Obstructive Sleep Apnea

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KEYWORDS

• Obstructive sleep apnea • Adult • Pediatric • Diagnosis • Treatment

KEY POINTS

- Obstructive sleep apnea (OSA) is a commonly underdiagnosed heterogenous condition that causes a significant burden of disease across the world.
- OSA causes significant long-term sequelae beyond just significant daytime sleepiness.
- Recognition of OSA and implementation of appropriate testing and treatment at the primary care level is crucial.
- Diagnostic methods for OSA, particularly at home testing options, are rapidly changing and becoming more convenient for patients.
- While positive airway pressure (PAP) therapy remains the mainstay of treatment for OSA, other exciting viable treatment options have emerged for those who cannot tolerate PAP therapy.

HISTORY

It is a common belief that sleep apnea syndrome was first described by Charles Dickens in his novel "The Pickwick Papers" (1836), which featured a character who exhibited obesity, loud snoring, and somnolence.[1] However, there were references to sleep disturbances dated back as far as 400 BC when Hippocrates noted the consequences of disrupted sleep on long-term health.[2] Until the late nineteenth century, research on sleep apnea symptoms focused solely on patients' obesity rather than the disordered breathing they experienced during sleep.

In 1965, the first polysomnography (PSG) was used by French researchers Gastaut, Tassinari, and Duron to observe apneic events during sleep, marking a crucial advancement in sleep apnea research. In 1970, the first sleep clinic was established by William Dement at Stanford University in California. Two years later, Christian Guilleminault joined the clinic and his research focused on respiratory disorders during sleep. Ever since, there has been an intense growth of sleep apnea research,

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especially during 1975 to 1980, with more than 300 articles on sleep apnea published in the literature.[3]

In 1980, Dr Colin Sullivan from Sydney, Australia, who devoted his career to studying respiratory control in dogs, invented the first continuous positive airway pressure (CPAP) machine, revolutionizing the treatment of sleep apnea. The treatment was not widely accepted by the public in the beginning. It was not until almost 20 years later, in 2000, when 4 separate papers were published in the same year that found significant sleep apnea-related health effects, a turning point in sleep apnea studies occurred. Later studies also showed a strong association between sleep apnea and an increased prevalence of a range of comorbidities such as hypertension, coronary heart disease, heart failure, and stroke.[3]

EPIDEMIOLOGY

Obstructive sleep apnea (OSA) is highly underdiagnosed in the United States, with 82% of men and 93% of women with OSA unaware of their conditions.[4] Estimates of prevalence vary depending on diagnostic methods, definitions of the disease, age, gender, body mass index (BMI), and racial backgrounds.[5]

The prevalence of OSA increases with age and appears to plateau in the elderly group. Men are twice as likely to develop OSA, although this difference declines in the middle to older age group, as menopause is a risk factor for OSA. Compared with Whites, the prevalence of OSA is higher in Blacks and similar in Asians. Despite generally having a lower BMI, differences in craniofacial bony structures predispose Asians to developing OSA. Although there is limited data on OSA prevalence rates among Hispanic and Native Americans, there is evidence that OSA prevalence has increased in these groups, likely due to rising obesity rates.[5]

In the pediatric population, the prevalence is estimated between 1% and 4%, although this is likely underestimated given the obesity epidemic. There is no gender difference in prevalence for prepubertal children, but it is more prevalent in adolescent males compared with females. OSA is more prevalent in Black children compared with White children, but the prevalence among other ethnicities has not been well established to this point.

PATHOPHYSIOLOGY

The 2 most common types of sleep apneas are obstructive and central sleep apnea. Central sleep apnea (CSA) results from dysfunction in the respiratory control centers of the brainstem, which fail to provide signals to inhale, causing the individual to miss one or more breathing cycles during sleep. In contrast, OSA occurs when there is a physical blockage of the upper airway despite the brain sending signals to breathe.[6]

The pathophysiology of OSA is complex but the underlying etiology involves the upper airway dilating muscles becoming insufficient to prevent narrowing and/or closure of the upper airway during sleep. During inspiration, the negative pressure generated in the lumen of the upper airway promotes closure, and pharyngeal dilating muscles must counteract this force to maintain airway patency.[7] Conditions that elevate negative pressure within the upper airway or impair the ability of dilating muscles to maintain airway patency disrupt this equilibrium, increasing the likelihood of upper airway obstruction.[8]

Upper Airway Narrowing

The most prominent factors contributing to upper airway narrowing are either excessive bulk of soft tissue (tongue, soft palate, and lateral pharyngeal walls) from obesity

or craniofacial anatomy, or both.[7] Adenotonsillar hypertrophy, often linked to obesity, is a major risk factor for pediatric OSA.[8]

Volume overload from congestive heart failure (CHF) or end-stage renal disease (ESRD) results in fluid redistribution to the parapharyngeal soft tissues in the recumbent position, which can increase upper airway resistance and collapsibility.[8]

Additional factors, such as nasal obstruction (as seen in rhinitis), the use of intranasal corticosteroids, and the supine posture, have also been identified as adverse influences on upper airway patency.[8]

In pediatric populations, the primary cause of airway narrowing is due to adenotonsillar hypertrophy, although abnormal neuromuscular control also contributes to the development of OSA, like adults.

Upper Airway Dilator Muscle Function

In rapid eye movement (REM) sleep, especially during phasic REM, there is an additional decrease in tone and phasic activity of the pharyngeal dilating muscle, which likely exacerbates the length and severity of apneas and hypopneas.[7]

Apnea Threshold

The apnea threshold is the partial pressure of carbon dioxide ($Paco_2$) at which respiratory effort ceases and apnea occurs and reflects the system's sensitivity to changes in carbon dioxide levels. Patients with OSA and a low apneic threshold have an increased likelihood of apneic events during sleep, as minor fluctuations in $Paco_2$ can trigger airway collapse or respiratory instability.[9]

Loop-Gain

Loop-gain refers to the ratio of the ventilatory response to the disturbance causing it and is a reflection of the sensitivity and responsiveness to changes in ventilation. In patients with OSA, a high loop-gain results in an exaggerated response to respiratory disturbances, which in turn causes unstable ventilation and oscillations in breathing resulting in recurrent hypoxia.[10,11]

Apnea Resolution

Apneas/hypopneas resolve due to 2 mechanisms: The first is increased upper airway muscle tone secondary to chemical (low Pao_2, high $Paco_2$) or mechanical stimuli (stretching of mechanoreceptors). The second mechanism is an arousal from sleep which causes a shift out of the sleep state, thus restoring muscle activity seen during the wake state.

Disease Course

Long-term population studies have shown that OSA severity gradually progresses over time.[7] Weight gain over time similarly results in worsened OSA severity. Conversely, weight loss results in decreased OSA severity. However, weight gain has a greater impact on OSA severity than weight loss, and weight impacts are greater in men compared with women.[7]

Long-term Complications of Obstructive Sleep Apnea

OSA has been identified as a significant risk factor in cardiovascular disease including essential hypertension, coronary artery disease, CHF, pulmonary hypertension, stroke, cardiac arrhythmias, and premature mortality.[7,12] The causes of these complications are multifactorial, including an increase in the sympathetic nervous system in response to oxygen desaturations and arousals from sleep, free radical production from re-oxygenation, and large negative intrathoracic pressures that in turn increases

the intramural pressure of vessels within the intrathoracic cavity. Additionally, OSA has clearly been shown to be related to the onset and recurrence of atrial fibrillation.[12]

OSA has also been linked with metabolic disorders.[13] New evidence suggests that untreated OSA is an independent risk factor for the development of Type 2 diabetes mellitus.[7]

Other risks of untreated OSA include depression, poor job performance, impaired family relationships, reduction in quality of life, and increased risk of motor vehicle accidents.

CLINICAL EVALUATION

Patients with OSA often present in clinic with an assortment of complaints related to nocturnal respiratory disturbances, fragmented sleep, and daytime sequelae. In other situations, patients may present not due to their own concern, but rather due to the concerns of a bed partner or family member.

Symptoms

In the evaluation of suspected sleep apnea, it is crucial to ascertain both nocturnal symptoms as well as those that occur during the day. In our experience, nocturnal symptoms are more reliably provided by a bed partner, and it is important to include the bed partner in the evaluation when possible.

Classic nighttime symptoms of OSA include:

- Loud Snoring: Frequently reported by bed partners, loud and disruptive snoring is a hallmark symptom of OSA due to upper airway obstruction during sleep.
- Witnessed Apneas: Partners or family members may observe episodes of breathing cessation or gasping during sleep, indicating significant airflow limitation.
- Excessive Daytime Sleepiness: Patients with OSA often experience excessive daytime sleepiness despite seemingly adequate nocturnal sleep, leading to impaired cognitive function, reduced productivity, and an increased risk of accidents or errors.
- Morning Headaches: Headaches upon awakening are commonly reported by individuals with OSA, attributed to nocturnal hypoxemia and hypercapnia leading to cerebral vasodilation and increased intracranial pressure.
- Nonrestorative Sleep: Patients may describe feeling unrefreshed or unrested despite spending an adequate duration in bed, reflecting disrupted sleep architecture and poor sleep quality.
- Nocturia: Frequent awakenings to urinate during the night, known as nocturia, are often reported by individuals with OSA.

Daytime symptoms of OSA include:

- Excessive daytime sleepiness: While it is the most common daytime symptom caused by OSA, it is important to recognize that many patients with diagnosed OSA do not report significant daytime sleepiness. Some studies have shown that less than 50% of patients with moderate to severe OSA have excessive daytime sleepiness.[5]
- Difficulty concentrating
- Memory difficulties
- Brain fog
- Irritability
- Hyperactivity
- Fatigue
- Drowsy driving.

Pediatric Considerations

In the pediatric population, daytime symptoms differ significantly from the adult population. At night, they similarly exhibit loud snoring or difficult breathing while asleep as well as significant sweating or restlessness at night.

During the daytime, particularly among younger pediatric patients, developmental, behavioral, and learning issues are most common. Many of the symptoms displayed may mimic those of attention deficit hyperactivity disorder.

Older children or teenagers may present with the symptoms of excessive daytime sleepiness, like adults.

Special attention should be paid to patients with Down syndrome or other neuromuscular disorders as there is a higher prevalence of OSA in these populations compared with the general pediatric population.[7]

Objective Tools to Use in Clinic

The Epworth Sleepiness Scale (**Box 1**) is an objective scale that is frequently used to assess the degree of sleepiness. A score higher than 10 indicates the patient is experiencing pathologic excessive daytime sleepiness.

A commonly used screening tool for OSA is the STOP-BANG score, which provides an objective tool to assess a patient's likelihood of having OSA.

The STOP-BANG mnemonic includes the following:

S: "Do you snore loudly, louder than talking, or enough to be heard through closed doors?"

T: "Do you feel tired or fatigued during the daytime almost every day?"

O: "Has anyone observed that you stop breathing during sleep?"

P: "Do you have a history of high blood pressure with or without treatment?"

B: BMI greater than 35 kg/m^2.

A: Age greater than 50 years.

N: Neck circumference greater than 43 cm (17 in).

G: Gender, male

The most recent 2017 guideline from the United States Preventive Services Task Force (USPSTF) states there is insufficient evidence to recommend the routine screening of OSA in asymptomatic patients.[14] Nevertheless, in patients where there is concern for

Box 1
Epworth Sleepiness Scale

How likely are you to doze off or fall asleep in the following situations, in contrast to feeling just tired? Use the following scale and indicate the most appropriate number for each situation. Answer choices for each situation range from 0 to 3. 0 = would never doze 1 = slight chance of dozing 2 = moderate chance of dozing 3 = high chance of dozing

Situation
- Sitting and Reading
- Watching TV
- Sitting, inactive in a public place (eg, a theater or meeting)
- As a passenger in a car for an hour without a break
- Lying down to rest in the afternoon when circumstances permit
- Sitting and talking with someone
- Sitting quietly after lunch without alcohol
- In a car, while stopped for a few minutes in traffic
- TOTAL (Range of 0–24)

OSA, the STOP-BANG score can be a helpful clinical tool to guide whether further testing or evaluation is warranted. An STOP-BANG score of 3 indicates an intermediate risk for moderate to severe OSA, whereas a score of 5 indicates a high risk for moderate to severe OSA. Specifically, with a score of 3 or higher, the sensitivity for OSA is 87% and the specificity for OSA is 31%.[5] The STOP-BANG tool can be accessed here.[15]

Risk Factors/Comorbid Medical Conditions

The major predisposing risk factor for OSA is excess body weight. In fact, it is estimated that ~60% of moderate to severe OSA is attributable to obesity.[7] In general, as the BMI increases, there is an increased risk of OSA. Among patients with morbid obesity, there is an extremely high prevalence of OSA.

Other risk factors to consider include middle-aged and older adults, postmenopausal status in women, patients with bony or soft structure abnormalities of the head and neck, endocrine disorders such as acromegaly or hypothyroidism, patients with Down syndrome or other neurologic disorders.

Patients who consume alcohol or sedating medications prior to bed are also at an increased risk of OSA.[7]

Physical Examination

The examination of patients with suspected OSA should include the following:

Anthropometric Measurements:

- BMI. BMI $\geq$ 30 kg/m^2, is a strong risk factor for OSA and contributes to airway narrowing and collapsibility
- Neck circumference. NC greater than 17 inches in men, 16 inches in women indicates an increased risk of OSA

Craniofacial Examination:

- Modified Mallampati Classification: This assesses the visibility of the oropharyngeal structures during phonation. Mallampati Scores of III and IV are associated with higher risk of upper airway obstruction.[16]
- Nasal patency: this can identify nasal obstruction that would contribute to mouth breathing. This in turn leads to an increased upper airway resistance during sleep.
- Oropharyngeal anatomy: Specific attention should be paid to tonsillar presence/size, uvula size, or whether the palate is high arching.
- Mandible: Position and size of the mandible should be assessed. Retrognathia and micrognathia are both associated with an increased risk of OSA.[5]
- Dentition: Quality and position of dentition as well as the presence of dentures should be assessed.
- Tongue size: Macroglossia is a common finding among patients with OSA. Patients with macroglossia may demonstrate scalloping of the tongue along the lateral edges.

Other important components of the physical examination:

- Cardiovascular examination: assess for murmurs or cardiac arrhythmias as well as edema or jugular venous distension as signs of heart failure.
- Pulmonary: assess for pulmonary vascular congestion.

TESTING OPTIONS FOR OBSTRUCTIVE SLEEP APNEA

OSA can only be diagnosed with objective testing. The primary testing options for OSA include nocturnal PSG and home sleep apnea testing (HSAT).

PSG is the gold standard for OSA diagnosis because it provides comprehensive monitoring of sleep architecture, respiratory parameters, as well as physiologic variables. Standard PSG monitoring includes electroencephalogram (EEG), facial electromyography (EMG), and electrooculogram (EOG), oronasal airflow, microphone for snoring or somniloquy, pulse oximetry, respiratory effort, body position, leg EMG, and single-lead electrocardiogram. While more thorough and comprehensive, patients may find laboratory testing to be more uncomfortable than their home environment, and this testing is significantly more expensive that HSAT.

HSAT is more accessible and cost-effective compared with PSG. It is reserved for those with high pretest probability for OSA and who lack comorbidities that would require an in-lab PSG. Examples of comorbidities that are a contraindication to HSAT include asthma, Chronic Obstructive Pulmonary Disease (COPD), CHF, comorbid sleep conditions, cognitive impairment, or morbid obesity (BMI >40 or 50). HSAT is generally more convenient for patients compared with in-laboratory PSG. HSAT options are numerous, and this is an area that continues to expand in terms of device options. Some HSAT options record oronasal airflow, respiratory effort, snore microphone, body position, and pulse oximetry. One newer HSAT option that has increased in use and popularity is the WatchPAT, which measures peripheral arterial tone to detect respiratory events and has no oronasal airflow monitoring. See **Table 1** for a summary of the comparison between PSG and HSAT options.

Overnight oximetry is sometimes used for screening purposes. While oximetry can give valuable insight into respiratory patterns or oxygen desaturation frequency during sleep, it is not capable of making a diagnosis of OSA and should not be used for diagnostic purposes.

Pediatric Considerations

In-laboratory PSG is currently the gold standard and only testing option to diagnose OSA in pediatrics populations. Current HSAT options are not approved or indicated for use in the pediatric population, and they should only have a PSG.[17]

DIAGNOSING OBSTRUCTIVE SLEEP APNEA

Regardless of testing type, all testing for OSA identifies the total number of abnormal respiratory events that occur for the duration of the night. Specifically, these events refer to the total number of apneas and hypopneas. An apnea is defined as a complete upper airway obstruction lasting for at least 10 seconds, whereas a hypopnea is defined as a partial upper airway obstruction lasting for at least 10 seconds and causing either an oxygen desaturation or arousal from sleep.[5,7,18] The total number of events is then divided by the number of hours of sleep. The result of this value is called the Apnea Hypopnea Index (AHI), and this is what is used to diagnose OSA. In adults, an AHI greater than 5 events per hour is considered diagnostic for OSA. See **Table 2** for how OSA severity is defined by the AHI in adults and **Table 3** for pediatrics. In the pediatric population, OSA is diagnosed if the AHI exceeds 1 event per hour.

TREATMENT OF OBSTRUCTIVE SLEEP APNEA

Numerous treatment options exist for OSA in adults and children, and they vary in both efficacy as well as patient convenience. The treatment of OSA requires frequent follow-up and close monitoring of response, and a patient-centered approach should always be prioritized.

Table 1
Diagnostic testing options for obstructive sleep apnea

Diagnostic Test	Description	Advantages	Limitations
Polysomnography (PSG)	Gold standard for OSA diagnosis; comprehensive assessment of sleep architecture, and respiratory parameters.	Provides detailed information on sleep stages, respiratory events, and physiologic variables including facial and limb electromyography, single-lead electrocardiogram.	More expensive than HSAT, requires overnight laboratory monitoring with a technician, may not be as readily accessible.
Home Sleep Apnea Testing (HSAT)	Portable monitoring devices used for out-of-center testing; simplified version of PSG with fewer parameters. Only used in patients with high pretest probability of OSA who lack significant comorbidities	Convenient, cost-effective, and accessible for patients; suitable for uncomplicated cases of OSA.	Limited ability to assess sleep architecture and comorbid sleep disorders; less comprehensive than PSG. May have reduced accuracy in patients with certain comorbidities.

Abbreviation: OSA, Obstructive sleep apnea.

Table 2
Obstructive sleep apnea severity by Apnea Hypopnea Index in adults

AHI	OSA Severity
0–4.9	Normal/none
5–14.9	Mild
15–29.9	Moderate
30 or higher	Severe

Abbreviations: AHI, Apnea Hypopnea Index; OSA, obstructive sleep apnea.

Positive Airway Pressure

Positive airway pressure (PAP) therapy is the mainstay of management of OSA in adults. PAP therapy works by maintaining upper airway patency by pressurizing the upper airway and thus prevents the collapse of the upper airway during sleep.

There are various types of PAP therapy that include CPAP, Automatically titration continuous positive airway pressure (Auto-CPAP), Bilevel positive airway pressure (BiPAP), noninvasive positive pressure ventilation, and adaptive servo ventilation. All PAP modalities are capable of detecting respiratory events including obstructive or central apneas as well as hypopneas. They report data including the AHI, average pressure used, and mask leak.

PAP therapy is recommended for all patients with moderate to severe range OSA and patients with mild OSA with significant daytime sleepiness.

Optimal PAP therapy is best identified with an in-laboratory titration PSG. In this study, the patient is placed on PAP therapy and various pressures and treatment modalities can be trialed until obstructive respiratory events resolve.

Sleep clinicians will frequently order a “split night” PSG. In this study, the first portion of the study is spent without the patient on no therapy and is used to diagnose OSA based on the criteria outlined above or based on criteria defined by insurance companies. If the patients meet the criteria for OSA, they are started on PAP therapy during the latter portion of the night to identify optimal PAP therapy. If optimal PAP therapy is not identified, they will then come back for a full night titration PSG.

Many patients diagnosed with OSA via HSAT are started on Auto-CPAP. This is a PAP modality that is programmed to provide a range of CPAP pressures. The machine will then detect airflow obstruction and increase the pressure to alleviate the obstruction. The benefit of Auto-PAP is that the patient avoids the inconvenience of an in-laboratory PSG. Pressures can be adjusted in follow-up appointments based on the data the machine reports back.

Common side effects of PAP therapy include mask leak, aerophagia, mask discomfort, claustrophobia, dry nose, and dry mouth.

Table 3
Obstructive sleep apnea severity by Apnea Hypopnea Index in children

AHI	OSA Severity
0–0.9	Normal/none
1–4.9	Mild
5–9.9	Moderate
10 or higher	Severe

Abbreviations: AHI, Apnea Hypopnea Index; OSA, obstructive sleep apnea.

Studies have shown PAP therapy to be effective in improving subjective symptoms of OSA. Of interest, studies examining improvements of objective measurements of OSA have not been as definitive. One aspect of PAP therapy that appears to denote subjective and objective benefit in patients with OSA is duration of treatment, with a usage of 7 hours or more showing significant improvement in both subjective and objective measurements.[5,19]

One of the main limitations of PAP therapy is nonadherence. Some studies show that after 1 year less than 50% of patients continue to use PAP therapy.[20] Other studies have shown that among patients who use CPAP regularly, the average use is only 58.7% of the total sleep time.[21] This is particularly problematic when one considers that the minimum number of hours of CPAP use per night needed to reap benefits from its use is between 5 and 6 hours per night. Numerous interventions have been implemented to attempt to increase PAP adherence including more comfortable therapy, new masks, and improved remote monitoring. While PAP therapy is first-line therapy for OSA, marked nonadherence is a significant barrier to management.

There is no generally accepted timeframe for when testing should be repeated. Common indications for repeat testing include persistent poor mask leak, aerophagia or discomfort with CPAP (for which BiPAP can be tried), worsened daytime sleepiness despite adequate PAP usage, persistent elevated leak or poor mask fit, or 10% weight loss or weight gain from the patient's weight at the time of their last study. Specifically, repeat testing for weight gain/loss is indicated to assess for a change in OSA severity or a need for increased or decreased PAP pressures.

Oral Appliances

The most effective oral appliance for the treatment of OSA is a mandibular advancement device (MAD). These are indicated for patients with mild to moderate OSA or patients with severe OSA that cannot tolerate PAP therapy. They work by protruding the mandible which in turn opens the airway by pulling the base of the tongue forward.

Patients who are most likely to benefit from a MAD include younger patients, patients with lower BMI, patients with positional (supine-dependent) OSA, and good dentition.[22] Patients with dentures are not candidates for MADs.

MADs should be fitted and managed by a dentist with sleep-specific training.

Patients who are using a MAD should undergo repeat testing once their device is felt to be at the optimal setting to determine whether OSA severity has been reduced by the MAD. Studies show the response to MAD in regards to OSA reduction to be more variable than with PAP therapy, and thus they should have close follow-up.[5]

Common side effects of MAD include temporomandibular joint dysfunction and chronic bite changes.

Hypoglossal Nerve Stimulator

In 2014, the FDA approved a novel therapy for OSA called Inspire. It is second-line therapy and can only be used or considered in patients who cannot tolerate PAP therapy. Inspire is a hypoglossal nerve stimulator. It is a permanently installed device that is implanted in the right side of the chest with leads that stimulate the hypoglossal nerve at the beginning of inspiration and causes tongue protrusion, which in turn brings the base of the tongue forward to open the airway.[5,23]

Inspire is currently approved for adult patients 18 years and older with an AHI of 15/h to 100/h with a BMI less than 40 kg/m^2. Additionally, patients must have less than 25% central respiratory events on sleep testing performed within the past 2 years. Prior to having the procedure, the patient must have consultation with an Inspire-trained otolaryngologist with a drug-induced sleep endoscopy to evaluate for concentric

collapse, which is a contraindication to PAP therapy. More information on Inspire can be found here.[24]

Studies have shown that 70% of patients with Inspire maintain a reduction in their AHI of 50% or more and AHI less than 20/h over time.[5,25] Additionally, patients who have Inspire tend to use therapy more often and for longer durations compared with patients using CPAP.[26]

While certainly a welcome and beneficial therapy, it is important to emphasize that for now this remains a second-line treatment option.

Other Surgical Therapies

In some patients, there may be specific anatomic abnormalities that can be targeted surgically in efforts to treat OSA. Historically, uvulopalatopharyngoplasty (UPPP) was performed as a surgical treatment for OSA. Long-term studies have shown that the benefits of UPPP tend to decrease over time to less than 50%, and thus UPPP is only performed in specific situations as per the recommendation of otolaryngology.[27] As a general principle, in a patient with refractory OSA or obvious anatomic abnormalities, further assessment by otolaryngology is recommended.

Bariatric surgery is commonly performed in patients with OSA given the significant overlap between obesity and OSA as discussed previously. Most studies have shown a reduction in AHI and BMI after bariatric surgery, but this does not guarantee resolution of OSA. Repeat sleep testing should be performed once the patient reaches a steady weight after surgery.[5]

Other Treament Recommendations

Positional sleep therapy is a common treatment recommendation used often in conjunction with other treatment strategies in patients with OSA that worsens in the supine position. Positional devices are typically worn and prevent inadvertently rolling into the supine position. They are not usually covered by insurance.

Alcohol use should be monitored in all patients with OSA given that alcohol is known to worsen OSA severity. Specifically, alcohol use should be avoided within 2 to 4 hours of sleep.[28]

Similarly, clinicians should avoid prescribing sedating medications such as benzodiazepines and opioids to patients with untreated OSA.

Pediatric Considerations

In the pediatric population, first-line therapy for OSA is tonsillectomy and adenoidectomy (T&A). In patients with confirmed OSA, a referral to ENT for evaluation for T&A is recommended as the first step. If the surgery is performed, repeat testing 3 to 6 months after treatment is recommended to evaluate for the resolution of OSA. The Childhood Adenotonsillectomy Trial (CHAT) study found significant improvements in quality of life and polysomnographic findings in patients who underwent tonsillectomy.[29] Specifically, in children who underwent tonsillectomy, the AHI dropped from 4.8/h to 1.3/h compared with a drop from 4.5/h to 2.9/h in patients who had watchful waiting. Those who underwent tonsillectomy also saw improvements in the Pediatric Quality of Life Inventory compared with those who underwent watchful waiting with an increase of 5.9 from baseline versus an increase of 0.9, respectively.[29]

If OSA persists after tonsillectomy or if the patient is found to not be a candidate for surgery, CPAP or BiPAP can be used in pediatrics following similar principles to those used in the adult population.

Weight loss should be prioritized in pediatric patients with obesity as well.

SUMMARY

OSA is an underdiagnosed heterogeneous condition that affects adults and children. Diagnosis and management of OSA can lead to significant subjective improvement in patient's lives. Given the significant burden of disease as well as significant medical comorbidities, accurate and prompt recognition of OSA by primary care physicians with appropriate work-up and management is crucial.

CLINICS CARE POINTS

- Snoring and daytime sleepiness are key indicators of OSA in adults.
- Attention or behavioral issues in children should raise suspicion for possible OSA.
- Resistant hypertension should raise suspicion for possible OSA.
- Daytime sleepiness and fatigue may be misattributed to other medical conditions such as depression. OSA should be considered in the differential for these common complaints.
- OSA is underdiagnosed in women and in patients who lack classic clinical features.
- While home sleep testing is becoming more common, a negative home sleep test does not mean a patient does not have obstructive sleep apnea.
- Lifestyle and behavioral changes can significantly improve or resolve OSA and should always be included in the treatment plan.

DISCLOSURE

None.

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