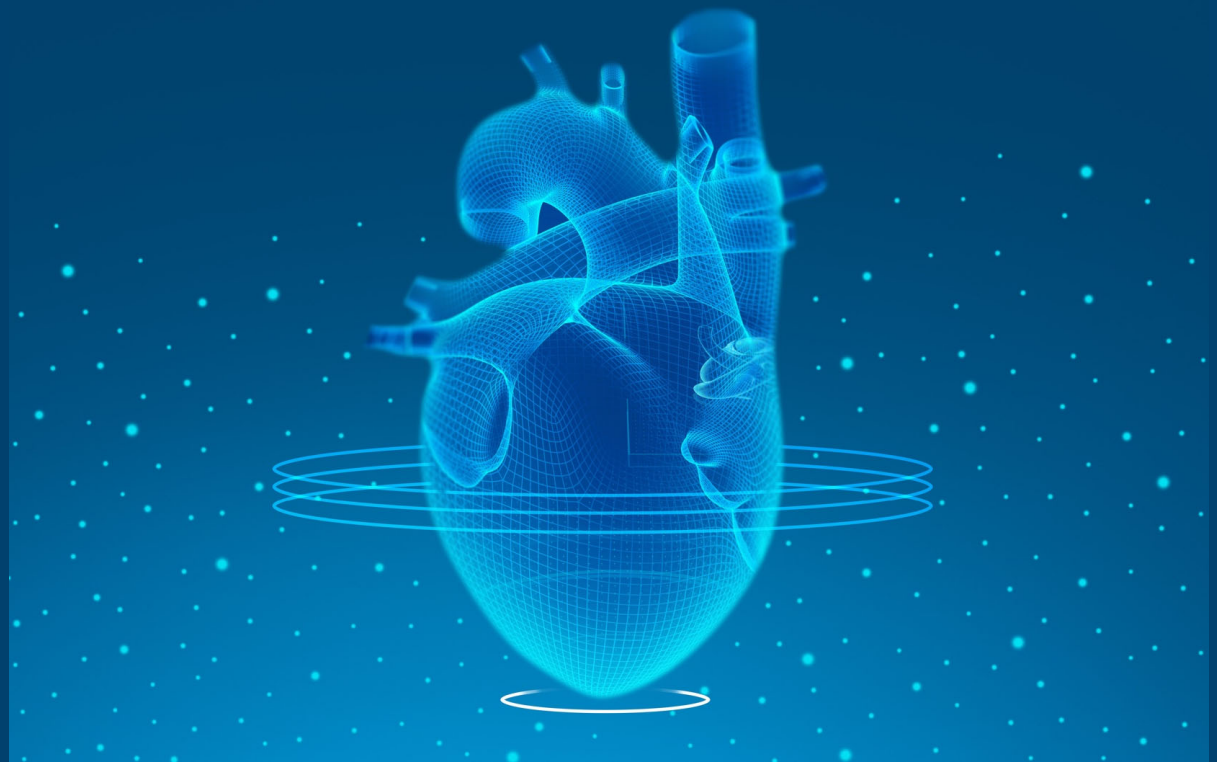
Minneapolis Heart Institute Foundation®

GRAND ROUNDS



MHI GRAND ROUNDS NOVEMBER 22, 2021

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EFFECT OF SLEEP-DISORDERED BREATHING ON CARDIOVASCULAR HEALTH

- No relevant conflict of interest pertaining to this topic.
- Served as primary investigator with: Novartis, GSK, AstraZeneca, Bayer, Merck, NeRRe, Pearl pharmaceutical, Shinogi, Jazz pharmaceutical, Theravance, Sunovion

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CASE

- 59 y.o. female with history of atrial fibrillation, asthma, Obesity, tobacco use disorder
- Admitted on 10/3/2021 following presentation to ANW emergency department for evaluation of worsening shortness of breath.
- suffering intermittent shortness of breath for several months, but worse the last several weeks.
- She quit smoking 2/2021. She reports having multiple steroid bursts in the last few months and reports that she usually feels better when she is on the steroids but that symptoms worsen again after stopping them.
- She was also given a course of cefdinir by her primary care physician 8/2021.

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- Patient reports that she has gained approximately 100 pounds in the last 2 years after switching jobs.
- Reports lower extremity edema, using as needed Lasix
- Feels that when her edema is better, her breathing is also better.
- She has a history of severe obstructive sleep apnea but does not use CPAP because she reports it previously caused her to have facial cellulitis and has had difficulty with the fit of nasal masks, although she admits she only tried one mask.
- Patient reports a history of atrial fibrillation but was previously told that anticoagulation was not indicated given her risk factors???

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- She was on diltiazem a few years ago when she was diagnosed with atrial fibrillation but had lower extremity edema and reports it made her thinking less clear so stopped it.
- She is hypoxic requiring BIPAP

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EXAM

- BP 120/92 | Pulse (!) 163 | Temp 99.6 °F (37.6 °C) | Resp (!) 26 | Ht 1.657 m (5' 5.25") | Wt (!) 138.3 kg (305 lb) | LMP 01/28/2011 | SpO2 98% (on 60% BiPAP) | BMI 50.37 kg/m² Temp (24hrs), Avg:99.6 °F (37.6 °C), Min:99.6 °F (37.6 °C), Max:99.6 °F (37.6 °C)
- Respiratory: Expiratory wheeze right base and bibasilar crackles Tachypnea mild distress
- Cardiovascular: Irregularly irregular HRs 120-160s, no murmurs appreciated
- Gastrointestinal: Protuberant, Normal BS, abdomen soft, obese, non-distended, non-tender
- Extremities: 2+ pitting edema bilaterally distal to the knees

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DIAGNSTIC DATA

	10/03/21 1817
SODIUM	138
POTASSIUM	3.8
CHLORIDE	103
CO2TOTAL	22
ANIONGAP	13
BUN	13
CREATININE	0.84
GLUCOSE	120*
CALCIUM	9.3
GFRAFRICAN	>60
GFRNOTAFRICA	>60

	10/03/21 1817
WBC	6.7
HGB	12.9
HCT	40.8
MCV	86
MCH	27.2
MCHC	31.6*
RDW	14.8
PLT	242

Cardiopulmonary | **Infection / Inflammatory** | **COVID-19**

COVID-19: Pending

	10/03/21 1817	10/03/21 1818
TROPONINI	0.025	--
BNP	638*	--
DDIMER	--	1.40

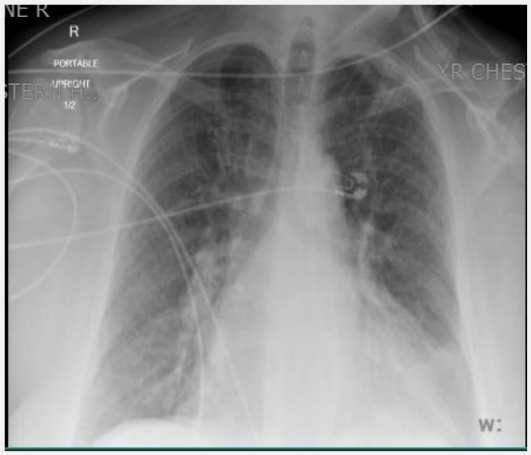
Recent Labs

	10/03/21 1818	10/03/21 1817
DDIMER	1.40	--
NRBC	--	0.0
ABSNRBC	--	0.0

Urinalysis | **Endocrine** | **Blood Gases**

	10/03/21 1817
PHVENOUS	7.43
PCO2VENOUS	39*
PO2VENOUS	70*
HCO3VENOUS	26

Chest Radiograph 10/3
No acute cardiopulmonary disease. Minor interstitial prominence diffusely not greatly changed. Heart size is upper normal. No bone finding of significance.



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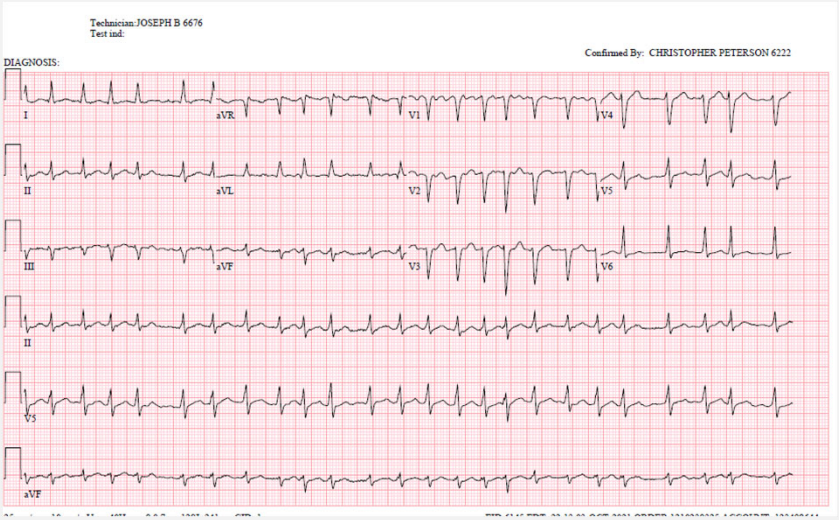
- ECG interpretation

Atrial fibrillation with rapid ventricular response
Septal infarct , age undetermined
Abnormal ECG



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CT CHEST 10/3

IMPRESSION:

No pulmonary emboli. Nonspecific mild right hilar and mediastinal adenopathy. Cardiomegaly.. Hepatomegaly.

ECHOCARDIOGRAM

Final Impressions:

1. Technically limited exam.
2. Mild to moderately increased LV size, mildly increased wall thickness, mildly reduced global systolic function with an estimated EF of 40 - 45%.
3. Right ventricular cavity size is mildly enlarged; global systolic RV function is mildly reduced.
4. Severely enlarged left atrium.
5. Moderate posteriorly directed mitral regurgitation.
6. Mild-moderate tricuspid regurgitation.
7. The inferior vena cava is dilated, respiratory size variation less than 50%.

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HOSPITAL COURSE

Cardiology and Pulmonary consulted.

The patient was managed with diltiazem drip and IV furosemide to achieve euvolemia.

Started steroid taper with optimization of inhalers with pulmonary follow up.

patient finally agreed to DOAC, although she would frequently try to drive her own cares.

Cardiology follow up scheduled

Otherwise had improvement in HR control and patient felt to be euvolemic.

She felt much improved symptomatically. She was discharged to home in stable condition with recommendation for outpatient PT (which she declined).

Was agreeable to undergo PAP titration study (scheduled but missed appointment November 5)

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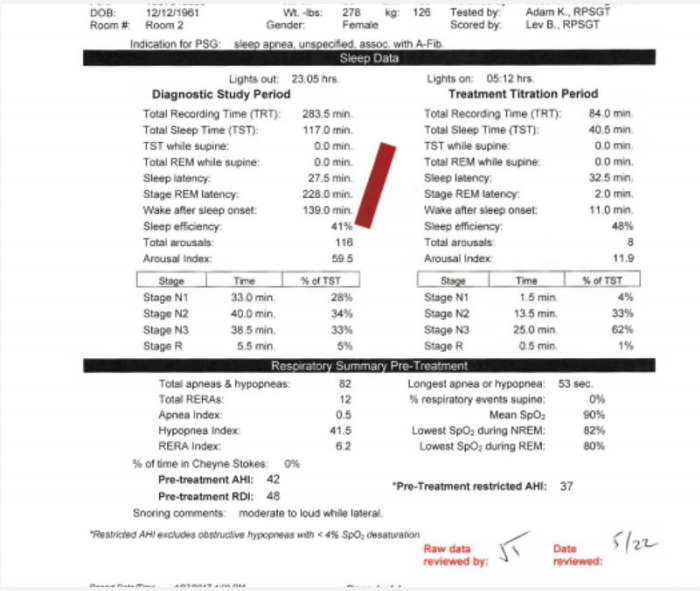
SPLIT NIGHT STUDY 2017



DOB: 12/12/1961 Wt. -lbs: 278 kg: 126 Tested by: Adam K., RPSGT
Room #: Room 2 Gender: Female Scored by: Lev B., RPSGT

Indication for PSG: sleep apnea, unspecified, assoc. with A-Fib.

Sleep Data

Diagnostic Study Period		Treatment Titration Period	
Lights out:	23:05 hrs.	Lights on:	05:12 hrs.
Total Recording Time (TRT):	283.5 min.	Total Recording Time (TRT):	84.0 min.
Total Sleep Time (TST):	117.0 min.	Total Sleep Time (TST):	40.5 min.
TST while supine:	0.0 min.	TST while supine:	0.0 min.
Total REM while supine:	0.0 min.	Total REM while supine:	0.0 min.
Sleep latency:	27.5 min.	Sleep latency:	32.5 min.
Stage REM latency:	228.0 min.	Stage REM latency:	2.0 min.
Wake after sleep onset:	139.0 min.	Wake after sleep onset:	11.0 min.
Sleep efficiency:	41%	Sleep efficiency:	48%
Total arousals:	116	Total arousals:	8
Arousal Index:	59.5	Arousal Index:	11.9

Stage	Time	% of TST	Stage	Time	% of TST
Stage N1	33.0 min.	28%	Stage N1	1.5 min.	4%
Stage N2	40.0 min.	34%	Stage N2	13.5 min.	33%
Stage N3	38.5 min.	33%	Stage N3	25.0 min.	62%
Stage R	5.5 min.	5%	Stage R	0.5 min.	1%

Respiratory Summary Pre-Treatment

Total apneas & hypopneas:	82	Longest apnea or hypopnea:	53 sec.
Total RERAs:	12	% respiratory events supine:	0%
Apnea Index:	0.5	Mean SpO_2	90%
Hypopnea Index:	41.5	Lowest SpO_2 during NREM:	82%
RERA Index:	6.2	Lowest SpO_2 during REM:	80%

% of time in Cheyne Stokes: 0%

Pre-treatment AHI: 42 ***Pre-Treatment restricted AHI:** 37

Pre-treatment RDI: 48

Snoring comments: moderate to loud while lateral.

**Restricted AHI excludes obstructive hypopneas with < 4% SpO_2 desaturation*

Raw data reviewed by: Date reviewed: 5/22

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EFFECT OF SLEEP-DISORDERED BREATHING ON CARDIOVASCULAR HEALTH

- Obstructive sleep apnea (OSA) has been associated with many different forms of CVD including hypertension, stroke, HF, coronary artery disease, and atrial fibrillation (AF).
- Adults with OSA not only have an increased risk of developing comorbid CVD but also have worse outcomes related to CVD. OSA is highly prevalent, estimated to affect 24% of men and 9-15% of women in the general population and 40% to 60% of patients with CVD.
- Furthermore, the prevalence is increasing, with these figures representing a 30% increase over the previous 2 decades, likely related to the obesity epidemic as well as an aging population.

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EFFECT OF SLEEP-DISORDERED BREATHING ON CARDIOVASCULAR HEALTH

- 15% of US population affected by a sleep breathing disorder
- Men AHI > 5/hr = 24% >15/hr = 9%
- Women AHI > 5/hr =9% >15/hr= 4% (significant increase postmenopausal)
- OSA in HTN = 30%
- OSA in CHF = 38% in men and 31% in women

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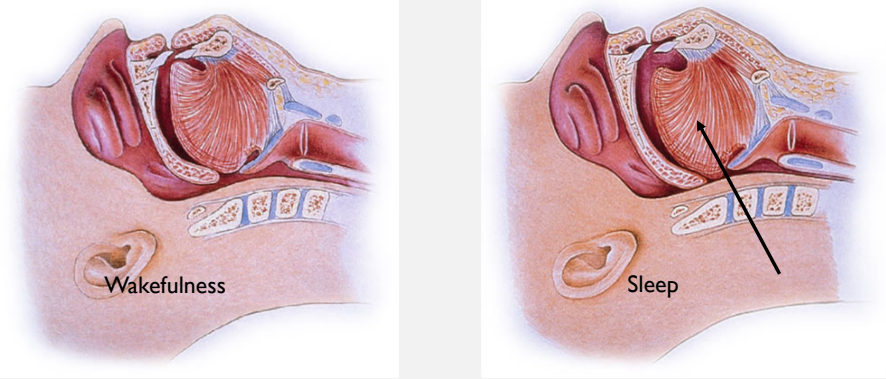
PATHOPHYSIOLOGY OF OBSTRUCTIVE APNEA



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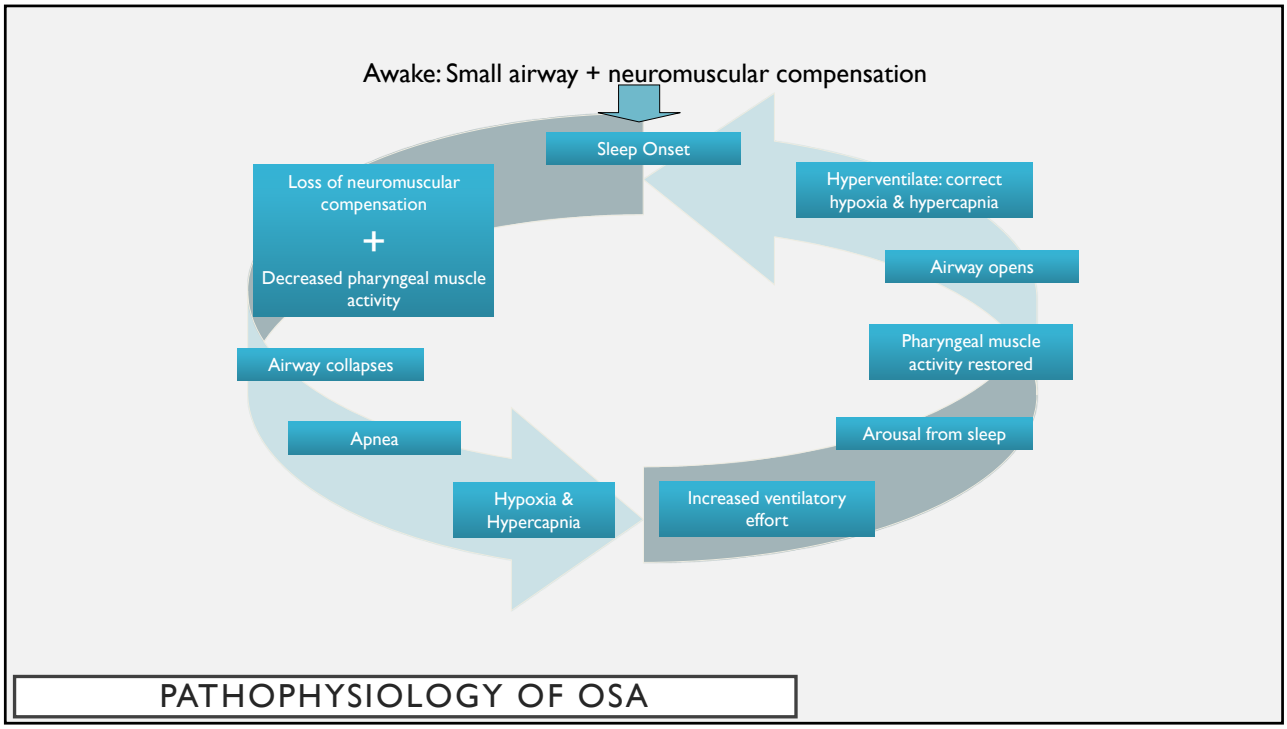
Awake: Small airway + neuromuscular compensation
Sleep Onset
Loss of neuromuscular compensation
+
Decreased pharyngeal muscle activity
Airway collapses
Apnea
Hypoxia & Hypercapnia
Increased ventilatory effort
Arousal from sleep
Pharyngeal muscle activity restored
Airway opens
Hyperventilate: correct hypoxia & hypercapnia
PATHOPHYSIOLOGY OF OSA

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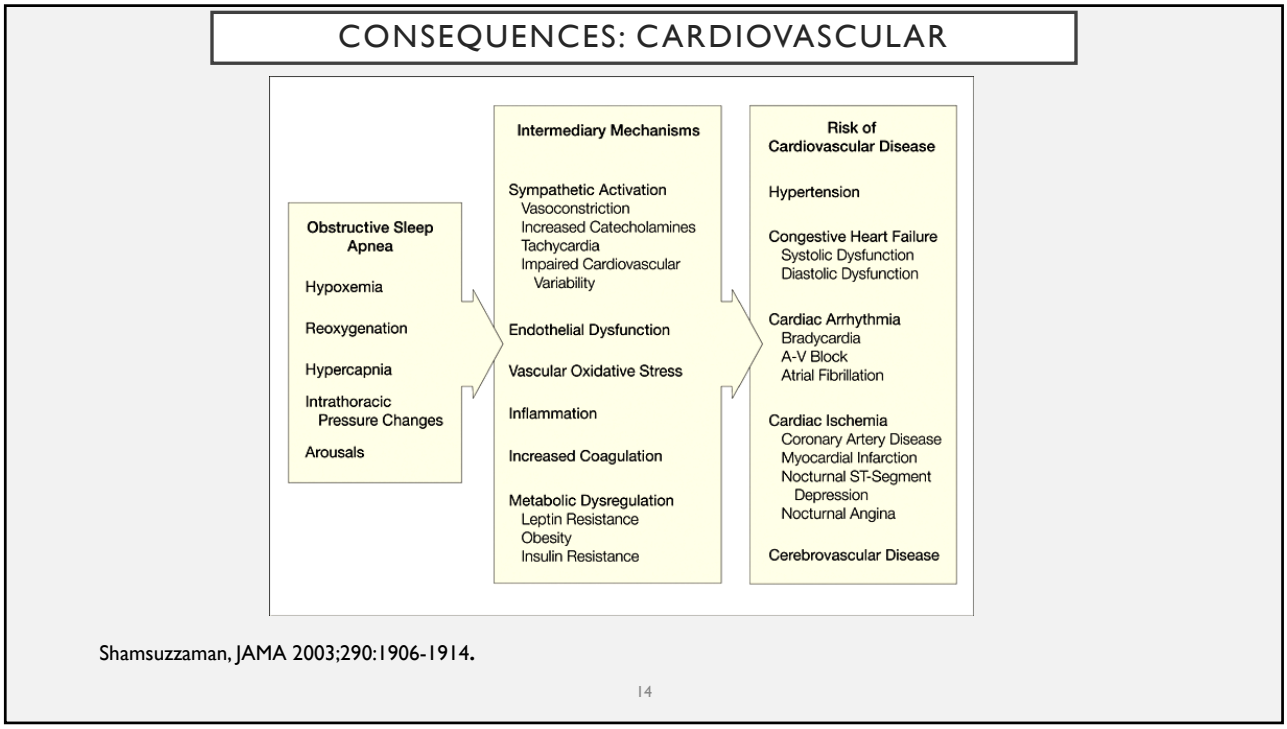
CONSEQUENCES: CARDIOVASCULAR
Obstructive Sleep Apnea
Hypoxemia
Reoxygenation
Hypercapnia
Intrathoracic Pressure Changes
Arousals
Intermediary Mechanisms
Sympathetic Activation
Vasoconstriction
Increased Catecholamines
Tachycardia
Impaired Cardiovascular Variability
Endothelial Dysfunction
Vascular Oxidative Stress
Inflammation
Increased Coagulation
Metabolic Dysregulation
Leptin Resistance
Obesity
Insulin Resistance
Risk of Cardiovascular Disease
Hypertension
Congestive Heart Failure
Systolic Dysfunction
Diastolic Dysfunction
Cardiac Arrhythmia
Bradycardia
A-V Block
Atrial Fibrillation
Cardiac Ischemia
Coronary Artery Disease
Myocardial Infarction
Nocturnal ST-Segment Depression
Nocturnal Angina
Cerebrovascular Disease
Shamsuzzaman, JAMA 2003;290:1906-1914.
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- Sleep apnea is characterized by repetitive episodes of apnea occurring during sleep.
- An apnea is defined as a cessation of inspiratory airflow lasting 10 seconds or more
- Hypopnea refers to a reduction in inspiratory airflow (by at least 30%) lasting 10 seconds or more with an associated drop in oxygen saturation equal or ≥ 3% or arousal from sleep.

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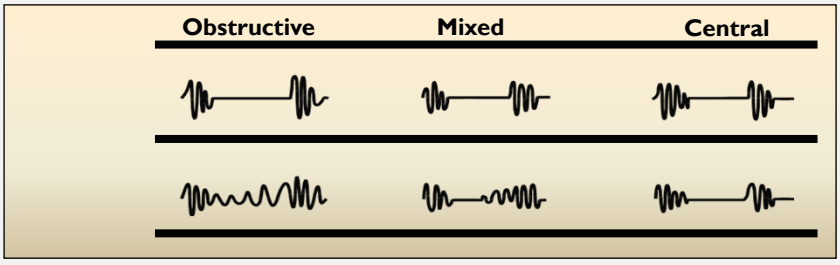
- The mechanism for apneas or hypopneas can be either obstructive, or central, in which both airflow and inspiratory efforts are absent. The term sleep-disordered breathing (SDB) encompasses OSA, central (CSA), and mixed apneas.



- Severity is also determined using the AHI, with 5 to 14 considered mild, 15 to 29 moderate, and ≥ 30 severe disease.
- Treatment is recommended for everyone with AHI >15. Or > 5 with signs/symptoms of sleep apnea or associated medical conditions (including hypersomnia, hypertension, HF, coronary artery disease, arrhythmias, and other forms of CVD).

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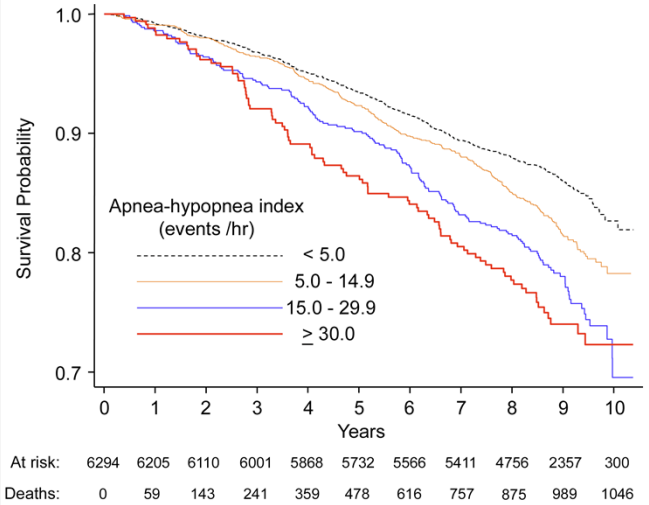
OSA & MORTALITY



Punjabi et al, *PLoS Medicine*. 2009.

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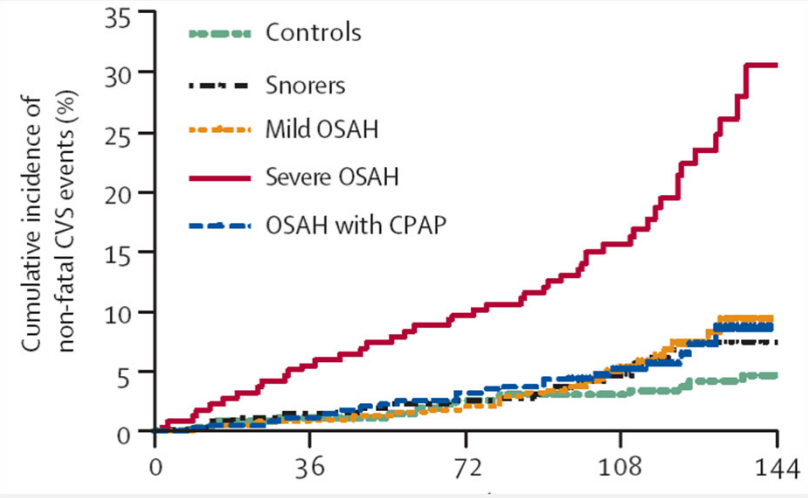
Non-fatal Cardiovascular Events



Marin, *Lancet* 2005; 365: 1046-53

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CARDIOVASCULAR CONDITIONS ASSOCIATED WITH OSA

- **Resistant Hypertension**
- **Heart Failure**
- **Atrial Fibrillation**
- **Other Arrhythmias**
- **Coronary Artery Disease, Cerebrovascular Disease, or Patients Without Established CVD Who Are at High Risk for Future Adverse Cardiovascular Events**
- **Perioperative Risk**
- **Pulmonary Hypertension**

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RESISTANT HYPERTENSION

- OSA, the relationship with hypertension is the best established. Multiple observational studies have demonstrated this association
- Resistant hypertension, commonly defined as inability to adequately control blood pressure despite use of 3 antihypertensive agents including a diuretic or adequate blood pressure control requiring ≥4 agents. For example, 1 study found the prevalence of OSA to be 71% in patients with resistant hypertension versus 38% in those with essential hypertension.
- ***A recent meta-analysis of 5 randomized trials enrolling 457 total patients found a significant reduction in 24-hour ambulatory blood pressure (4.78 mm Hg [95% CI, 1.61–7.95] systolic and 2.95 mm Hg [95% CI, 0.53–5.37] diastolic).***

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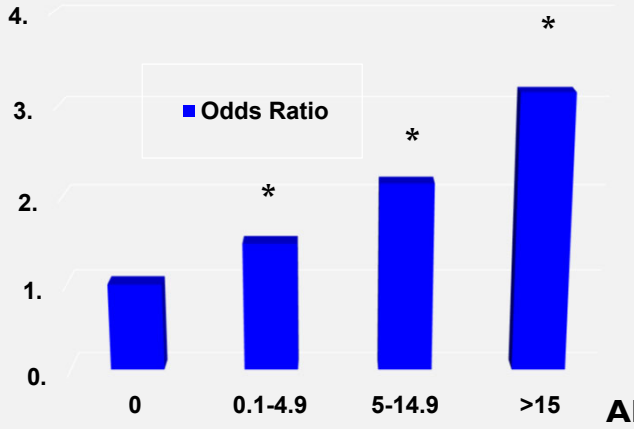
Hypertension and OSA by AHI



*OR adjusted for age, sex, ethnicity, BMI, neck & waist circumference, smoking and alcohol use

WISCONSIN SLEEP COHORT STUDY: ADJUSTED ODDS RATIOS FOR HYPERTENSION AT
4-YEAR FOLLOW-UP PARTICIPANTS WHO WERE NORMOTENSIVE AT BASELINE

Peppard PE et al. *N Eng J Med* 2000 May 11; 342(19): 1378-84

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OSA AND HYPERTENSION

TABLE 2. ADJUSTED ODDS RATIOS* OF INCIDENT HYPERTENSION AT FOLLOW-UP IN RELATION TO BASELINE APNEA-HYPOPNEA INDEX AMONG 2,470 SLEEP HEART HEALTH STUDY SUBJECTS WITHOUT HYPERTENSION AT BASELINE

Baseline AHI	n	Model 1†	Model 2‡	Model 3§
0–4.9	1,511	—	—	—
5–14.9	629	1.13 (0.90–1.43)	0.92 (0.72–1.17)	0.94 (0.73–1.22)
15–29.9	234	1.54 (1.12–2.11)	1.12 (0.80–1.56)	1.09 (0.77–1.54)
≥30	97	2.19 (1.39–3.44)	1.51 (0.93–2.47)	1.50 (0.91–2.46)

Definition of abbreviations: AHI = apnea-hypopnea index; BMI = body mass index.

* Estimated by generalized estimating equation models with each subject contributing one or two follow-up intervals.

Values are odds ratio (95% confidence interval) or n.

† Adjusted for age, sex, race, and time since baseline.

‡ Adjusted for factors in model 1 plus BMI.

§ Adjusted for factors in model 2 plus waist/hip ratio and neck girth.

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HEART FAILURE

Sleep apnea is highly prevalent in patients with HF

HSAT devices have not been validated to diagnose CSA, current guidelines recommend polysomnography

Sleep apnea is prevalent in patients with asymptomatic left ventricular dysfunction as well as those with clinically overt HF,

Coexisting sleep apnea has been associated with increased risk of adverse outcomes, including mortality, in patients with HF, increased risk of arrhythmias, sudden cardiac death, and an elevated risk of coronary events.

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BENEFITS OF PAP THERAPY IN HF

- Reductions in sympathetic nervous system signaling have been observed in patients with both OSA and CSA treated with CPAP.
- PAP also reduces the pro-arrhythmic state associated with OSA, having been shown to decrease ventricular ectopy as well as the risk of recurrent AF.
- Furthermore, CPAP results in favorable hemodynamic effects, including improved left ventricular systolic and diastolic function in OSA patients with high wedge pressures.
- Post hoc analysis suggested a reduction in mortality among patients for whom CPAP therapy resulted in a significant improvement in AHI (to <15 events/h).

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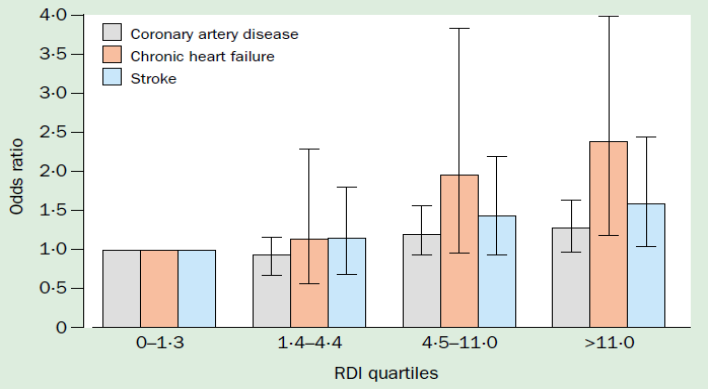
SLEEP HEART HEALTH STUDY: CROSS-SECTIONAL ANALYSIS



Adjusted Relative Odds of Prevalent Coronary Heart Disease, Heart Failure, or Stroke, by Quartile of SDB

Shahar E et al. Am J Respir Crit Care Med 2001

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OSA AND SUDDEN CARDIAC DEATH

10,701 consecutive adults undergoing their first diagnostic polysomnogram between July 1987 and July 2003.

SCD was best predicted by:

- age >60 years (HR: 5.53)
- apnea-hypopnea index >20 (HR: 1.60)
- mean nocturnal O_2sat <93% (HR: 2.93)
- lowest nocturnal O_2sat <78% (HR: 2.60; all $p < 0.0001$).

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OSA AND SUDDEN CARDIAC DEATH

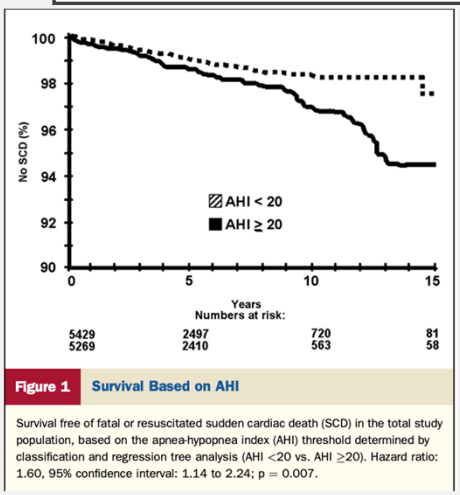

Figure 1 Survival Based on AHI

Survival free of fatal or resuscitated sudden cardiac death (SCD) in the total study population, based on the apnea-hypopnea index (AHI) threshold determined by classification and regression tree analysis (AHI <20 vs. AHI ≥20). Hazard ratio: 1.60, 95% confidence interval: 1.14 to 2.24; p = 0.007.

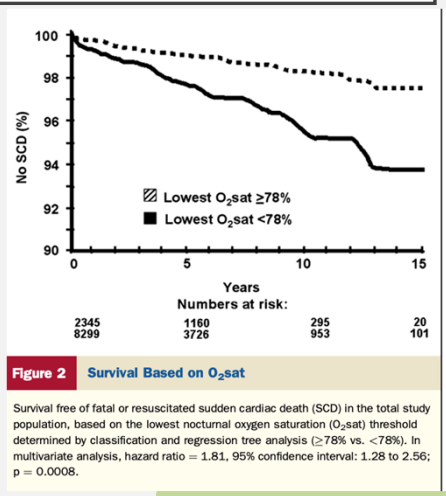

Figure 2 Survival Based on O_2sat

Survival free of fatal or resuscitated sudden cardiac death (SCD) in the total study population, based on the lowest nocturnal oxygen saturation (O_2sat) threshold determined by classification and regression tree analysis (≥78% vs. <78%). In multivariate analysis, hazard ratio = 1.81, 95% confidence interval: 1.28 to 2.56; p = 0.0008.

Survival decreases as severity of obstructive sleep apnea increases.

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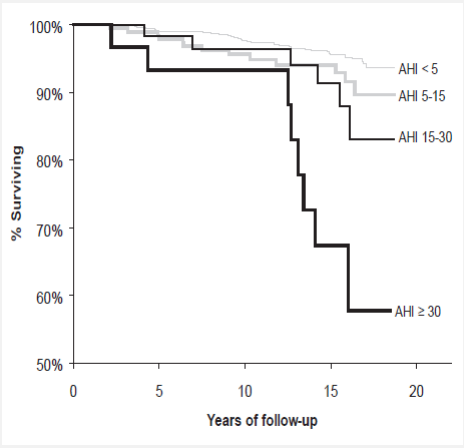
OSA AND SUDDEN CARDIAC DEATH



Survival decreases as severity of obstructive sleep apnea increases.

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ATRIAL FIBRILATION

- Similar to OSA, AF is common in the general population, with a prevalence of 1% to 2%.
- **In patients with OSA, the prevalence of AF is ≈5%, while notably the prevalence of OSA in patients diagnosed with AF has been reported as high as 32% to 39%.**
- Independent association between the 2 disorders exists even after controlling for confounding conditions such as systemic hypertension, obesity, and HF.
- Alterations in both sympathetic and parasympathetic system regulation have been observed in association with OSA.

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ATRIAL FIBRILATION

- But incomplete protection against apnea-associated AF induction suggests that additional mechanisms beyond neurohormonal activation are relevant.
- Over time, OSA promotes structural remodeling of both the ventricles and atria, providing an additional pro-arrhythmic mechanism. Exaggerated swings in intrathoracic pressure, as are seen during apneic events, have been shown to cause acute atrial dilation as well as increased frequency of premature beats
- Significant slowing of atrial conduction also occurs during hypercapnia, even in the absence of hypoxia. The susceptibility to AF among patients with OSA likely reflects the combined effects of these various mechanisms

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ATRIAL FIBRILATION

- Electrophysiologic studies of dilated left atria following repetitive apneic events have revealed slowed atrial conduction, reduced electrogram amplitudes, and complex fractionated atrial electrograms providing mechanistic support for atrial remodeling in AF related to OSA

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ATRIAL FIBRILLATION

- Suggest referral and diagnostic testing in patients with suspected sleep apnea following comprehensive sleep assessment based on nocturnal symptoms and/or risk factors when catheter ablation is planned.
- This recommendation is consistent with the 2017 Heart Rhythm Society
  - Meta-analisis 4572 patients AF after successful catheter ablation were enrolled. Compared to patients without OSA, the pooled OR of recurrent AF in patients with OSA was 1.70 (95% CI)
  - *Among OSA patients with AF after successful catheter ablation, the use of CPAP was significantly associated with decreased risk of recurrent AF with pooled OR of 0.28*

Effect of obstructive sleep apnea and its treatment of atrial fibrillation recurrence after radiofrequency catheter ablation: A meta-analysis
Evid Based Med. 2018 Aug;11(3):145-151.

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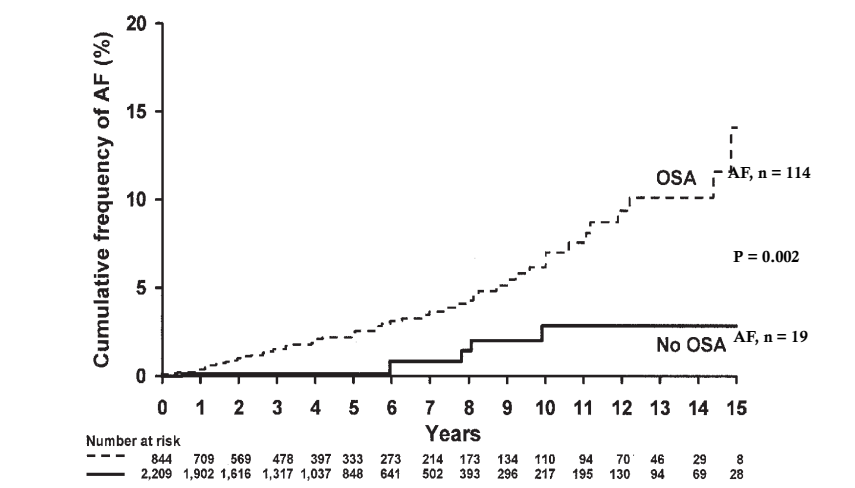
OSA AND ATRIAL FIB



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CORONARY ARTERY DISEASE & CEREBROVASCULAR DISEASE.

Among the pathophysiologic mechanisms linking OSA with CAD and cerebrovascular disease include:

- Increased sympathetic nervous system activity,
- Oxidative stress,
- Predilection to poorly controlled and/or resistant hypertension.
- Endothelial dysfunction
- Promotion of a procoagulable state
- Metabolic dysregulation characterized by insulin resistance

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Obstructive Sleep Apnea as a Risk Factor for Stroke and Death

Table 3. Trend Analysis for the Relationship between Increased Severity of the Obstructive Sleep Apnea Syndrome and the Composite Outcome of Stroke or Death from Any Cause (N=1022).*

Severity of Syndrome	Stroke or Death		Mean Follow-up Period	Hazard Ratio (95% CI)
	No. of Events	No. of Patients		
			yr	
AHI ≤3 (reference score)	13	271	3.08	1.00
AHI 4–12	21	258	3.06	1.75 (0.88–3.49)
AHI 13–36	20	243	3.09	1.74 (0.87–3.51)
AHI >36	34	250	2.78	3.30 (1.74–6.26)

* P=0.005 by the chi-square test for linear trend. AHI denotes apnea–hypopnea index, and CI confidence interval.

Obstructive sleep apnea as a risk factor for stroke and death. Yaggi HK, Concato J, Kernan WN, Lichtman JH, Brass LM, Mohsenin V N Engl J Med. 2005;353(19):

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Treatment of OSA with CPAP has been shown to mitigate these processes

Meta-analysis data revealed that in the 4 RCTs achieving median adherence >4 hours/night, PAP therapy was associated with a significantly lower risk of adverse cardiovascular events (relative risk 0.58; 95% CI, 0.34–0.99)

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PULMONARY HYPERTENSION

- OSA is strongly associated with pulmonary hypertension (PH) and may play a causative role in its pathophysiology. Whereas ≈10% to 20% of patients with moderate-to-severe OSA have coexisting PH.
- The prevalence of OSA in patients with PH diagnosed by right heart catheterization has been estimated to be 70% to 80%.
- Pulmonary hypertension resulting solely from OSA is generally mild;
- Recommended overnight oximetry testing in patients with PH
- Patients with suspected sleep apnea should be referred for sleep consultation followed by formal polysomnography.

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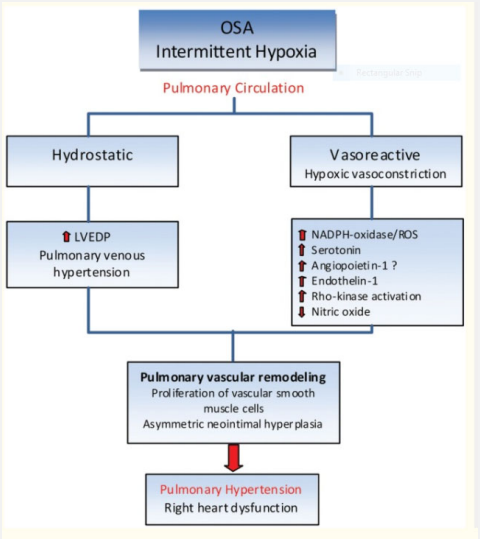

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OTHER ARRHYTHMIAS

- 22% prevalence of prolonged pauses and bradycardia in patients with moderate-to-severe OSA who received long-term monitoring with an implanted loop recorder.
- Significantly higher prevalence of nonsustained ventricular tachycardia (5.3% versus 1.2%, *P*=0.004) among patients with severe OSA compared with controls.
- Similarly, severe OSA was associated with a significantly higher overall risk of complex ventricular ectopy

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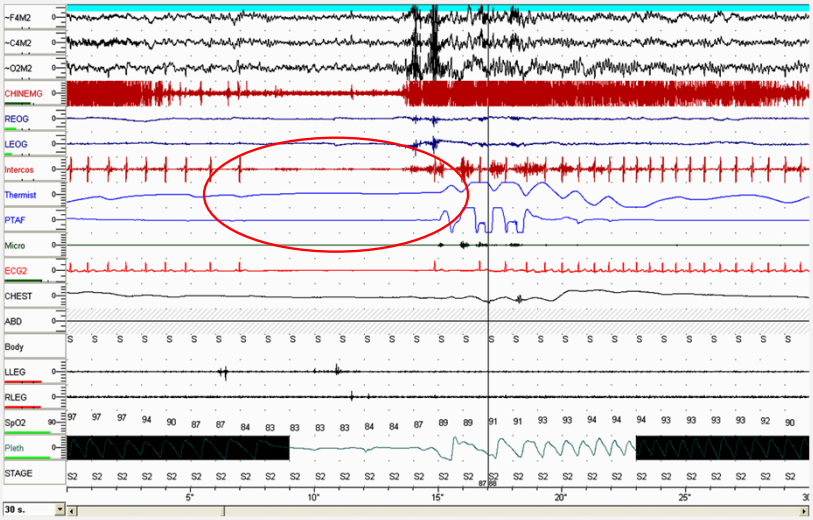
CONSEQUENCES: ARRHYTHMIAS



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DIAGNOSING SLEEP APNEA

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OBSTRUCTIVE SLEEP APNEA RISK FACTORS

- Obesity
  - Increasing age
  - Male gender
  - Craniofacial anatomic abnormalities
- Alcohol or sedative use
- Smoking
- CV disease
- Family history
- Conditions decreasing upper airway tone

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CENTRAL SLEEP APNEA

- Less common than obstructive sleep apnea
- Central respiratory control abnormality
- Heterogeneous conditions
  - Congestive Heart Failure
  - Normal individuals at high altitude
  - Primary Central Sleep Apnea
  - Structural brain or chest wall disease
  - Chronic narcotic use

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DIAGNOSIS: HISTORY

- Snoring (most often loud, habitual)
- Apneas, nocturnal gasping or choking
  - Ask bed partner (witnessed apneas, but can be unreliable)
- Assess risk factors & common co-morbidities
- Daytime symptoms:
  - sleepiness, poor concentration
- Sleep symptoms:
  - insomnia, sleep disruption
  - Night sweats, nocturia

Sleep Apnea: Is Your Patient at Risk? NIH Publication, No 95-3803.

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DIAGNOSIS: OSA EXAM

- **Vitals:** Blood Pressure, O2 saturation
- **Obesity:** BMI >35
- **Neck** circumference
  - >17" men, >16" women
- **Oral pharynx:** upper airway crowding
  - Lateral wall narrowing
  - Large tongue, high based
  - Large uvula and tonsils
  - High arched palate
  - Modified Mallampati

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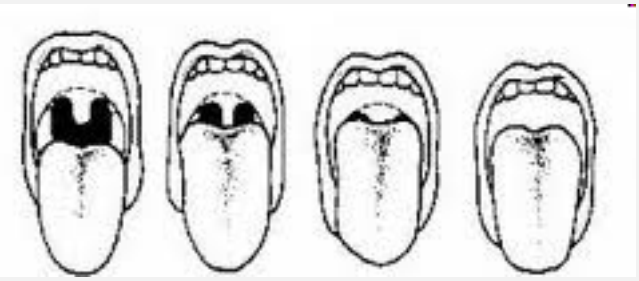

Class 1: Full visibility of tonsils, uvula and soft palate

Class 2: Visibility of hard and soft palate, upper portion of tonsils and uvula

Class 3: Soft and hard palate and base of the uvula are visible

Class 4: Only hard palate visible

UPPER AIRWAY EXAM

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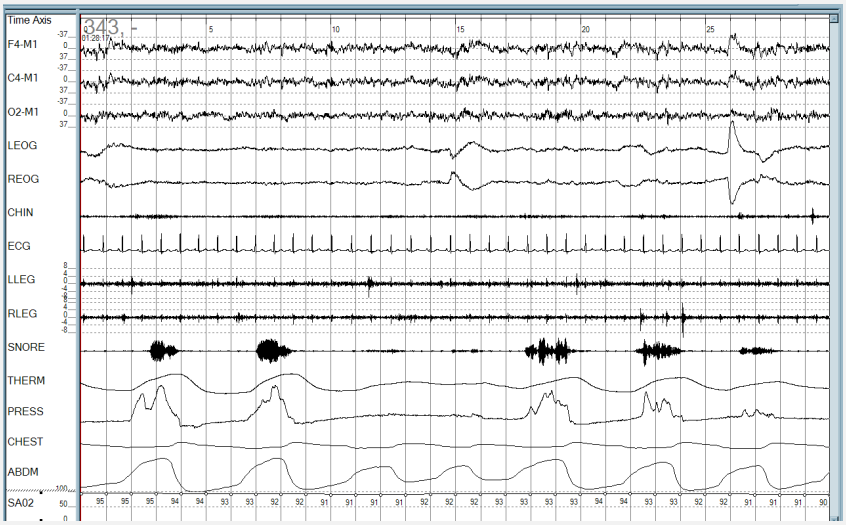
POLYSOMNOGRAM (PSG)



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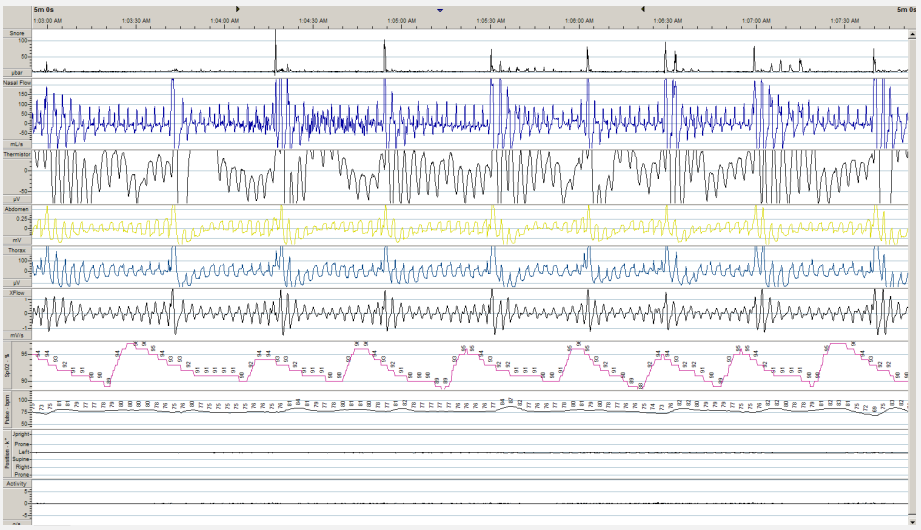
HOME SLEEP APNEA TEST (HSAT) TRACING



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DIAGNOSTIC CONCLUSIONS

- **Signs and symptoms**
  - Excessive daytime sleepiness
  - Hypertension and other cardiovascular sequelae
- **Sleep study results**
  - Apnea / hypopnea frequency
  - Sleep fragmentation (PSG)
  - Oxyhemoglobin desaturation

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TREATMENT OBJECTIVES

- Reduce morbidity and mortality
  - Reduce sleepiness
  - Decrease cardiovascular, metabolic and other systemic consequences
- Improve quality of life

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THERAPEUTIC APPROACH

- Risk counseling
  - Motor vehicle crashes
  - Job-related hazards (Pilots, Truck drivers , heavy machinery)
  - Judgment impairment
- Apnea and co-morbidity treatment
  - Behavioral
  - Medical
  - Surgical

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BEHAVIORAL INTERVENTIONS

- Encourage patients to:
  - Lose weight (if overweight)
  - Avoid alcohol and sedative-hypnotics
  - Avoid sleep deprivation
  - Avoid supine sleep position in susceptible patients
  - Stop smoking
  - Maintain clear nasal passages

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WEIGHT LOSS

- Should be strongly encouraged for all obese patients
- Can be curative but has low success rate
- Other treatment is required
  - Until optimal weight loss is achieved
  - And sleep apnea is proven to be resolved

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MEDICAL INTERVENTIONS

- Positive airway pressure
  - Continuous positive airway pressure (CPAP)
  - Fixed CPAP
  - Auto-positive airway pressure or APAP
  - Bi-level positive airway pressure
  - Fixed bi-level positive airway pressure (BPAP)
  - Variable positive airway pressure (VPAP)
- Oral appliances
- Hypoglossal nerve stimulation
- Other (limited role): medications, oxygen

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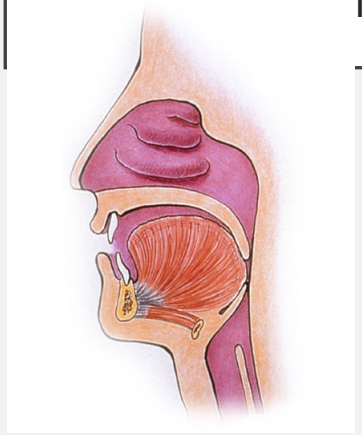
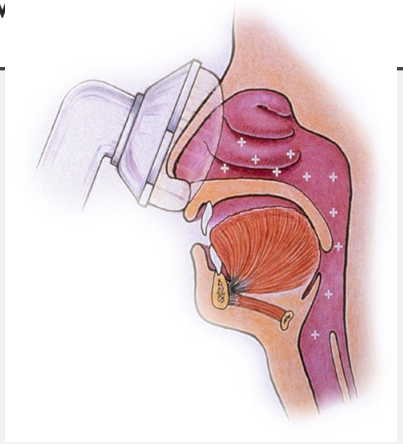
IRV





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POSITIVE AIRWAY PRESSURE

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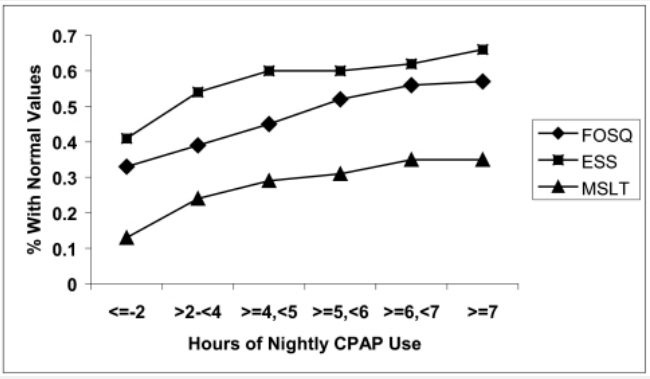
BENEFITS OF CPAP: SLEEPINESS & FUNCTIONAL OUTCOMES



FOSQ = Functional Outcomes of Sleep Questionnaire
ESS = Epworth Sleepiness Scale
MSLT = Multiple Sleep Latency Test

Weaver TE, et al. Sleep. 2007;30(6):715.

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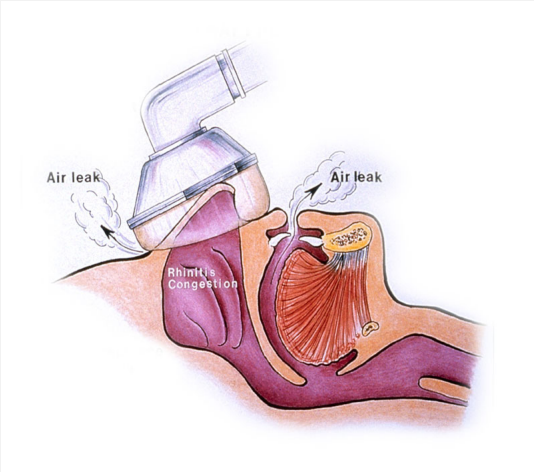
POSITIVE AIRWAY PRESSURE: PROBLEMS

Mask Discomfort



Patient Acceptance
Claustrophobia
Aerophagia
Chest Discomfort

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CPAP COMPLIANCE: PREDICTORS

- Predict Good Compliance
  - Increased AHI
  - Increased daytime sleepiness
  - Perception of benefit
- Predict Poor Compliance
  - Lack of EDS
  - Lack of perceived benefit
  - Nasal obstruction
  - Side effects
  - Claustrophobia
  - PTSD

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STRATEGIES TO IMPROVE COMPLIANCE

- Patient Education
- Frequent and early follow-up
- Desensitization
- Machine-patient interfaces
  - Masks
  - Nasal pillows
  - Chin straps
- Humidifiers
- Ramp

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CPAP MASKS





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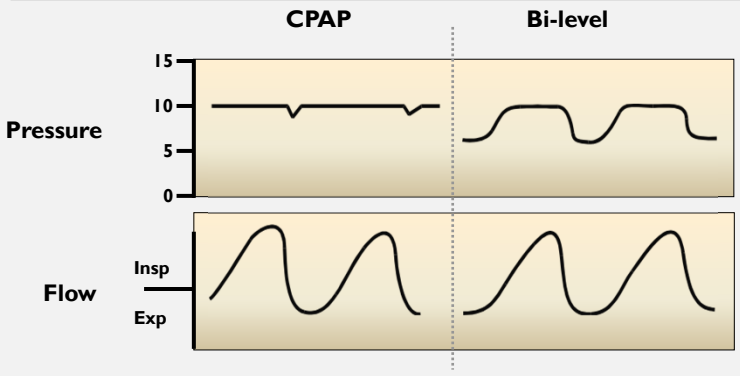
BI-LEVEL POSITIVE AIRWAY PRESSURE

Positive Pressure Therapy



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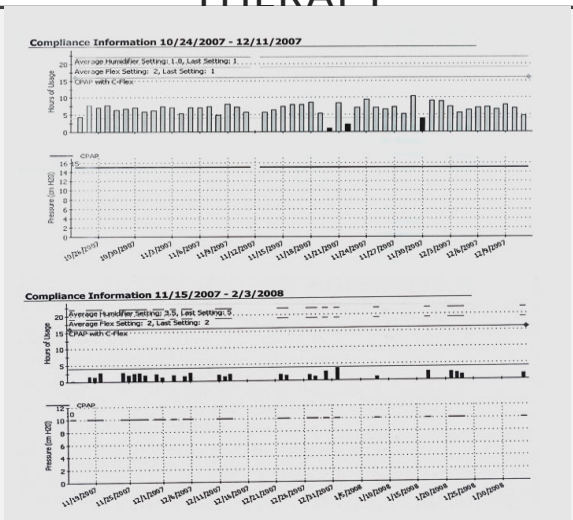
MONITORING ADHERENCE TO PAP THERAPY



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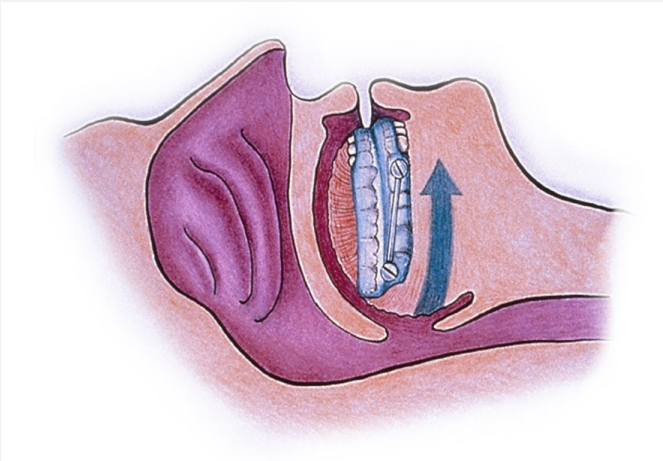
ORAL APPLIANCE
GENERAL PRINCIPLE



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ORAL APPLIANCES
TREATMENT PREREQUISITES

- OSA status must be clarified
  - Oral appliances indicated for primary snorers and mild-moderate OSA
- Qualified dental expertise crucial
  - Assess candidacy for oral appliance
  - Adequate healthy teeth
  - No important TMJ disorder
  - Adequate jaw range of motion
  - Adequate manual dexterity and motivation to position device
  - Fit and monitor oral appliance

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ORAL APPLIANCES
TREATMENT CONSIDERATIONS

- Adherence: varies between studies
  - 77% at 1 year
- Side effects: frequency varies between studies
  - Pain: TMJ; myofascial; tooth; gum
  - Dry-mouth, salivation, morning occlusal changes
- Combined dental and sleep follow-up important
  - Sleep study after optimal fit to look for residual OSA
  - Periodic dental visits to assess device, oral health

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MEDICAL THERAPY: SUPPLEMENTAL OXYGEN

- Not a primary treatment for obstructive sleep apnea- not standard of care and not routinely recommended
- Does not improve daytime sleepiness
- May prolong apneas
- Reduces oxygen desaturation during apneas
- Reduces arrhythmias

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SURGICAL TREATMENT OF OSA

- OSA status must be clarified pre-operatively
- Multidisciplinary patient assessment important
  - Identify anatomic surgical targets
  - Assess for significant co-morbidities
  - Determine patient's desire for surgery
  - Counsel on options, benefits, risks, and alternatives
- Potential candidates
  - Primary therapy: mild OSA + severe, but surgically remediable, obstructing anatomy
  - Secondary therapy: unsuccessful response to PAP and/or OA
  - Adjunctive therapy: increase tolerance of PAP or OA
- Post-operative sleep specialist follow-up recommended

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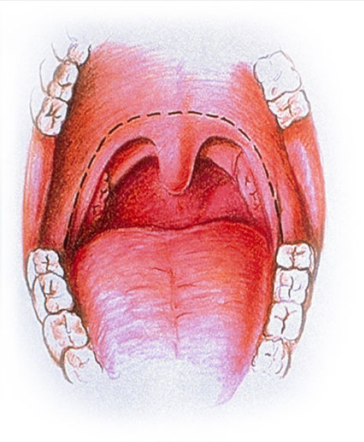
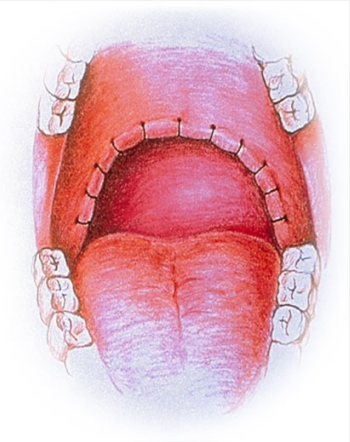
UVULOPALATOPHARYNGOPLASTY (UPPP)





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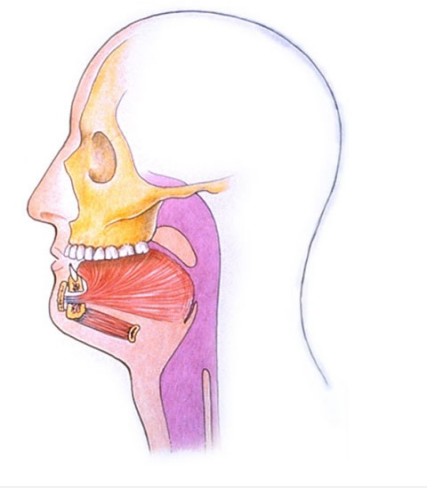
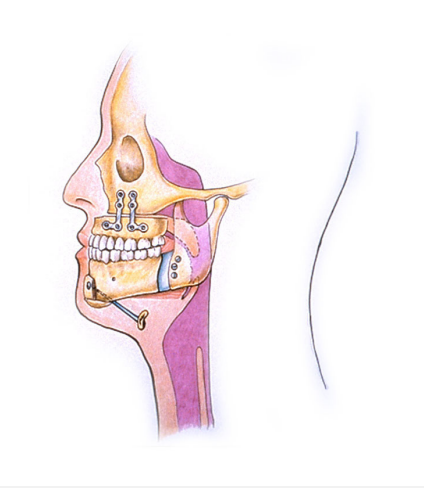
MAXILLOFACIAL SURGERIES



Genioglossal advancement



Maxillomandibular advancement

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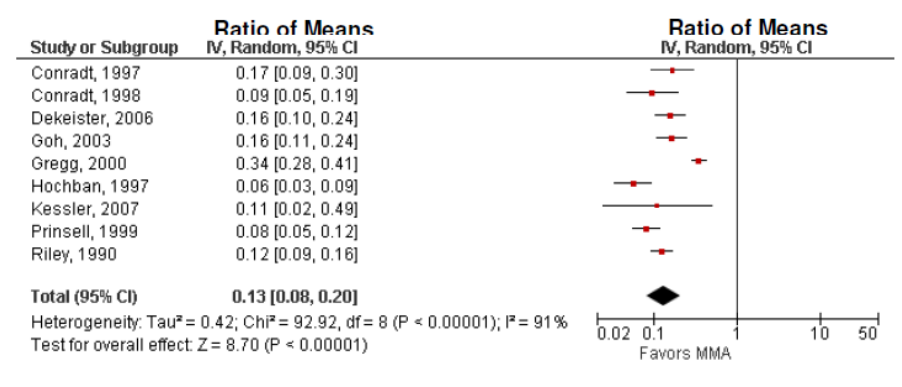
META-ANALYSIS OF MAXILLOMANDIBULAR ADVANCEMENT (MMA)



Pooled reduction in AHI with MMA: 87%

Caples SM, et al. Sleep 2010;33

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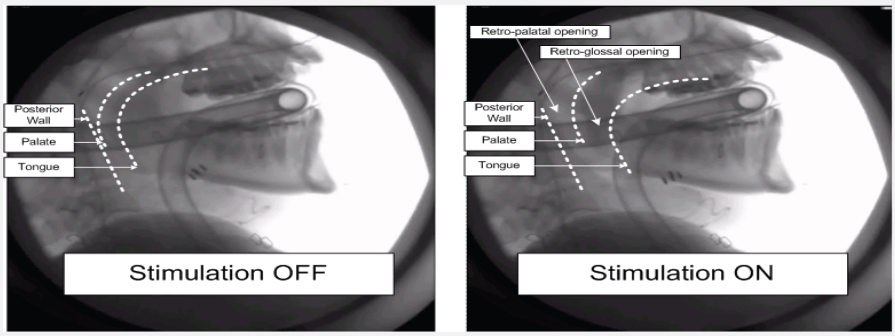
HYPOGLOSSAL NERVE STIMULATOR

- Neurostimulator delivers electrical pulses to the hypoglossal nerve (stimulation lead)
- The pulses are synchronized with ventilation (sensing lead)
- 3 incisions are required



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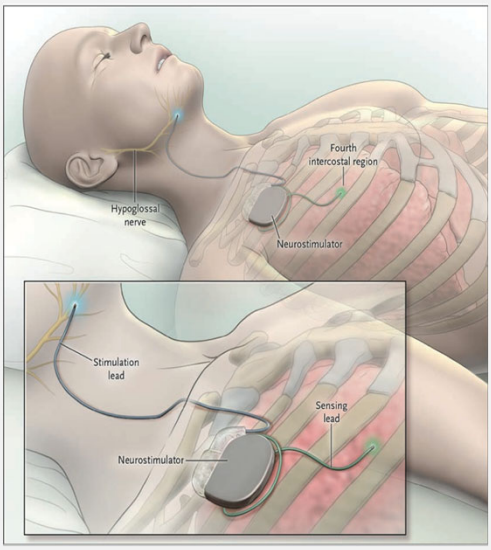
Newer Therapies

Upper airway pacing

Consists of three components: an implantable generator, a breathing sensor lead, and a stimulation lead

Controlled by a handheld sleep remote.

Turn the therapy on at night before bed, and off in the morning when you wake up.



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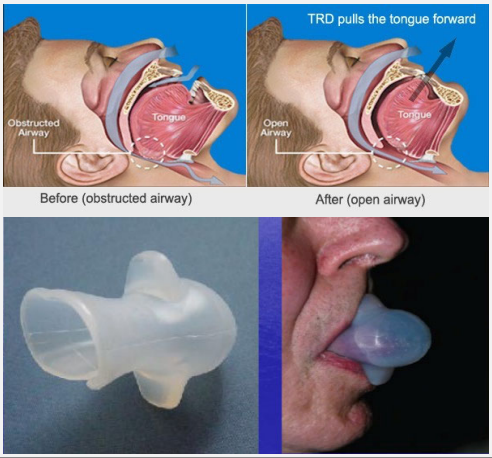
TONGUE REPOSITIONING DEVICE



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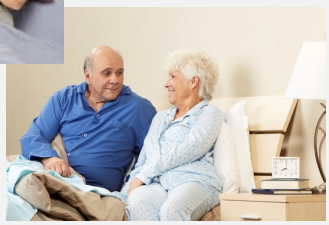
POSITIONAL THERAPIES





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OBSTRUCTIVE SLEEP APNEA

VS

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QUESTIONS?

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