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Review Article

Snoring, Choking, Gasping, Awakening, and Falling Asleep Again: Getting to Know the Symptoms of Obstructive Sleep Apnea and the Treatment Options Available for OSA

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Abstract

Obstructive sleep apnea (OSA) is a common sleep disorder characterized by episodes of apnea or hypopnea that occur during sleep and lead to decreased oxygen and awakening. Persistent sleep deprivation and fall in blood oxygen levels can lead to certain long-term complications, such as cerebrovascular accidents, myocardial infarction, hypertension, depression, and increased daytime sleepiness. Common types of OSA are adult OSA and pediatric OSA. Risk factors include obesity, family history, craniofacial abnormalities, and certain lifestyle habits such as smoking, alcohol and sedatives use.

Polysomnography (sleep study) is the gold standard for confirming an OSA diagnosis. Treatment options for OSA include lifestyle modifications, use of devices, and pharmacotherapy. Lifestyle modifications consist of smoking cessation, weight loss in obese patients, shunning alcohol, and avoiding neuromuscular blockers and spasmolytics. Devices range from the use of positive pressure therapy (CPAP) to undergoing surgical intervention, such as uvulopalatopharyngoplasty (UPPP) and radiofrequency ablation (RFA). Rarely utilized is pharmacotherapy (such as paroxetine and acetazolamide). CPAP is considered the first-line treatment for OSA.

Keywords: *Ablation; Daytime Sleepiness; Hypoapnea; Obesity; Rapid Eye Movement; Tongue*

Abbreviations

AHI: Apnea-Hypopnea Index; BMI: Body Mass Index; CPAP: Continuous Positive Airway Pressure Therapy; CSA: Central Sleep Apnea; EEG: Electroencephalogram; ENT: Ear-Nose-Throat; ICSD-3: International Classification of Sleep Disorders–Third Edition; MARPE: Mini-Implant-Assisted Rapid Palatal Expansion; NREM: Non-Rapid Eye Movement; OSA: Obstructive Sleep Apnea; OSAHS: Obstructive Sleep Apnea-Hypopnea Syndrome; OSAS: Obstructive Sleep Apnea Syndrome; PSG: Polysomnography; REM: Rapid Eye Movement; RFA: Ra-

diofrequency Ablation; UPPP: Uvulopalatopharyngoplasty; USFDA; US Food and Drug Administration; VPAP: Variable Positive Airway Pressure

Introduction

The first mention of obstructive sleep apnea (OSA) in the medical literature was in 1965, although the condition was already known [1–3]. The initial reports of OSA described in the medical literature included a specific type of symptomatology due to severe episodes of OSA. Commonly described cases had similar presentations, such as decreased blood oxygen levels, increased carbon dioxide production, and congestive cardiac failure [1–4].

Long before 1965, a physician coined the term Pickwickian syndrome for modern-day OSA in the early 20th century. The term Pickwickian syndrome probably came from the description by the famous English writer Charles Dickens in his novel *The Pickwick Papers*. Dickens appropriately described the symptoms of OSA in this book.

Before 1965, physicians considered OSA a condition characterized by simple intermittent closure of the upper airway. Thus, treatment focused on removing obstruction of the upper airway. Despite OSA's first appearance in the medical literature, it took about fifteen years to optimize suitable treatment options. Before the 1980s, the only effective treatment available for OSA was tracheostomy to bypass the obstruction in the upper airway.

However, in 1981, the introduction of continuous positive airway pressure therapy (CPAP) through a nasal mask was hailed as a landmark in the treatment of OSA and, thus, renewed interest in the field of sleep medicine [5]. Since the 1980s, researchers worldwide have made significant progress in understanding the features and long-term and short-term sequelae of OSA. The condition is now recognized as a significant health issue.

Discussion

With a better understanding of the condition, its treatment options also evolved. From the first days of tracheostomy, OSA treatment options have multiplied. In 1981, CPAP was introduced as a non-surgical option for OSA management. However, CPAP machines were bulky and noisy during those days, making them notably troublesome for patients. Nevertheless, by the late 1980s, these devices were replaced by quieter and sleeker versions of CPAP machines [2,6].

The availability of affordable treatment options for OSA led to a push by sleep experts to raise awareness among physicians and the community in general regarding OSA, thus revolutionizing the management and diagnosis of sleep disorders, including OSA. A seminal study published in 1993 compiled data from the Wisconsin Sleep Cohort Study explored the prevalence of undiagnosed sleep disorders among the adult population and addressed public health associations that explored its impact on individuals and their families [7].

Overnight polysomnography from a random sample of 602 men and women aged 30–60 years was undertaken to estimate the frequency of apnea and hypopnea during each hour of sleep (the apnea-hypopnea score). According to this study, 9% of women and 24% of men had abnormal sleep breathing [7]. Furthermore, 2% of women and 4% of middle-aged men met the minimal diagnostic criteria for OSA.

Being male and obese are two significant contributing factors associated with OSA. Also, people with a history of habitual snoring irrespective of their sex are at increased risk of having higher apnea-hypopnea scores (15 or higher). The study concluded that the estimated prevalence of OSA is much higher among women than previously thought; also, men are at increased risk of suffering from OSA [8,9]. Researchers also concluded that daytime hypersomnolence is common with OSA [6,8,9].

More recent studies have estimated that the prevalence of OSA is significantly greater in people in high-income countries than had been noted in previously published studies [10,11]. Growing obesity and dependence on technology have been cited as possible causes of the increase in OSA prevalence. However, specific Latin American countries, such as Brazil, and Asian countries also have higher OSA

prevalence than certain high-come countries—although there is a lower obesity prevalence in these countries than in some developed high-income countries. In addition, there is a lower prevalence of obesity in these countries [10–12].

Several famous personalities suffer from sleep apnea: Shaquille O'Neal (considered to be one of the greatest basketball players of all time), Rosie O'Donnell (a US television personality), Randy Jackson (a judge on the show American Idol), Amy Poehler (an American actress), Reggie White (a famous footballer and member of the Football Hall of Fame—who died in 2004 from a heart attack, a direct complication of sleep apnea as pointed out by his physicians), and many more.

OSA is characterized by frequent episodes of complete or partial obstruction of the upper airway, resulting in decreased breathing during sleep. These episodes of complete or partial breathlessness are called apnea or hypoapnea, respectively.

In both scenarios mentioned above, the affected person may suffer from decreased blood oxygen level, disturbed sleep, or both. Frequent episodes of apnea and hypopnea can interfere with restorative sleep, leading to decreased blood oxygen levels, poor quality of life, and negative health consequences [13,14].

Sometimes, the terms obstructive sleep apnea syndrome (OSAS) and obstructive sleep apnea-hypopnea syndrome (OSAHS) are used in specific patients to refer to OSA. These patients may suffer from symptoms, such as excessive daytime sleepiness and poor cognitive functioning due to a lack of sleep [13–15].

Most patients with OSA are usually unaware of their symptoms, such as difficulty breathing during sleep, even after awakening in the middle of the night. A family member or sleeping partner often reports symptoms, such as frequent loud snoring, sudden stoppage of breathing, and episodes of gasping and choking while sleeping [13–15].

People who live alone or sleep alone are more likely to be unaware of their symptoms. Therefore, it might take years for a diagnosis of OSA to occur. These patients may also be accustomed to the resulting daytime sleepiness and fatigue and, therefore, do not know or report to physicians about their symptoms.

According to the Third Edition of the International Classification of Sleep Disorders (ICSD-3), OSA is classified under Sleep-Related Breathing Disorders and described in adult OSA and pediatric OSA [7]. Additionally, there is another type of sleep apnea, known as central sleep apnea (CSA).

OSA is differentiated from CSA by certain characteristic features. CSA is characterized by episodes of reduced or complete breathing cessation due to decreased respiratory effort. However, in OSA, breathing is decreased or completely stopped due to obstruction of the upper airways [5].

Thus, to differentiate between the two conditions and for confirmation of diagnosis, the respiratory effort of the patients must be assessed [6]; in OSA, the inspiratory effort (diaphragmatic activity) continues or even increases during the entire episode of apnea [16]. In patients with apnea and hypopnea, the term obstructive sleep apnea-hypopnea is used, while in patients in whom, in addition to apnea and hypopnea, there is daytime sleepiness, the condition is called obstructive sleep apnea-hypopnea syndrome [17].

According to the diagnostic criteria for OSA, a patient must have hypopnea that meets one or more of the symptoms, namely snoring during the episode of hypopnea, increased flattening of the oronasal flow, and paradoxical thoracoabdominal respiration during the attack of hypopnea [16]. However, in patients with central hypopnea, none of those mentioned above criteria is met.

Pathogenesis

Under normal physiological conditions, when a person transits from wakefulness to a sleeping state (REM sleep or NREM sleep), there is a reduction in upper airway muscle tone [18–21]. Significantly during REM sleep, the tone of the throat and neck muscles is attenuated along with other skeletal muscles. Therefore, especially during REM sleep, the tongue and soft palate relax, airway patency is reduced,

and airflow inside the lungs during the inspiratory phase is diminished. Therefore, if reduced ventilation is associated with low blood oxygen levels or requires increased high breathing efforts, the neurological system of the affected person could lead to sudden interruption of sleep, called neurological arousal. This type of awakening rarely leads to complete awakening; however, it can negatively impact sleep quality.

One consequence for patients with significant OSA is sleep deprivation due to repeated episodes of sleep disturbances and poor sleep recovery. In addition, interrupted sleep in stage 3 (during slow-wave sleep) and REM sleep can negatively impact standard growth patterns, healing, and immune response—specifically in children and younger adults.

The leading underlying cause of OSA is blockade of the upper airways, especially behind the tongue and epiglottis. Therefore, in the awake patient, the airway is open, but during sleep in a supine position, the airway is prone to collapse due to the loss of pharyngeal muscle tone [19,20].

During the initial stage of sleep, when the person is in a lighter stage of sleep, muscle tone remains intact and breathing is noiseless (as airflow is laminar). As sleep deepens muscle tone, the upper airway gradually collapses and airway obstruction progressively becomes apparent, characterized by noisy breathing as airflow becomes turbulent and snoring appears due to the Venturi effect. The entire cycle of steady airway collapse worsens, leading to decreased blood oxygen saturation and total airway obstruction, prompting a sudden stop of all sleep sounds and complete apnea that may last for a few minutes in some patients [19–23].

Eventually, the affected person awakens from deep sleep to lighter sleep, thus facilitating a general muscle tone response. The transition from deep sleep to light sleep, then again to deep sleep, can be documented by electroencephalogram (EEG) monitoring. During the lighter phases of sleep, muscle tone becomes usual and hence the airway opens up spontaneously, leading to the resumption of noiseless breathing and normalization of blood oxygen levels. Eventually, the patient reenters the deep sleep phase where there is again a loss of upper airway muscle tone, resulting in airway obstruction. This cycle of blockage and opening of the airways is repeated throughout the sleep cycle.

For quantitative analysis, the total episodes of apnea and hypopnea during any hour are counted and a score is assigned. If the number of episodes of apnea and hypopnea is greater than five in one hour, the patient is diagnosed with a mild form of OSA. In severe OSA, the number of episodes of apnea and hypopnea is more than thirty episodes per hour. There are specific pathophysiological models of OSA. However, the underlying causes of spontaneous upper airway blockage have not yet been established unequivocally—specialists from different fields of medicine offer differing hypotheses.

According to classic pulmonologists and neuro medicine specialists, brain injury, poor muscle tone, an increase in airway soft tissues and a long history of snoring can contribute to OSA pathogenesis. In addition to the elderly, OSA can occur among newborns with Pierre Robin syndrome. However, in most OSA patients, there is no history of brain injury [24]. Drug abuse, including alcoholism, could cause poor muscle tone, although there is no history of drug abuse in most OSA patients. Chronic snoring could injure local nerves in the pharynx, contributing to the OSA etiopathogenesis [23]. Although an increase in soft tissues in the upper airways is considered a contributing factor, it might not be present in many OSA patients.

According to ear-nose-throat (ENT) specialists, certain anatomical structural features can narrow the airways; these are enlargement of the tonsil or posterior part of the tongue or accumulation of fatty deposits in the neck region. Furthermore, other factors, such as defective nasal breathing, epiglottis collapse, and difficulty breathing, could also precipitate OSA.

Finally, according to maxillofacial surgeons, OSA occurs primarily due to mandibular hypoplasia—a condition termed glossoptosis. Thus, according to these specialists, a better outcome can be achieved for patients with OSA than other treatment options through orthognathic surgery.

Symptoms

The main symptoms of OSA are listed in Table 1.

• Frequent loud snoring
• Family members reporting periods of apnea, choking, or gasping by the individual
• Daytime sleepiness
• Fatigue (otherwise unexplained)
• Morning-after headaches
• Poor concentration
• Irritability

Table 1: *Cardinal symptoms of OSA [2,8,9].*

Common presenting OSA symptoms include unexplained daytime sleepiness, interrupted and disturbed sleep, and loud snoring followed by intermittent phases of silence and gasping or choking.

Other less commonly occurring symptoms include morning-after headaches, difficulty falling asleep (insomnia), poor concentration, irritability, anxiety, depression, loss of memory, tachycardia, hypertension, substandard libido, unexplained weight gain, increased frequency of urination (polyuria)—especially during the nighttime (nocturia), regular attacks of heartburn (gastroesophageal reflux), and episodes of night sweats (sleep hyperhidrosis) [15–17].

In some instances, OSA symptoms appear intermittently due to underlying transitory causes, such as an upper respiratory infection or acute tonsillitis [25,26]. For example, viral infection due to the Epstein-Barr virus can increase the size of the lymphoid tissue, leading to the sudden onset of OSA in infectious mononucleosis. Furthermore, chronic and excessive alcohol intake can lead to a loss of muscle tone, precipitating OSA symptoms.

Adult and pediatric OSA

In adults, increased daytime sleepiness is considered the hallmark of OSA. The affected person may fall asleep during rest, sitting, or in severe cases, while engaged in conversation. Their partners often inform them about these symptoms. Since snoring is thought of as a stigma, women are less likely to be informed by their partners regarding such. Also, fewer women seek medical consultation on their own [15–17].

Excessive sleeping can also occur in children, although it is not considered a characteristic symptom of pediatric OSA. The relatively common symptoms of OSA among children are hyperactive or overtired behavior [25,26]. Compared to adult OSA patients, pediatric OSA patients have different body structures. Adult OSA patients are typically overweight, short in stature, with bulky necks. Young children with OSA are thinly built with difficulty to thrive. In children, OSA generally occurs due to enlarged tonsils or adenoids. Therefore, tonsillectomy and adenoidectomy can correct OSA in these patients [25,26]. Thus, tonsillectomy and adenoidectomy can correct OSA in these patients [25,26]. When OSA manifests in an overweight or obese child, their symptoms will be similar to those of adult variant OSA [27].

Obesity-associated narrowing of the airways due to the accumulation of fatty tissue in the cervical area increases the risk of pharyngeal collapse, even in children. Obesity can be compounded by OSA because of severe daytime sleepiness. In such a circumstance, people tend to increase food intake, contributing to excessive body weight [15–17,27].

According to the presence or absence of obesity in children, two types of OSA are observed: the type I variety is associated with lymphatic hypertrophy without oobesity; the type II variety is associated with obesity and mild lymphadenopathy. Specific studies have revealed that weight loss in the type II variety of childhood OSA can relieve OSA symptoms [27].

Risk factors

Commonly implicated risk factors include obesity, age, poor muscle tone, unhealthy lifestyle, genetic anomalies, craniofacial abnormalities, and postoperative complications [8–13]. The chief risk factors for OSA are listed in Table 2.

• Obesity; BMI > 35 kg/m^2
• Family history of OSA
• Retrognathia
• Down syndrome
• Craniofacial syndrome

Table 2: *Major risk factors of OSA [2,8,9].*

Obesity: Obese adults and young people are at increased risk of developing OSA due to an accumulation of fatty tissue in the neck region. However, body mass index (BMI) may remain normal [28]. Although these patients might show a significant amount of neck fat—as seen on a DEXA scan—it is posited that these patients with normal BMI are likely to have poor muscle tone that could precipitate OSA symptoms [28]. CPAP use often provides partial or complete relief from OSA symptoms, therefore suggesting that airway collapse is the main problem in OSA.

Age: Older adults often lose muscle tone and experience diminished neurological function of the upper respiratory tract [29].

Drugs, including alcohol: Alcoholic beverages and sedatives might precipitate OSA symptoms in vulnerable people [30].

Brain injury: Brain trauma can result in permanent or temporary muscle tone loss, triggering OSA symptoms [15–17].

Muscle tone: Poor muscle tone has been identified as a contributing factor to OSA [16].

Gender: Males, especially those of middle age, are at increased risk of suffering from OSA more than females of the same age group. Possible causes could be that males have more muscle mass in the torso and neck. Females in the premenopausal age group generally experience less OSA, which could be due to female hormones (especially progesterone). However, the prevalence of OSA increases in postmenopausal women and is similar to men in that age group. Furthermore, women are at increased risk of OSA during pregnancy [15–17].

Lifestyle: Certain lifestyle habits, mainly smoking, can precipitate OSA symptoms [15–17]. The chemical irritants present in cigarettes can cause inflammation of the soft tissue of the upper airway, fluid retention, and narrowing of the upper airway, thereby triggering OSA symptoms. Cigarette smoking cessation can reverse the symptoms of OSA if smoking is the only precipitating cause of OSA.

Co-morbidities: Allergy and asthma are two primary conditions associated with OSA pathogenesis.

Genetic predisposition: People with a family history of OSA are at increased risk of suffering from OSA.

Craniofacial syndromes: A recent hypothesis is that a shorter lower jaw (neoteny) along with downward displacement of the tongue or its retraction (glossoptosis) can cause OSA.

Down syndrome: In this genetic disorder (chromosome 21 trisomy), several characteristic features is might induce OSA. These characteristic features are poor muscle tone, a narrow nasopharyngeal passage, and an oversized tongue. OSA risk is much higher in those with Down syndrome than in the general population. More than 50% of patients with Down syndrome express OSA [31].

Cleft palate: In patients with cleft palate, if the nasal cavity is narrow, the open palate facilitates breathing; therefore, palatoplasty in these patients could lead to OSA symptoms [32].

Specific surgeries: Specific surgical procedures can increase OSA occurrence—the most identified surgical procedure is pharyngeal flap surgery [33].

Prognosis

Cerebrovascular stroke and cardiovascular disease are related to OSA, especially among patients under 70 years of age [34]. The risk of heart attack (myocardial infarction) increases by approximately 30% in patients with OSA compared to the general population [35]. In severe cases of OSA, heightened pulmonary pressure is transmitted to the right heart, resulting in cor pulmonale. Hypertension is another cardiovascular complication of OSA. OSA-associated hypertension has a distinguishing characteristic compared to essential hypertension. In OSA-associated hypertension, blood pressure does not fall during sleep although if it might increase.

Sleep deprivation and inadequate oxygen concentration can increase the risk of other diseases, namely myocardial infarction, hypertension, aortic aneurysm, weight gain, obesity, and death. In addition, severe sleep deprivation associated with OSA can cause deficits in reasoning, attention, learning, and memory.

Epidemiology

Senaratna., *et al*. (2017) published a systematic review on OSA prevalence among the adult population [29]. The researchers included 24 studies. According to their review, OSA prevalence in the general population was estimated to be between 9% and 38%; OSA was also reported more frequently among men than women. The researchers concluded that the OSA prevalence is much higher among obese men and women.

OSA is considered an underdiagnosed condition as it is not always associated with daytime sleepiness, and nighttime apnea or hypopnea, and thus, can go unnoticed. Some studies have reported that OSA is more likely among the Hispanic and African American populations than the Caucasian population [36]. Polysomnography performed among American adults is suggesting that 1 in 5 American adults suffer from at least a mild type of OSA.

Diagnosis

In 2017, the US Preventive Services Task Force published a systematic review, concluding that there was no certainty regarding the utility of the currently applied OSA screening tools. The researchers noted inadequate evidence on the risk-benefit ratio for OSA screening among asymptomatic adults [34].

According to the ICSD-3, there are four criteria for diagnosing OSA: these are excessive sleepiness, nonrestorative sleep, fatigue, and insomnia. However, as there is the possibility of night-to-night variability of the symptoms, the diagnosis of OSA is perplexing. Thus, in some instances, multiple tests might be required to achieve an accurate diagnosis [32].

Polysomnography (PSG) is considered the gold standard for the confirmation of OSA diagnosis. During the night in the PSG laboratory, patients are continuously monitored with EEG, pulse oximetry, temperature, blood pressure, and nasal and oral airflow. (A respiratory plethysmograph or a similar type of resistance belt is placed around the chest and abdomen of the subject being tested [15].) The apnea-hypopnea index (AHI) is measured to categorize the severity of OSA. An less than five is considered normal, and an AHI more than 30 is considered a severe form of OSA.

Measurement of oxygen saturation by pulse oximetry can be applied to identify patients who are at increased risk of suffering from OSA. However, home oximetry cannot identify apneic episodes or produce AHI scores [15].

Differential diagnosis

Differential diagnoses of OSA include Cheyne-Stokes breathing, narcolepsy, insufficient sleep, primary snoring, nocturnal panic attack, sleep-related laryngospasm, and chronic fatigue [14–16].

Treatment

There are several treatment options available for OSA as follows [15–17]:

Lifestyle modification: Cigarette smoking, alcohol, or drugs that lead to poor muscle tone should be avoided. Weight loss should be attempted through exercise and a balanced diet.

Surgical intervention: In specific patients, tonsillectomy or adenoidectomy is performed.

Physical intervention: The most critical measure in OSA management is by employing using positive airway pressure via various devices, such as CPAP, variable positive airway pressure (VPAP, also called BiPAP), nasal EPAP, and auto-CPAP.

It is often difficult to motivate people with moderate to severe OSA to use CPAP devices—these devices require a change in sleeping behavior [15,16]. Studies have shown that more than 8% of people who chose to use CPAP stop doing so after the first night, while 50% of people who use CPAP to manage moderate to severe OSA symptoms quit in the first year [15]. Increasing awareness and providing a caring and robust support system tend to increase OSA-patient compliance regarding CPAP use.

In some cases, oral appliances are recommended; however, they are not as effective as CPAP [38]. Oral appliances or splints act as a mouth-guards (similar to those used by athletes in some sports), holding the lower jaw down and forward compared to the relaxed neutral position of the jaw. Also, some OSA patients benefit from using a recliner during sleep. A recliner provides an elevation of about 30 degrees to the upper part of the body, preventing a collapse of the airway due to gravity. For OSA patients, it is also recommended to sleep on the side rather than sleep in a supine position [16,18,37,38].

Rapid palatal expansion: This mode of treatment is mainly adopted and useful in pediatric OSA, but not in adult OSA as the palatal sutures are already fused in adults. However, recently, mini-implant-assisted rapid palatal expansion (MARPE) can be used in adults as an OSA nonsurgical treatment option. The resultant beneficial changes are near permanent with minimal complications.

Surgery

Although surgery is not considered first-line treatment for adult OSA, different surgical options might benefit an OSA patient. The choice of surgery depends on an individual's severity of the symptoms and needs of the patient, such as specific anatomical and physiologial issues and the patient's preference. Several clinical trials and meta-analyses have studied the benefit of uvulopalatopharyngoplasty (UPPP) in patients whose symptoms are resistant to conservative treatment options [18,28,34].

Other than UPPP, the scientific evidence for other types of sleep surgery is relatively weak. In addition to UPPP, surgical options include the following: septoplasty, laser-assisted uvulopalatoplasty, turbinectomy, reduction of the tongue base, advancement of the genioglossus, advancement of the genioglossus maxillomandibular, advancement of the genioglossus, and advancement of the maxillomandibular.

In morbidly obese patients where obesity is the leading cause of OSA, bariatric surgery can be attempted if other weight-loss measures fail.

Pediatric typically results from enlarged tonsils and adenoids. In these patients, tonsillectomy and adenoidectomy are the first lines of treatment options. However, in some countries, only the protruding tonsillar section is removed. to avoid complications associated with the complete removal of the tonsils [25].

Neurostimulation

In 2014, the US Food and Drug Administration (USFDA) approved neurostimulation—in the form of upper airway stimulation—in patients adverse to CPAP. This upper airway stimulation system can sense episodes of apnea or hypopnea and sends mild electrical stimulation to the hypoglossal nerves to improve muscle tone so that the tongue does not fall rearward into the airway. The device comes with a hand-held controller, which the patient can use to activate the device before sleep [34,38].

Radiofrequency ablation (RFA)

Radiofrequency ablation (RFA) delivers low frequency (300 kHz to 1 MHz) radio waves to reduce enlarged neck tissues. Although the American Academy of Otolaryngology recognizes its safety and efficacy in OSA management in mild to moderate cases of OSA, there is meager scientific evidence for the adoption of this method for routine use in OSA [34,38].

RFA is typically performed in the outpatient setting as it takes less than three minutes. The target tissue, like the tongue or the palate, is approached via the oral cavity. However, in certain patients, the approach is through assisted imaging techniques. Complications are rare (less than 1%) but can include ulceration, infection, numbness, and localized swelling.

Pharmacotherapy

Scientific evidence regarding OSA pharmacotherapy is sparse. Nevertheless, certain drugs—such as fluoxetine, paroxtine,and acetazolamide—show preliminary promise in symptom management [34,38]. Currently, the role of cannabinoids OSA management is under investigation. However, there have been no consistent findings to date. Therefore, more studies are needed in this regard [15,16]. Treatment of OSA is usually performed by sleep specialists, pulmonologists, neurologists, and in some instances by maxillofacial surgeons.

Conclusion

Although the diagnosis of OSA is somewhat complicated, OSA-affected patients experience characteristic symptoms such as snoring, awakening, sudden choking, gasping, waking up, and falling asleep again. The start of OSA treatment should not be delayed as long-term untreated OSA can lead to several life-threatening conditions, such as cerebrovascular accidents and myocardial infarction. CPAP use during bedtime remains the gold standard of treatment in adult OSA, while in pediatric OSA, tonsillectomy or adenoidectomy may be sufficient to alleviate symptoms.

Conflict of Interest Statement

The authors declare that this paper was written without any commercial or financial relationship that could be construed as a potential conflict of interest.

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