comparing the effects of 2 weeks of CPAP versus sham-CPAP versus supplemental nocturnal oxygen on 24 hour ambulatory blood pressure in 46 patients with moderate to severe OSA (both normotensives and hypertensives)
Nasal CPAP

Daytime

Nighttime

Change in BP (mm Hg)

SBP  MAP  DBP

Placebo  CPAP  Oxygen

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Nasal CPAP

1. Two weeks of CPAP therapy significantly lowered nighttime diastolic, mean, and systolic BP and daytime mean and diastolic BP in patients with OSA.
2. Supplemental oxygen did no result in a lowering of daytime or nighttime BP.
3. Two weeks of sham-CPAP did not result in a significant change in daytime BP and actually raised nighttime systolic BP.
Sleep Apnea and Coronary Artery Disease

• The incidence of OSA is 2 to 3 fold higher in patients with symptomatic CAD relative to age and gender matched healthy individuals.

• There is a high prevalence of OSA-associated nocturnal myocardial ischemia in patients with both CAD and moderate to severe OSA.

• A direct causal relationship between OSA and CAD has yet to be firmly established.
OSA and Cardiac Arrhythmias

• Tachyarrhythmias
  – Atrial Fibrillation / Flutter
  – Ventricular ectopy

• Bradyarrhythmias
  – Sinus arrest / AV block / Sinus Brady
  – Related to hypoxemia / cessation of breathing
  – Secondary to increased vagal tone
Sleep Apnea and Cardiovascular Disease Summary

• There is a nighttime / daytime blood pressure disturbance in patients in OSA.
• Disturbance is mediated by sympathetic nervous stimulation and hypoxemia.
• Disturbance is attenuated by CPAP
• OSA – independent risk factor for HTN, CAD, Cardiac Dysrhythmia