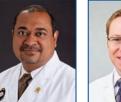
Obstructive Sleep Apnea Diagnosis and Management

by Munish Goyal, MD & Jeremy Johnson, DO

Obstructive Sleep Apnea is a serious illness affecting about 12% of adults in the United States. The majority of patients are undiagnosed posing serious health care and economic burden.





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Abstract

The diagnosis and treatment of obstructive sleep apnea is discussed in the context of recent diagnostic and therapeutic advances.

Introduction

Obstructive sleep apnea (OSA) is characterized by episodes of breathing cessation or shallow breathing in sleep. These episodes are due to complete or partial collapse of upper airway. Most of the time, the respiratory events are associated with snoring, oxygen desaturations and brief arousal from sleep.

Sleep apnea is usually worse during supine and Rapid Eye Movement (REM) sleep. There could be exacerbation of snoring and OSA with alcohol consumption or ingestion of sedative medications.

Epidemiology

According to the Wisconsin Sleep Cohort Study preformed on random middle aged state employees of Wisconsin in 1993, Young et al. reported an estimated prevalence of OSA (AHI \geq 5) in 9% of women and 24% of men. However, the prevalence of symptomatic OSA or OSA syndrome was only 2% in women versus 4% in men.¹ Due to increasing prevalence of obesity, the current estimated prevalence in North America is close to 20-30% in men and 10-15% in women.² According to a recent report by American Academy of Sleep Medicine (AASM), OSA affects 12% of US adult population

totaling 29.4 million individuals and 80% of these are undiagnosed. The annual economic burden due to the undiagnosed sleep apnea is estimated around \$150 billion in year 2015.³

Obesity is a major risk factor for OSA. OSA is seen in up to 70% of morbidly obese individuals.⁴ Other risk factors include advancing age and craniofacial deformities. OSA is also common in smokers, individuals with family history of OSA and in postmenopausal females. Prevalence of OSA increases with coexisting medical conditions like – pregnancy, atrial fibrillation, stroke, congestive heart failure (CHF), Chronic Obstructive Pulmonary Disease (COPD), hypothyroidism, and Polycystic Ovary Syndrome (PCOS).

Diagnostic Criteria

Modified from International Classification of Sleep Disorders – Third edition (ICSD-3)⁵ (A and B) or C satisfies the criteria:

A. The presence of one or more of the following:

1. The patient complains of sleepiness, nonrestorative sleep, fatigue, or insomnia.

2. The patient wakes with breath holding, gasping, or choking.

3. The bed partner or an observer reports habitual snoring or breathing interruptions in sleep.

4. The patient has hypertension, a mood disorder, cognitive dysfunction, coronary artery disease, stroke, congestive heart failure, atrial fibrillation, or type 2 diabetes mellitus.

B. Polysomnography (PSG) or out-of-center sleep testing (OCST) demonstrates:

Five or more predominantly obstructive respiratory events (obstructive apneas, hypopneas, or respiratory effort related arousals [RERAs]) per hour of sleep during a PSG or per hour of monitoring (OCST).

C. PSG or OCST demonstrates:

Fifteen or more predominantly obstructive respiratory events (apneas, hypopneas, or RERAs) per hour of sleep during a PSG or per hour of monitoring (OCST).

Definition or Scoring of Respiratory Events During PSG

In adults, various respiratory events are defined and scored per the criteria laid out by the AASM Manual for the Scoring of Sleep and Associated Events.⁶

An obstructive apnea is scored if there is a drop in the respiratory effort signal by \geq 90% of pre-event baseline, for \geq 10 seconds and continued or increased inspiratory effort from chest and/or abdomen.

A hypopnea is defined when there is drop in the respiratory effort by \geq 30% of pre-event baseline for \geq 10 seconds, associated with oxygen desaturation by \geq 3% (AASM criteria) or by \geq 4% (CMS criteria).

A Respiratory Effort-Related Arousal (RERA) is scored if there is flattening of the inspiratory nasal pressure for ≥ 10 seconds causing an arousal from sleep, but not meeting criteria for an apnea or hypopnea.

The severity of OSA is determined by an index – Apnea Hypopnea Index (AHI) or Respiratory Disturbance Index (RDI), if PSG is preformed, or Respiratory Event Index (REI) if OCST is performed.

- AHI = number of Apneas+Hypopneas/total sleep time
- RDI = number of Apneas + Hypopneas + RERAs/ total sleep time
- REI = number of Apneas + Hypopneas/ monitoring time
- AHI or REI < 5/hour = normal (for adults);
 5-14.9/hour = mild OSA; 15-29.9/hour = moderate OSA; and ≥30/hour = severe OSA.

Signs and Symptoms

Snoring and daytime sleepiness are the most common symptoms in patients with OSA. Other nighttime symptoms include witnessed apneas by the bedpartner, waking up choking or gasping, restless sleep, frequent arousals, nocturia, and erectile dysfunction. Patients with OSA may experience non refreshing sleep, early morning headaches, lack of energy, poor concentration, fatigue or bad mood.

OSA can be associated with lack of attention, loss of productivity, and accidents at work or on the road. OSA increases the risk of a motor vehicle crash by two to three times. Significant reduction in this risk can be achieved with treatment of OSA such as Continuous positive airway pressure (CPAP) therapy.⁷

Examination

As obesity is a major risk factor for OSA, measurement of Body Mass Index (BMI) should be performed. Neck circumference of \geq 17 inches in men and \geq 16 inches in women is associated with higher risk of OSA. Upper airway abnormalities such as macroglossia, enlarged tonsils, large uvula, retrognathia, nasal septal deviation, turbinate hypertrophy; predispose one for OSA. Airway narrowing can be clinically quantified by Mallampati score.

Screening Tools

To identify the patients at risk, there are several screening tools such as Berlin questionnaire, STOP-BANG questionnaire, and Preoperative questionnaire. Epworth Sleepiness Scale (ESS) is a subjective measure of sleepiness, and is commonly used to screen for OSA. STOP-BANG is an easily administrable tool with high sensitivity. (See Table 1.) Patients with a score of 0 to 2 have low risk of OSA whereas those with score of 5 to 8 have high risk of moderate – severe OSA (AHI > 15).⁸

Diagnostic Tests

The sleep tests are classified depending on the number of channels being monitored during the test. Type 1 test is the in-lab attended sleep study or polysomnography (PSG), which is the gold standard test to diagnose OSA. Out-of-center sleep testing (OCST) or Home Sleep Apnea Test (HSAT), a type 3 test, is an acceptable alternative, when PSG is not feasible. The HSAT does not measure sleep as there is no EEG recording. AASM recommends HSAT in patients with high pretest probability after comprehensive sleep evaluation. HSAT should not be used as a screening tool and is not appropriate in patients with comorbid severe pulmonary disease, congestive heart failure, neuromuscular disease or other sleep disorders like periodic limb movement disorder (PLMD), narcolepsy, or parasomnias. A negative or technically inadequate HSAT should be followed by an in-lab PSG in a symptomatic patient with high index of suspicion for OSA.9

Table	1	
STOP	BANG	score

	Question	Yes or No
S	Do you Snore loudly?	
Т	Do you feel Tired or sleepy during the day?	
0	Has anyone Observed apneas or choking during sleep?	
Р	Do you have high Blood Pressure	
В	BMI > 35	
А	Age > 50	
Ν	Neck circumference ≥17 inches (men); ≥16 inches (women)	
G	Gender - male	

Adapted from Chung F, Abdullah HR, Liao P. STOP-Bang Questionnaire: A practical approach to screen for obstructive sleep apnea. CHEST Journal. 2016;149(3):631-8.⁽⁸⁾

Management of Obstructive Sleep Apnea

Moderate or severe OSA should always be treated due to significantly increased risk of cardiovascular and all-cause mortality if untreated.¹⁰⁻¹² Treatment of mild OSA should be considered in symptomatic patients (insomnia, daytime fatigue or sleepiness) or if there are co-morbid illnesses such as - heart failure, ischemic heart disease, atrial fibrillation, hypertension, stroke, pulmonary hypertension, metabolic syndrome, diabetes mellitus, cognitive impairment, and mood disorders. The AASM recommends positive airway pressure (PAP) as the treatment of choice for all severities of OSA.¹³ PAP is primarily employed as continuous positive airway pressure (CPAP); however, autotitrating (APAP) and bilevel (BPAP) modes of PAP are also available for select patients. Alternative therapies to CPAP include oral appliances, surgical procedures, and behavioral treatments. (See Table 2.) Recently several new and novel therapies have been introduced.

Continuous Positive Airway Pressure (CPAP)

Continuous fixed positive pressure (CPAP) applied to the upper airway acts as a pneumatic splint and prevents collapse of the upper airway during inspiration. Nasal CPAP was first described in 1981¹⁴ and has since become the first line of therapy for OSA. The Centers for Medicare & Medicaid Services (CMS) approves CPAP use in patients with moderate or severe OSA (AHI \geq 15) and in patients with mild OSA (AHI \geq 5 – 14.9) that have documentation of one of the following: excessive daytime sleepiness, hypertension, ischemic heart disease, history of stroke, impaired cognition, mood disorder, or insomnia.¹⁵

Behavioral Treatment

Behavioral treatments include weight loss, positional therapy, and avoidance of alcohol and/or sedatives before bedtime. These therapies are primarily adjunctive in the treatment of OSA, but they can also be primary treatment of mild OSA. A substantial amount of weight loss to potentially have an effect on OSA is generally considered to be at least 10% of the body weight. Weight loss to a BMI of 25 or less is also considered ideal.

Positional therapy is the avoidance of sleeping in a supine position to counter the gravitational effect of supine sleep on the tongue and narrowing of the airway. There are various ways to attempt to stay off the back during sleep. Cost effective ways include placing tennis balls in a tube sock and attaching the tube sock vertically in the center on the back of a sleep shirt or by wearing a filled backpack to bed. There are also commercially available, FDA approved, positional therapy devices (Zzoma) and sleep positioner alarm systems (Night ShiftTM)

Oral Appliances

Oral appliances (OA) or mandibular repositioning appliances (MRA) are designed to treat OSA by enlarging the upper airway by advancing the mandible anteriorly. Although not as efficacious as CPAP therapy, oral appliance therapy (OAT) can be an acceptable alternative treatment for snoring, mild OSA, and even moderate OSA. Due to inferior efficacy, OAT should only be used as second line therapy in patients with severe OSA after they have been given a trial of CPAP and failed. There are many OAs on the market; however, only a few are approved by Medicare such as the Herbst, Narval CC,

Table 2	
Treatment of OSA based o	n Severity

	Mild OSA	Moderate OSA	Severe OSA
	(AHI/RDI 5 - 14.9)	(AHI/RDI 15 - 29.9)	(AHI/RDI <u>></u> 30)
Primary treatment	Observe (Asymptomatic) Positional Therapy Oral appliance Surgery – UPPP	РАР	РАР
Secondary treatment	РАР	Oral appliance Or Surgery - UPPP	Surgery – MMA Or Oral appliance
Adjunctive	Weight loss	Weight loss	Weight loss
	Positional Therapy	Positional Therapy	Positional Therapy

AHI – Apnea Hypopnea Index; RDI – Respiratory Disturbance Index; PAP – positive airway pressure; UPPP – uvulopalatopharyngoplasty; MMA – maxillomandibular advancement

Adapted from Epstein LJ, Kristo D, Strollo Jr PJ, Friedman N, Malhotra A, Patil SP, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med. 2009;5(3):263-76.⁽¹³⁾

and TAP. The OA should be custom fit by a dentist knowledgeable in the treatment of OSA. Patients on OAT need to be monitored closely for temporal mandibular joint pain, headaches, gum irritation, and changes in occlusion.

Surgical Treatment

There are many surgical options for the treatment of OSA aimed at bypassing the site of obstruction (tracheostomy), modifying the site of obstruction (majority of procedures), and indirectly treating OSA (bariatric surgery). In general a tracheostomy is considered the gold standard treatment of OSA but has social implications and is undesired by most patients. Uvulopalatopharyngoplasty (UPPP) is the most common surgery. It consists of a tonsillectomy and excision of the uvula and part of the soft palate. UPPP corrects retropalatal obstruction but does not address retroglossal (retrolingual) obstruction. The success rates for UPPP are only approximately 40% - 50%.^{16, 17}

After tracheostomy, maxillomandibular advancement (MMA) surgery is the most successful surgical procedure for OSA and reported to be curative in 80% to 90% of patients.¹⁷ Therefore MMA is an alternative treatment for severe OSA.¹³ MMA involves moving forward the mandible, maxilla, upper teeth, and lower teeth to increase the retrolingual space and the retropalatal space.

New and Novel Therapies for OSA

Although CPAP is extremely effective, one fourth to one half of patients with OSA have substantial difficulty tolerating CPAP.¹⁸⁻²⁰ Due to the importance of treating OSA, several new therapies have recently been introduced. These include oral pressure therapy, nasal expiratory positive airway pressure, and upper airway stimulation.

Oral Pressure Therapy (OPT) Winx®

The Winx[®] Sleep Therapy System provides a light negative pressure in the oral cavity through an individually fitted soft mouthpiece connected to a quiet vacuum by tubing. OPT circumferentially increases the size of the retropalatal and retrolingual airways by pulling forward the soft palate and the tongue.²¹

Nasal Expiratory Positive Airway Pressure (Provent®)

Nasal Expiratory Positive Airway Pressure (Provent[®]) is another new and novel device that consists of disposable, single night use, one-way valves or resistors that are held in place over the nasal passages by a hypoallergenic adhesive. The one-way valve allows for low resistance on inspiration but high resistance during exhalation thus creating positive pressure throughout the exhalation cycle. This splints open the upper airway creating resistance to collapse during the following inspiration.²² Long-term studies have demonstrated efficacy in treating all severities of OSA.²³

Upper Airway Stimulation (Inspire[®])

Another promising emerging treatment of OSA involves stimulation of the upper airway via an implantable neurostimulator. This device stimulates the hypoglossal nerve and therefore the genioglossal muscle during patient's breathing leading to protrusion of the tongue and palate. This increases the airway diameter maintaining an open airway. This FDA approved device has been validated as an alternative treatment in carefully selected patients with moderate to severe OSA and a BMI below $32^{24, 25}$; however, currently it is only being performed in select centers.

Conclusion

OSA is a serious illness affecting about 12% of adults in the US. The majority of patients are undiagnosed posing serious healthcare and economic burden. STOP-BANG score can be used as a reliable screening tool to identify patients with probable OSA. Early diagnosis with an in-lab PSG or HSAT should be followed by an appropriate therapeutic modality. Currently CPAP remains the treatment of choice for moderate and severe OSA. Although not as efficacious as CPAP, there are acceptable alternative therapies for the treatment of mild OSA and more severe OSA in patients that cannot tolerate or adhere to CPAP. As the search for the most efficacious and best tolerated treatment continues, new and novel therapies are emerging.

References

 Young T, Palta M, Dempsey J, Skatrud J, Weber S, Badr S. The occurrence of sleep-disordered breathing among middle-aged adults. The New England journal of medicine. 1993;328(17):1230-5. Epub 1993/04/29.

2. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. American journal of epidemiology. 2013;177(9):1006-14.

 Watson N. Health care savings: the economic value of diagnostic and therapeutic care for obstructive sleep apnea. J Clin Sleep Med. 2016;12(8):1075-7.

4. Ravesloot MJL, Van Maanen J, Hilgevoord A, Van Wagensveld B, De Vries N. Obstructive sleep apnea is underrecognized and underdiagnosed in patients undergoing bariatric surgery. European Archives of Oto-Rhino-Laryngology. 2012;269(7):1865-71.

5. Medicine AAoS. International classification of sleep disorders third edition (ICSD-3). Darien, Illinois. 2014.

6. AASM Manual for the Scoring of Sleep and Associated Events, Version 2.3 - April 1, 2016.

7. Karimi M, Hedner J, Häbel H, Nerman O, Grote L. Sleep apnea related risk of motor vehicle accidents is reduced by continuous positive airway pressure: Swedish traffic accident registry data. Sleep. 2015;38(3):341.

8. Chung F, Abdullah HR, Liao P. STOP-Bang Questionnaire: A

practical approach to screen for obstructive sleep apnea. CHEST Journal. 2016;149(3):631-8.

9. Collop NA, Anderson WM, Boehlecke B, Claman D, Goldberg R, Gottlieb D, et al. Clinical guidelines for the use of unattended portable monitors in the diagnosis of obstructive sleep apnea in adult patients. J Clin Sleep Med. 2007;3(7):737-47.

 Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, et al. Sleep disordered breathing and mortality: eighteen-year followup of the Wisconsin sleep cohort. Sleep. 2008;31(8):1071-8.
 Marin JM, Carrizo SJ, Vicente E, Agusti AG. Longterm cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. The Lancet. 2005;365(9464):1046-53.

12. Punjabi NM, Caffo BS, Goodwin JL, Gottlieb DJ, Newman AB, O'Connor GT, et al. Sleep-disordered breathing and mortality: a prospective cohort study. PLoS Med. 2009;6(8):e1000132.

13. Epstein LJ, Kristo D, Strollo Jr PJ, Friedman N, Malhotra A, Patil SP, et al. Clinical guideline for the evaluation, management and long-term care of obstructive sleep apnea in adults. J Clin Sleep Med. 2009;5(3):263-76.

 Sullivan C, Berthon-Jones M, Issa F, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. The Lancet. 1981;317(8225):862-5.
 CMS update March 2008. 2008; Available from: www.cms.gov.
 Caples SM, Rowley JA, Prinsell JR, Pallanch JF, Elamin MB, Katz SG, et al. Surgical modifications of the upper airway for obstructive sleep apnea in adults: a systematic review and meta-analysis. Sleep. 2010;33(10):1396-407.

17. Elshaug A, Moss J, Southcott A. Redefining success in airway surgery for obstructive sleep apnea: a meta analysis and synthesis of the evidence. SLEEP 2007; 2007.

18. Kohler M, Smith D, Tippett V, Stradling JR. Predictors of longterm compliance with continuous positive airway pressure. Thorax. 2010;65(9):829-32.

19. Kribbs NB, Pack AI, Kline LR, Smith PL, Schwartz AR, Schubert NM, et al. Objective measurement of patterns of nasal CPAP use by patients with obstructive sleep apnea. American Review of Respiratory Disease. 1993;147(4):887-95.

20. McArdle N, Devereux G, Heidarnejad H, Engleman HM, Mackay TW, Douglas NJ. Long-term use of CPAP therapy for sleep apnea/ hypopnea syndrome. American Journal of Respiratory and Critical Care Medicine. 1999;159(4):1108-14.

21. Colrain IM, Black J, Siegel LC, Bogan RK, Becker PM, Farid-Moayer M, et al. A multicenter evaluation of oral pressure therapy for the treatment of obstructive sleep apnea. Sleep medicine. 2013;14(9):830-7.

 Berry RB, Kryger MH, Massie CA. A novel nasal expiratory positive airway pressure (EPAP) device for the treatment of obstructive sleep apnea: a randomized controlled trial. Sleep. 2011;34(4):479-85.
 Kryger MH, Berry RB, Massie CA. Long-term use of a nasal expiratory positive airway pressure (EPAP) device as a treatment for obstructive sleep apnea (OSA). J Clin Sleep Med. 2011;7(5):449-53B.
 Strollo Jr PJ, Soose RJ, Maurer JT, De Vries N, Cornelius J, Froymovich O, et al. Upper-airway stimulation for obstructive sleep apnea. New England Journal of Medicine. 2014;370(2):139-49.
 Woodson BT, Gillespie MB, Soose RJ, Maurer JT, De Vries N, Steward DL, et al. Randomized controlled withdrawal study of upper airway stimulation on OSA short-and long-term effect. Otolaryngology--Head and Neck Surgery. 2014;151(5):880-7.

Disclosure

None reported.