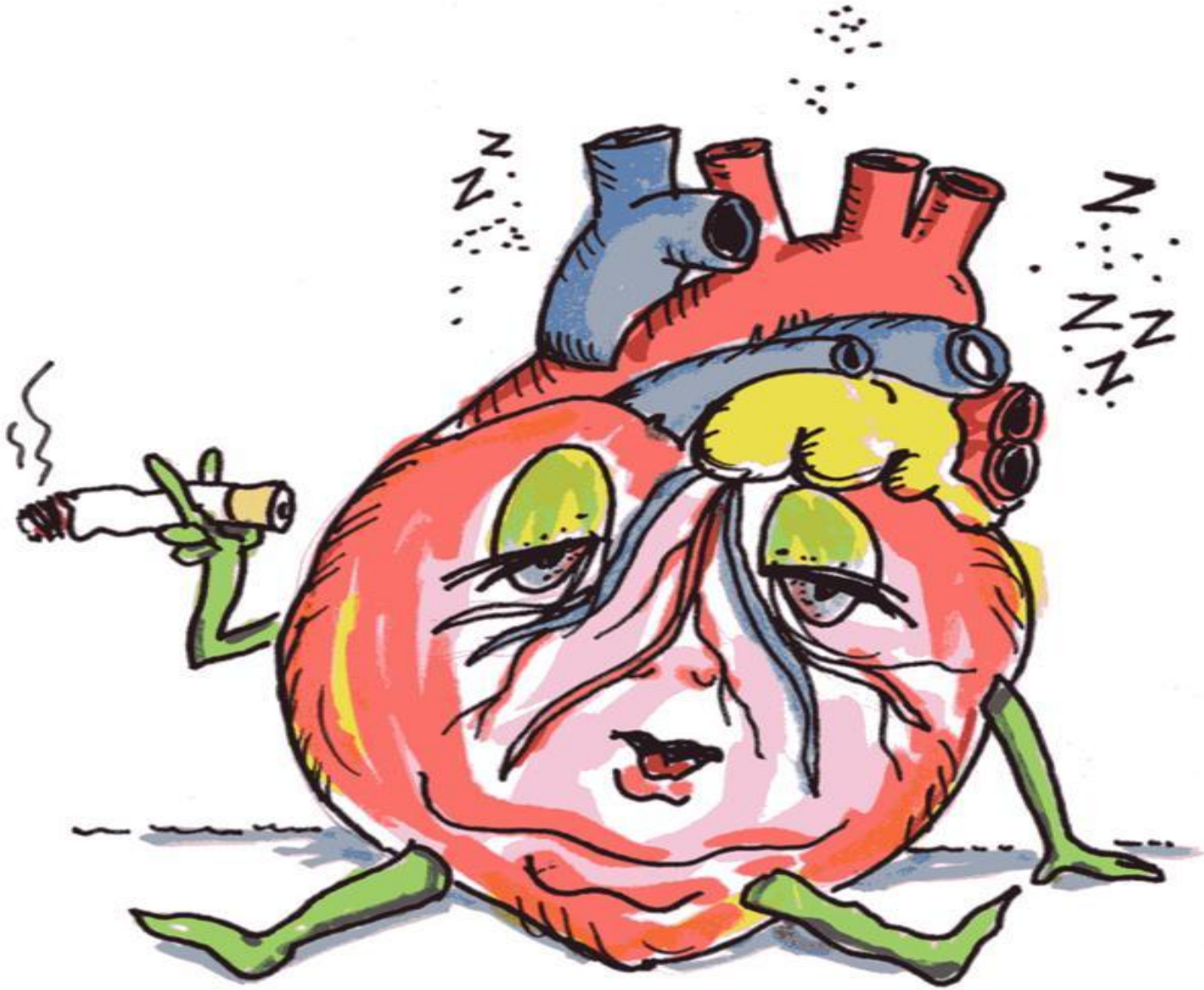


***Cardiovascular  
Consequences of Sleep  
Disordered Breathing:  
Mechanisms***

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# Arousals

- Cause increased sympathetic activation and decreased parasympathetic activity
  - Increased HR
  - Increased BP
  - Increased O<sub>2</sub> consumption
  - Increased afterload



# Dysfunction created by Sleep Disordered Breathing

Hypoxemia & reoxygenation

Hypercapnea – hypocapnea

Large negative swings in intrathoracic pressure,

↑ afterload, ↑ atrial transmural pressure leading to atrial stretch and A Fib, LVH

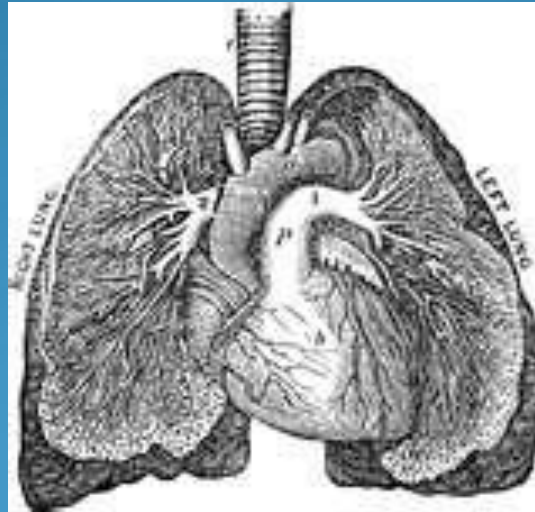
Arousals cause sympathetic activation &

↓ parasympathetic activity

↑ HR, ↑ BP

↑ O<sub>2</sub> Consumption, ↑ afterload


# Currently accepted cause and effect



# Nocturnal & diurnal hypertension

- Decreases and increases in wall tension
- Alterations in CBF
- Adhesion molecules
- Inflammation
- Transcription Factors
- Oxidative Stress
- Platelet aggregation/coagulopathy/thrombosis
- Metabolic dysregulation
- Leptin resistance

# Resultant cardiovascular derangements

- 
- Endothelial dysfunction
  - Systemic Hypertension
  - Pulmonary Hypertension
  - Systolic Heart Failure
  - Diastolic heart failure
  - Arrhythmias
  - Atherosclerosis, CAD, CVA

# HBV



# HF



- 50% of OSA patients are hypertensive
- 30% of hypertensive patients have OSA
- Drug resistant hypertension = 83% have OSA
  - Non dippers (no nocturnal drop in BP) have more OSA and higher 1.3 hazard ratio for mortality
  - LVH = non dipping
- Mechanisms: 1. intermittent hypoxemia ,  
2. sympathetic stimulation, 3. renin angiotension activation
- Therapy with CPAP benefits hypertensives more than normals. Severe OSA = Most benefit
- More studies needed

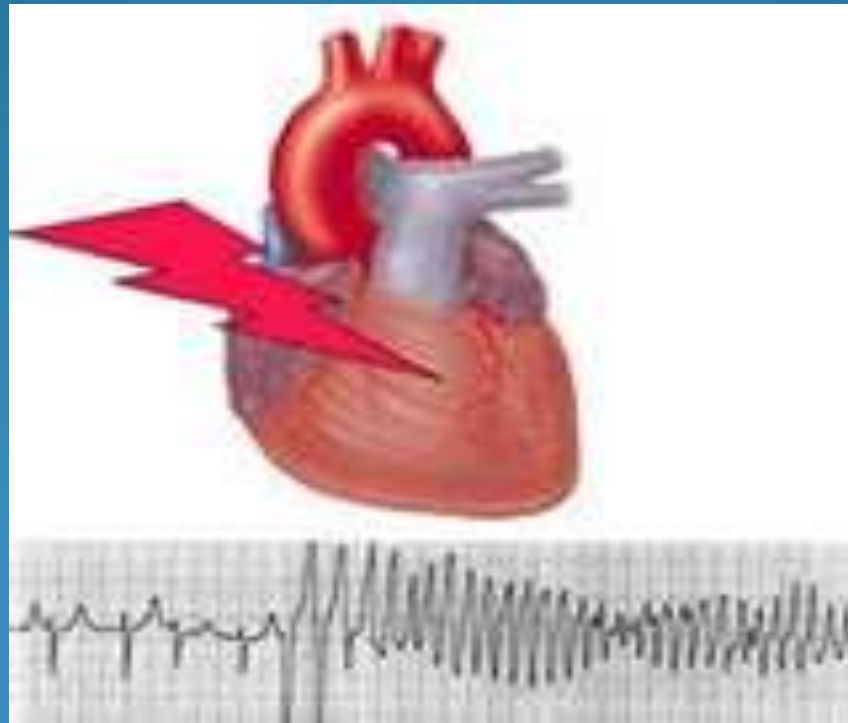
- OSA seen in 37% of patients with systolic failure. Only a minority have excessive daytime sleepiness (EDS) - more in men, more in diastolic failure, and CPAP helps
- Mechanisms: HBP worsens systole, hypoxemia worsens diastole. BMI correlates with CHF. LVH may result from OSA independently of HBP.
- Hypoxemia & RV afterload
- ↑ Risk of myocardial infarction
- Response to therapy with CPAP, up to 9-12% increase in EF in 3 mos, lowers HR, BP possibly mortality reduction

# CVA



- 62% of TIA patients have AHI > 10/hr
- Of 132 stroke patients in rehab, 53 had OSA, CSA or mixed openers
- Compliance on CPAP after CVA is 15-70%
- Severity of OSA correlates with risk of CVA, and oximetry results after CVA correlate with mortality
- Mechanisms: Oxidative stress, hypoxemia, cytokines, A Fib, HBP

# Arrhythmias



Nocturnal arrhythmias occur in 50% of OSA pts: NSVT, sinus arrest, second degree heart block, PVCs, 2-4 fold increase in severe OSA patients.

PVCs seen in 66% of OSA pts.

VTach seen in sats < 60%

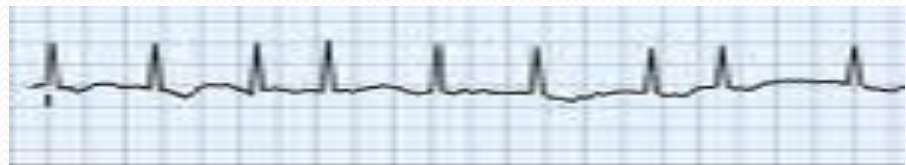
Bradycardia- due to dive reflex after prolonged apnea, REM sleep, hypoxemia. CPAP reverses it.

- 59% of pacemaker pts have OSA
- OSA seen in 68% of pts with AV block
- Mechanisms: Desaturation, dive reflex, REM sleep, sympathetic stimulation, organic heart disease,
  - 58% reduction with CPAP therapy

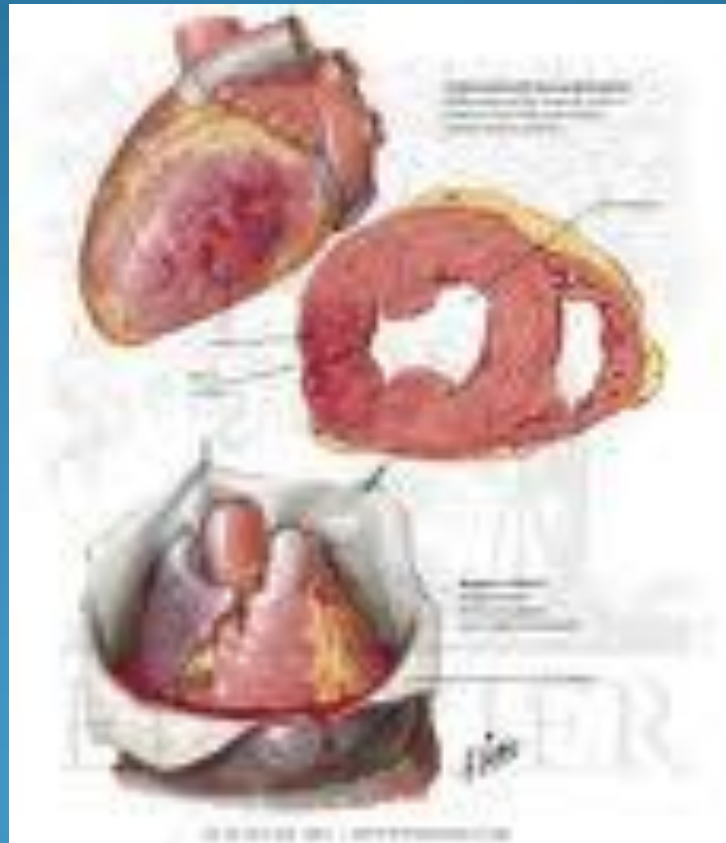


# Atrial Fibrillation

- Obesity and hypoxemia are independent predictors of A Fib in pts < 65.
- 82% recurrence of Afib of OSA untreated after cardioversion
- 50% of pts presenting for cardioversion have OSA (30% in general cardiol population)
- A Fib role in CVA in OSA pts undetermined
- Mechanisms: LA stretch more common in OSA, recurrent hypoxemia, intermittent wide catediol swings, arousal related startle, Sats dramatically low, atrial pacing of no benefit



# MI / CAD

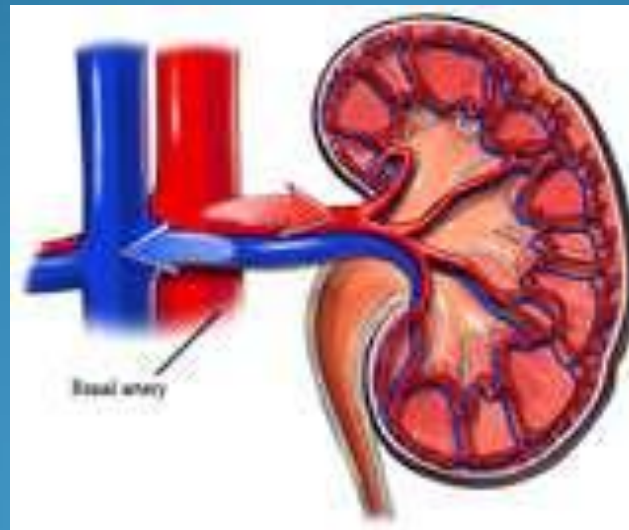


- CAD twice as likely to occur in OSA pts

Mechanism: intermittent hypoxemia, acidosis, BP rise, sympathetic vasoconstriction, changes in transmural and transthoracic pressures, all seen in OSA.  
(Chronically, endothelial dysfunction and inflammation)

- Coronary calcium score correlates with OSA
- SDB pts with CAD have more MI's, CVA's, and mortality is higher
- In sudden cardiac death, OSA pts die 10 PM - 6 AM, and non OSA die 6 AM - 11 AM
- CPAP therapy reduces CV events, to same as simple snorers

# End Stage Renal Disease



- EDS common symptom in CRD. OSA 40-60%
- Obstructive and Central apneas

Mechanism:

Chronic BP elevations

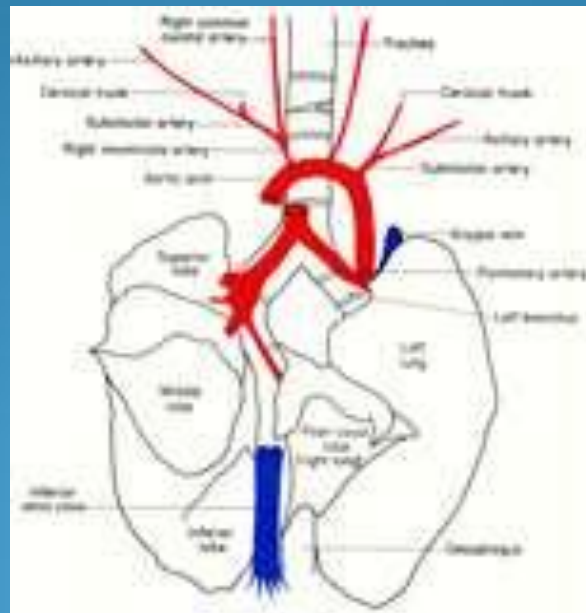
Sympathetic nerve discharge

Glomerular hyperfiltration

Periodic limb movement are associated with  
premature mortality

Uremia may destabilize central ventilatory control  
and cause airway edema

# Pulmonary Hypertension



- 17% of pts with AHI>20 had PAP > 20 most being obese with hypoxemia, hypercapnic

### Mechanisms:

- Hypoxemia → Acute increase in pressure
- CO & wedge usually normal
- Contributors, obesity, hypovent syndrome and COPD
- Marked intrathoracic negative pressure during apnea brings ↑ venous return to right heart and pulmonary circuit
- CPAP helps lower pressures, by Echo

**Other**

- Sleep attacks vs syncope
- Central Sleep Apnea
- Cheyne Stokes Breathing
- Diabetes (Leptin and weight)
- Pregnancy, and women in general
- Mortality
- Future research opportunities





# Summary Thoughts

**Questions?**

# References

Sleep and Cardiovascular disease, Sleep Medicine Clinics, 2007

ACCF/AHA Scientific Statement on Sleep Apnea and Cardiovascular Disease; Circulation, 2008.

Practice Parameters in Sleep Medicine; SLEEP, 2005.  
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