

Chapter

Evaluation and Management in Patients with Obstructive Sleep Apnea

Yuliia Dieieva and Oleksandr Naumenko

Abstract

The theme of conservative and surgical management of obstructive sleep apnea syndrome (OSAS) revolves around the various approaches and strategies used to treat this common sleep disorder. OSAS is a sleep disorder characterized by breathing pauses due to upper airway obstruction. The treatment of OSAS involves conservative and surgical approach. Conservative management uses non-invasive techniques and lifestyle changes, such as weight loss, positional therapy, and CPAP. Surgical management corrects anatomical abnormalities causing the obstruction, including uvulopharyngopalatoplasty and other upper airway surgeries. Conservative management is usually the first treatment, while surgery is for severe or refractory OSAS. In this chapter, we provide information about possible options for OSAS management and treatment.

Keywords: obstructive sleep apnea, polysomnography, uvulopharyngopalatoplasty, OSAS, snoring

1. Introduction

Obstructive Sleep Apnea Syndrome (OSAS) manifests as a recurring series of occurrences wherein the upper airway undergoes episodic collapse and obstruction during sleep, giving rise to arousals, often accompanied by oxygen desaturations. This intricate interplay results in the fragmentation of sleep patterns.

Symptomatology associated with OSAS is diverse, reflecting individual variability in presentation. Snoring, recognized as a prominent hallmark, underscores the turbulent airflow dynamics characteristic of the condition during sleep cycles. In the wakeful domain, daytime symptoms of OSAS encompass an array of manifestations. Chief among these is excessive daytime sleepiness, defined by periods of pronounced drowsiness and somnolence. Distinct from this is the sensation of fatigue, indicative of a broader sense of exhaustion, depleted vigor, and a lack of motivation. Pertinently, individuals may endure a feeling of non-rejuvenation despite adhering to recommended sleep durations, thereby punctuating the multifaceted symptomatology of OSAS [1].

Snoring is a specific process accompanying the act of breathing, mainly during sleep, and manifests as a characteristic low-frequency vibrating sound. According to statistical data, approximately 20% of men and 5% of women suffer from snoring at the age of 30. These numbers increase significantly with age, and by the age of 60, about 60% of men and 40% of women face this issue. Snoring can indicate a potential obstructive sleep apnea syndrome or be its precursor.

Among the factors contributing to the risk of developing OSAS, the following can be highlighted: gender (men are affected 4–6 times more often than women), age (as mentioned earlier, the prevalence increases after the age of 30), weight (including neck circumference and lipid deposition characteristics), genetic and ethnic factors (individuals of African and Latino populations are more prone to OSAS than those of European descent; in Japanese individuals, the correlation between obesity and apnea is less pronounced; genetically determined anatomical characteristics of the lower jaw; presence of chromosomal defects, etc.), alcohol consumption or use of sedative and hypnotic drugs, smoking, certain medical conditions, such as adenotonsillar hypertrophy, acromegaly, stroke, myopathy, Parkinsonism, and others [2, 3].

During the study of snoring and the apnea syndrome, various hypotheses have been proposed regarding their development mechanisms. Initially, snoring was perceived as a natural phenomenon associated with healthy sleep rather than a medical anomaly. However, further analysis revealed that snoring is characteristic of individuals with specific body structures and can be accompanied by pathological symptoms. Subsequent research indicated that snoring can occur not only in individuals with excessive weight but also in people with a normal body mass index.

Regarding the mechanism of OSAS development, two main types are recognized: central, associated with central nervous system activity disturbances, and peripheral (obstructive), which arises due to obstruction of the airways.

Central origin apnea syndrome accounts for less than 10% of cases and is often linked to complex neurological disorders. As for obstructive apnea syndrome, it develops due to the relaxation of throat and soft palate muscles during sleep, leading to the obstruction of the upper airways by the tongue or other throat and larynx tissues. This results in blocked air passages, and the diaphragm continues to contract, creating negative pressure in the airways, exacerbating the obstruction. This “vicious cycle” can lead to oxygen deficiency and increased carbon dioxide levels in the blood, resulting in hypoxia and subsequent awakening [4].

Minin Y.V. and colleagues developed a classification system for upper airway obstruction. They divided this syndrome into four stages based on the severity of obstruction symptoms [5].

- The first stage is characterized by snoring that typically occurs after alcohol consumption or the use of muscle relaxants while lying on the back.
- The second stage involves persistent snoring in both the supine and lateral positions, with normal oxygen hemoglobin levels.
- The third stage shows compromised blood oxygenation during sleep and constant snoring in any position, affecting the patient’s psychological state.
- The fourth stage is marked by constant snoring, obstructive apnea during sleep, and alterations in other organs and systems, particularly the cardiovascular system.

Developers divided upper airway narrowing mechanisms into functional (resulting from physiological changes) and organic (caused by local anatomical changes). Depending on the level of obstruction, narrowing can occur in the nasal cavity, nasopharynx, oropharynx, hypopharynx, or simultaneously affect multiple segments [5].

Frequent obstruction episodes lead to sleep disruption and fragmentation, insufficient blood oxygenation, and result in clinical manifestations such as daytime sleepiness, reduced attention and productivity, alterations in the function of internal organs and systems, and an increased risk of developing or exacerbating conditions like arterial hypertension, ischemic heart disease, arrhythmias, heart failure, stroke, and diabetes [6].

Episodes of apnea lasting over 10 seconds, occurring frequently (10 times or more per hour), hold particular importance. Some of these conditions can worsen the course of apnea syndrome, especially in the presence of obesity.

Recent research indicates that the mechanism of developing arterial hypertension and cardiovascular diseases involves sympathetic nervous system activation due to insufficient oxygen saturation. Additionally, there is a connection between leptin levels in the blood, associated with systolic arterial pressure, and insulin and C-reactive protein levels. Studies suggest that patients with obstructive apnea have elevated leptin levels, which increase with the severity of obstruction [7].

The relationship between obesity and OSAS is reciprocal [8]. Observations of middle-aged men have shown a connection between OSAS and acquired obesity. On the one hand, patients with excess weight often experience breathing disturbances during sleep. On the other hand, individuals with OSAS experience rapid weight gain, with fat accumulating in the waist and neck regions [9].

In the general population, individuals with OSAS are more likely to have excess weight (about 32% more often, on average) and insomnia. Sleep disturbances can lead to various problems such as irritability, mental disorders, and depression, complicating weight loss efforts in cases of obesity. Some studies indicate that OSAS treatment with CPAP therapy can improve psychological well-being and has a limited impact on body parameters.

An additional contributory mechanism in the genesis of metabolic perturbations within the context of obstructive sleep apnea syndrome is the modulatory impact of sleep and its temporal patterns on the endocrine glands. Notably, disturbances in the profound sleep phase have been associated with a surge in cortisol levels and the modulation of hypothalamo-pituitary-adrenal (HPA) axis activity, thereby imparting alterations in leptin, an integral hormone governing appetite regulation [10].

Furthermore, erectile dysfunction (ED) afflicts over half of OSAS patients [11]. By employing polysomnographic scrutiny, we can predict the risk for ED, especially in cases of moderate to severe OSAS where a higher predisposition is observed [12].

Among other symptoms of OSAS, it is worth mentioning nocturia and reports of nocturnal ptyalism [13]. Moreover, the influence of OSAS on the immune system has been a possible way of investigation [14].

It is notable that a significant proportion of OSAS patients resort to sleeping in separate beds from their partners [15].

2. Diagnostic measurements

In the diagnostic realm, polysomnography and cardiorespiratory monitoring persist as the most efficacious methodologies for OSAS assessment. Alternative modalities encompass sleep bio-radar monitoring [16].

Critical diagnostic parameters encompass the tally of apneic and hypopneic episodes, the apnea-hypopnea index (AHI) [17].

In the context of ascertaining the imperative for orthodontic intervention, the application of cephalometric diagnostic methodologies proves instrumental in scrutinizing craniofacial dimorphism within cohorts manifesting obstructive sleep apnea syndrome, precipitated by dysmorphic attributes. Notably, during pharmaceutically induced somnolence, the feasibility of executing upper airway endoscopy surfaces for the purpose of pinpointing obstructions spanning disparate tiers of the upper airways, all within settings that closely approximate the natural somnolent state [18].

In the intricate landscape where metabolic disturbances intersect with obstructive sleep apnea syndrome, it becomes evident that the disruption of sleep architecture reverberates beyond hormonal modulation to intricately intertwine with metabolic processes. The attenuation of slow-wave sleep (SWS) and rapid eye movement (REM) sleep, pivotal in orchestrating energy equilibrium and glucose metabolism, assumes a crucial role in the risk of insulin resistance and metabolic perturbations. This postulate finds empirical reinforcement through investigations revealing anomalous glucose metabolism, compromised insulin sensitivity, and perturbed lipid profiles among OSAS-affected individuals. This interplay between sleep architecture modifications and hormonal dysregulation underscores OSAS's multi-layered influence on metabolic dynamics [19].

The burgeoning body of evidence unfolds the adverse implications of OSAS on cardiac structure and function, accompanied by an elevated susceptibility to hypertension, atrial fibrillation, and myocardial infarction. Chronic intermittent hypoxia, a cardinal feature of OSAS, is posited to instigate oxidative stress and inflammation, culminating in endothelial dysfunction and consequent cardiovascular sequelae. It is increasingly clear that OSAS transcends the confines of isolated organ systems, evolving into a systemic ailment with repercussions spanning metabolic, cardiovascular, and neurobiological domains [20].

In the clinical domain, while polysomnography persists as the gold standard for OSAS diagnosis, technological advancements have engendered innovative modalities. Portable sleep monitoring devices, wearable sensors, and computational algorithms offer promising avenues for swift and cost-effective screening and continuous surveillance of OSAS, both in clinical settings and the comfort of one's home [21]. This paradigm shift toward accessible and uninterrupted monitoring holds significant promise in bolstering early detection and the customization of therapeutic strategies.

As the complicated pathogenesis of OSAS continues to unfold, the important call for interdisciplinary cooperation reverberates more compellingly than ever. The amalgamation of expertise spanning pulmonology, otolaryngology, sleep medicine, endocrinology, cardiology, and biomedical engineering holds the potential to cultivate a comprehensive understanding of OSAS. This, in turn, catalyzes the development of interventions that target not only the symptomatic facets but also the foundational mechanistic intricacies underpinning this complex syndrome.

Summarizing the aforementioned research methods, we have compiled them into **Table 1**, which provides a brief overview of each method and its purpose.

It is worth noting that one should not disregard the patient examination data, as the diagnostic value of this examination method remains quite significant. During the examination, attention should be paid to the patient's general characteristics, nasal and nasal cavity features, throat, larynx, and neck. Below, in **Table 2**, summarized information about possible diagnostic findings during an otolaryngologist's examination is provided.

Step	Diagnostic measure	Indication
1	Medical history and patient questionnaire	Identify symptoms, complaints, and risk factors, genetics predisposing
2	Otolaryngological examination	Evaluate anatomical aspects of upper airways
3	Biometric measurement	Determine body metrics, including body mass index
4	CT (MRI) scan	In case of nasal anatomical abnormalities due to ENT examination
5	Cardiorespiratory monitoring	Detect potential heart rhythm and breathing issues at home during sleep
6	Polysomnography	Establish presence and severity of apnea episodes in a hospital
7	Sleep video endoscopy	Detail level of obstruction aspects during sleep

Table 1.

Diagnostic measure of OSAS.

General physical characteristics	Obesity
	Skeletal/craniofacial abnormalities
Neck	Enlarged neck circumference
Nose/nasal cavity	External nasal deformity
	Septal deviation
	Turbinate hypertrophy
	Mucosal hypertrophy
Pharynx	Nasal polyps or any pathological discharge
	Tongue abnormalities
	Palatal/uvular elongation
	Tonsillar hypertrophy
Larynx	Lateral wall collapse
	Epiglottis characteristics (shape, form factor, etc.)
	Glossoptosis
	True vocal cord function
	Arytenoid location/dislocation

Table 2.

Diagnostic findings during otolaryngologist's examination.

3. Management of patients with obstructive sleep apnea

In contemporary medical practice, the therapeutic management of obstructive sleep apnea can be categorized into two primary domains: conservative interventions and surgical approaches.

Within the realm of conservative interventions, the Continuous Positive Airways Pressure (CPAP) therapy stands as a widely embraced strategy. Conceived and introduced in 1981 by Australian Scholar Professor Colin Sullivan, CPAP therapy

involves the administration of a constant stream of air through a nasal mask, tethered by a flexible tube to an air compressor. This method effectively prevents the collapse of the upper airway during sleep, maintaining adequate oxygen levels, reducing apnea episodes, decreasing sleep fragmentation, and fostering psychological and physiological well-being. This encompasses factors like weight reduction, diminished depressive symptoms, reduced somnolence, and ameliorated cardiovascular, and endocrine function. The substantiation of CPAP's effectiveness rests on a body of evidence comprising subjective reports and objective polysomnographic data, particularly in cases of sustained and consistent use [22, 23].

Nonetheless, CPAP therapy is not without its limitations. The financial burden of acquiring high-quality equipment, patient resistance to mask use, social discomfort, oral and nasal dryness, and skin irritation due to mask usage are noteworthy constraints [24]. Moreover, the supposed weight-reduction benefits of CPAP therapy have been met with skepticism in some investigations [24].

Alternative solutions involve intraoral devices, primarily suitable for mild to moderate OSAS cases. These devices operate by stimulating reflexive muscle contractions in the tongue and oropharynx, augmenting their tonal resilience and mitigating vibrational tendencies. Similarly, other devices advance the mandible, thereby expanding the upper airway dimensions within the oropharynx. For pediatric cases attributed to anatomical snoring causes, specialized caps are employed to stimulate mandibular growth and enhance the tonicity of oropharyngeal muscles and the tongue.

It is vital to note that the mitigation of snoring does not necessarily correlate with an equivalent reduction in the severity of obstructive apneic events during sleep. Thus, when considering intraoral interventions, a systematic evaluation of treatment efficacy is paramount. In cases of moderate to severe OSAS, recourse to more efficacious therapeutic modalities is advisable [25].

Behavioral interventions focus on addressing modifiable risk factors contributing to OSAS. These interventions primarily revolve around optimizing metabolic profiles through weight management, achieved via personalized dietary interventions and increased physical activity. Notably, a weight loss ranging from 10 to 17% has demonstrated a significant reduction in apnea/hypopnea indices, substantiating this approach [26, 27].

Smoking cessation, moderation in alcohol consumption, and abstaining from sedative agents have also exhibited a contributory role in attenuating apnea occurrences [28].

Pharmacotherapy pursuits have explored the "Good night" preparation as a noteworthy example. This preparation incorporates essential oils that, when conveyed through nasal and oral conduits, induce reflexive enhancement in the contractility of pharyngeal dilator muscles [29].

Various pharmacological classes (serotonin receptor antagonists, progesterone derivatives, methylxanthines, and others) have undergone scrutiny; however, the conservative therapy for OSAS remains constrained due to a dearth of medication validation and discernible clinical impact [30, 31].

Distinct structural regimens targeting the oropharyngeal musculature and soft palate have been devised to counteract collapsibility tendencies during sleep [32].

Uvulopalatopharyngoplasty (UPPP) presently stands as the quintessential surgical intervention for OSAS. Initiated by T. Ikematsu in Japan in 1952, UPPP involves a wedge-like resection of the mucosal lining of the posterior soft palate contiguous to the palatine tonsil base. Subsequent steps encompass excision of the

intervening mucosa between the anterior and posterior palatal arches, followed by sutural reapproximation utilizing nodal sutures, and a minor partial resection of the uvula [33].

Further expanding on this groundwork, S. Fujita advocated for an augmented version termed uvulopalatopharyngoplasty to optimally enhance oropharyngeal expansiveness. Based on research comprising 66 subjects, disseminated in 1985, a substantial 76% reported notable subjective enhancements following the procedure [34, 35].

UPPP is particularly endorsed in instances where superfluous soft tissue within the oral cavity prevails, when CPAP proves ineffective, or when patient preference diverges from continual positive airway pressure adherence. Notably, UPPP is usually sidestepped in pediatric cohorts. In the domain of severe affliction, UPPP is often subsumed within a comprehensive therapeutic regimen. While UPPP can lead to a cessation of snoring, its capacity to wholly ameliorate the syndrome of nocturnal apnea remains circumscribed. Empirical investigations elucidate a 40 to 60% enduring efficacy rate for UPPP, prompting a judicious approach considering its potential drawbacks [34].

UPPP is a widely accepted and generally safe surgical approach for managing OSAS. Regrettably, UPPP does not consistently maintain its initial success, and some patients who initially saw improvements in their OSAS severity may experience relapses. While UPPP is recognized for its safety and established use, its long-term effectiveness tends to decrease over time. Success rates decline as patients move beyond the immediate postoperative period, especially when factors like an increase in Body Mass Index play a role. This highlights the importance of continuous monitoring and the potential consideration of additional surgical interventions that target specific areas of the airway to comprehensively address instances where UPPP does not achieve the desired outcome [36].

Furthermore, a common observation is the resurgence of snoring and apneic episodes with regained body mass, negating the efficacy of UPPP in this context. Despite a body of scientific studies suggesting substantial UPPP efficacy even in long-term observations extending beyond a year post-intervention, the limitations of the procedure remain evident [37].

A significant development during the 1980s was the introduction of a surgical method employing laser technology for soft palate interventions, offering advantages such as shorter postoperative recovery periods, increased surgical precision, reduced blood loss, and fewer postoperative complications. However, it is pertinent to acknowledge the technique's drawbacks, particularly the larger necrotic zone compared to classical scalpel interventions [38].

In the mid-1990s, the utilization of radiofrequency thermoreduction (RFTR) gained traction; nevertheless, its widespread adoption was hampered by its limited effectiveness in moderate to severe cases of obstructive sleep apnea syndrome [39, 40].

Beyond traditional UPPP methods, contemporary practice explores less invasive techniques, notably those involving microdebriders for excising small portions of the soft palate through minimal incisions [17].

Worthy of note is the comparatively less invasive nature of cold plasma or electrocautery-assisted UPPP. Furthermore, the integration of coblation technology is progressively making headway in modern otolaryngological surgery [41].

A notable study conducted in Brazil compared standard UPPP to lateral pharyngoplasty, incorporating Z-plasty, for preventing posterior soft palate collapse. Results

indicated a clear superiority of lateral pharyngoplasty over UPPP, characterized by a reduction in the apnea-hypopnea index from 41.2 to 9.5, substantial reduction in daytime sleepiness and snoring, improved overall patient condition, and an extension in deep sleep duration [42].

In a notable instance, in 2012, Kolyadych et al. evaluated the effectiveness of treating OSAS using palate implants. This approach induced inflammation and fibrous capsule formation, leading to increased soft palate rigidity and dampening vibrational fluctuations during breathing. This method exhibited a minimally invasive profile, culminating in a 67.7% reduction in snoring intensity and notable alleviation of daytime sleepiness [43].

Additionally, surgical interventions for obesity, including the excision of fat deposits in the neck region, are also pertinent within the therapeutic spectrum [44].

Historically, in the 1970s, tracheostomy emerged as a common recourse for managing OSAS. Presently, it is reserved for severe cases refractory to alternative interventions [45].

In cases of upper airway obstruction, remedial strategies encompass septoplasty, turbinectomy, and uvulopalatopharyngoplasty tailored to address the underlying causative factors.

Curiously, despite the array of therapeutic options for managing obstructive sleep apnea syndrome, the challenge remains due to the modest efficacy, often hovering around 60% when scrutinized against objective benchmarks. Consequently, the exploration of innovative surgical ways for enhancing OSAS management remains an imperative trajectory for ongoing research endeavors.

Considering a range of factors in the pathogenesis of obstructive sleep apnea syndrome, the approach to treatment should be personalized and comprehensive, involving collaborative efforts of interdisciplinary specialists. Treatment options may encompass both conservative and surgical approaches, which can be employed concurrently. According to our research and practice, in the majority of cases, emphasis should be placed on weight reduction and surgical interventions, specifically nasal cavity surgeries (septoplasty, turbinate reduction, polypectomy, adenoidectomy), as well as pharyngeal surgeries, including soft palate procedures. However, the AHI should be taken into account, along with the benefit-risk assessment of such interventions. To facilitate a better understanding of the indications for potential treatment, an infographic in the form of **Figure 1** is provided below.

The most common and typical surgical intervention for obstructive sleep apnea syndrome is uvulopalatopharyngoplasty [46]. The traditional uvulopalatopharyngoplasty procedure was carried out with the patient under endotracheal anesthesia. The surgeon made an incision in the soft palate along its free edge, about 7–10 mm away from the front arch, going through the base of the uvula and continuing to the other side. At the same time, they performed a bilateral tonsillectomy from the back, regardless of whether the patient had chronic tonsillitis or not. The front arches were removed along with the tonsils, while the back arches were kept for later shaping of the side of the throat. To make it easier to stitch the mucous membranes of the soft palate after partially removing a section, the nasal side was left a bit longer than the oral side by about 6–8 mm. The stitches on the edge of the soft palate were done using a gentle needle, spaced about 1–1.5 cm apart, with special thread.

To provide better results, one of the possible approaches is removing a rectangular strip of mucous membrane along with a layer beneath it from the soft palate, all

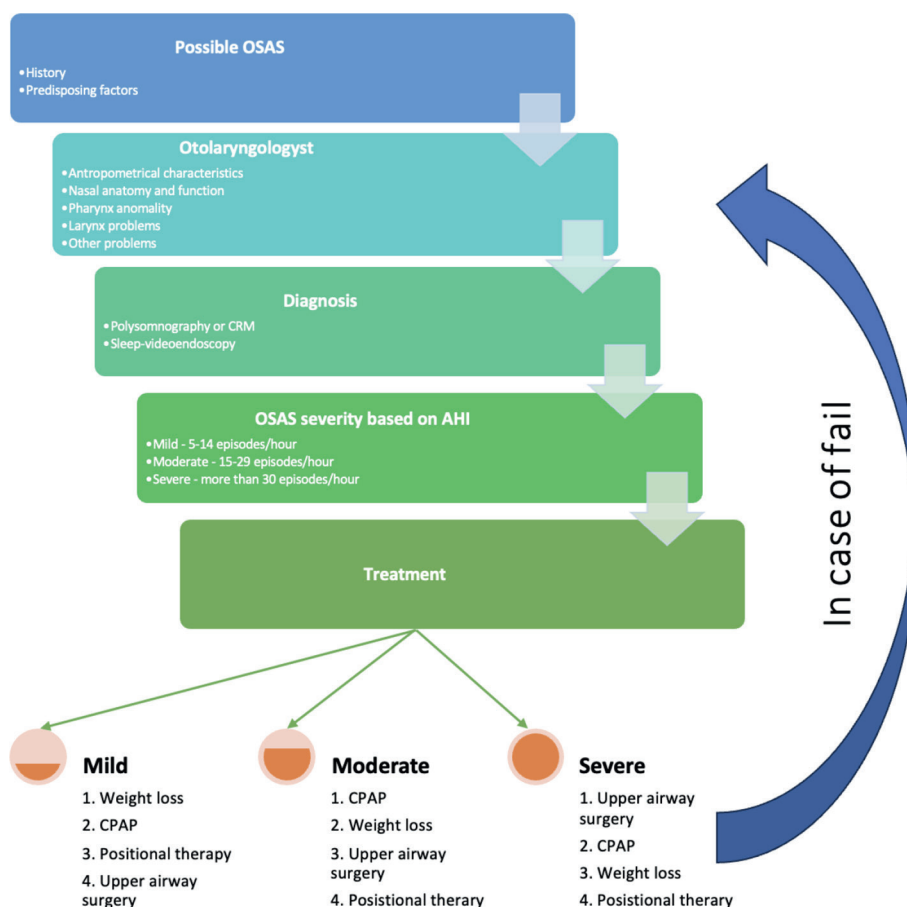


Figure 1.
 Management of OSAS.

without harming the muscles of the soft palate. Then, we can use a special V-Loc™ 180 thread made of poliglecaprone to stitch three times. This thread had circular notches that helped securely fasten the soft palate to the fibrous ring of the hard palate using P-shaped stitches. This technique prevented the stitches from pulling apart during talking or swallowing. Over the course of 120 days, as the thread gradually dissolved, scar tissue formed at the site of these stitches. This scar tissue provided extra stability and reduced vibrations in the soft palate as the patient healed in the weeks and months following the surgery. This technique, as illustrated in **Figure 2**, can be used like first-line surgery [47].

Additionally, **Table 3** displays a range of sleep surgery procedures commonly employed to address obstructive sleep apnea. Ahead of undergoing surgery, it is recommended, according to the guidelines set by the American Academy of Sleep Medicine, that patients eligible for surgical options should receive counseling regarding the potential complications and success rates associated with relevant surgical methods. Traditionally, the success of these surgeries has been gauged by using the apnea-hypopnea index as the main metric. However, there is a growing trend toward incorporating patient-centered outcomes like sleepiness, quality of life, and improvement in other concurrent health issues [48].

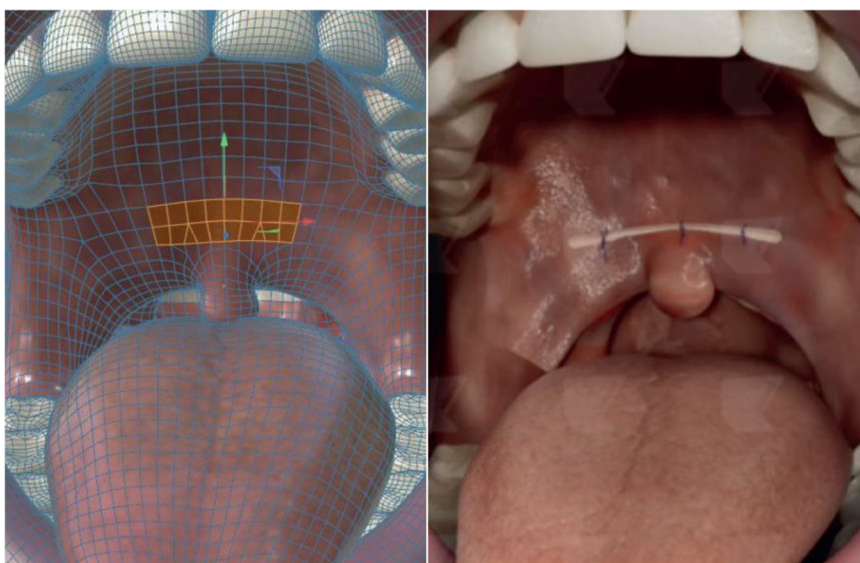


Figure 2. UPPP operative technique. On the left image illustrated possible mucous membrane removing zone and, on the right is a final result with suturing.

Nasal surgery	Septoplasty
	Turbinoplasty
	Polypectomies
	Rhinoplasty
Oral/palatal	UPPP and their modifications (laser-, cold plasm-, radiofrequency induced)
	Tonsillectomy
Hypopharyngeal	Radiofrequency ablation of the tongue
	Midline glossectomy
	Lingual tonsillectomy
	Hyoid suspension
Other	Maxillofacial surgery
	Hypoglossal nerve stimulation

Table 3. Surgical measurements for OSAS treatment – upper airway surgery.

4. Conclusion

In conclusion, the assessment and management of patients grappling with obstructive sleep apnea syndrome epitomize a multidimensional challenge that warrants a nuanced and personalized approach. OSAS, characterized by recurrent upper airway collapse during sleep, underscores the intricate tapestry of considerations in diagnosis and treatment.

In the diagnostic realm, a comprehensive evaluation regimen involving polysomnography and home sleep apnea tests unravels the intricacies of OSAS severity and

phenotypic expression. Augmented by anatomical insights via methodologies such as drug-induced sleep endoscopy, the diagnostic phase unveils the unique facets of each patient's condition.

The therapeutic landscape encompasses an array of interventions, encompassing lifestyle adaptations, intermediate measures such as continuous positive airway pressure therapy and oral appliances, and the burgeoning domain of sleep surgery. This surgical avenue, ranging from nasal interventions to oropharyngeal procedures, provides a tailored arsenal to address the intricate mosaic of anatomical and functional complexities.

Nonetheless, a holistic perspective is essential, recognizing the multifaceted nature of OSAS. Embracing patient education, managing comorbidities, and fostering adherence to therapeutic modalities are integral tenets. As the realm of OSAS continues to unravel its intricacies, an individual-centric strategy, harmonized with empirically grounded practices, underscores the bedrock of effective management.

In the ultimate analysis, the evaluation and management of OSAS mandate a collaboration between healthcare practitioners and patients, epitomizing the synergy of clinical acumen and the patient's lived experience. As we voyage deeper into the annals of understanding and technological innovation, the optimization of OSAS management holds the potential to elevate the quality of life for those ensnared by this sleeping disease.

Conflict of interest


The authors declare no conflict of interest.

Author details

Yuliia Dieieva* and Oleksandr Naumenko
Bogomolets National Medical University, Kyiv, Ukraine

*Address all correspondence to: deevanmu@gmail.com

IntechOpen

© 2023 The Author(s). Licensee IntechOpen. This chapter is distributed under the terms of the Creative Commons Attribution License (<http://creativecommons.org/licenses/by/3.0>), which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited. 

References

- [1] Rundo JV. Obstructive sleep apnea basics. *Cleveland Clinic Journal of Medicine*. 2019;**86**(9 Suppl 1):2-9. DOI: 10.3949/ccjm.86.s1.02
- [2] Redline S, Kirchner HL, Quan SF, et al. The effects of age, sex, ethnicity, and sleep-disordered breathing on sleep architecture. *Archives of Internal Medicine*. 2004;**164**(4):406-418
- [3] Palmer LJ, Buxbau SG, Larkin EK, et al. A whole genome scan for obstructive sleep Apnea and obesity in African-American families. *American Journal of Respiratory Critical Care Medicine*. 2004;**169**(12):1314-1321
- [4] Tan SN, Abdullah B. Phenotypes of obstructive sleep apnea and direct targeted therapy: A literature review. *Current Respiratory Medicine Reviews*. 2020;**16**:76-83
- [5] Minin YV. Clinic, diagnosis and surgical treatment of snoring and obstructive sleep apnea: Abstract. Dissertation work for MD, specialty: 14.00.04 / Minin Yuri Viktorovich. Kyiv. 1994. pp. 1-41
- [6] The Health Institute (THI), International Resource Center (IRC) for Health Care Assessment. Scoring Exercise for the MOS SF-36 Health Survey. Boston, MA: MOS Trust; 1992
- [7] Somers VK, Dyken ME, Clary MP, Abboud FM. Sympathetic neural mechanisms in obstructive sleep apnea. *The Journal of Clinical Investigation*. Oct 1995;**96**(4):1897-1904. DOI: 10.1172/JCI118235
- [8] Janson C, Lindberg E, Gislason T, Elmasry A, Boman G. Insomnia in men-a-10-year prospective population based study. *Sleep*. 2011;**24**(4):425-430
- [9] Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: A population health perspective. *American Journal of Respiratory and Critical Care Medicine*. 1 May 2002;**165**(9):1217-1239. DOI: 10.1164/rccm.2109080. PMID: 11991871
- [10] Singareddy R, Vgontzas AN, Fernandez-Mendoza J, Liao D, Calhoun S, Shaffer ML, et al. Risk factors for incident chronic insomnia A general population prospective study. *Sleeping Medicine*. 2012;**13**(4):346-353
- [11] Gurbuz C, Okur H, Demir S, Ordu S, Çaşkurlu T. Pure obstructive sleep apnea syndrome and erectile dysfunction. *Balkan Medical Journal*. 2011;**2011**:435-439. DOI: 10.5152/BALKANMEDJ.2011.011
- [12] Margel D, Cohen M, Livne P, Pillar G. Severe, but not mild, obstructive sleep apnea syndrome is associated with erectile dysfunction. *Urology*. 2004;**63**(3):545-549. DOI: 10.1016/J.UROLOGY.2003.10.016
- [13] Wimms A, Woehrle H, Ketheeswaran S, Ramanan D, Armitstead J. Obstructive sleep apnea in women: Specific issues and interventions. *BioMed Research International*. 2016;**2016**:1764837. DOI: 10.1155/2016/1764837. Epub 2016 Sep 6. P 1-9
- [14] Bollinger T, Bollinger A, Skrum L, et al. Sleep-dependent activity of T cells and regulatory T cells. *Clinical and Experimental Immunology*. 2008;**155**(2):231-238

- [15] Doherty L, Kiely J, Lawless G, McNicholas W. Impact of nasal continuous positive airway pressure therapy on the quality of life of bed partners of patients with obstructive sleep apnea syndrome. *Chest*. 2003;**124**(6):2209-2214. DOI: 10.1378/CHEST.124.6.2209
- [16] Kushida CA, Littner MR, Morgenthaler T, et al. Practice parameters for the indications for polysomnography and related procedures: An update for 2005. *Sleep*. 2005;**28**(4):499-521
- [17] Aittokallio T, Saaresranta T, Polo-Kantola P, et al. Analysis of inspiratory flow shapes in patients with partial upper-airway obstruction during sleep. *Chest*. 2001;**119**(1):37-44
- [18] Andersson L, Brattström V. Cephalometric analysis of permanently snoring patients with and without obstructive sleep apnea syndrome. *International Journal of Oral and Maxillofacial Surgery*. 1991;**20**(3):159-162. DOI: 10.1016/S0901-5027(05)80007-4
- [19] Vyazovskiy V, Riedner B, Cirelli C, Tononi G. Sleep homeostasis and cortical synchronization: II. A local field potential study of sleep slow waves in the rat. *Sleep*. 2007;**30** 12:1631-1642. DOI: 10.1093/SLEEP/30.12.1631
- [20] Wang J, Yu W, Gao M, Zhang F, Gu C, Yu Y, et al. Impact of obstructive sleep apnea syndrome on endothelial function, arterial stiffening, and serum inflammatory markers: An updated meta-analysis and metaregression of 18 studies. *Journal of the American Heart Association: Cardiovascular and Cerebrovascular Disease*. 2015;**4**:1-18. DOI: 10.1161/JAHA.115.002454
- [21] Jiang P, Zhu R. Dual tri-axis accelerometers for monitoring physiological parameters of human body in sleep. *IEEE Sensors*. 2016;**2016**:1-3. DOI: 10.1109/ICSENS.2016.7808735
- [22] Oslen S, Smith S, Oei T. Adherence to continuous positive airway pressure therapy in obstructive sleep apnoea sufferers: A theoretical approach to treatment. *Clinical Psychology Review*. 2008;**28**:1355-1371
- [23] Sarrell EM, Chomsky O, Shechter D. Treatment compliance with continuous positive airway pressure device among adults with obstructive sleep apnea (OSA): How many adhere to treatment? *Harefuah*. 2013;**152**(3):140-144
- [24] Broström A, Nilssen P, Johansson P, Ulander M, Strömberg A, Svanborg E, et al. Putative facilitators and barriers for adherence to CPAP treatment in patients with obstructive sleep apnea syndrome: A qualitative content analysis. *Sleep Medicine*. 2010;**11**(2):126-130. DOI: 10.1016/j.sleep.2009.04.010
- [25] Pevernagie D, Aarts R, Meyer M. The acoustics of snoring. *Sleep Medicine Reviews*. 2010;**14**(2):131-144. DOI: 10.1016/j.smrv.2009.06.002
- [26] Strobel RJ, Rosen RC. Obesity and weight loss in obstructive sleep apnea: A critical review. *Sleep*. 1996;**19**:104-115
- [27] Malhotra A, White DP. Obstructive sleep apnoea. *Lancet*. 2002;**360**:237-245
- [28] Javaheri S, Shukla R, Wexler L. Association of smoking, sleep apnea, and plasma alkalosis with nocturnal ventricular arrhythmias in men with systolic heart failure. *Chest*. 2012;**141**(6):1449-1456. DOI: 10.1378/chest.11-1724
- [29] Kuna S, Sant' Ambrogio FB, Sant' AG. Effect of airway- surface liquid

composition on laryngeal muscle activation. *Sleep*. 1996;**19**(10):180-183

[30] Atwood CW, Strollo PJ, Givelber R. Medical therapy for obstructive sleep apnea. In: Kryger MH, Roth T, Dement WC, editors. *Principles and Practice of Sleep Medicine*. Philadelphia, PA, USA: W.B. Saunders; 2011

[31] Smith I, Lasserson TJ, Wright J. Drug therapy for obstructive sleep apnea in adults. *Cochrane Database of Systematic Reviews*. 2006;**19**(2):CD003002

[32] Azarbarzin A, Sands S, Taranto-Montemurro L, Marques M, Genta P, Edwards B, et al. Estimation of pharyngeal collapsibility during sleep by peak inspiratory airflow. *Sleep*. 2017;**40**:1-11. DOI: 10.1093/sleep/zsw005

[33] Ikematsu T. Clinical study of snoring for the past 30 years. In: Ikematsu T, editor. *New Dimensions in Otorhinolaryngology – Head and Neck Surgery*. Vol. 1. North Holland: Elsevier Science Publishers, B.V; 1985. pp. 199-202

[34] Fujita S et al. Evolution of the effectiveness of uvulopalatopharyngoplasty. *Laryngoscope*. 1985;**95**:70-74

[35] Fujita S et al. Surgical correction of anatomic abnormalities in OSAS: Uvulopalatopharyngoplasty. *Otolaryngology Head and Neck Surgery*. 1981;**89**:923-934

[36] Sheen D, Abdulateef S. Uvulopalatopharyngoplasty. *Oral and Maxillofacial Surgery Clinics of North America*. 2021;**33**(2):295-303. DOI: 10.1016/j.coms.2021.01.001

[37] Janson C, Hillerdal G, Larsson L, Hultcrantz E, Lindholm C,

Bengtsson H, et al. Excessive daytime sleepiness and fatigue in nonapnoeic snorers: Improvement after UPPP. *The European Respiratory Journal*. 1994;**7**(5):845-849. DOI: 10.1183/09031936.94.07050845

[38] Berger G, Finkelstein Y, Ophir D. Histopathologic changes of the soft palate after laser-assisted uvulopalatoplasty. *Archives of Otolaryngology – Head and Neck Surgery*. 1999;**125**:786-790. DOI: 10.1001/ARCHOTOL.125.7.786

[39] Malis L. Electrosurgery. *Journal of Neurosurgery*. 1996;**85**:970-975

[40] Powell NB et al. Radio frequency volumetric tissue reduction of the soft palate in subjects with sleep-disordered breathing. *Chest*. 1998;**113**(5):1163-1174

[41] Olhoffer IH, Leffell DJ. What's new in electro surgery? Coblation: A new method for facial resurfacing. *Aesthetic Dermatology Cosmetic Surgery*. 1999;**1**:31-33

[42] Yamaguchi K, Lonic D, Lee C, Yun C, Lo L. Modified furrow palatoplasty using small double-opposing Z-Plasty: Surgical technique and outcome. *Plastic and Reconstructive Surgery*. 2016;**137**:1825-1831. DOI: 10.1097/PRS.0000000000002181

[43] Kolyadych ZhV. The program method selection and forecasting effectiveness of surgery interventions in patients with the syndrome of obstructive sleep apnea. *Journal of Ear, Nose and Throat Diseases*. 2016;(1):54-61

[44] Uçok K, Ayçiçek A, Sezer M, Genç A, Akkaya M, Çağlar V, et al. Aerobic and anaerobic exercise capacities in obstructive sleep Apnea and associations with subcutaneous fat distributions. *Lung*. 2008;**187**:29-36. DOI: 10.1007/s00408-008-9128-0

- [45] Borowiecki B, Sassin J.
Surgical treatment of sleep apnea.
Archives of Otolaryngology.
1983;**109**(8):508-512. DOI: 10.1001/
ARCHOTOL.1983.00800220014004
- [46] Crampette L, Carlander B,
Mondain M, Billiard M, Guerrier B,
Dejean Y. Surgical alternatives to
uvulopalatopharyngoplasty in sleep
apnea syndrome. *Sleep*. 1992;**15**(6
Suppl):S63-S68. DOI: 10.1093/SLEEP/15.
SUPPL_6.S63
- [47] Denisenko R, Yu, Dikhtyaruk OV,
Naumenko OM. Effect of modified
uvulopalatopharyngoplasty on
body weight indicators and quality
of night sleep in patients with
low compliance to cpap therapy.
Otorhinolaryngology . 2020;**4**(3):73-78.
DOI: 10.37219/2528-8253-2020-4-73
- [48] Olszewska E, Rutkowska J,
Czajkowska A, Rogowski M. Selected
surgical managements in snoring
and obstructive sleep apnea patients.
*Medical Science Monitor : International
Medical Journal of Experimental and
Clinical Research*. 2012;**18**:CR13-CR18.
DOI: 10.12659/MSM.882193