



RESONEA

Obstructive Sleep Apnea: Effective Intervention & Care
Provider Training Modules

Jonathan Freudman, MD

Provider Support: OSA Training Modules

- RESONEA will support providers with a training program in sleep health and OSA management.

Obstructive Sleep Apnea: Effective Intervention & Care

- Slide presentation Module 1 – Epidemiology & Physiology of OSA
 - Slide presentation Module 2 – Confirming the Clinical Diagnosis of OSA
 - Slide presentation Module 3 – Therapy for OSA
 - Slide presentation Module 4 – Effective Intervention to Influence Therapy Adherence
 - Case Studies
- RESONEA will support providers with patient education tools to enhance consumer knowledge about OSA and to help set expectations for successful treatment.
 - All provider training modules, educational materials, and resources (STOP/BANG and Epworth Sleepiness Scale questionnaires, as well as key clinical publications) will be available via the Provider Portal on the RESONEA website: www.resonea.com.



Provider Support: Patient Intervention Tools & Materials

- RESONEA will also support providers with educational materials to dispense to patients.
- Materials will be determined by further discussions with providers, however the following topics are anticipated for coverage in materials:
 - What is Sleep Breathing Health and why is it important?
 - What are the long-term health risks of untreated sleep apnea?
 - Symptoms & risk factors
 - How do I know if I am at risk? How do I know if my risk is changing?
 - If I have sleep apnea, what are my treatment options?
 - If I don't have sleep apnea, but have other risk factors, how can I lower my risk of getting sleep apnea?
 - How can I improve the restfulness of my sleep and my daytime alertness?
 - When should I retest and how do I monitor symptoms and risks of sleep breathing health?
 - How do I improve my long-term health risk benefits once treated?
- All patient educational materials will be available via a Patient Portal on the RESONEA website:
www.resonea.com



Obstructive Sleep Apnea: Effective Intervention & Care - Overview

- **Module I – Pathophysiology & Epidemiology of OSA**
 - Definition
 - Symptoms
 - Pathophysiology
 - Epidemiology
 - Impact on other conditions
- **Module II – Confirming The Clinical Diagnosis of OSA**
 - Index of suspicion
 - Screening
 - Diagnosis
- **Module III – Therapy for OSA**
 - Therapeutic goals
 - Treatment options
 - Follow-up
- **Module IV – Effective Intervention To Influence Therapy Adherence**
 - What patients need to know to succeed
 - Best practices
 - Monitoring
- **Module V – Case Studies**





RESONEA

Module I: Pathophysiology & Epidemiology of OSA

Module I: Case #1

A 58 year old woman has been followed for 8 years at the primary care clinic of a prestigious US medical school. She had poorly controlled hypertension despite being treated with multiple medications in a variety of combinations, including triple drug therapy. Workup for secondary forms of hypertension included normal renal arteriogram and endocrine evaluations that ruled out pheochromocytoma and hyperaldosteronism. She was thought to be compliant with her medications. Her BMI was 29.

- After about 6 years of poorly controlled blood pressure, she was asked her if she snored. She said her husband frequently complained about her loud snoring.
- She was referred to sleep medicine and had a sleep study, which suggested severe obstructive sleep apnea (OSA).
- She was placed on continuous positive airway pressure (CPAP) therapy.
- Over the next several months:
 - ✓ **Her blood pressure dropped and was controlled with 2 medications.**
 - ✓ **She commented that she had more energy and felt better than she had in a long time.**
 - ✓ **Several symptoms that she attributed to menopause – mood changes, headaches, and lack of energy – had improved since treatment with CPAP.**



Module I: Case #1 – Comments & Take Away Points

This case is not unusual.

- OSA is not limited to overweight men.
- OSA can be insidious in its onset and progression.
- OSA frequently defies diagnosis unless the clinician is looking for it.
- OSA is characterized by recurrent obstruction of the pharyngeal airway during sleep, with resultant hypoxia, sleep fragmentation, and other adverse effects.
- The four most common and important symptoms of OSA are:
 - 1) snoring,
 - 2) choking or gasping for breath during sleep,
 - 3) interruptions in breathing during sleep, and
 - 4) excessive daytime sleepiness.
- However, not all patients exhibit all of these symptoms.
- OSA can contribute to or amplify a variety of other symptoms commonly seen in medical practice.
- Untreated OSA diminishes the patient's quality of life and increases the risk for **hypertension, cardiovascular disease, type II diabetes, depression** and other common and serious medical problems.
- OSA has several effective treatments, including CPAP, weight reduction and oral appliances.



Module I: Case #2

A 54 year old man decided to go on the internet to read about obstructive sleep apnea, because his wife was complaining more about his snoring. She had commented that lately he seemed to choke at times while sleeping.

In the previous 3 years, his weight had increased by twenty-five pounds and he attributed this to a bad knee that forced him to stop jogging. Also his law practice had become busier and this meant eating fast food at his desk for lunch. His work days had become longer and he stopped taking his walks after dinner, because it was too late or he was too tired.

At a medical check up a few months earlier, his blood pressure was “borderline” (142/90) for the first time. When asked how he was feeling, he admitted that lately he felt somewhat stressed, irritable, and had less energy. He attributed this to his work schedule and “getting lazy and fat”. His BMI was 32.

“I’ve always been in good shape and this is embarrassing.”

- His physician suggested more exercise and provided a few dietary tips for weight reduction.
- He was not asked about his snoring and the patient did not bring this up.
- When he went on the internet to read about OSA, he was shocked and dismayed to see this image of CPAP...

“That’s what they hooked my dad up to in the ICU ... before he died!”

“No way would I ever use that to treat my snoring.”



Module I: Case #2 – Comments & Take Away Points

This is not an unusual case. It illustrates common barriers to the efficient detection and eventual treatment of OSA.

- Patients do not always volunteer that they snore –
 - If they live alone, they may not know that they snore, or may not know the frequency and volume of snoring.
 - In some cases, if they know they snore, they may be ashamed and see it as a consequence of weight gain.
 - If they are aware of snoring, they may believe it is a normal sign of sound sleep.
- Patients rarely know if their snoring is benign (primary snoring) or potentially dangerous (OSA).
- Physicians seldom ask about snoring or sleep quality, nor do they routinely screen for OSA risk factors.
- Patients have misconceptions about CPAP, believing it to be uncomfortable, embarrassing, and intrusive in their lifestyle.
- Patients also may have negative impressions of sleep testing, especially in-lab polysomnography (PSG).
- Physicians may not realize the importance of their role in helping patients to overcome perceptual hurdles about OSA diagnosis and treatment.



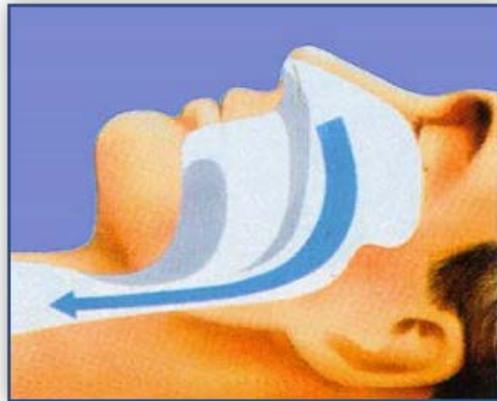
Module I: Intro to Continuous Positive Airway Pressure (CPAP)

Continuous Positive Airway Pressure (CPAP) will be covered in more depth in Module III, however a brief introduction may be useful here.

- CPAP works -- it is safe and its efficacy is well-documented in randomized, controlled trials.
- CPAP is a chronic therapy, rather than a cure.
- Invented by Dr. Colin Sullivan in Australia in 1980.
- CPAP treatment involves the introduction of pressurized air into the airway while the patient sleeps.
 - A small pump provides the pressurized air. (Dr Sullivan initially used a vacuum cleaner.)
 - Tubing connects the pump to a mask, which is fitted to the patient's nose and mouth.
 - Pressurized air passes through the nares into the patient's upper airway.
 - In patients with OSA, the pressurized air acts like a "splint" to "prop" the airway open.
- CPAP is painless, but does require attitude adjustment on the part of the patient, and a willingness to try it. Spousal or family support may also play a key role in determining successful outcomes.
- The quantity of air pressure delivered by the CPAP machine is determined for each patient in the sleep lab during a "titration study".
- Newer "auto-titrating" CPAP machines adjust the pressure automatically and continuously during treatment, which many patients find easier to tolerate than fixed-pressure CPAP.
- **In people with OSA, treatment with CPAP:**
 - ✓ **Lowers blood pressure**
 - ✓ **Lowers Hemoglobin A1c in patients with diabetes**
 - ✓ **Reduces daytime sleepiness**
 - ✓ **Usually reduces or eliminates snoring**



Obstructive Sleep Apnea (OSA) is characterized by recurrent obstruction of the pharyngeal airway during sleep, with resultant hypoxia, sleep fragmentation, and other adverse effects.



Open Airway



Obstructed Airway



Module I: Hallmark Symptoms of OSA

Most common and important symptoms:

- Loud, frequent snoring
- Choking or gasping for breath during sleep
- Interruptions in breathing during sleep
- Excessive daytime sleepiness (difficulty maintaining wakefulness)

Snoring – vibration of the soft palate from air being forced through a partially collapsed airway

- ✓ Most people with OSA snore, however
- ✓ Many people who snore do not have OSA



Module I: Additional Common Symptoms of OSA

Other symptoms and historical features include:

- Awakening with a sensation of choking, gasping, or smothering
- Awakening with a dry mouth or sore throat
- Non-restorative sleep
- Moodiness or irritability
- Cognitive impairment (concentration or memory)
- Decreased libido and impotence
- Nocturia
- Awakening with angina pectoris
- Depression
- Erectile dysfunction
- Symptoms of fibromyalgia
- Gastro-esophageal reflux disease (GERD)
- History of medical conditions: hypertension, obesity, cardiovascular disease, cerebrovascular disease, type 2 diabetes, renal disease, or polycystic ovary disease



Module I: Physical Findings Associated With OSA

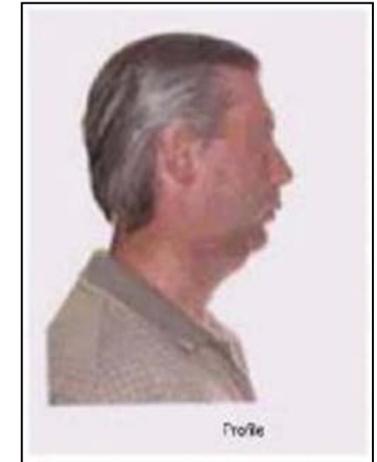
- Obesity: BMI >30, but the higher the BMI, the more likely OSA is present
- Elevated blood pressure
- Large neck circumference: ≥ 17 inches for men, ≥ 16 inches for women
- Narrow or crowded oropharyngeal airway (e.g. large tonsils)



Module I: Pathophysiology of OSA

Why does the upper pharyngeal airway collapse?

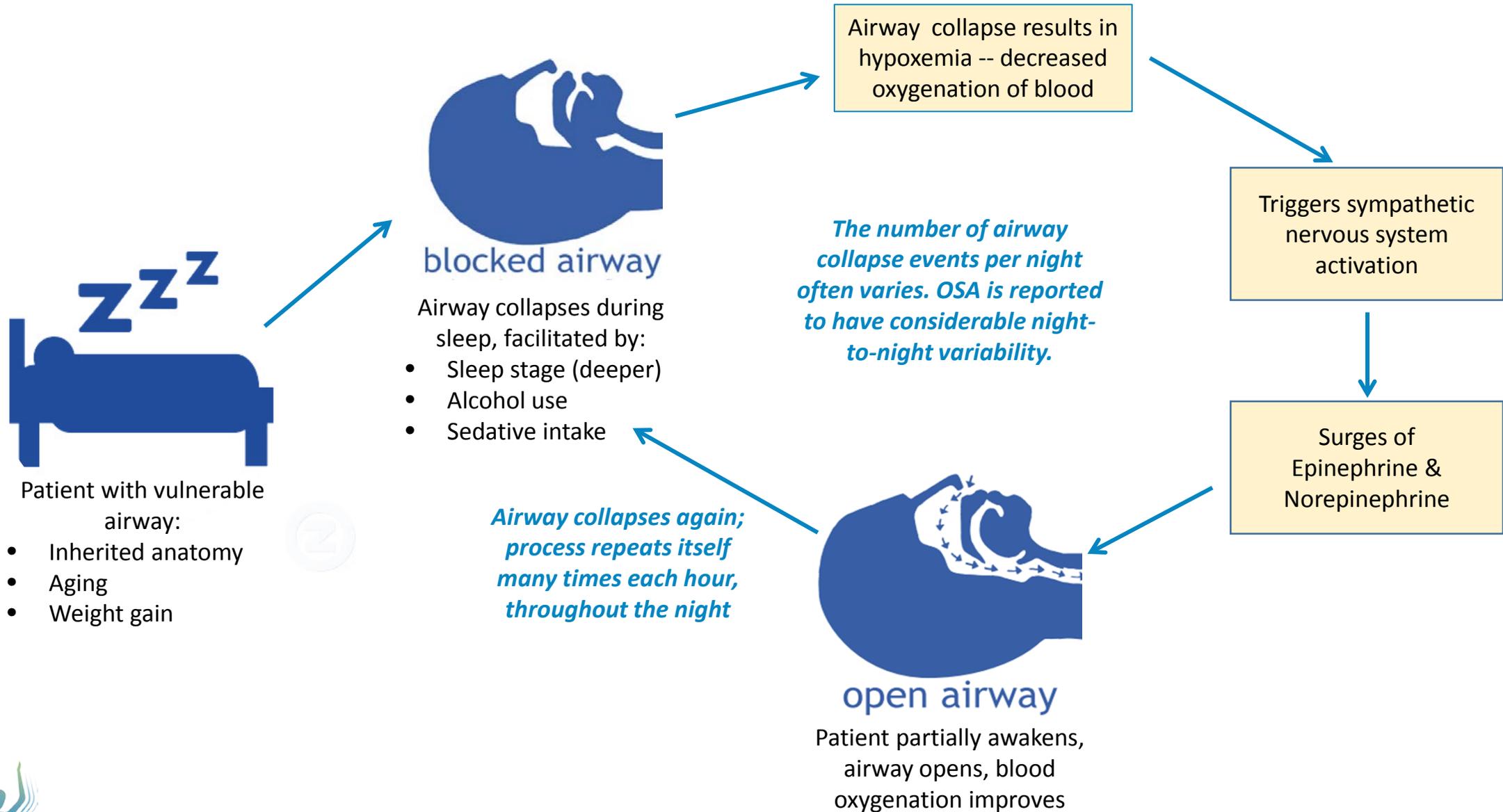
- Changes during sleep create the potential for a vulnerable airway to collapse:
 - Lumen of the airway is narrowed
 - Airway muscle tone is diminished
 - Mechanics of breathing are altered, with less ventilatory effort expended to maintain airway patency
- Patient factors which make the upper airway more vulnerable to collapse during sleep:
 - Anatomic features
 - ✓ Retrognathia – posterior recess of the maxilla or mandible
 - ✓ Hypertrophy of tonsils or adenoids
 - Obesity - increased volume of soft tissue compressing the airway
 - Aging – reduced stiffness of the upper airway
 - ✓ Decreased neural stimulation of supporting muscles



Retrognathia



Module I: Pathophysiology - Sequence



Module I: Pathophysiology - Consequences

Repetitive partial awakening results in fragmented sleep.



Deficit in stage 4/REM sleep.



Sleep is non-restorative and leads to:

- Daytime sleepiness
- Irritability
- Cognitive impairment
- Depression
- Headaches
- Increased likelihood for motor vehicle and other accidents



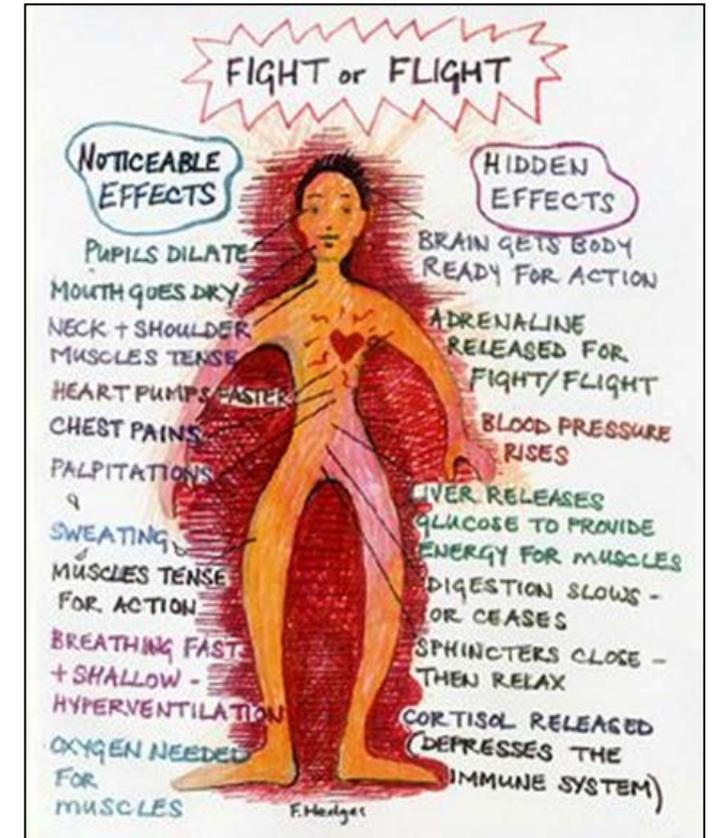
Module I: Pathophysiology - Consequences (Continued)

Repetitive epinephrine/norepinephrine surges have the following effects:

- Elevate the heart rate
- Increase blood pressure
- Increase inflammatory mediators
- Create a hypercoagulable state
- Trigger an increase in cortisol release

Sympathetic nervous system /epinephrine release - components of the “Fight or Flight” response:

- Instinctive survival mechanism in the wild, but
- Maladaptive to humans in modern times



Module I: Pathophysiology Consequences – Elevated Health Risks

Epinephrine excess:

- Elevations in pulse and blood pressure increase the risk for myocardial infarction, heart failure, cardiac arrhythmia and stroke.
- Increases inflammatory mediators - implicated in the development of vascular endothelial dysfunction that may lead to hypertension and atherosclerosis.
- Cortisol increases blood sugar and appetite, making type II diabetes more likely.

Repeated episodes of tissue hypoxia:

- Exacerbate the metabolic syndrome
- Augment insulin resistance
- Increase non-alcoholic fatty liver disease



Module I: Classification of Airway Collapse Events

Apnea

- Cessation, or near cessation, of airflow.
- Scoring an apnea on sleep testing requires documentation of a 90 percent or greater decrease in airflow, compared with preceding signals, for a minimum of 10 seconds.

Hypopnea

- An airflow event that, while insufficient to meet the definition of an apnea, meets the following criteria:
 - Airflow decreases at least 30 percent compared with the pre-event baseline.
 - The diminished airflow event lasts at least 10 seconds.
 - The event is associated with either oxygen de-saturation $\geq 3\%$ from baseline and/or an EEG arousal (or $\geq 4\%$ de-saturation, if arousals not counted).

Over the years, the criteria for a hypopnea have varied.

Medicare now considers 4% oxygen de-saturation the cut-off and most sleep labs report OSA studies using 4%).



Module I: Apnea/Hypopnea Index

AHI (Apnea/Hypopnea Index)

The number of apneas and hypopneas identified during a sleep study, divided by the total sleep time in hours:

5 -15 per hour = mild OSA

15-30 per hour = moderate OSA

>30 per hour = severe OSA

- AHI is a crude measure of OSA disease severity, since not all apneas and hypopneas have the same impact on oxygen desaturation, but they are counted equally in the AHI score.
- AHI is not a good predictor of response to OSA therapy
- Nonetheless, AHI remains the standard measure used by industry, payers and the sleep medicine community.
- When assessing the severity of OSA in a patient, it is important to look beyond the AHI and also consider the degree of OSA-related symptoms (e.g. excessive sleepiness), as well as the patient's co-morbid conditions (e.g. hypertension).



Module I: Epidemiology of OSA

Prevalence depends on the population segment & how OSA is defined:

In North American adults, age 30-70: *

When OSA defined as AHI > 5
(Mild OSA)

- 20-30% Males have OSA
- 10-15% Females have OSA

When OSA defined as AHI ≥ 15
(Moderate - Severe OSA)

- 15% Males have OSA
- 5% Females have OSA

- Men are 2x to 3x more likely to have OSA than women.
- OSA is more prevalent in African American males < 35 years of age, compared with Caucasians of the same age group, independent of body weight.
- OSA is as prevalent in Asians as in Americans, despite the lower rate of obesity.
- The prevalence of OSA in the United States appears to be increasing due to rising rates of obesity.



* Wisconsin Sleep Cohort, Young 2009, Peppard 2013

Module I: Epidemiology of OSA – Major Risk Factors

Major risk factors for developing OSA:

- **Age** – prevalence progressively increases from young adulthood onward and levels off at age 70
- **Men are 2-3 times** more likely than women (reason not known)
- **Obesity** – progressive increase in risk with greater BMI, neck circumference and waist-to-hip ratio
- **Craniofacial and soft tissue abnormalities**, including wide craniofacial base, abnormal maxilla, short mandible, hypertrophy of tonsils or adenoids

Other risk factors for developing OSA:

- Nasal congestion
- Smoking
- Menopause
- Prevalence of OSA is increased in several medical conditions, including
 - pregnancy,
 - end stage renal disease,
 - heart failure,
 - chronic obstructive lung disease,
 - stroke/transient ischemic attack,
 - acromegaly,
 - hypothyroidism, and
 - polycystic ovary syndrome.



Module I: Epidemiology – Risk For Other Conditions

The impact of OSA on other medical conditions has been extensively investigated in many studies*

- People with OSA utilize **significantly more medical resources** and experience more medical disability than those without OSA.
- **Motor vehicle accidents occur 2-3X** more frequently in those with OSA vs. those without OSA.
- The duration and severity of hypoxemia in OSA has been shown to independently adversely affect performance of cognitive tasks
 - There is a high prevalence of OSA in those with **dementia** and OSA may aggravate cognitive dysfunction in dementia **
- Patients with OSA have approximately 2X the incidence of **depression** compared with matched controls without OSA.
- Patients with OSA have an increased prevalence of **type 2 diabetes** and insulin resistance. Several studies have confirmed this association, which is independent of obesity.

The impact of treatment on these risks will be discussed in the Module III.



* Lee et al 2008

** Ancoli-Israel 2008

Module I: Epidemiology – Risk For Cardiovascular Conditions

Patients with untreated OSA, especially if moderate or severe, are at increased risk for cardiovascular morbidities, including:

- **Hypertension** - Population studies document that OSA independently increases the risk for hypertension.
 - A longitudinal prospective population-based study compared patients with and without OSA and controlled for known confounders (age, body habitus, baseline hypertension, alcohol and smoking): Adjusted odds ratio for prevalent hypertension was 2.03 for mild OSA and 2.89 for moderate or worse OSA¹
- **Stroke** - Independent association between OSA and stroke documented in several large observational studies:
 - Sleep Heart Study² adjusted odds ratio for stroke = 1.58, compared with patients without OSA.
 - Wisconsin Sleep Cohort³ found adjusted odds ratio 4.33 for patients with AHI >20.
- **Coronary artery disease (CAD)** -
 - Sleep Heart Study subjects with the highest quartile of AHI (AHI > 11), had an adjusted odds ratio for angina or myocardial infarction of 1.27, relative to subjects with no OSA.²
 - In patients with documented CAD, the prevalence of OSA has ranged from 30-57% and several prospective studies have documented an increased rate of adverse events in patients with CAD and OSA.⁴
- **Congestive heart failure (CHF)** - high prevalence of OSA in heart failure patients is well established.
 - Sleep Heart Study subjects with OSA (AHI>11) had odds ratio of 2.38 for congestive heart failure vs. those without OSA.²
- **Arrhythmias** – association between OSA and increased prevalence has been documented in several studies.

“... mounting evidence from clinic-based and epidemiologic population-based studies have established a strong and independent association between OSA and various cardiovascular conditions.”⁴



¹ Peppard 2000 ² Arzt 2005 ³ Shahar 2001 ⁴ Lee 2008

Module I: Pathophysiology & Epidemiology of OSA - Summary

○ Pathophysiology

- Recurrent collapse of airway during sleep, causing hypoxia & fragmented sleep
- Hallmark symptoms: loud, frequent snoring; choking or gasping for breath during sleep; observed interruptions in sleep breathing; excessive daytime sleepiness
- Repeated episodes trigger sympathetic nervous system activation
- Epinephrine & Norepinephrine surges increase blood pressure
- Increased cortisol release can impact weight, immune function, and risk of chronic diseases
- Untreated OSA causes or worsens numerous diseases & increases risk of early death

○ Epidemiology

- Among adults 30 to 70 years of age, 15% of men and 5% of women have moderate-to-severe OSA, need treatment
- Key risk factors are male gender, age ≥ 50 , and obesity
- According to the literature, 80-90% of people with OSA remain undiagnosed & untreated*
- Patients with untreated OSA are at significantly increased risk for cardiovascular morbidities, including hypertension, stroke, CAD and CHF
- Patients with untreated OSA utilize significantly greater healthcare resources, with double the expenditures of people with no OSA or treated OSA



* Finkel 2009 Young 2009 Peppard 2013

Module I: References

- Anconi-Israel S, Palmer B, Cooke J et al; Cognitive Effects of Treating Obstructive Sleep Apnea in Alzheimer's Disease: A Randomized Controlled Study *J Am Geriatr Soc*. 2008 Nov 1; 56(11): 2076–2081.
- Arzt M, Young T, Finn L, Skatrud et al; Association of sleep-disordered breathing and the occurrence of stroke . *Am J Respir Crit Care Med* 2005;172:1447–1451.
- Berry, R; Sleep Medicine Pearls Second Edition *Mosby* 2003
- Drager L, Togeiro S, Polotsky V et al; Obstructive Sleep Apnea: A Cardiometabolic Risk in Obesity and the Metabolic Syndrome *J Am Coll Cardiol*. 2013 August 13; 62(7): 569–576.
- Epstein L, Kristo D, Strollo P et al; Clinical guideline for evaluation and management of obstructive sleep apnea in adults Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine *J Clin Sleep Med* 2009;5(3):263-276
- Finkel KJ, et al; Prevalence of undiagnosed obstructive sleep apnea among adult surgical patients in an academic medical center; *Sleep Medicine* 2009; 10(7): 753-758
- Kine L (Collop N, Finlay G, editors); Clinical presentation and diagnosis of obstructive sleep apnea in adults *UpToDate* September 29, 2016
- Lee W, Nagubadi S, Kryger M, and Mokhlesi; Epidemiology of Obstructive Sleep Apnea: A population-based perspective *Expert Rev Respir Med*. 2008 June 1; 2(3): 349–364.
- Peppard PE, Young T, Barnet JH, et al; Increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol* 2013; 177:1006.
- Peppard PE, Young T, Palta M, Skatrud J; Prospective study of the association between sleep- disordered breathing and hypertension. *N Engl J Med* 2000;342:1378–1384.
- Shahar E, Whitney CW, Redline S, et al; Sleep-disordered breathing and cardiovascular disease: cross-sectional results of the Sleep Heart Health Study . *Am J Respir Crit Care Med* 2001;163:19– 25
- Strohl K. (Collop N, Finlay G, editors); Overview of obstructive sleep apnea in adults *UpToDate* June 24, 2016
- Young T, Palta M, Dempsey J, et al; Burden of sleep apnea: rationale, design, and major findings of the Wisconsin Sleep Cohort study. *WMJ* 2009; 108:246.

