IntechOpen

Obstructive Sleep Apnea

New Insights in the 21st Century

Edited by Marco Carotenuto

Obstructive Sleep Apnea - New Insights in the 21st Century

Edited by Marco Carotenuto

Published in London, United Kingdom

Obstructive Sleep Apnea - New Insights in the 21st Century
http://dx.doi.org/10.5772/intechopen.111133
Edited by Marco Carotenuto

Contributors
Gregory Carter, Yuliia Dieieva, Oleksandr Naumenko, Elizabeth Jones, Michael Greaves, Dwight McLeod, Ignacio Christian Marquez, Gabriel Santos De Freitas, Isabel Portela Ferreño

First published in London, United Kingdom, 2024 by IntechOpen
IntechOpen is the global imprint of INTECHOPEN LIMITED, registered in England and Wales, registration number: 11086078, 5 Princes Gate Court, London, SW7 2QJ, United Kingdom

British Library Cataloguing-in-Publication Data
A catalogue record for this book is available from the British Library

Additional hard and PDF copies can be obtained from orders@intechopen.com

Obstructive Sleep Apnea - New Insights in the 21st Century
Edited by Marco Carotenuto
p. cm.
Print ISBN 978-0-85466-362-0
Online ISBN 978-0-85466-361-3
eBook (PDF) ISBN 978-0-85466-363-7

Meet the editor

Professor Marco Carotenuto obtained a master's degree in medicine and surgery at the University of Campania "Luigi Vanvitelli," Italy, in 2000. He became a specialist in child neuropsychiatry in 2005 after a long period of training in the United Kingdom. He also obtained a Ph.D. in Behavioral Sciences and Learning Disorders in 2008. In 2018, after further training at prestigious Italian research centres to delve deeper into diagnostic, therapeutic, and rehabilitative issues in developmental age, Dr. Carotenuto became a Professor of Child Neuropsychiatry and Director of the Child Neuropsychiatry Unit at the University of Campania "Luigi Vanvitelli." Since 2022, he has been the president of the National Commission for Neurotherapy and Psychomotor Therapy in Developmental Age (TNPEE). Since 2022, he has been Director of the School of Specialization in Child Neuropsychiatry, University of Campania "Luigi Vanvitelli." Dr. Carotenuto has authored more than 200 scientific journal articles. His main areas of research interest include the diagnostic evaluation and therapeutic management of neurodevelopmental disorders, with particular attention to childhood autism, sleep disorders, pediatric headaches and epilepsies, and neurocognitive and behavioral rehabilitation in children.

Contents

Preface

Obstructive sleep apnea syndrome (OSAS) is a multifaceted complex disease that impacts all aspects of a person's daily life regardless of their age and sex. The effects of untreated and unrecognized OSAS can be very serious on all systems of the body, especially the cardiovascular and central nervous systems. Currently, the therapeutic strategies available are varied and multiple, including surgery. Early temporal recognition can limit long-term damage that may even be irreversible.

This edited volume is a collection of reviewed and relevant research chapters on the developments within OSAS. *Obstructive Sleep Apnea – New Insights in the 21st Century* includes scholarly contributions written by and edited by experts in the field. It is designed for scholars and specialists in the field.

Chapter 1: "The Many Faces of Obstructive Sleep Apnea" by Gregory Carter

Chapter 2: "Evaluation and Management in Patients with Obstructive Sleep Apnea" by Yuliia Dieieva and Oleksandr Naumenko

Chapter 3: "Epidemiology of Childhood Sleep Apnea" by Elizabeth Jones

Chapter 4: "Perspective Chapter: Role of the Oral Healthcare Team in the Management of Obstructive Sleep Apnea" by Michael Greaves, Dwight McLeod and Ignacio Christian Marquez

Chapter 5: "The Role of Midline Glossectomy with Coblation in Obstructive Sleep Apnea" by Gabriel Santos De Freitas

Chapter 6: "Changes in the Level of Anxiety and Depression in the Couples of Patients with Obstructive Sleep Apnea after One Year of CPAP Therapy" by Isabel Portela Ferreño

Marco Carotenuto
University of Campania "Luigi Vanvitelli",
Caserta, Italy

Chapter 1

The Many Faces of Obstructive Sleep Apnea

Gregory Carter

Abstract

Obstructive sleep apnea is a common disorder with many different comorbidities. Patients can present with these comorbidities even when sleep apnea has not been diagnosed due to the low diagnostic rates for obstructive sleep apnea worldwide. This presents a concern for clinicians as unless sleep apnea is treated along with a presenting comorbidity, the patient will not have an optimal health outcome. This chapter addresses several of the most common and worrisome comorbidities of sleep apnea and discusses the relationships and pathophysiology of comorbidities including insomnia, treatment resistant hypertension, restless legs syndrome, depression, gastroesophageal reflux, asthma, cognitive disorder, REM sleep behavioral disorder and paroxysmal atrial fibrillation. Clinical studies documenting the relationships of each of these comorbidities to obstructive sleep apnea are presented with putative pathophysiologic discussion of how obstructive sleep apnea aggravates or leads to the development of each of these illnesses.

Keywords: obstructive sleep apnea, OSA, comorbidities, insomnia, hypertension, restless legs, depression, GERD, asthma, cognitive disorder, REM behavioral disorder, atrial fibrillation

1. Introduction

There are many comorbidities associated with obstructive sleep apnea, several of which will be discussed in this chapter. The problem for clinicians providing care to patients is that patients may present with the comorbidity rather than the sleep related breathing disorder. Indeed, underdiagnosis of sleep related breathing disorders has been shown in epidemiological studies [1, 2]. There are thus many patients who present for medical care of a comorbidity rather than the causative or aggravating sleep related breathing disorder.

The reason for the low diagnosis and treatment rates for sleep related breathing disorders (SRBD) is speculative as most of the literature deals with the problems of continuous positive airway pressure (CPAP) management rather than resistance to presentation for evaluation and treatment for the symptoms of SRBD [3–5]. Anecdotal hints are provided by primary care physician comments and patient encounters in the author's 35 years of evaluating and managing patients with SRBD [6]. After a lecture to primary care physicians one of the physicians raised patient concerns about cost, especially if the evaluation did not show a SRBD. This cost issue

discouraged him from referring patients who did not have a high probability of SRBD. One of our fellows, whose family immigrated from Vietnam described a cultural concept that viewed snoring positively as a sign of deep restorative sleep, not a phenomenon requiring medical evaluation. The author has seen a lack of knowledge of the risks associated with untreated SRBD among patients presenting to the sleep clinic. In addition, anxiety about the ability to tolerate a CPAP mask secondary to claustrophobia [7] or cosmetic concerns, i.e., head gear producing indentations on their cheeks or forehead or loss of romantic attractiveness in bed. Some non-compliant patients in my clinic report that they feel that they sleep well, and it is only the concern of their family and friends that brought them to the sleep center. The author had an enlightening experience in London. I and my wife were having dinner at a pub by Victoria Station in 2018. The pub was filled with Americans, and we were sitting with another couple from Minnesota. I had mentioned that I was a sleep medicine physician and I'm not sure if I was overheard. Across the room a Caucasian gentleman stood up to leave with his group. He appeared to be in his mid-sixties with an estimated body mass index (BMI) of 33. He complained in a loud voice that he had been referred by his cardiologist for a sleep study due to a heart rhythm problem (I assumed paroxysmal atrial fibrillation from his description). He stated "I sleep fine. I don't need a sleep study. Sleep studies are just another way for doctors to make money." All these examples are anecdotal. Improving technology may lower the cost of diagnostic evaluations and hopefully one of the barriers to treatment of patients.

There are several comorbidities of SRBD. This chapter will review those comorbidities that are common or concerning in the author's clinical practice. These consist of insomnia [8, 9], hypertension [10, 11], depression [12, 13], restless legs syndrome [14, 15], REM sleep behavioral disorder [16, 17], nocturnal asthma [18, 19], cognitive disorders [20, 21], gastroesophageal reflux disease [22, 23], and finally, paroxysmal atrial fibrillation [24, 25]. Additional comorbidities occur, such as nocturia, sleepwalking, diabetes, metabolic syndrome, and congestive heart failure, but are not discussed in this chapter to allow a more thorough discussion of the listed comorbidities. This chapter discusses the relationship in some detail between each listed comorbidity and SRBD and the need to consider SRBD in various diagnostic differentials.

2. Insomnia

The relationship with chronic insomnia and sleep related breathing disorder or obstructive sleep apnea is the most complex of the list. Insomnia is defined in the International Classification of Sleep Disorders, Third Edition [26] as requiring three components, including persistent sleep difficulty, adequate sleep opportunity, and associated daytime dysfunction. The recognized daytime symptoms of fatigue, decreased mood or irritability, general malaise and cognitive impairment overlap with the daytime symptoms of SRBD [27]. Impaired social or job/school performance, heightened risk of automobile and work accidents and cardiovascular disorders also overlap [8]. Chronic insomnia disorder of adults is present in an estimated 10% of the population but is more common in women, those with secondary insomnia from medical, psychiatric, and substance abuse disorders and in lower income individuals. Cho et al. [28] performed a clinical research study in South Korea utilizing two university sleep centers and enrolling 476 patients with polysomnogram documented obstructive sleep apnea (OSA). The investigators used Korean versions of a health survey and the Insomnia Severity Index (ISI-K) [29, 30], the Pittsburgh Sleep Quality

Index (PSQI-K) [31], the Epworth Sleepiness Scale (ESS-K) [32], and the Beck Depression Inventory (BDI-K) [33]. The investigators used the ISI-K to separate the subjects with OSA plus insomnia (OSA + I) and OSA without insomnia (OSA-I) based on an ISI-K score of less than 15 for the OSA-I group. Of the 476 patients 29.2% or 139 were in the OSA + I group. Whereas females accounted for 24.2% of the total number of subjects, females accounted for 35.3% of the OSA + I group. There was also a significant difference in the BDI-K scale indicating an increase in depressive mood in the OSA + I group. The OSA + I group also showed a statistically significant increase in heart disease. While both OSA and chronic insomnia have documented cardiovascular risk, this study supported prior concerns that the combined risk is greater. This Korean study pointed to three conclusions. Women are more likely to have combined OSA and insomnia and individuals with OSA plus insomnia are more likely to have depression and heart disease.

The causation of OSA plus insomnia is speculative. These two sleep disorders are very common, however, the prevalence of insomnia found in OSA is higher than the prevalence of insomnia in the general population [28]. Several authors [8, 34–36] have reviewed the relationship of co-morbid insomnia and sleep apnea. Over the past 5 years the abbreviation OSA + I has evolved to the eponym 'COMISA.' One clear fact is that these patients are more difficult to manage [37, 38], requiring treatment of both sleep disorders in order to reach an optimal clinical improvement. This suggests that COMISA is not a complication of OSA alone, though the sleep fragmentation and activation of the sympathetic nervous system and hypothalamic-pituitary-adrenal axis appear to be aggravating factors. Chronic sleep deprivation of insomnia may play an additional role, compromising upper airway dilator muscle tone [39, 40] thus worsening OSA.

The treatment of COMISA is more complex than management of OSA without insomnia. COMISA patients have a greater prevalence of psychiatric disorders, including anxiety and claustrophobia that reduce CPAP adherence. Several authors [9, 38, 41] recommend treating insomnia first or concomitantly with cognitive behavioral therapy for insomnia (CBT-I) and/or non-benzodiazepine sedative hypnotics such as zolpidem and eszopiclone. CPAP produces improvement in sleep maintenance insomnia, but not sleep onset insomnia at two-year follow-up [42].

3. Hypertension

Obstructive sleep apnea is the leading contributor to treatment resistant hypertension [43]. The exact prevalence of resistant hypertension is estimated at 20–30% [44]. The strongest risk factors are older age and obesity. Logan et al. [45] performed a study with 41 participants (24 men and 17 women) who completed an overnight polysomnographic study. Drug-resistant hypertension was defined as a clinic blood pressure greater or equal to 140/90 mmHg while on a sensible combination of three or more antihypertensive drugs in maximally recommended doses. A long list of exclusionary criteria included a prior diagnosis of obstructive sleep apnea, 'white coat' hypertension, a history of poor compliance with drug treatment, use of substances that raise blood pressure or interfere with antihypertensive agents, renal insufficiency, excessive alcohol use, liver enzyme levels greater than twice the upper limit of normal, anatomic abnormalities of the upper airway, pregnancy, and significant aortic or mitral valve disease. The participants had a mean age of 57.2 ± 1.6 years, were 85% white, and had a mean body mass index 34.0 ± 0.9 kg/m^2. They were taking an

average of 3.6 ± 0.1 different antihypertensives. The mean office blood pressure was a systolic of 168 ± 4.4 over a diastolic of 94.0 ± 2.3 mm Hg. The overall prevalence of OSA in this resistant hypertension group was 83%. There was a gender difference with OSA being seen in 96% of the male participants compared to 65% of the females. This gender difference was also reflected in the severity of OSA. The average apnea hypopnea index was 32.2 ± 4.5 events per hour in the men (severe OSA is greater than 30 events per hour). This compared to 14.0 ± 3.1 events per hour in the women (mild OSA is 5–15 events per hour).

The pathogenesis whereby OSA is causative of resistant hypertension has been addressed by several authors [10–11, 46–49]. The etiology appears to be multifactorial. Clearly, there is persistent sympathetic activation in both experimental animals [50] and humans [51, 52] from recurrent episodes of hypoxia. Activation of the renin-angiotensin-aldosterone system occurs through mechanisms that are not completely understood [53, 54]. In addition, oxidative stress has been shown to impair endothelial-dependent vasodilation [55].

The causal relationship of OSA to treatment resistant hypertension is one of the clearest relationships in this chapter.

4. Restless legs syndrome

Sleep apnea is often comorbid with restless legs syndrome (RLS) and the associated polysomnographic finding of periodic limb movements of sleep [14]. There is also an overlap with COMISA discussed previously. Pistoris et al. [14] investigated 202 patients retrospectively who had been studied in the Sleep Disorder Center in Regensburg, Germany from 2015 to 2016. Inclusion criteria were mild symptomatic obstructive sleep apnea (OSA) with an apnea hypopnea index (AHI) greater than five events per hour but less than 15 events per hour or moderate to severe OSA (AHI of greater than 15 events per hour) with or without symptoms. Patients with RLS were diagnosed clinically in a face-to-face interview and examination conducted by a sleep specialist. Criteria from the International Classification of Sleep Disorders [56] were used including (1) urge to move the legs, usually accompanied by discomfort in the lower limbs, (2) occurrence or worsening of symptoms in situations of rest or inactivity, (3) partial or total relief of symptoms by movement, and (4) symptoms that are worse or solely occur in the evening. Patients were excluded from the retrospective study if they had severe psychiatric disorders including psychosis, cognitive disorder, alcohol abuse, Parkinson's disease, of difficulties with the German language. Patients with a prior diagnosis of RLS or who were treated with CPAP were also excluded. Fourteen patients were excluded for the above reasons plus another 20 were excluded due to inadequate information on retrospective review. The patients' mean age was 55 ± 11 years. Women were 35% of the total number of patients. The mean body mass index (BMI) was 31 ± 6 kg/m^2. Of the 202 patients in the retrospective review 42 patients (21%) had comorbid RLS. The percentage of 21% is higher than the 5–10% prevalence of RLS in North American and European populations [56]. Of these 42 patients 25 or 60% were women, significantly higher than the percentage of women (29%) in the entire group of 202 patients.

The etiology of an increase in the prevalence of RLS in OSA is speculative and not without controversy. Lakshminarayanan et al. [57] showed prevalence of RLS in 60 sequential OSA patients with an AHI of greater than 10 events per hour of only 8.3%, within the expected range for the general population. Gothi et al. [58] reported

findings like Pistoris et al. [14] described above. Of Gothi's 249 OSA patients 61 patients had comorbid RLS for an increased prevalence of 28.5%.

The pathophysiology of RLS is a decrease of dopamine receptor density [59]. Some patients report a family history and younger age of onset. Familial genetic linkage studies [60] have revealed some sequence variants, but how these genes affect the pathogenesis of RLS is unclear. In addition, RLS can be secondary to iron deficiency, renal failure, pregnancy, peripheral neuropathy, chronic myelopathy and a variety of medications including tricyclic antidepressants, selective serotonin re-uptake inhibitors, lithium, antipsychotic drugs and dopamine receptor antagonists [15].

Though there are rationales for the pathophysiology of RLS in some of the afore mentioned, the rationale for an increased prevalence of RLS in OSA is unclear. The overlap with chronic insomnia suggests a common pathophysiology in the relationship with OSA. In the author's personal experience, and as described by Gothi et al. [58], the combination of RLS and OSA (as with insomnia and OSA) makes these patients more complex to manage. This is especially true as two options for the management of treatment resistant RLS, benzodiazepines and opiates, are respiratory suppressants. These drugs suppress arousals, thus prolonging apneas and deepening oxygen desaturations in patients not using CPAP every night [61]. Rodrigues et al. [61] reported that 17 patients with RLS + OSA improved both their OSA and RLS symptoms with CPAP. Myc et al. [62] reported similar improvement in a single case report. The long-term results of consistent CPAP use in RLS + OSA patients need further research.

5. Depression

Several investigators [13, 63, 64] have surveyed newly diagnosed patients with OSA for depressive and anxiety symptoms. Velescu et al. [63] utilizing the Patient Health Questionnaire-9 Depression Scale found a prevalence of depressive symptoms in 48.48% of 99 consecutive new patients. At 1 year follow-up of CPAP treatment 92.9% of their patients had experienced significant improvement. Akberzie et al. [64] examined 45 patients' records who presented for polysomnography over a five-month period and were diagnosed with obstructive sleep apnea (AHI greater than 5). The patients had completed a Hospital Anxiety and Depression Scale. There was a female predominance with 29 of the 45 patients being female. Of the 45 patients 29 were positive for depression (64.4%). Shoib et al. [13] performed the mini international neuropsychiatric interview plus scale and Hamilton Depression Rating Scale (HAM-D) on 182 patients undergoing polysomnography over a 2 year period. Patients were excluded if they were on nocturnal oxygen supplementation, CPAP or mandibular advancement devices, had upper airway surgery or had unstable cardiopulmonary, neurological, or psychiatric disease. Of the 182 patients, 47 had depression. Of the depressed patients 44 or 93.6% had either mild (3 or 6.8%), moderate (18 or 40.9%), or severe (23 or 52.3%) OSA. The HAM-D scale was significantly greater ($p = 0.0001$) in the depressive patients than the non-depressive patients.

Patients with OSA have a higher prevalence of depression [12, 65]. These depressive symptoms may gradually improve with CPAP treatment [66, 67]. Zheng et al. [68] showed a statistically significant ($p = 0.031$) reduced odds of depression care in the Sleep Apnea Cardiovascular Endpoints (SAVE) trial of 2410 patients with moderate to severe OSA and established cardiovascular disease in patients randomly allocated to CPAP plus usual care versus usual care alone and followed for 3–7 years.

The pathophysiology of OSA and depression's association is speculative. Direct pathophysiology through aggravation of each other's symptom complexes, as well as, indirect pathophysiology through molecular processes (hormonal and inflammatory) have been postulated.

6. Gastroesophageal reflux

Nocturnal gastroesophageal reflux (nGER) is common in patients with obstructive sleep apnea (OSA) [23, 69–71]. Green et al. [22] studied 331 patients diagnosed with OSA over a seven-year period from 1993 to 2000 who graded their nGER frequency from 1 (never) to 5 (always). All their patients were prescribed CPAP. Prior to treatment nGER was present in 204 patients (62%). Follow-up was obtained from 181 patients. Of these 181 (91%) were still using CPAP and 16 (9%) were not using CPAP. The compliant group's frequency score fell from a mean of 3.38 to 1.75 ($p < 0.001$). Patients not using CPAP had no significant change, 3.56–3.44. ($p = 0.55$). Interestingly, there was a correlation between CPAP pressure and improvement in the frequency score ($r = 0.70$; $p < 0.001$).

Ing et al. [69] studied 63 patients with OSA (AHI > 15) and 41 patients without OSA (AHI < 5) with esophageal pH monitoring simultaneous with polysomnography. Patients with OSA had more gastroesophageal reflux events than patients without OSA (115 vs. 23; $p < 0.001$). In addition, OSA patients spent a higher percentage of their recording time at a pH of less than 4.0 (21.4 vs. 3.7%; $p < 0.001$).

The pathophysiology of the relationship of nGER to OSA is suggested by the effect of CPAP pressure. Intrathoracic negative pressure, as compared to abdominal pressure can show marked negativity with attempts to breath against a closed upper airway. CPAP treatment not only removes upper airway obstruction, thus reducing negative intrathoracic pressure, but also increases intrathoracic pressure with the use of higher CPAP pressures. This has a direct effect as esophageal pressure exceeds gastric pressure.

7. Asthma

A bidirectional link between bronchial asthma and obstructive sleep apnea (OSA) has been reported since the initial single case report by Hudgel and Shucard in 1979 [72].

Teodorescu et al. [73] recruited 472 subjects out of the allergy and pulmonary subspecialty clinics of the University of Wisconsin-Madison with a diagnosis of asthma who did not have co-morbid lung disease or were not under treatment for OSA. The subjects completed the Asthma Control Questionnaire (ACQ) [74] and the Sleep Apnea scale of the Sleep Disorders Questionnaire (SA-SDQ) [75]. Abnormal scores were ≥ 1.5 for the ACQ and ≥ 36 (men) or ≥ 32 for women for the SA-SDQ. High SA-SDQ scores were associated with 3.60 times higher odds of having a high ACQ score. With adjustments for obesity, race, nasal polyps, GERD, and psychopathology the odds of not-well-controlled asthma were still 2.87 times higher in the high OSA risk group.

Teodorescu et al. [76] performed a prospective study with 547 participants recruited from a random sample of Wisconsin state employees who were enrolled in the Wisconsin Sleep Cohort Study [2]. The presence or absence of OSA was assessed by polysomnography performed on enrollment and every 4 years thereafter. None of

the 547 participants had OSA on their first polysomnogram. The presence of asthma in 81 participants was assessed by a questionnaire. At their first 4-year interval 22 (27%) participants with asthma had developed OSA while 16% of the asthma-free participants had developed OSA. This was statistically significant ($p = 0.02$).

The pathophysiology that links asthma and OSA are hypothetical. Dixit [77] postulated pathways that included increased parasympathetic tone, hypoxia-related reflex bronchospasm, altered nocturnal neurohormonal secretion, increased inflammatory mediators, gastroesophageal reflux, and obesity. Also included were adverse actions of inhaled corticosteroids producing increased upper airway adiposity or localized steroid myopathy of dilator muscles. The pathophysiology of this overlap continues to be discussed [19, 78].

8. Cognitive disorder

Several authors have reviewed the association of obstructive sleep apnea (OSA) with cognitive disorders [79–81]. The risk of OSA for the future development of Alzheimer's disease remains prominently discussed [79]. The question of whether treatment of OSA with CPAP will improve cognitive status and improve risk of further deterioration is only beginning to be answered.

Dalmases et al. [82] performed a randomized, evaluator-blinded, parallel-group, single center study of 31 patients (69.7% male) who were at least 65 years old. The two arms of the study looked at newly diagnosed OSA patients (apnea hypopnea index mean average of 55.49 ± 17.63 events per hour). Patients were excluded if they had a Mini Mental Status Examination of less than 24, respiratory failure, neurologic or psychiatric disorders, chronic heart failure, unstable illnesses, other sleep disorders, contraindications for MRI, and inability to respond to questionnaires. Out of the 51 patients assessed, 18 patients were excluded (3 for declining to participate). The 33 eligible patients were divided into two groups. One group was managed with sleep hygiene and dietary counseling and the second group was placed on continuous positive airway pressure (CPAP) in addition to sleep hygiene and dietary counseling. All patients received neuropsychological examination and T1 high resolution MRI and 5-minute resting state functional MRI testing at baseline and at 3 months. One patient in each group was lost to follow-up and not included in the final assessment. While there were no significant differences between the two groups at baseline, the CPAP treatment group showed improvement in short-term memory ($p = 0.032$) and executive functioning ($p = 0.014$). The CPAP treatment group showed improvement in the speed of mental processing ($p = 0.007$) and mental flexibility ($p = 0.008$). Functional MRI revealed a significant increase in the intensity of connectivity ($p = 0.012$) between the posterior cingulate cortex and precuneus, the parahippocampal gyrus, and the middle and medial frontal gyrus ($p = 0.013$). There were no significant changes in the Epworth Sleepiness Scale (ESS) between groups.

Werli et al. [83] looked specifically at the effect of residual excessive daytime sleepiness (ESS > 10) in patients (ages from 30 to 65 years old) with moderate to severe OSA (AHI > 20) all of whom were being treated with CPAP. The comparison groups were 15 patients with excessive daytime sleepiness (EDS) versus a control group of 15 patients without excessive daytime sleepiness. There was no significant difference in the baseline AHI and ESS, however the multiple sleep latency test did reveal the average sleep latency in the control group to be 9.4 ± 2.7 minutes versus the average sleep latency in the EDS group of 2.9 ± 2.34. There was no significant difference in CPAP adherence to

therapy. The EDS group performed poorly in executive functions, including such functions as handling of information, inadequate planning, judgment, and decision making, plus inflexibility, impulsivity, and difficulty maintaining motivation. The control group did not show evidence of executive function deficits. The prognostic implications of excessive daytime sleepiness in OSA continue to be an area of study and discussion.

There are several rationales for the finding of cognitive deficits in OSA [84–86]. Edwards et al. [84] showed that higher night-time cortisol levels were associated with greater cognitive impairment in patients with moderate to severe OSA (mean AHI 30.3 ± 21.7 events per hour). Ciccone and Mehra [85] presented an unpublished study showing the number of minutes during the sleep period with low oxygen levels was significantly associated with lower scores on the Montreal Cognitive Assessment (MOCA) test. There is a growing literature on cognition and OSA [20, 21]. OSA must be considered as an aggravating factor in patients presenting with a mild cognitive disorder.

9. REM sleep behavioral disorder

Rapid eye movement (REM) sleep behavioral disorder is a sleep parasomnia associated with dream enactment. It has been associated with neurodegeneration of long tracts originating in pontomedullary regions that send excitatory signals to the glycinergic neurons of the spinal ventral horns to hyperpolarize spinal motor neurons producing paralysis due to loss of muscle tone [16]. Idiopathic REM sleep behavioral disorder (RBD) is caused by a group of neurodegenerative disorders called the α-synucleinopathies, including Parkinson's disease, multiple system atrophy, and dementia with Lewy bodies [87].

Secondary disorders mimicking idiopathic RBD include dream enactment behaviors caused by certain drugs, such as selective serotonin reuptake inhibitors and brainstem injuries, tumors, vascular lesions, or inflammation [16]. Iranzo and Santamaria [88] described 16 patients who were identified with dream enactment behaviors in addition to snoring and excessive daytime sleepiness. Polysomnograms did not show REM sleep without atonia but did show severe obstructive sleep apnea (OSA) with mean apnea hypopnea index (AHI) of 67.5 ± 18.7 events per hour. The abnormal sleep behaviors occurred only during apnea-induced arousals. The authors felt that the polysomnographic finding of REM sleep without atonia in the neurodegenerative disorders allowed dream enactment of neurodegenerative disorders to be distinguished fromdream behaviors of severe OSA. Dream enactment associated with OSA gained the name "pseudo-RBD." Gabryelska et al. [17] mailed a questionnaire to 120 patients (85% male) with diagnosed RBD. One hundred and seven (89.2%) of the patients had a diagnosis of OSA with an AHI greater than 5. Of the 72 patients who responded to the questionnaire, 27 were using CPAP. Of the 27, 45.8% reported CPAP therapy improved their dream enactment.

Pseudo-RBD can present as RBD, however, the treatment of pseudo-RBD with clonazepam or another benzodiazepine can be contraindicated due to suppression of arousals to breathe, especially in the absence of CPAP.

10. Paroxysmal atrial fibrillation

Paroxysmal atrial fibrillation has been recognized as having a strong association with obstructive sleep apnea (OSA) [24, 25, 89, 90]. Atrial fibrillation (chronic and

paroxysmal) is the most common cardiac arrhythmia. It can result in embolism to the brain and body. The Sleep Heart Health Study compared 228 patients with sleep-disordered breathing (SDB) (mean AHI 44.7 ± 13.1; 50.88% male) with 338 patients without SDB (mean AHI 2.7 ± 1.4; 47.00% male). This comparison showed a higher incidence of atrial fibrillation in patients with OSA than in the general population [91]. If adjustments were made for age, body mass index (BMI), hypertension, and congestive heart failure, the odds ratio for atrial fibrillation in the OSA group was 4.02.

Anter et al. [24] studied 86 patients with paroxysmal atrial fibrillation including 43 patients with moderate to severe OSA (AHI ≥ 15) and 43 patients without OSA (AHI < 5). The two groups underwent detailed electrophysiologic mapping and ablation protocol which included pulmonary vein (PV) isolation plus ablation of extra-PV triggers. An atrial fibrillation trigger site was defined as a site that produces an atrial premature depolarization (APD) triggering episodes of atrial fibrillation (AF) lasting ≥30 seconds. At baseline the PV was the most frequent trigger site in both groups and PV isolation was achieved in all patients. On repeating the protocol to identify additional triggers 18 of 43 patients with OSA had residual extra-PV triggers compared to 5 of 43 patients without OSA. The association of AF and OSA was felt to be secondary to electric and structural bi-atrial remodeling predominantly in the anterior septum.

The pathophysiology of atrial fibrillation is becoming clearer from animal studies. The heart has dual autonomic nervous system innervations. The sympathetic nervous system causes the heart to speed up and vagal nerve parasympathetic nervous system causes the heart to slow down among other effects. Occlusion of the upper airway leads to exaggerated negative intrathoracic pressure swings, intermittent hypoxia, atrial stretching, and cortical arousals with proarrhythmic cycles of parasympathetic and sympathetic nervous activities.

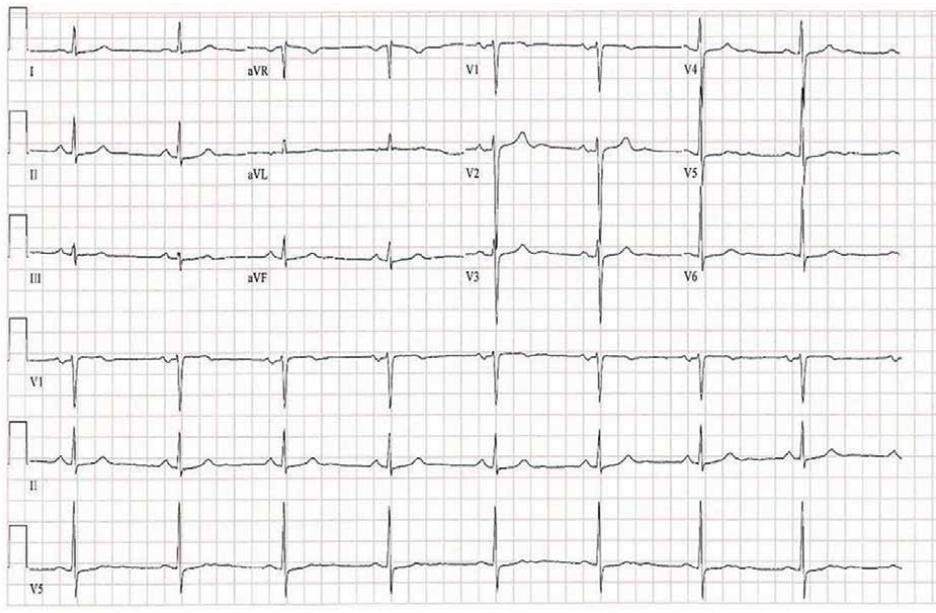

Figure 1.
This is the baseline electrocardiogram showing normal sinus rhythm with no atrial abnormality.

Figure 1 is a routine electrocardiogram (ECG) from a patient in our laboratory. The ECG is normal without any evidence of an abnormality that would predispose to a cardiac arrhythmia. **Figure 2** is the initial recording from the diagnostic polysomnogram. **Figure 3** shows the appearance of prolonged apneas, however, there is

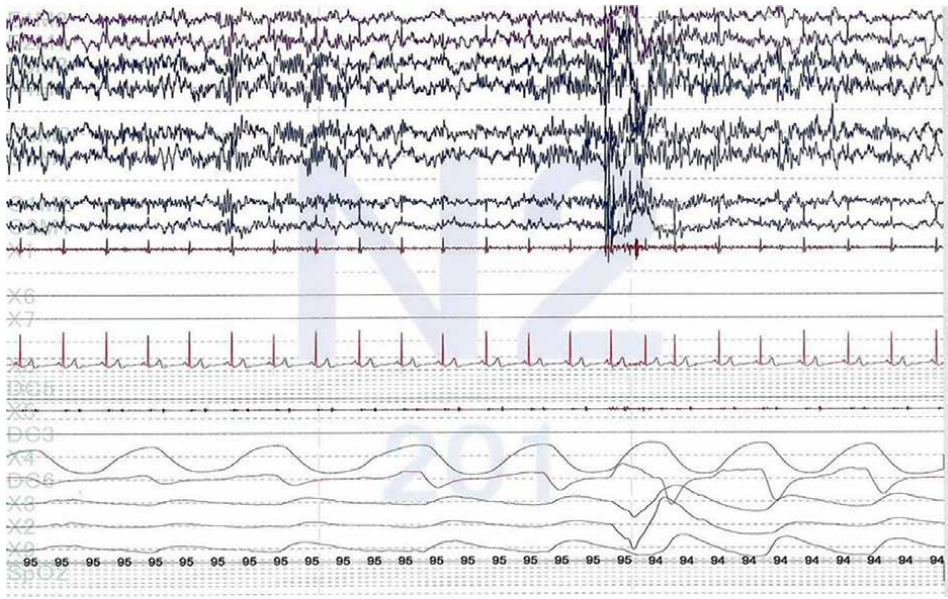

Figure 2.
The polysomnogram begins and the patient falls asleep with early signs of respiratory arousals.

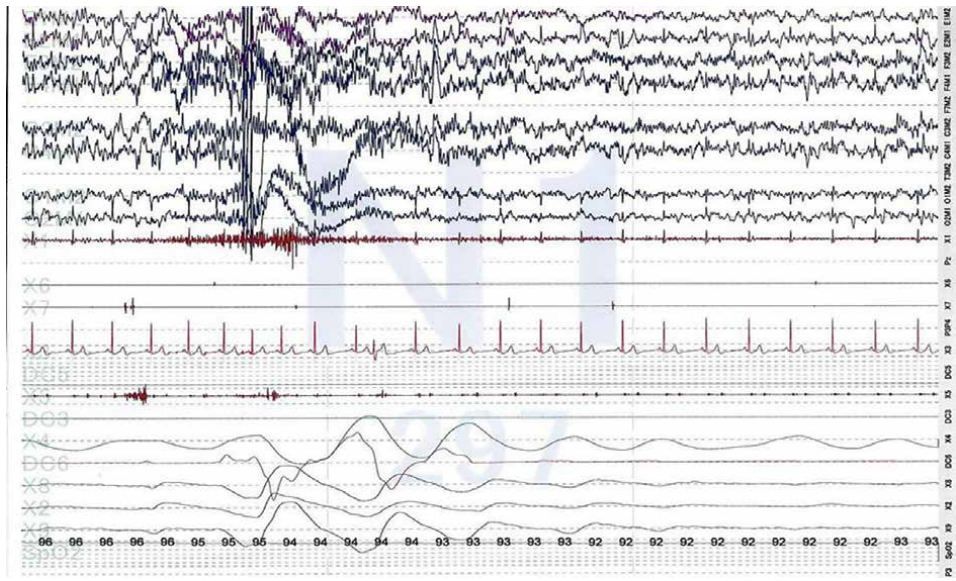

Figure 3.
Prolonged apneas develop with occasional premature beats.

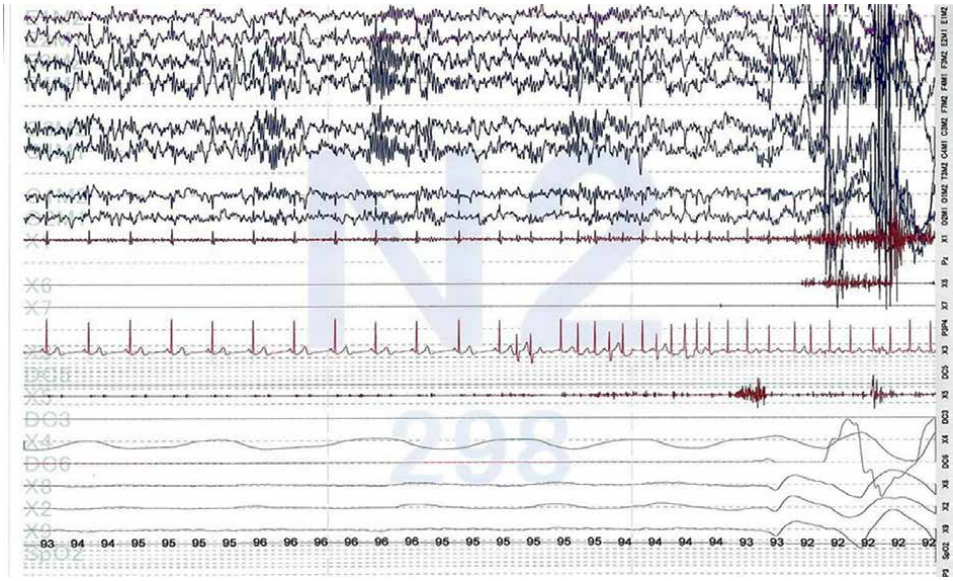

Figure 4.
After two premature beats occurring consecutively, the rhythm changes to atrial flutter.

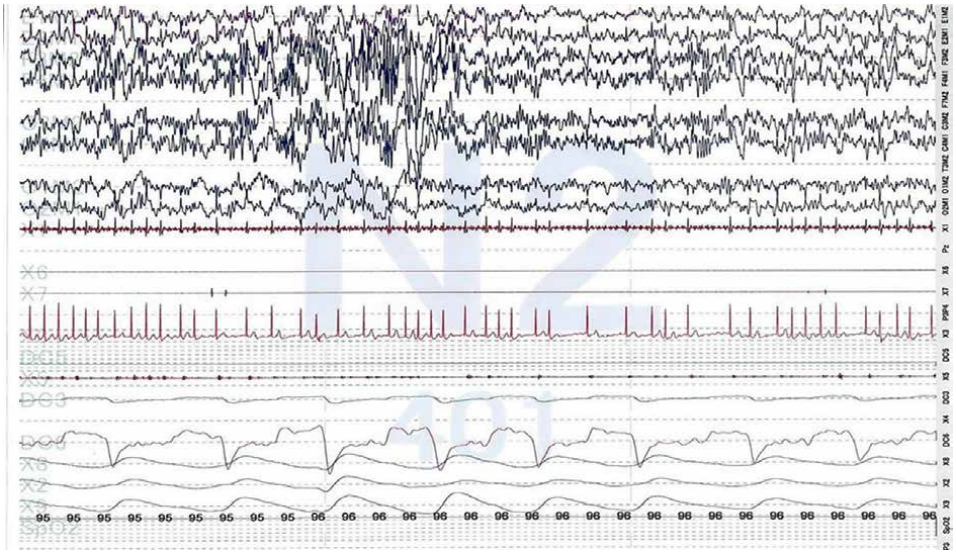

Figure 5.
CPAP is initiated. Atrial flutter with a rapid ventricular response changes to atrial fibrillation.

continuation of normal sinus rhythm. **Figure 4** shows the appearance of two atrial premature contractions occurring quickly during a prolonged apnea with initiation of atrial flutter. **Figure 5** shows atrial flutter deteriorating to atrial fibrillation. **Figure 6** is post initiation of CPAP and atrial fibrillation spontaneously converts to normal sinus rhythm.

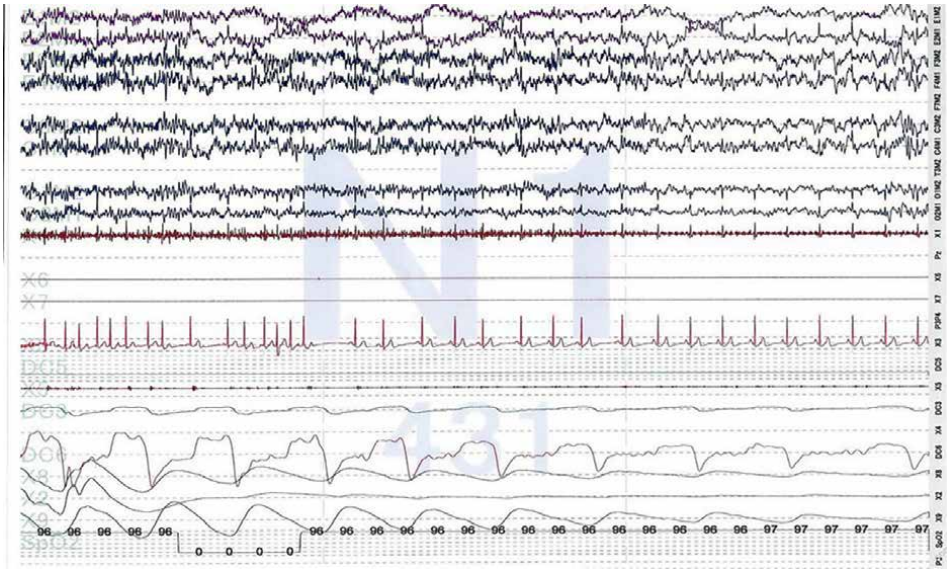

Figure 6.
Spontaneous conversion back to normal sinus rhythm on CPAP at `5 cm of water. The patient would need to be treated with a final CPAP pressure of 10 cm of water.

11. Conclusion

Obstructive sleep apnea is a common illness that often flies under the radar of clinicians, but the comorbidities discussed above may be common in clinical practice. The danger for clinicians is that patients may not mention their sleep problems in the outpatient evaluation and management unless specifically asked. Patients may also be concerned about the cost of testing and treatment and resistant to referral. Treatment of the comorbidity alone may fail to resolve the co-morbidity in the absence of treating sleep related breathing, underlying or aggravating the presenting complaint.

The increasing use and sophistication of inexpensive personal monitors and home testing devices may be very helpful for the cost concern, however, public education about the risks of sleep related breathing disorders and their comorbidities will require continuing efforts.

Conflict of interest

The author declares no conflict of interest.

DOI: http://dx.doi.org/10.5772/intechopen.1003062

Author details

Gregory Carter
The University of Texas Southwestern Medical Center at Dallas, Dallas, Texas, United States of America

*Address all correspondence to: gregory.carter@utsouthwestern.edu

IntechOpen

References

[1] Chen X, Wang R, Zee P, Lutsey PL, Javaheri S, Alcántara C, et al. Racial/ethnic differences in sleep disturbances: The multi-ethnic study of atherosclerosis (MESA). Sleep. 2015;**38**(6):877-888

[2] Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, et al. Sleep disordered breathing and mortality: Eighteen-year follow-up of the Wisconsin sleep cohort. Sleep. 2008;**31**(8):1071-1078

[3] Campos-Rodriguez F, Martinez-Alonso M, Sanchez-de-la-Torre M, Barbe F. Long-term adherence to continuous positive airway pressure therapy in non-sleepy sleep apnea patients. Sleep Medicine. 2016;**17**:1-6

[4] Lin H-S, Prasad AS, Pan C-JG, Rowley JA. Factors associated with non-compliance to treatment with positive airway pressure. Archives of Otolaryngology--Head & Neck Surgery. 2007;**133**:69-72

[5] Gabryelska A, Sochal M, Wasik B, Szczepanowski P, Bialasiewicz P. Factors affecting long-term compliance of CPAP treatment–A single centre experience. Journal of Clinical Medicine. 2022;**11**:139

[6] Author's personal experience at the University of Texas Southwestern Medical Center at Dallas Clinical Center for Sleep and Breathing Disorders, 2007-2023

[7] Watenpaugh DE. CPAP intolerance is a sweeping, misused phrase. Sleep Review. 2023;**25**(5):10-11

[8] Sweetman A, Lack L, Bastien C. Co-morbid insomnia and sleep apnea (COMISA): Prevalence, consequences, methodological considerations, and recent randomized controlled trials. Brain Sciences. 2019;**9**:371

[9] Ragnoli B, Pochetti P, Raie A, Malerbe M. Comorbid insomnia and obstructive sleep apnea (COMISA): Current concepts of patient management. International Journal of Environmental Research and Public Health. 2021;**18**:9248

[10] Bangash A, Wajid F, Poolacherla R, Mim FK, Rutkofsky IH. Obstructive sleep apnea and hypertension: A review of the relationship and pathogenic association. Cureus. 2020;**12**(5):e8241. DOI: 10.7759/cureus.8241

[11] Wang AY-M. Sleep-disordered breathing and resistant hypertension. Seminars in Nephrology. 2014;**34**(5):520-531

[12] Ejaz SM, Khawaja IS, Bhatia S, Hurwitz TD. Obstructive sleep apnea and depression: A review. Innovations in Clinical Neuroscience. 2011;**8**(8):17-25

[13] Shoib S, Malik JA, Masoodi S. Depression as a manifestation of obstructive sleep apnea. Journal of Neurosciences in Rural Practice. 2017;**8**(3):346-351

[14] Pistoris F, Geisler P, Wetter TC, Crönlein T. Sleep apnea syndrome comorbid with and without restless legs syndrome: Differences in insomnia specific symptoms. Sleep & Breathing. 2020;**24**(3):1167-1172

[15] Roux FJ. Restless legs syndrome: Impact on sleep-related breathing disorders. Respirology. 2012;**18**(2):238-245

[16] Roguski A, Rayment D, Whone AL, Jones MW, Rolinski M. A neurologist's

guide to REM sleep behavior disorder. Frontiers in Neurology. 2020;**11**:610

[17] Gabyryelska A, Roguski A, Simpson G, Maschauer EL, Morrison I, Riha RL. Prevalence of obstructive sleep apnoea in REM behaviour disorder: Response to continuous positive airway pressure therapy. Sleep & Breathing. 2018;**22**:825-830

[18] Alkhalil M, Schulman E, Getsy J. Obstructive sleep apnea syndrome and asthma: What are the links? Journal of Clinical Sleep Medicine. 2009;**5**(1):71-78

[19] Ragnoli B, Pochetti P, Raie A, Malerba M. Interrelationship between obstructive sleep apnea syndrome and severe asthma: From endo-phenotype to clinical aspects. Frontiers in Medicine. 2021;**8**:640636

[20] Gagnon K, Baril A-A, Gagnon J-F, Fortin M, Décary A, Lafond C, et al. Cognitive impairment in obstructive sleep apnea. Pathologie Biologie. 2014;**62**:233-240

[21] Lai C, Ayappa I, Ayas N, et al. The link between obstructive sleep apnea and neurocognitive impairment: An Official American Thoracic Society workshop report. Annals of the American Thoracic Society. 2022;**19**(8):1245-1256

[22] Green BT, Broughton WA, O'Connor JB. Marked improvement in nocturnal gastroesophageal reflux in a large cohort of patients with obstructive sleep apnea treated with continuous positive airway pressure. Archives of Internal Medicine. 2003;**163**:41-45

[23] Mahfour R, Barchuk A, Obeidat AE, Mansour MM, Hernandez D, Darweesh M, et al. The relationship between obstructive sleep apnea (OSA) and gastroesophageal reflux disease (GERD) in inpatient settings: A nationwide study. Cureus. 2022;**14**(3):e22810. DOI: 10.7759/cureus.22810

[24] Anter E, Di Biase L, Contreras-Valdes FM, et al. Atrial substrate and triggers of paroxysmal atrial fibrillation in patients with obstructive sleep apnea. Circulation: Arrhythmia and Electrophysiology. 2017;**10**:e005407

[25] Lin C-H, Timopfeeva M, O'Brien T, Lyons OD. Obstructive sleep apnea and nocturnal attacks of paroxysmal atrial fibrillation. Journal of Clinical Sleep Medicine. 2022;**18**(5):1279-1286

[26] American Academy of Sleep Medicine. Insomnia. In: International Classification of Sleep Disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014. pp. 19-48

[27] American Academy of Sleep Medicine. Sleep related breathing disorders. In: International Classification of Sleep Disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014. pp. 49-141

[28] Cho YW, Kim KT, Moon H-J, Korostyshevskiy VR, Motamedi GK, Yang KI. Comorbid insomnia with obstructive sleep apnea: Clinical characteristics and risk factors. Journal of Clinical Sleep Medicine. 2018;**14**(3):409-417

[29] Cho YW, Song ML, Morin CM. Validation of a Korean version of the insomnia severity index. Journal of Clinical Neurology. 2014;**10**(3):210-215

[30] Morin CM, Belleville G, Belanger L, Ivers H. The insomnia severity index: Psychometric indicators to detect insomnia cases and evaluate treatment response. Sleep. 2011;**34**(5):601-608

[31] Sohn SI, Kim DH, Lee MY, Cho YW. The reliability and validity of the

Korean version of the Pittsburgh sleep quality index. Sleep & Breathing. 2012;**16**(3):803-812

[32] Cho YW, Lee JH, Son HK, Shin C, Johns MW. The reliability and validity of Korean version of the Epworth sleepiness scale. Sleep & Breathing. 2011;**15**(3):377-384

[33] Sung H, Kim J, Park Y, Bai D, Lee S, Ahn H. A study on the reliability and the validity of Korean version of the Beck depression inventory II (BDI-II). Journal of the Korean Society of Biological Therapies in Psychiatry. 2008;**14**:201-212

[34] Ong JC, Crawford MR. Insomnia and obstructive sleep apnea. Sleep Medicine Clinics. 2013;**8**(3):389-398

[35] Benetó A, Gomez-Siurana E, Rubio-Sanchez P. Comorbidity between sleep apnea and insomnia. Sleep Medicine Reviews. 2009;**13**:287-293

[36] Luyster FS, Buysse DJ, Strollo P Jr. Comorbid insomnia and obstructive sleep apnea: Challenges for clinical practice and research. Journal of Clinical Sleep Medicine. 2010;**6**(2):196-204

[37] Nguyen XL, Chaskalovic J, Rakotonanahary D, et al. Insomnia symptoms and CPAP compliance in OSAS patients: A descriptive study using data mining methods. Sleep Medicine. 2010;**11**(8):777-784

[38] Wallace DM, Vargas SS, Schwartz SJ, et al. Determinants of continuous positive airway pressure adherence in a sleep clinic cohort of South Florida Hispanic veterans. Sleep & Breathing. 2013;**17**(1):351-363

[39] Krakow B, Melendrez D, Ferreira E, et al. Prevalence of insomnia symptoms in patients with sleep-disordered breathing. Chest. 2001;**120**(6):1923-1929

[40] Al-Jawder SE, Bahammam AS. Comorbid insomnia in sleep-related breathing disorders: An underrecognized association. Sleep & Breathing. 2012;**16**(2):295-304

[41] Pieh C, Bach M, Popp R, et al. Insomnia symptoms influence CPAP compliance. Sleep & Breathing. 2013;**17**(1):99-104

[42] Bjornsdottir E, Janson C, Sigurdsson JF, et al. Symptoms of insomnia among OSA patients before and after 2 years of positive airway pressure treatment. Sleep. 2013;**36**(12):1901-1909

[43] Pedrosa RP, Drager LF, Gonzaga CC, et al. Obstructive sleep apnea: The most common secondary cause of hypertension associated with resistant hypertension. Hypertension. 2011;**58**:811-817

[44] Calhoun DA, Jones D, Textor S, et al. Resistant hypertension: Diagnosis, evaluation, and treatment: A scientific statement from the American Heart Association Professional Education Committee of the Council for high blood pressure research. Hypertension. 2008;**51**:1403-1419

[45] Logan AG, Perlikowski SM, Mente A, Tisler A, Tkacova R, Niroumand M, et al. High prevalence of unrecognized sleep apnoea in drug resistant hypertension. Journal of Hypertension. 2001;**19**:2271-2277

[46] Konecny T, Kara T, Somers VK. Obstructive sleep apnea and hypertension: An update. Hypertension. 2014;**63**:203-209

[47] Kapa S, Kuniyoshi FHS, Somers VK. Sleep apnea and hypertension: Interactions and implications for management. Hypertension. 2008;**51**:605-608

[48] Dopp JM, Reichmuth KJ, Morgan BJ. Obstructive sleep apnea and hypertension: Mechanisms, evaluation, and management. Current Hypertension Reports. 2007;**9**(6):529-534

[49] Patel AR, Patel AR, Singh S, Singh S, Khawaja I. The association of obstructive sleep apnea and hypertension. Cureus. 2019;**11**(6):e4858. DOI: 10.7759/cureus.4858

[50] Fletcher EC, Bau G, Miller CC III. Effect of recurrent episodic hypocapnic, eucapnic, and hypercapnic hypoxia on systemic blood pressure. Journal of Applied Physiology. 1995;**78**:1516-1521

[51] Foster GE, Brugniaux JV, Pialoux V, et al. Cardiovascular and cerebrovascular responses to acute hypoxia following exposure to intermittent hypoxia in healthy humans. The Journal of Physiology. 2009;**587**:3287-3299

[52] Tamisier R, Pepin JL, Remy J, et al. 14 nights of intermittent hypoxia elevate daytime blood pressure and sympathetic activity in healthy humans. The European Respiratory Journal. 2011;**37**:119-128

[53] Pratt-Ubunama MN, Nishizaka MK, Boedefeld RL, Cofield SS, Harding SM, Calhoun DA. Plasma aldosterone is related to severity of obstructive sleep apnea in subjects with resistant hypertension. Chest. 2007;**131**:453-459

[54] Goodfriend TL. Obesity, sleep apnea, aldosterone, and hypertension. Current Hypertension Reports. 2008;**10**:222-226

[55] Kato M, Roberts-Thompson P, Phillip BG, Haynes WG, Winnicki M, Accurso V, et al. Impairment of endothelium-dependent vasodilation of resistance vessels in patients with obstructive sleep apnea. Circulation. 2000;**102**:2607-2610

[56] American Academy of Sleep Medicine. Sleep related movement disorders. In: International Classification of Sleep Disorders. 3rd ed. Darien, IL: American Academy of Sleep Medicine; 2014. pp. 281-337

[57] Lakshminarayanan S, Paramassivan KD, Walters AS, Wagner ML, Patel S, Passi V. Clinically significant but unsuspected restless legs syndrome in patients with sleep apnea. Movement Disorders. 2005;**20**:501-503

[58] Gothi D, Kumar S, Patro M, Malhotra N, Vaidya S. A study on the prevalence of RLS in OSA and the consequences of co-occurrence. Lung India. 2023;**40**(4):321-326

[59] Connor JR, Wang XS, Allen RP, Beard JL, Wiesinger JA, Felt BT, et al. Altered dopaminergic profile in the putamen and substantia nigra in restless leg syndrome. Brain. 2009;**132**:2403-2412

[60] Winklemann J, Schormair B, Lichtner P, et al. Genome-wide association study of restless legs syndrome identifies common variants in three genomic regions. Nature Genetics. 2007;**39**:1000-1006

[61] Delgado Rodrigues RN, de Abreu ESARAA, Krieger J. Outcome of restless legs severity after continuous positive air pressure (CPAP) treatment in patients affected by the association of RLS and obstructive sleep apneas. Sleep Medicine. 2006;**7**:235-239

[62] Myc LA, Churnin IT, Jameson MJ, Davis EM. Treatment of comorbid sleep apnea by upper airway stimulation results in resolution of debilitating symptoms of restless legs syndrome. Journal of Clinical Sleep Medicine. 2018;**14**(10):1797-1800

[63] Velescu DR, Marc M, Manolescu D, Trăilă D, Oancea C. CPAP therapy on depressive and anxiety symptoms in patients with moderate to severe

obstructive sleep apnea syndrome. Medicina. 2022;**58**:1402

[64] Akberzie W, Hesselbacher S, Aiyer I, Surani S, Surani ZS. The prevalence of anxiety and depression symptoms in obstructive sleep apnea. Cureus. 2020;**12**(10):e11203. DOI: 10.7759/cureus.11203

[65] Jehan S, Auguste E, Pandi-Perumal SR, Kalinowski J, Myers AK, Zizi F, et al. Depression, obstructive sleep apnea and psychosocial health. Sleep Medicine and Disorders: International Journal. 2017;**1**(3):1-13

[66] Schwartz DJ, Kohler WC, Karatinos G. Symptoms of depression in individuals with obstructive sleep apnea may be amenable to treatment with continuous positive airway pressure. Chest. 2005;**128**(3):1304-1309

[67] Sánchez AI, Buela-Casal G, Bermúdez MP, Casas-Maldonado F. The effects of continuous positive air pressure treatment on anxiety and depression levels in apnea patients. Psychiatry and Clinical Neurosciences. 2001;**55**(6):641-646

[68] Zheng D, Xu Y, You S, et al. Effects of continuous positive airway pressure on depression and anxiety symptoms in patients with obstructive sleep apnoea: Results from the sleep apnoea cardiovascular endpoint randomized trial and meta-analysis. eClinicalMedicine. 2019;**11**:89-96

[69] Ing AJ, Ngu MC, Breslin AB. Obstructive sleep apnea and gastroesophageal reflux. The American Journal of Medicine. 2000;**108**(4a):120S-125S

[70] Shaker A, Magdy M. Frequency of obstructive sleep apnea (OSA) in patients with gastroesophageal reflux disease (GERD) and the effect of nasal continuous positive airway pressure. The Egyptian Journal of Chest Diseases and Tuberculosis. 2016;**65**:797-803

[71] Wang X, Wright Z, Wang J, Song G. Obstructive sleep apnea is associated with an increased risk of developing gastroesophagel reflux disease and its complications. Journal of Respiration. 2023;**3**:75-85

[72] Hudgel DW, Shucard DW. Coexistence of sleep apnea risk with asthma control in adults. JAMA. 1979;**242**:2789-2790

[73] Teodorescu M, Polomis DA, Hall SV, Teodorescu MC, Gangnon RE, Peterson AG, et al. Association of obstructive sleep apnea risk with asthma control in adults. Chest. 2010;**2010**(138):543-550

[74] Teodorescu M, Barnet JH, Hagen EW, Palta M, Young TB, Peppard PE. Association between asthma and risk of developing obstructive sleep apnea. JAMA. 2015;**313**(2):156-164

[75] Juniper EF, Svensson K, Mörk AC, Ståhl E. Measurement properties and interpretation of the three shortened versions of the asthma control questionnaire. Respiratory Medicine. 2005;**99**(5):553-558

[76] Douglas AB, Bornstein R, Nino-Murcia G, Keenan S, Miles L, Zarcone VP Jr, et al. The sleep disorders questionnaire. I: Creation and multivariate structure of SDQ. Sleep. 1994;**17**(2):160-167

[77] Dixit R. Asthma and obstructive sleep apnea: More than an association (editorial). Lung India. 2018;**35**:191-192

[78] Prasad B, Nyenhuis SM, Imayama I, Siddiqi A, Teodorescu M. Asthma and

obstructive sleep apnea overlap: What has the evidence taught us? American Journal of Respiratory and Critical Care Medicine. 2020;**201**(11):1345-1357

[79] Pan W, Kastin AJ. Can sleep apnea cause Alzheimer's disease? Neuroscience and Biobehavioral Reviews. 2014;**47**:656-669

[80] Legault J, Thompson C, Martineau-Dussault M-E, André C, Baril A-A, Villar GM, et al. Obstructive sleep apnea and cognitive decline: A review of potential vulnerability and protective factors. Brain Sciences. 2021;**11**:706

[81] Krysta K, Bratek A, Zawada K, Stepańczak R. Cognitive deficits in adults with obstructive sleep apnea compared to children and adolescents. Journal of Neural Transmission. 2017;**124** (Suppl. 1):S187-S201

[82] Dalmases M, Solé-Padullés C, Torres M, Embid C, Nuñez MD, Martinez-Garcia MA, et al. Effect of CPAP on cognition, brain function, and structure among elderly patients with OSA: A randomized pilot study. Chest. 2015;**148**(5):1214-1223

[83] Werli KS, Otuyama LJ, Bertolucci PH, Rizzi CF, Guilleminault C, Tufik S, et al. Neurocognitive function in patients with residual excessive sleepiness from obstructive sleep apnea: A prospective, controlled study. Sleep Medicine. 2016;**26**:6-11

[84] Edwards KM, Kamat R, Tomfohr LM, Ancoli-Israel S, Dimsdale JE. Obstructive sleep apnea and neurocognitive performance: The role of cortisol. Sleep Medicine. 2014;**15**:27-32

[85] Ciccone I, Mehra R. Lower oxygen levels in obstructive sleep apnea are associated with worsening mild cognitive impairment and Alzheimer disease. NeurologyLive. 2023;**6**(4):8-9. (Presented at the 2023 SLEEP Annual Meeting; 3-7 June 2023; Indianapolis)

[86] Mubashir T, Abrahamyan L, Niazi A, Piyasena D, Arif AA, Wong J, et al. The prevalence of obstructive sleep apnea in mild cognitive impairment: A systematic review. BMC Neurology. 2019;**19**:195

[87] Iranzo A, Fernández-Arcos A, Tolosa E, et al. Neurodegenerative disorder risk in idiopathic REM sleep behavior disorder: Study in 174 patients. PLoS One. 2014;**9**:e89741. DOI: 10.1371/journal.pone.0089741

[88] Iranzo A, Santamaria J. Severe obstructive sleep apnea/hypopnea mimicking REM sleep behavior disorder. Sleep. 2005;**28**(2):203-206

[89] Zhang L, Hou Y, Po SS. Obstructive sleep apnoea and atrial fibrillation. Arrhythmia and Electrophysiology Review. 2015;**4**(1):14-18

[90] Marulanda-Londoño E, Chaturvedi S. The interplay between obstructive sleep apnea and atrial fibrillation. Frontiers in Neurology. 2017;**8**:668

[91] Mehra R, Benjamin EJ, Shahar E, et al. Association of nocturnal arrhythmias with sleep-disordered breathing: The sleep heart health study. American Journal of Respiratory and Critical Care Medicine. 2006;**173**(8):910-916

Chapter 2

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 an 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
	Nasal polyps or any pathological discharge
Pharynx	Tongue abnormalities
	Palatal/uvular elongation
	Tonsillar hypertrophy
	Lateral wall collapse
Larynx	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

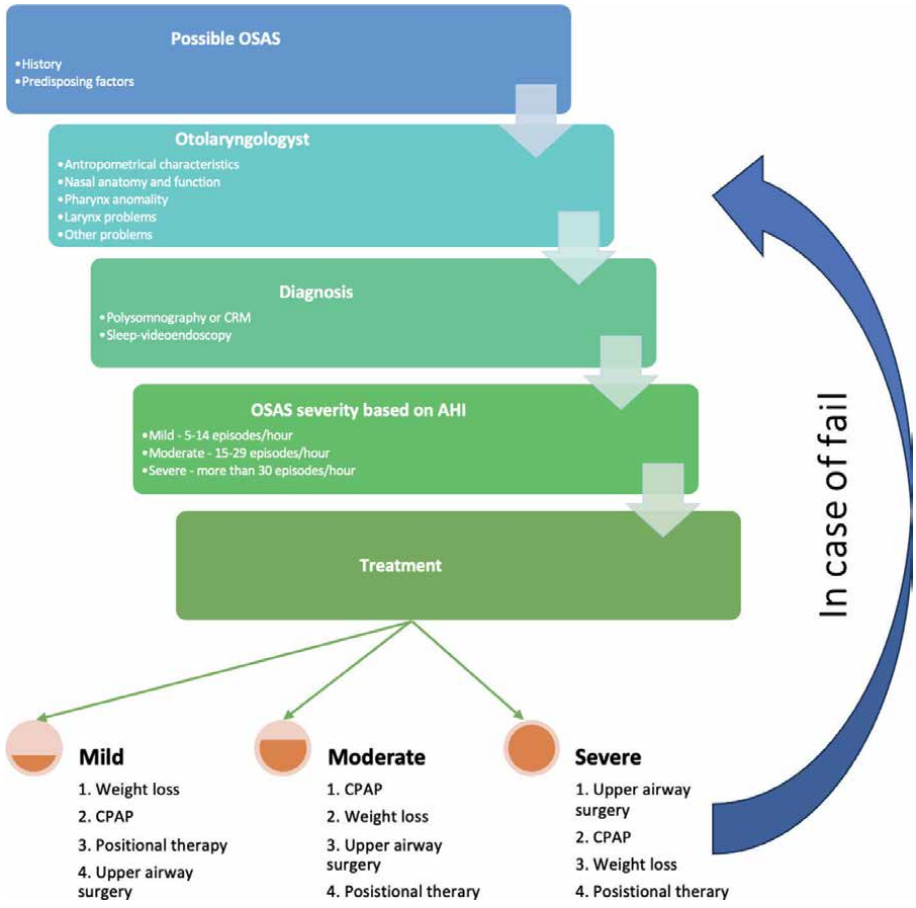

Figure 1.
Management of OSAS.

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].

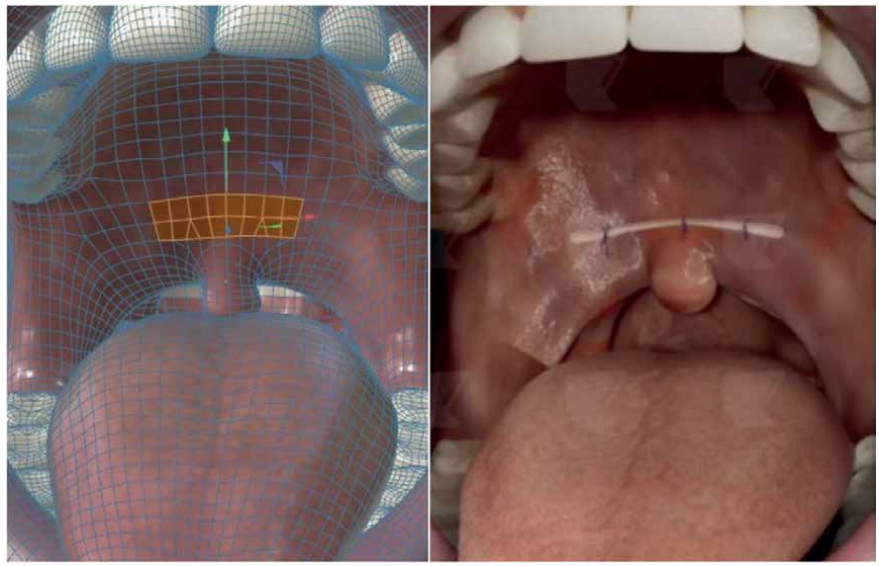

Figure 2.
UPPP operative technique. On the left image illustrated possible mucous membrane removing zone and, on the right is a final result with suturing.

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

DOI: http://dx.doi.org/10.5772/intechopen.1002896

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

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] MininYV. 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, Nilsen 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' Ambrodgino FB, Sant' AG. Effect of airway- surface

liquid composition on laryngeal muscule 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 uvulopalatopharingoplasty. Larynioscope. 1985;**95**:70-74

[35] Fujita S et al. Surgical correction of anatomic abnormalities in OSAS: Uvulopalatopharingoplasty. 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 furlow 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

Chapter 3

Epidemiology of Childhood Sleep Apnea

Elizabeth Jones

Abstract

Childhood sleep apnea is characterized as airway obstruction because of upper airway impairment due to reduced oxygen levels or awakening from sleep in children. In children, only 1–5% of children suffer from sleep apnea, which equals to about 20 million–100 million children, globally. However, childhood sleep apnea tends to be underdiagnosed with about 90% of children not being properly diagnosed with sleep apnea. Sleep apnea is a major concern due to the harmful clinical manifestations associated with the condition, such as abnormal breathing during sleep, frequent awakenings or restlessness, frequent nightmares, nocturnal enuresis, difficulty getting up in the morning, excessive daytime sleepiness (EDS), ADHD-like symptoms, daytime mouth breathing, and abnormal sleep patterns. There are also certain risk factors that can increase the risk of childhood sleep apnea. These risk factors are tonsillar hypertrophy, adenoid hypertrophy, obesity, craniofacial anomalies, and neuromuscular disorders. Childhood risk factors are important to consider because they can increase risk and severity. To aid an efforts to relieve, prevent, and diagnose childhood sleep apnea, there needs to be more efforts to promote, to educate, and to implement diagnostic testing for sleep apnea as a standard of care in pediatric patients.

Keywords: childhood, sleep apnea, airway obstruction, epidemiology, apnea risk

1. Introduction

Childhood sleep apnea is characterized as airway obstruction because of upper airway impairment due to reduced oxygen levels or awakening from sleep in children. Childhood sleep apnea is less commonly diagnosed and can severely affect functioning, growth, behavior, and development. Knowledge of childhood sleep apnea is very important because it allows adults to understand the source of certain behaviors, promote awareness, and to ensure the proper diagnosis of children with sleep apnea. This chapter introduces several concepts including, prevalence of sleep apnea, risk factors, clinical manifestations, diagnosis, screening, and recommendations.

2. Prevalence of sleep apnea in children

"Globally, around 1 billion people suffer from sleep apnea" [1]. "In children, only 1–5% of children suffer from sleep apnea, which equals to about 20 million–100 million children, globally" [2]. While the prevalence of sleep apnea is lower in children than adults, childhood sleep apnea is usually not diagnosed as frequently as in adults. "About 90% of children are underdiagnosed for sleep apnea" [3]. Childhood sleep apnea is usually underdiagnosed because of the difficulty detecting the condition in children.

2.1 Prevalence of sleep apnea in children by sex

"The prevalence of childhood sleep apnea is equal in boys and girls, which equivocates to a prevalence of 10 million–50 million for both boys and girls" [4]. "Both boys and girls, who have asthma or are obese have an increased risk of childhood sleep apnea" [5]. While childhood asthma and obesity have been shown to increase the risk of childhood sleep apnea, the role that asthma and obesity play in sleep apnea severity is unknown in both boys and girls. "Other risk factors that are associated with childhood sleep apnea in both boys and girls are down syndrome, facial birth defects, birth defects in the skull, cerebral palsy, sickle cell disease, neuromuscular disease, low birth weight, and family history of sleep apnea" [6].

2.2 Prevalence of sleep apnea in children by age and race

Table 1 indicates that the prevalence of sleep apnea for children between the ages 0–4 years is 0.66%, the prevalence of sleep apnea for children between the ages of 5–9 years is 1.37%, the prevalence of sleep apnea for children between the ages of 10–14 years is 1.56%, and the prevalence of sleep apnea for children between the ages of 15–19 years is 1.14% [7]. **Table 1** shows that children between the ages of 10–14 years have a higher prevalence of sleep apnea. Typically, children between the ages of 10–14 are experiencing puberty. At times, puberty has been shown to increase the risk of sleep apnea because growth spurts can cause sleep disorder breathing.

Table 1 also indicates that the prevalence of sleep apnea in Black children is 47.1%, the prevalence of sleep apnea in Hispanic children in 12.5%, and the prevalence of sleep apnea in White children is 40.4%. "**Table 1** shows Black children have a higher prevalence of sleep apnea" [8]. "Studies have shown that

	Age	Race
Sleep apnea prevalence (%)	0–4 years: 0.66% 5–9 years: 1.37% 10–14 years: 1.56% 15–19 years: 1.14%	Black: 47.1% Hispanic: 12.5% White: 40.4%
Total number of sleep apnea cases:	0–4 years: 13,200,000 5–9 years: 27,400,000 10–14 years: 31,200,000 15–19 years: 22,800,000	Black: 942,000,000 Hispanic: 250,000,000 White: 808,000,000

Table 1.
Sleep apnea by age & race in children.

Black children have a higher odds of developing sleep apnea and often have a 20% increase in the severity of sleep apnea" [8].

3. Background: snoring in children

Snoring is a severe sound that obstructs air through the nose and pharynx. "Generally, snoring is quite common in children" [9]. Since snoring is seen as common, it is not seen as a concern. "However, habitual snoring can be a sign of sleep apnea. "Habitual snoring is defined as snoring 3 or more nights per week" [10, 11].

4. Prevalence of snoring in children

"Globally, 60,000,000 million–240,000,000 million children snore, accounting for a prevalence of 3–12%" [12]. In children, "African Americans have a higher prevalence of snoring" [13]. **Figure 1** also indicates the prevalence of habitual snoring by age. Children between the ages of 11–13 have a higher prevalence of habitual snoring.

5. Risk factors in children

"These risk factors are tonsillar hypertrophy, adenoid hypertrophy, obesity, craniofacial anomalies, and neuromuscular disorders" [14, 15]. Childhood risk factors are important to consider because they can increase risk and severity.

5.1 Tonsillar hypertrophy

Tonsillar hypertrophy is a condition characterized by swollen or enlarged tonsils. Tonsillar hypertrophy is commonly diagnosed in children. Tonsillar hypertrophy

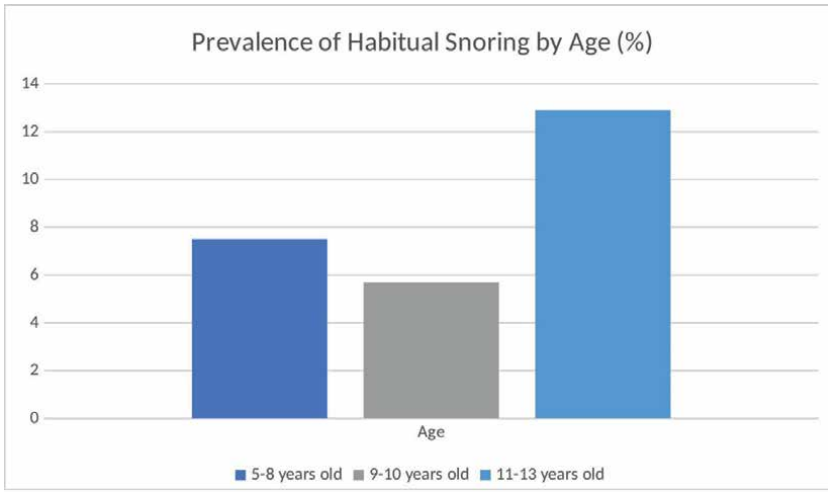

Figure 1.
Habitual snoring by age children.

increases the risk of childhood sleep apnea because the condition causes the narrowing of the upper airways, which can lead to airway obstruction.

5.2 Adenoid hypertrophy

Adenoid hypertrophy is characterized by enlarged or swollen adenoids. When adenoid hypertrophy and reduced muscle tones are both present simultaneously in children, the conditions significantly increase the risk of childhood sleep apnea. Both conditions simultaneously cause severe airway obstruction.

5.3 Obesity

Obesity is characterized as a body mass index of 30 or higher. Body mass index is the measure of body fat by height and weight. Obesity is a very common risk factor in children diagnosed with sleep apnea. Fat build-up in the upper respiratory track reduce airway flow, which leads to decrease muscle activity. The reduction of muscle activity results in hypoxia and apneic episodes, which causes sleep apnea.

5.4 Craniofacial anomalies

Craniofacial anomalies are characterized by facial and head deformities in children. "Generally, certain craniofacial anomalies put children more at risk of sleep apnea including, maxillary hypoplasia, mandibular hypoplasia, poor motor tone, crowded oropharynx, and macroglossia" [16]. These conditions tend to cause air narrowing and obstruction.

5.5 Neuromuscular disorders

Neuromuscular disorders are characterized as peripheral nervous system diseases. "Specifically, polymyositis, dermatomyositis, and inclusion body myositis are neuromuscular conditions that cause sleep apnea in children" [17]. These conditions result in sleep apnea due to a loss of muscle tone, respiratory muscles weakness, and movement impairment.

6. Prevalence of risk factors in children

6.1 Prevalence of tonsillar hypertrophy by age, sex, and race

1–5% of children with sleep apnea have tonsillar hypertrophy. Usually, children between the ages of 2–8 years old have a higher prevalence of tonsillar hypertrophy. The prevalence of tonsillar hypertrophy is also associated with sex in children. Boys diagnosed with tonsillar hypertrophy have a higher prevalence of sleep apnea compared to girls. The prevalence of tonsillar hypertrophy in boys is 55.4% compared to 44.6% in girls. However, there is no known association between tonsillar hypertrophy in childhood sleep apnea cases and race.

6.2 Prevalence of adenoid hypertrophy by age, sex, and race

1–5% of children with sleep apnea have adenoid hypertrophy. Usually, children ages 2–8 years old have a higher prevalence of adenoid hypertrophy. However, sex and

race do not play a role in the relationship between adenoid hypertrophy and childhood sleep apnea.

6.3 Prevalence of obesity by age, sex, and race

"13–59% of children with sleep apnea are obese" [18]. Typically, boys, who are obese have a higher prevalence of sleep apnea compared to girls, who are obese. In terms of age, obese adolescents have a higher prevalence of sleep apnea than obese children of other ages. However, the relationship between race and obesity in childhood sleep apnea cases vary.

6.4 Prevalence of craniofacial anomalies by age, sex, and race

"Around 16% of children with craniofacial anomalies have sleep apnea" [16]. "However, children with craniofacial anomalies are 30 times more likely to be diagnosed with sleep apnea compared to children without craniofacial anomalies" [16]. Currently, there is no known association between age, sex, and race in children with craniofacial anomalies and sleep apnea diagnosis.

6.5 Prevalence of neuromuscular disorders by age, sex, and race

"40% of children with neuromuscular disorders have childhood sleep apnea" [18]. "Generally, children between the ages of 10–12 years old with neuromuscular disorders have a higher prevalence of sleep apnea" [18]. However, the relationship between sex and race in children diagnosed with neuromuscular disorders and sleep apnea vary.

7. Genetic risk factors in children

A genetic risk factor is the probability of having a genetic mutation or disorder associated with a disease. Genetic risk factors in children such as, down syndrome, prader-willi syndrome (PWS), smith-magensis syndrome, and angelman syndrome (AS) often significantly increase the risk of obstructive sleep apnea compared to children without these conditions. Sleep apnea in children with down syndrome, PWS, smith-magensis syndrome, or AS can result in very harmful health outcomes both physically, mentally, and developmentally.

7.1 Down syndrome

Children with down syndrome have an increased risk of sleep apnea. Down syndrome is a genetic disease associated with an extra chromosome 21. Individuals with down syndrome have certain physical features associated with their genetic condition, such as enlarged adenoids, tongue, tonsils, flat face, short neck, and poor muscle tone. These physical features are the reason for their increased risk of sleep apnea compared to other children.

7.2 Prader-Willi syndrome

Commonly, children with prader-willi syndrome (PWS) have an increased risk of sleep apnea. PWS is a genetic condition that results in numerous physical, mental,

and behavioral challenges. The most common symptom associated with PWS is experiencing constant hunger. Due to constant hunger being the most common symptom of the condition, children with PWS are usually obese. Being obese increases the risk of childhood sleep apnea.

7.3 Smith-Magenis syndrome

Children with smith-magenis syndrome have higher rates of sleep apnea than children without the condition. Smith-Magenis syndrome is a developmental disorder associated with emotional challenges, behavioral challenges, and difficulty with learning processes. Individuals with Smith-Magenis syndrome also have certain phenotypic facial features that increase their risk of childhood sleep apnea.

7.4 Angelman syndrome

Children diagnosed with AS also have an increased risk of sleep apnea. AS is a genetic disease associated with developmental challenges, problems with balance and speech, and seizures. Individuals with AS often have a happy disposition and are easily excitable. Generally, children with AS have difficulty falling asleep, remaining asleep, and have various other sleep related conditions.

8. Prevalence of genetic risk factors in children

8.1 Prevalence of sleep apnea in children with down syndrome by age, sex, and race

"The prevalence of sleep apnea in children with down syndrome is 53–76% compared to children without down syndrome, who have disease prevalence of 1–5%" [19]. Prevalence of sleep apnea is also higher in younger children with down syndrome compared to older children with down syndrome. However, the prevalence of sleep apnea does not differ by sex or by race and ethnicity in children with down syndrome.

8.2 Prevalence of sleep apnea in children with prader-willi syndrome by age, sex, and race

"The prevalence of sleep apnea in children with PWS is 57%" [20]. Prevalence of sleep apnea is also higher in older children (>2 years) with PWS compared to younger children with the condition [20]. However, the prevalence of sleep apnea does not differ by sex or by race and ethnicity in children with PWS.

8.3 Prevalence of sleep apnea in children with angelman syndrome by age, sex, and race

"The prevalence of sleep apnea in children with AS is 20–80%" [21]. Prevalence of sleep apnea is higher for children between 2 and 6 years and diminishes as a child progresses through late childhood [21]. However, the prevalence of sleep apnea does not differ by sex or by race and ethnicity in children with AS.

9. Clinical manifestations

Clinical manifestations vary for all children. Clinical manifestations for childhood sleep apnea include abnormal breathing during sleep, frequent awakenings or restlessness, frequent nightmares, nocturnal enuresis, difficulty getting up in the morning, excessive daytime sleepiness (EDS), daytime mouth breathing, abnormal sleep patterns, non-rapid eye movement (NREM) parasomnias, ADHD-like syndrome, and cognitive and neuropsychological conditions. Clinical manifestations present in children tend to make day to day activities difficult for parents and children.

9.1 Abnormal breathing during sleep

"Abnormal breathing during sleep is characterized by the ceasing of breathing during sleep" [22]. Breathing cessation is usually the result of airway blockage from enlarged tonsils or adenoids in the upper airway. Abnormal breathing can be observed in children by noticing brief pauses during sleep.

9.2 Frequent awakenings or restlessness

Frequent awakenings or restlessness is characterized by disruptions from sleep or awakening or inability to sleep. Frequent awakenings are a result of lacking oxygen, which result in the initiation of the survival reflex. The survival reflex is used to allow the child to return to normal breathing by waking-up.

9.3 Frequent nightmares

Frequent nightmares are characterized by unpleasant dreams during sleep. Children, who suffer from sleep apnea experience abnormal or fragmented sleep patterns that can cause high alert in the brain. By the brain being in high alert, it can result in mental distress that can be expressed as nightmares in children.

9.4 Nocturnal enuresis

Nocturnal enuresis is bedwetting that occurs at night. Enuresis is a secondary condition that occurs due to exhaustion from the inability to sleep due to childhood sleep apnea. "Children, who experience exhaustion or extreme fatigue may not be able to control their bladder as well as children, who are not suffering from sleep apnea" [23].

9.5 Difficulty getting up in the morning

Children with sleep apnea may have trouble getting up in the morning due to irritability from the lack of a normal sleep pattern caused by childhood sleep apnea. The irritability that a child may experience during awakening can last throughout the day, which can result in issues in the classroom.

9.6 Excessive daytime sleepiness

EDS is characterized as the inability to remain awake or alert during the daytime. EDS in children can result in failing grades, emotional disturbances, and

inattentiveness. Obese children with sleep apnea are more likely to suffer from EDS than non-obese children with EDS at any given severity level of sleep apnea.

9.7 ADHD-like symptoms

ADHD-like symptoms are characterized as disruptive behavior in children and is a mimicry condition to ADHD. ADHA-like symptoms are associated with childhood sleep apnea. ADHD-like symptoms make it difficult for children to function and concentrate properly during the day.

9.8 Daytime mouth breathing

Children with sleep apnea tend to suffer from adenoid hypertrophy and tonsillar hypertrophy. These conditions usually cause daytime mouth breathing. Daytime mouth breathing is characterized as a child breathing through their mouth instead of their nose during the daytime hours. Daytime mouth breathing can cause harm to a child's mental and oral health. Mentally, daytime breathing can result in issues with problem solving and concentration. In terms of oral health, long-term daytime mouth breathing can cause gum disease, caries, crowded teeth, and cracked lips.

9.9 Sleep patterns

Sleep patterns are characterized as the typical sleep cycle that individuals undergo each night. "A typical sleep pattern has 4 to 6 cycles each night. These cycles last 80–100 minutes and involve two phases of sleep" [24]. "Children with moderate sleep apnea have an abnormal sleep pattern that results in children waking up 120–239 minutes per 8 hours of sleep" [25]. "Children with severe sleep apnea may wake-up 30 or more times per hour" [25].

9.10 Non-rapid eye movement parasomnias

NREM parasomnias is characterized as "sleep walking and confusional arousals during the first half of the night" [26]. Children with sleep apnea may experience NREM parasomnias. NREM parasomnias are caused by cortical arousals and/or sleep inertia caused by abnormalities in arousal mechanisms due to sleep apnea.

9.11 Deficits of IQ and executive function

Children diagnosed with sleep apnea also suffer from cognitive and neuropsychological effects. Children with sleep apnea may have lower IQs and poor executive functioning. Developing children with untreated sleep apnea can have their cognitive function altered permanently.

10. Diagnosis

Clinical symptoms of sleep apnea do not manifest in the same manner in children as adults. For children, clinical manifestations tend to affect behavior, which can result in hyperactivity and inability to concentrate. While the clinical manifestation of symptoms differs for adults and children, symptoms of sleep apnea fluctuate for

all children. Due to fluctuation of clinical symptoms in children, diagnosis criteria, clinical criteria, and the assessment of severity play a major role in understanding the extent of sleep apnea in children.

10.1 Diagnosis criteria

Diagnosis criteria for childhood sleep apnea are factors used in medical practice to direct the care of young patients through the assessment of symptoms, signs, and tests. Generally, diagnosis criteria are very important aspects of medical practice and patient care because it ensures that patients receive the best possible care. In children, the diagnosis criteria for sleep apnea are vital in preventing, understanding, and identifying declines in oxygen levels, or increases in carbon dioxide levels.

According to the American Academy of Family Physicians (AAFP), "the golden standard for the diagnosis criteria of sleep apnea in children is measured using the apnea-hypopnea index (AHI)" [27]. The AHI is the average rate of breathing disturbances or apneas or shallow or unusually slow breathing patterns or hypopneas. The AAFP has set the golden standard for the diagnosis criteria of childhood sleep apnea as an "AHI of greater than 1 or an average of 0.1–0.5 events per hour or a minimum oxygen saturation level of less than 92%" [27].

10.1.1 Clinical criteria

Clinical criteria are guidelines that are used to assess a patient's condition to establish the patient's medical needs. In children with sleep apnea, clinical criteria are used to assess a child's condition to lead to a diagnosis. The clinical criteria of childhood sleep apnea are "snoring, labored, paradoxical, or obstructed breathing during sleep, sleepiness, hyperactivity, behavioral problems, or learning problems" [28].

10.1.2 Polysomnography

Polysomnography (PSG) is a test that is used to diagnose sleep apnea in adults and children. PSG diagnoses sleep disorders by measuring AHI or respiratory disturbance index (RDI). Typically, the most recommended environment for the use of PSG in children is to use an overnight PSG in a sleep laboratory. **Figure 2** depicts PSG in a child with severe obstructive sleep apnea.

10.2 Assessment of severity

The assessment of severity is used to describe the intensity of sleep apnea cases in children. There are three grades of severity- mild obstructive sleep apnea, moderate obstructive sleep apnea, and severe obstructive sleep apnea. "Mild sleep apnea is defined as an AHI of greater than 1 to less than 5" [29]. "Moderate sleep apnea is defined as an AHI of greater than or equal to 5 or less than 10" [29]. "Severe sleep apnea is defined as an AHI of greater than or equal to 10" [29].

10.3 Preliminary classification using respiratory disturbance index/apnea-hypopnea index

Both RDI and AHI can be used as a diagnosis and assessment tool for obstructive sleep apnea in children. While both RDI and AHI are similar, RDI also includes

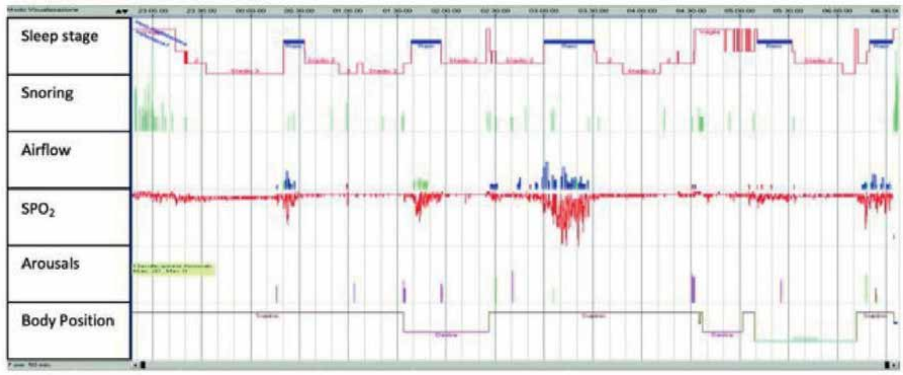

Figure 2.
PSG in a child with severe obstructive sleep apnea [21].

respiratory effort-related arousal (RERAs) as a standard for diagnosing and assessing severity of sleep apnea. RERAs is a disorder caused by airflow reduction in the upper airways and leads to increases in respiratory effort that settles by the appearance of arousal.

11. Screening

Screening is the application of tools used to detect potential disease occurrence in patients, who are without symptoms. Medically, symptoms are considered the subjective experience of patients, who are dealing with a health concern. The screening process for childhood sleep apnea involves the use of the pediatric sleep questionnaire (PSQ) to identify potential risk. Followed by using Nocturnal Polysomnogram (PSG) or the Nocturnal Pulse Oximetry to physically confirm cases of childhood sleep apnea. Generally, Nocturnal Polysomnogram is conducted in a sleep laboratory, while Nocturnal Pulse Oximetry is conducted at home.

11.1 Pediatric sleep questionnaire

"The Pediatric Sleep Questionnaire (PSQ) is a 22-item survey that accesses for apneas, snoring, sleepiness, and other childhood sleep apnea symptoms" [30]. PSQ is scored using 0 and 1 s with 0 s meaning that the symptom is absent and 1 s meaning that the symptom is present. "A final score of greater than or equal to 0.33 is used to indicate a risk of childhood sleep apnea" [30].

11.2 Nocturnal polysomnogram

Nocturnal PSGs conducted in sleep laboratories are considered the gold standard for detecting childhood sleep apnea. A nocturnal PSG can identify both obstructive events and assess severity. However, the process of scheduling nocturnal PSGs for pediatric patients is rather complex. Nocturnal PSGs for children are generally ordered based on several factors, including the decision of the healthcare provider, age, comorbidities, the preference of the parent or guardian, tolerance of sensors, and the availability and accessibility of PSG testing.

The technical process of nocturnal PSG testing involves the use of sensors to track brain waves, oxygen levels, breathing, heart rate, and physical movement, such as eye and leg movement. Typically, nocturnal PSGs are performed in sleep laboratories within hospitals or sleep centers. The set-up time for PSGs can be timely and the detection of nocturnal seizures can also extend the time further.

11.3 Nocturnal pulse oximetry

A nocturnal pulse oximetry is a device that is used to detect oxygen levels in the blood as a means for indicating the presence of childhood sleep apnea. Typically, nocturnal pulse oximetry is used at home during the night and is easy to use. The device has an apparatus that connects to one finger, which makes it easy to set-up and easy to manage for parents of young patients.

12. Summary and recommendations

Childhood sleep apnea is a very serious condition in children. While childhood sleep apnea may not be as common as adult sleep apnea, the effects of the disease is still very harmful and is often underdiagnosed in children. The effects of childhood sleep apnea tend to affect the day-to-day activities of children. Certain populations of children are also more at risk of sleep apnea due to genetic factors, and certain conditions, such as obesity, neuromuscular disorders, and craniofacial anomalies. To aid an efforts to relieve, prevent, and diagnose childhood sleep apnea, there needs to be more efforts to promote the commonality of the disease in children, to educate parents on identifying the symptoms, to explore new treatment options [31], andto promote the implementation of diagnostic testing for sleep apnea as a standard of care in pediatric patients.

Author details

Elizabeth Jones
Department of Epidemiology and Biostatistics, Jackson State University, Jackson, MS, USA

*Address all correspondence to: eakjones@yahoo.com

References

[1] Lyons MM, Bhatt NY, Pack AI, Magalang UJ. Global burden of sleep-disordered breathing and its implications. Respirology. 2020;**25**(7):690-702. DOI: 10.1111/resp.13838

[2] Cleveland Clinic Medical. Is it sleep apnea? [Internet]. Available from: https://my.clevelandclinic.org/health/diseases/14312-obstructive-sleep-apnea-in-children

[3] Science X. Up to 15 percent of children have sleep apnea, yet 90 percent go undiagnosed [Internet]. Medical Xpress; 2019. Available from: https://medicalxpress.com/news/2019-02-percent-children-apnea-undiagnosed.html#:~:text=Up%20to%2015%20percent%20of%20children%20have%20some%20form%20of,to%20psychological%20or%20emotional%20issues

[4] Togeiro S, Santos C, Tufik S, Smith A, Moreira G. 0814 obstructive sleep apnea in asthmatic children: highly prevalent though no identifiable risk factors. Sleep. 2023;**46**(Supplment_1):00049-9. DOI: 10.1093/sleep/zsad07 7.0814

[5] American Lung Association. The link between asthma and weight [Internet]. Available from: https://www.lung.org/blog/the-link-between-asthma-weight

[6] Pediatric obstructive sleep apnea [Internet]. Mayo Foundation for Medical Education and Research; 2023. Available from: https://www.mayoclinic.org/diseases-conditions/pediatric-sleep-apnea/symptoms-causes/syc-20376196

[7] Lumeng JC, Chervin RD. Epidemiology of pediatric obstructive sleep apnea. Sleep and Breathing in Children. 2008;**5**(2):429-450. DOI: 10.3109/9781420060836-23

[8] Dudley KA, Patel SR. Disparities and genetic risk factors in obstructive sleep apnea. Sleep Medicine. 2016;**18**:96-102. DOI: 10.1016/j.sleep.2015.01.015

[9] Tan Y, How C, Chan Y, Teoh O. Approach to the snoring child. Singapore Medical Journal. 2020;**61**(4):170-175. DOI: 10.11622/smedj.2020054

[10] Isaiah A, Ernst T, Cloak CC, Clark DB, Chang L. Association between habitual snoring and cognitive performance among a large sample of preadolescent children. JAMA Otolaryngology–Head & Neck Surgery. 2021;**147**(5):426. DOI: 10.1001/jamaoto.2020.5712

[11] Pediatric sleep-disordered breathing [Internet]. 2022. Available from: https://www.enthealth.org/conditions/pediatric-sleep-disordered-breathing/

[12] Children with sleep apnea [Internet]. 2022. Available from: https://www.pinnacledentalgroupmi.com/obstructive-sleep-apnea-in-children-dont-ignore-a-childs-snoring/

[13] Song S. Snoring at night may affect kids' daytime behavior [Internet]. Time; 2012. Available from: https://healthland.time.com/2012/08/13/how-a-childs-nighttime-snoring-may-affect-daytime-behavior/

[14] Baidas L, Al-Jobair A, Al-Kawari H, AlShehri A, Al-Madani S, Al-Balbeesi H. Prevalence of sleep-disordered breathing and associations with orofacial symptoms among Saudi primary school children. BMC Oral Health. 2019;**19**(1):43. DOI: 10.1186/s12903-019-0735-3 81

[15] Xu Z, Wu Y, Tai J, Feng G, Ge W, Zheng L, et al. Risk factors of obstructive sleep apnea syndrome in children. Journal of Otolaryngology - Head & Neck Surgery. 2020;**49**(1):11. DOI: 10.1186/ s40463-020-0404

[16] Lam DJ, Jensen CC, Mueller BA, Starr JR, Cunningham ML, Weaver EM. Pediatric sleep apnea and craniofacial anomalies: a population-based case-control study. The Laryngoscope. 2010;**120**(10):2098-2105. DOI: 10.1002/ lary.21093

[17] Lacomis D. Neuromuscular disorders in critically ill patients: review and Update. Journal of Clinical Neuromuscular Disease. 2011;**12**(4):197-218. DOI: 10.1097/ cnd.0b013e3181b5e14d

[18] Kinimi L. Clinical Profile of Children with Suspected Sleep Apnea [Internet]. 2022. Available from: https://journals.lww.com/jpp/_layouts/15/oaks.journals/PageNotFound.aspx

[19] Sleep & down syndrome [Internet]. 2023. Available from: https://ndss.org/resources/sleep-down syndrome#:~:text=Children%20 syndrome%20are,Facial%20 structure%20differences

[20] Hedstrom S. Sleep apnea in people with PWS [Internet]. 2017. Available from: https://www.fpwr.org/blog/sleep-apnea-in-people-with pws#:~:text=Approximately%20 57%25%20of%20PWS%20 patients,suffer%20from%20 obstructive%20sleep%20apnea

[21] Lo, Bue A, Salvaggio A, Insalaco G. Obstructive sleep apnea in developmental age. A narrative review. European Journal of Pediatrics. 2020;**179**(3):357-365. DOI: 10.1007/ s00431-019-03557-8

[22] Tobin M. Breathing abnormalities during sleep. Archives of Internal Medicine. 1983;**143**(6):1221-1228. DOI: 10.1001/archinte.143.6.1221

[23] Claire McCarthy M. A tired child? what you should know [Internet]. 2020 Available from: https://www.health.harvard.edu/blog/a-tired-child-heres-what-to-think-about-2018041713672

[24] Stages of sleep: What happens in a sleep cycle [Internet]. 2023 Available from: https://www.sleepfoundation.org/stages-of-sleep

[25] Professional CC medical. Sleep apnea: What it is, causes, symptoms & treatment [Internet]. 2022. Available from: https://my.clevelandclinic.org/health/diseases/8718-sleep-apnea

[26] Parasomnias in children [Internet]. 2012. Available from: https://bestpractice.bmj.com/topics/en-us/117 7

[27] Understanding the apnea-hypopnea index (AHI) [Internet]. 2023. Available from: https://www.sleepfoundation.org/sleep-apnea/ahi#:~:text=Mild%3A%20 Children%20with%20an%20 AHI,diagnosed%20with%20severe%20 sleep%20apnea

[28] Gipson K, Lu M, Kinane B. Sleep-disordered breathing. Surgery for Sleep-Disordered Breathing; **1**(1):1-2. DOI: 10.1007/3-540-27608-4_1

[29] Savini S, Ciorba A, Bianchini C, Stomeo F, Corazzi V, Vicini C, et al. Assessment of obstructive sleep apnoea (OSA) in children: an update. Acta Otorhinolaryngologica Italica. 2019;**39**(5):289-297. DOI: 10.14639/0392-100x-n0262

[30] Umano GR, Rondinelli G, Luciano M, Pennarella A, Aiello F, di Santo M, et al. Pediatric sleep

questionnaire predicts moderate-to-severe obstructive sleep apnea in children and adolescents with obesity. Children. 2022;**9**(9):1303. DOI: 10.3390/children9091303

[31] Gozal D, Tan H-L, Kheirandish-Gozal L. Treatment of obstructive sleep apnea in children: Handling the unknown with precision. Journal of Clinical Medicine. 2020;**9**(3):888. DOI: 10.3390/jcm9030888

Chapter 4

Perspective Chapter: Role of the Oral Healthcare Team in the Management of Obstructive Sleep Apnea

Michael Greaves, Dwight McLeod and Ignacio Christian Marquez

Abstract

Obstructive sleep apnea (OSA) represents a significant health issue with numerous social and health ramifications caused by disturbed sleep. Additionally, it is well-known that OSA has an influence on cardiovascular diseases. OSA has a multifactorial etiology, and therefore requires a multidisciplinary approach for both diagnosis and treatment. Traditionally, dentists have treated OSA at the request of physicians and do not routinely contribute to diagnosis and treatment; however, the awareness of OSA is increasing within the healthcare profession and the potential role of dental practitioners in its treatment is an emerging field. The dental profession is in a unique position to work with their medical colleagues in providing treatment which can dramatically improve quality of life.

Keywords: obstructive sleep apnea, dentist, oral healthcare team, mandibular advancement device, oral appliance therapy

1. Introduction

Oral healthcare professionals can play an important role in the screening, evaluation for clinical signs, and clinical management of obstructive sleep apnea (OSA). Dentists and other oral healthcare professionals are trained to evaluate for the clinical signs of obstructive sleep apnea, such as large tonsils, micro or retrognathia, bruxism, TMD, etc. Working with a patient's team of medical professionals, the oral healthcare team can help provide treatment for mild to moderate OSA that includes the fabrication of oral appliances, management of dental considerations, and follow-up care to maximize treatment efficacy. In instances where patients cannot tolerate other treatment modalities, oral appliance therapy can also help in managing patients with severe OSA. Due to the high rates of patient adherence to oral appliance therapy and its efficacy, oral appliance therapy is similar in effectiveness to CPAP therapy in the treatment of mild to moderate OSA.

2. Relevant anatomy

2.1 Oropharynx

The pharynx is located in the midline of the neck and connects the oral cavity to the gastrointestinal tract and the trachea. It is funnel-shaped with the upper portion being larger and is at the base of the skull, while the lower end is narrower and located at the level of the sixth cervical vertebra (C6). This is where the pharynx meets the larynx and the esophagus. It is formed of muscles and mucous membranes, which allows it to serve several functions related to both digestion and respiration, including food swallowing, air conduction, and voice production [1–3].

The pharynx is divided into three parts from superior to inferior: the nasopharynx, located behind the nasal conchae, the oropharynx, located posterior to the oral cavity, and the laryngopharynx, which is inferior to the epiglottis. The nasopharynx is part of the respiratory tract and conducts air from the nasal passages. Also, the lateral surface of the posterior wall of the nasopharynx includes two openings, which are called the auditory (or Eustachian/pharyngotympanic) tubes. These tubes are connected to the middle ears posteriorly. Their main function is to equalize pressure and to drain secretions of the middle ears. The oropharynx is a continuation of the oral cavity and serves to pass the bolus of food to the laryngopharynx.

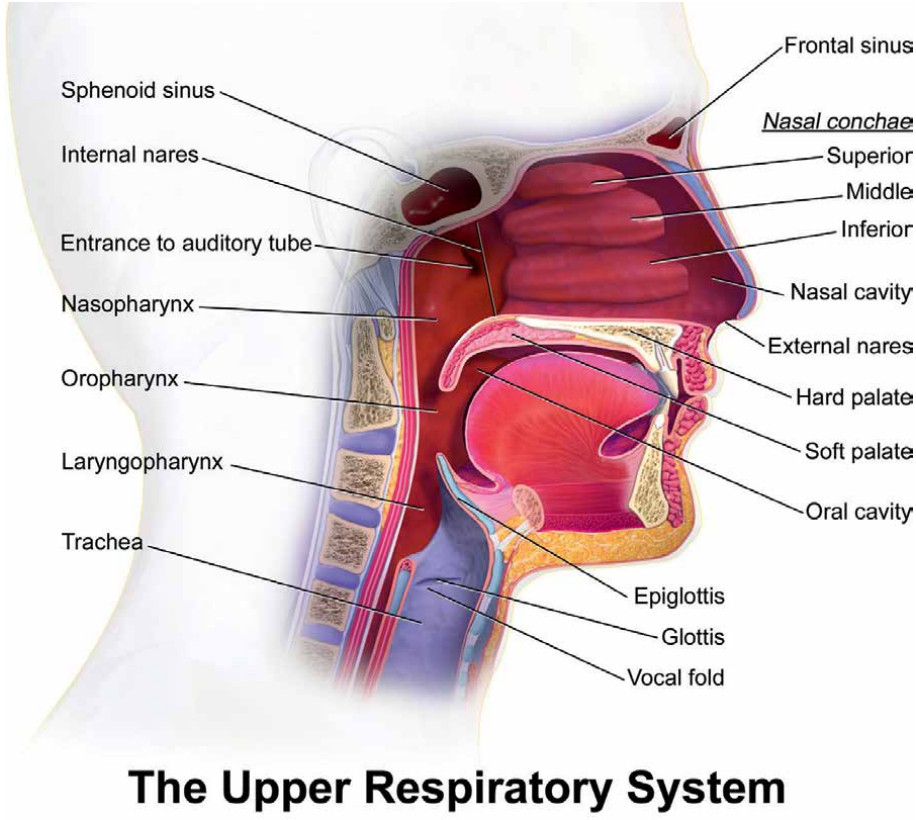

Figure 1.
Anatomy of the upper airway [4].

The muscles of the soft palate contract to close the nasal cavity as the bolus passes from the oral cavity. This prevents the bolus from entering the nasal cavity. At the same time, the epiglottis (which is cartilage located at the superior portion of the larynx) is pushed anteriorly to close the opening to the airway and prevent food from entering. Finally, the laryngopharynx receives the bolus and moves it into the esophagus to continue digestion. Alternatively, air moves from either the nasal cavity to the nasopharynx or from the oral cavity to the oropharynx and enters the laryngopharynx to the trachea to continue its path on the respiratory tract (**Figure 1**) [2, 5–7].

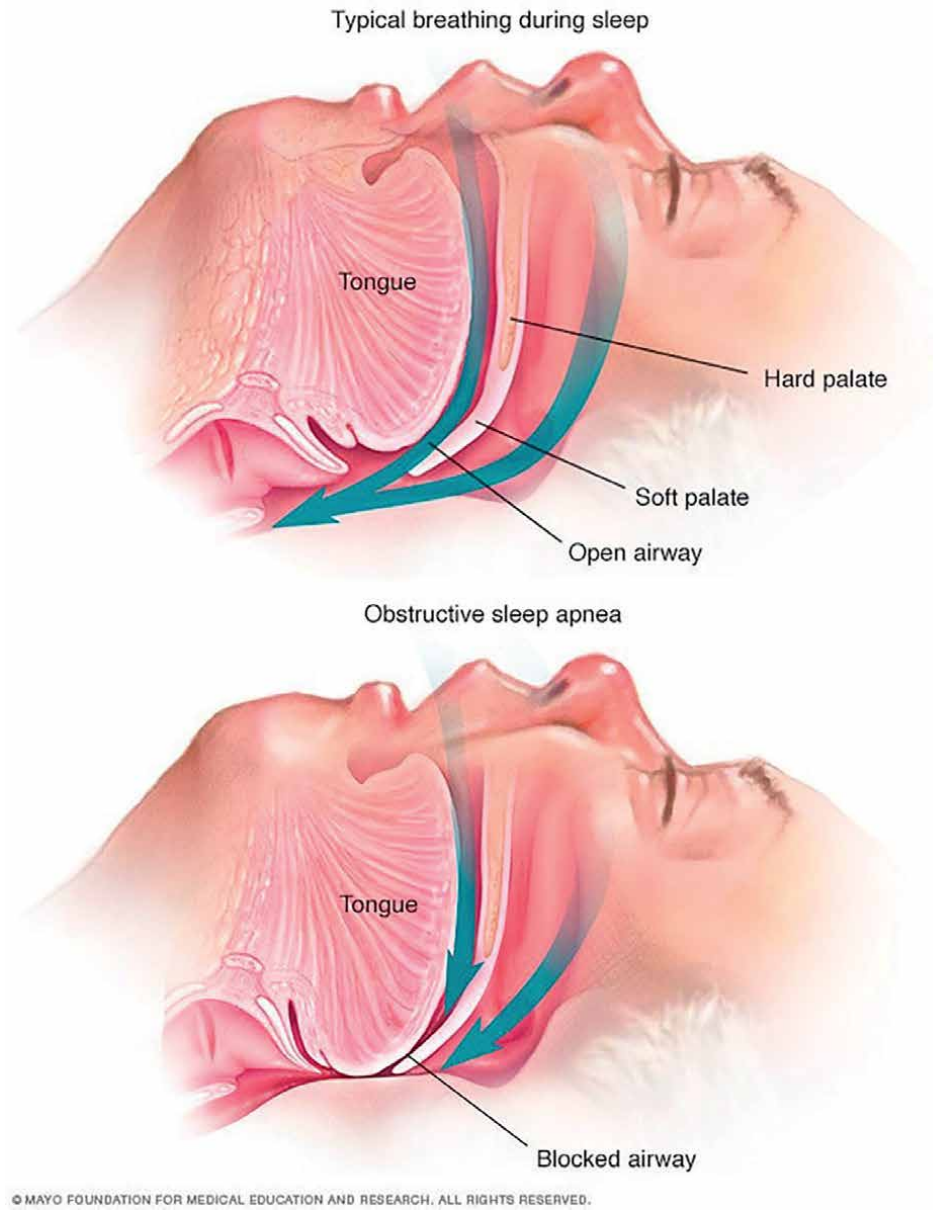

Figure 2.
Blockage of the airway in obstructive sleep apnea [16].

2.2 Pathophysiology of obstructive sleep apnea

The pathophysiology of OSA contains many factors and differs between individuals. The major contributing components that impact the severity of the obstructive sleep apnea include anatomic obstruction of the upper airway, low respiratory arousal threshold, high loop gain (unstable respiratory control), and poor upper airway dilator muscle responsiveness [8].

The most prominent feature of the treatment of OSA is a widening of the lateral diameter of the airway, especially in the area behind the soft palate [9, 10], likely through soft tissue connections between the lateral airway wall muscles and the mandible [11]. This lateral widening of the airway can be brought about by the advancement of the mandible.

In dentistry, however, there are signs possibly related to OSA that can be observed: a hard palate that is narrow or exhibits a high arch, relationship between the maxillary and mandibular jaws, relative tongue size and other soft tissue characteristics (e.g., enlarged uvula and narrowing of the airway from the tonsillar area) [12–14].

Obstructive sleep apnea (OSA) is defined as repetitive and intermittent blockage of the upper airway during sleep [15]. The pharyngeal walls collapse, leading to this constrictor or blockage of the airway. A complete closure of blockage leads to apnea and a partial closure results in hypopnea. There can be significant consequences and changes in the nervous and circulatory systems from continued apnea events. Snoring is caused by a narrowing of the pharynx. The soft portions of the upper airway (the pharyngeal walls, uvula, and soft palate) vibrate, leading to the snoring sound (**Figure 2**).

3. Common oral signs/symptoms

Dental professionals can identify many intraoral signs and symptoms associated with OSA during routine appointments in the dental office. Examples of the intraoral signs associated with OSA include redness of the soft palate and uvula area, narrow palate, enlarged tongue and bilateral mandibular tori. Symptoms may include dry mouth (xerostomia) and bruxism (grinding of the teeth). These signs and symptoms are not necessarily indicative of OSA, however, identification by dental professionals may serve the dental professional in identifying the need for further evaluation of OSA for the patient [17, 18]. There are several clinical indices, for example, the Mallampati index, that can be used to determine the risk level of the patient for OSA [19]. There are also several questionnaires/surveys, for example, the STOP-Bang, that can also be evaluated by dental professionals to help screen their patients for OSA [20–22]. OSA can negatively impact a patient's oral and overall health, therefore dental professionals serve as an important resource within health care for identifying patients at risk for OSA and identifying non-adherent CPAP or OAT users.

A patient that exhibits a neck circumference greater than 40 centimeters, macroglossia, Mallampati score of Class 3 or 4, and a deep palatal vault have been shown to be predictive for a high risk for OSA [23]. When the dental professional identifies a patient exhibiting one or more of these findings, the dental professional should discuss the patient's sleep history and screen the patient with a validated questionnaire and refer the patient for further evaluation/diagnosis if appropriate.

OSA should be seen as a condition that needs to have the signs/symptoms identified in dental appointments and should be included in a routine oral exam [24]. The inter-professional collaboration between dental and medical professionals to

promote quality care for patients with potential OSA should also be brought to the attention of dental/medical professionals in their education. This is in line with the new oral health definition [20], which brings a holistic view of different domains of overall health that impact oral health. Driving determinants, one domain of oral health, are factors that affect oral health: genetic and biological factors, social environment, physical environment, health behaviors, and access to care. This new definition and framework are used "to explain the multidimensions of oral health to our patients, other healthcare professionals, policy makers, and those others we seek to collaborate with and inform" [20]. Thus, understanding associations between oral health and OSA and interprofessional collaborations within healthcare is of importance. Increased training on OSA is required for oral health professionals. This additional knowledge is an important step that would improve patient communication, education, and treatment in the dental field along with increasing interprofessional collaboration between the oral health professionals and medical professionals.

4. Impact of OSA on oral health and outcomes of oral healthcare

OSA is a widely prevalent problem in the general population [25]. Untreated OSA is associated with long-term health consequences including hypertension, heart disease, diabetes, depression, metabolic disorders, and stroke. The high risk group of OSA include patients with ischemic heart disease, heart failure, arrhythmias, cerebrovascular diseases, and type II diabetes [26]. Untreated OSA has been shown to be associated with cognitive dysfunction, impaired productivity in the workplace, and an increased risk of motor vehicle accidents, which could result in increased risk for injury or death. Impaired vigilance, daytime somnolence, performance deficits, morning headaches, mood disturbances, neurobehavioral impairments, and general malaise are reported in individuals with OSA [27]. These long-term health consequences can drastically reduce the quality of life and wellbeing, leading to premature death. Whether diagnosed or undiagnosed, OSA is a serious threat to the overall health and longevity in those individuals who are burdened by this widely prevalent sleep-related breathing disorder [28–32].

Obstructive sleep apnea is characterized by frequent episodes of airflow obstruction associated with a reduced caliber of the upper airway and is vulnerable to further narrowing and collapse. Both acute and continued effects of apnea and hypopnea include oxygen desaturation [33], reduction in intrathoracic pressure, excessive daytime sleepiness, impaired cognitive function and central nervous system arousals [27, 34]. Obesity is one of the major predisposing factors [35]. Three types of apneas have been recognized, obstructive, central and mixed with Obstructive sleep apnea being the most common [36]. Weight loss can lead to improvement in OSA. Treatments for OSA are based upon a thorough medical and physical examination and a sleep study or polysomnography. Medications are not effective in the management of sleep apnea but could help with reducing the effects of pathogenic mechanisms [22, 36–38]. **Table 1** highlights some of the potential risk factors/pathogenic mechanisms for OSA as outlined by Jordan et al. Addressing individual pathogenic mechanisms may be alternative treatments even though it is understood that Constant Positive Airway Pressure (CPAP) is the acceptable treatment for Obstructive Sleep Apnea.

This table is a modification of **Figure 3**. Risk factors, pathogenic mechanism, and possible treatments for obstructive sleep apnea as presented by Jordan et al. [37].

Risk factors	Pathogenic mechanism	Possible treatment
Nasal Congestion	Small Upper Airway Lumen	Surfactant
Breathing	Surface Forces	
Genetic & Ethnic Origin	Small Upper Airway Lumen	Mandibular Advancement Device
Craniofacial Structure	Small Upper Airway Lumen	Unknown
Obesity	Low Lung Volume	Oxygen or Drugs
	Respiratory Instability	
Sex	Small Upper Airway Lumen	Mandibular Advancement Device
	Low Lung Volume	Unknown
	Respiratory Instability	Oxygen or Drugs
Age	Low Lung Volume	Unknown
	Respiratory Instability	Oxygen or Drugs
	Poor Airway Muscle Function	Hypoglossal Nerve Stimulation
	Low Arousal Threshold	Sedatives

Table 1.
Potential risk factors/pathogenic mechanism for obstructive sleep Apnea & Treatment.

Phrase	Question
Snore	Do you snore loudly?
Tired	Do you feel tired during the daytime often?
Observed	Has anyone observed you
Pressure (Blood Pressure)	Are you being treated or have you been treated for high blood pressure?
Body Mass Index	Is your BMI greater than 35?
Age	Are you over 50 years of age?
Neck Circumference	Is your neck circumference greater than 40 cm?
Gender	Are you male?

Figure 3.
STOP-bang sleep apnea questionnaire [39].

5. Oral-related conditions associated with OSA

Mouth breathing is a common finding in patients diagnosed with OSA or those who show signs of OSA but are not diagnosed. Xerostomia is often associated with mouth breathers and can predispose to caries and periodontal disease through complete or partial reduction of protective salivary flow to portions of the dentition during sleep or during the day. OSA and periodontal disease share some of the same risk factors and it is not uncommon to see a greater periodontal disease prevalence in patients with OSA [40]. Common risk factors may include obesity, gender, male more than female, age, smoking and mouth breathing resulting in xerostomia, and alcoholism. Additionally, increased adrenocorticosteroids stemming from stress and fatigue of inadequate sleep can put a patient at risk for periodontal disease.

The association between xerostomia and OSA has been poorly investigated. Nocturnal xerostomia or sleep-related xerostomia causes discomfort or dryness in the mouth or throat making it difficult to swallow. In a clinical study investigating xerostomia and hyposalivation in patients with OSA, the authors reported the clinical implication that OSA patients showed a decreased pH value of oral fluid which may put patients at risk for the development of dental caries. They further demonstrated that dry mouth upon awakening is a common symptom with OSA and 22 (73.3%) of the 30 patients evaluated had dry mouth and the prevalence of sleep-related xerostomia is correlated with OSA severity. In the majority of patients, dry mouth was correlated with mouth breathing and not salivary hypofunction and only 20% of the patients exhibited objective signs of hyposalivation [41].

The quantity and quality of saliva serve as a protective mechanism for the hard and soft tissue of the periodontium while allowing optimal physiologic functions of phonation, mastication and homeostasis. When the quantity and quality of saliva are affected, the tendency for disease is increased because of loss of the beneficial elements in saliva and a likelihood for increase in bacterial plaque accumulation on soft and hard tissue structures leading to inflammation of soft tissues and the potential for the development of periodontal disease and dental caries. Duplancic and colleagues, evaluating salivary parameters and periodontal inflammation in OSA patients, showed that patients with hyposalivation and reduced salivation had higher concentrations of salivary electrolytes and lower salivary pH than subjects with normal salivation and that patients with severe OSA tended to have a higher clinical attachment levels and plaque volume. The authors noted that multiple interactions might impact salivary flow and electrolyte composition and that complex interrelationships might affect the integrity of oral health, especially considering OSA severity, inflammation, concomitant diseases and medication [40].

5.1 Temporomandibular joint disease

Individuals suffering from OSA complain of snoring, experienced apnoeas, waking up with a choking sensation, excessive sleepiness [42], fatigue or exhaustion, morning headache [43], and even temporomandibular joint pain [44]. A recent systematic review, linking an association between temporomandibular disorders (TMD) and their association with sleep disorders in adults, concluded that there is inclusive evidence between the relationship of TMD and sleep bruxism (SB) and insufficient evidence regarding the relationship with obstructive sleep apnea (OSA). There is consistent evidence that supports a link between TMD and sleep quality [45]. In a review of over 706 reports on tooth wear and the mentioned dental sleep disorders, the authors concluded that tooth wear is associated with the dental sleep disorders orofacial pain, dry mouth, GERD, and sleep bruxism. As these dental sleep disorders are interlinked, it is difficult to determine the significant consequences of each individual disorder and leads more to indirect associations [46].

6. Screening for OSA in the dental office

In this era where interprofessional education and interprofessional collaboration care are embedded in the curriculum and taught in most professional schools to a certain degree, obstructive sleep apnea (OSA) is often a common topic that brings professional teams together for the health benefit and wellbeing of the

patient. Dental and dental hygiene students are learning more about how to incorporate assessment tooling to screen for OSA. There is evidence that the dental team spends more time with their patients than their medical counterparts. According to the ADA Health Policy Resources Center, in any given year 27 million Americans visit a dentist but do not see a physician. Another 108 million visit a physician but do not see a dentist, including more than 60 percent of children aged 1 through 4 years. Increased interprofessional collaboration between dentists and medical professionals will help to raise the awareness of providers and their patients to oral and overall health [47].

Dentists and dental hygienists are exceptionally educated to counsel patients on oral health and wellness topics. Dentists and dental hygienists' in-depth knowledge of the anatomy of the head and neck, in particular the oral cavity and oropharyngeal area makes them well suited to assisting the medical team in identifying high risk patients [48] and referring them for definitive diagnosis and treatment. Berggren et al. reported that dental professionals, including dentists and hygienists, in general dentistry experience with OSA varies widely which leads to oral and overall health problems (such as OSA) not being recognized regularly. This lack of experience can be attributed to lack of knowledge about OSA and of validated indices/questionnaires that can be used to determine OSA risk or detect patients at risk of OSA [49].

It is important to train oral health professionals about OSA as an oral health determinant and which may promote interprofessional collaboration between dental and medical professionals. Practicing dental hygienist and dentists can play even a greater role in screening by the incorporation of screening questions as part of their routine medical history, identifying potential risk factors (retrognathia, high arched palate, enlarged tonsils or tongue, enlarged tori, high Mallampati score, poor sleep, supine sleep position, obesity, hypertension, morning headache or orofacial pain, bruxism) and collaborating with the medical team in making referrals [50]. The earlier undiagnosed cases are referred to the medical team from screening the dental office, the sooner cases can be diagnosed and treated which may reduce the morbidity and possible mortality of patients, especially those with severe OSA [51]. The dentist and dental team should be proactive in screening patients for OSA and making timely referrals for diagnosis and treatment.

The **Table 2** outlines some surgical considerations to manage snoring and OSA or both. Surgical interventions are not always effective and predictable in the management of OSA and/snoring, but there are notable advantages to surgery which may widen airway spaces allowing better flow of oxygen and breathing [36, 60].

CPAP is the gold standard for treatment of OSA [61], and other treatment options can be considered for treating mild to moderate OSA cases such as oral appliances [51]. The dental team is suited to play a role in the treatment and management of OSA when oral appliances are recommended by the sleep physician. Referrals can be reciprocated back to the dental team from the medical team for those patients who are diagnosed but cannot tolerate continuous positive airway pressure (CPAP) therapy and could benefit from oral appliance, including tongue positioning and mandibular advancing devices. OSA is the most common sleep disorder [25] and with increasing awareness and diagnosis, healthcare providers can work with dentists [60], given their knowledge of the structures and function of the oral cavity, to provide a customized treatment plan that is cost effective, elicits compliance and improves sleep quality, culminating in a better quality of life.

Tonsillectomy [52]	**Recommended for enlarged tonsils, reducing obstruction to breathing**
Genioglossus Tongue Advancement [52]	Creates a wider space at the posterior base of the tongue for improved breathing
Uvulopalatopharyngoplasty [53, 54]	Effective in snoring reduction but not a predictive therapy for OSA
Laser-assisted Uvulopalatoplasty [55]	More applicable for snoring reduction and not a predictive therapy for OSA
Maxillomandibular Advancement [56, 57]	A more aggressive treatment consideration when other treatment procedures are not effective in controlling OSA
Radio frequency or Somnoplasty [58]	A treatment consideration which shrinks internal tissue leaving external tissues intact and requires multiple treatment sessions. Effective against snoring and OSA
Hyoid Suspension [52, 56]	Leads to a wider airway and is effective an effective treatment for OSA
Tracheostomy [52, 59]	Old treatment method which is considered when OSA is severe, CPAP is ruled out and cardio-pulmonary failure has developed
Bilateral Mandibular Torectomy	Prevents posterior displacement of tongue

Table 2.
Obstructive sleep apnea and its surgical management [36].

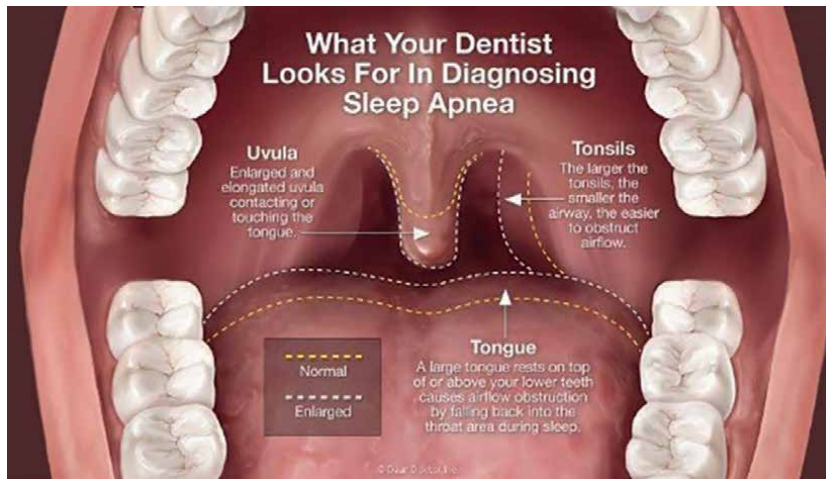

Figure 4.
Intraoral signs of obstructive sleep apnea [62].

Oral appliances, mainly the mandibular advancement devices which move the mandible forward and open the airway during sleep are used to treat mild to moderate OSA, can be beneficial for patients who do not tolerate CPAP, patients who are at high risk for surgery and patients who decline surgery as a treatment option. Treatment approach for oral appliances is simple, noninvasive, cost effective and reversible. Observed oral changes associated with oral appliances in the management of OSA include tooth mobility and repositioning, temporomandibular joint pain, tenderness of teeth, sore oral soft tissues and muscle pain (**Figure 4**).

7. Oral appliance therapy

7.1 Goals and mechanism of action of oral appliance therapy

Oral appliance therapy is an effective therapy for the treatment of obstructive sleep apnea. The goal of therapeutic interventions for OSA varies in literature but has a common factor in the decrease in the apnea-hypopnea index (AHI) [63]. The AHI is defined as the average number of apneas/hypopneas that occur per hour of sleep [64]. Mild OSA exhibits an AHI of 5–15, moderate OSA exhibits an AHI of 15–30, and severe OSA is classified as an AHI > 30 [64]. Generally, successful OSA treatment is recognized as an AHI <5, a 50% reduction in AHI, to a combination of a decrease in the AHI from baseline and final AHI [63]. Approximately two-thirds of patients treated with OAT will achieve a decrease in AHI greater than 50%, with at least one-third of these patients showing a complete response (AHI < 5) [10].

Oral appliances help treat OSA in three ways: maintaining the mandible in a more closed position, maintaining the mandible in a more anterior position (protrusion), and anterior movement of the tongue [63, 65, 66].

Protrusion of the mandible is the primary mechanism of action in oral appliances fabricated for the treatment of OSA [63]. The protrusion of the mandible leads to an increase in the space of the airway, created by a widening of the airway laterally in the velopharynx (posterior surface of the hard palate to the posterior wall of the pharynx) [63]. The advancement of the mandible and the protrusion of the tongue lead to an improvement, or decrease, in the collapsibility of the airway [66, 67], likely due to the airway enlargement [63]. This reduction in collapsibility of the airway decreases the number of episodes of complete or partial collapse of the airway, thereby improving the AHI of the patient [68]. Patients with mild OSA generally respond better to oral appliance therapy, as their airways are less collapsible and they tend to show a greater increase in the size of their pharynx with OAT [69]. It is not entirely clear why the protrusion of the mandible and tongue leads to stretching of the soft tissue connections between the mandible, tongue, lateral pharyngeal walls, and soft palate, which leads to an overall enlargement of the velopharynx as shown in **Figure 5** [70].

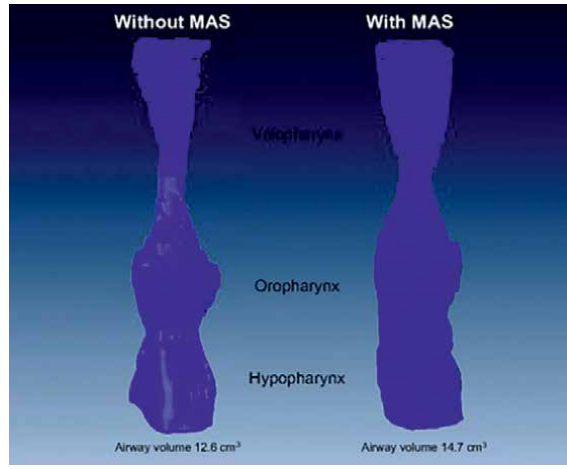

Figure 5.
Enlargement of the Velopharynx with mandibular advancement [70].

Maintaining the mandible in a more closed position allows for a reduction in the collapsibility of the airway [71]. Mouth breathing not only leads to an increase in airway collapsibility, it compromises the adherence of patients to OAT as compared to patients that breathe through their nose [72]. This leads to a decrease in overall efficacy of the treatment as the patients are not only wearing the appliances less but the appliances are less effective when they are worn.

7.2 Classifications of oral appliances

Oral appliances fabricated for the treatment of OSA are classified into two categories: mandibular advancement splits (MAS) or mandibular advancement devices (MAD) and tongue retaining devices (TRD) [63, 73]. MAS devices act by advancing the mandible forward, therefore reducing the collapsibility of the airway and increasing the size of the airway [73]. Tongue retaining devices work by suctioning the tongue in an anterior position [63, 73]. Since TRD's have limited evidence showing their efficacy in the treatment of OSA [74], clinical guidelines relate to mandibular advancement devices (**Figures 6** and **7**) [63, 77].

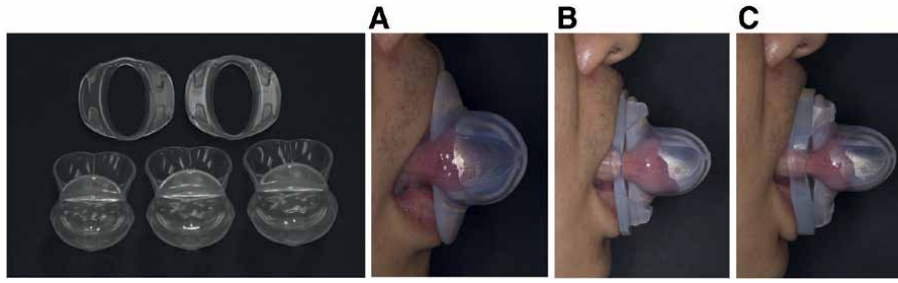

Figure 6.
Titratable tongue retaining device [75].

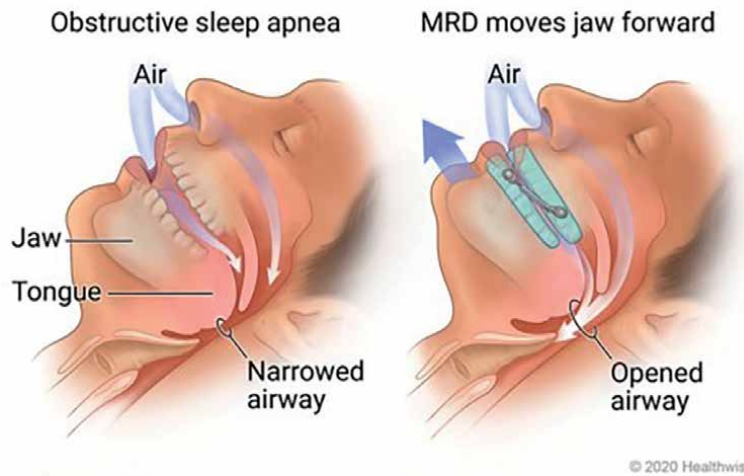

Figure 7.
Mandibular advancement device [76].

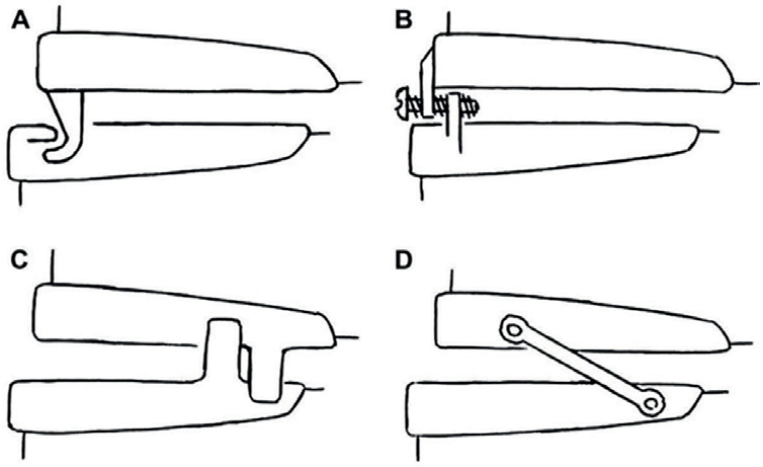

Figure 8.
Example schematics of mandibular advancement devices [78].

There are a wide variety of MADs, ranging from custom-made to prefabricated [78]. Prefabricated MADs are made from thermoplastic material and are generally a lower cost alternative, as they are not custom fitted using bite registrations or plaster casts [78]. Custom-made appliances are fabricated using casts and a bite registration made by the oral healthcare team [78]. Evidence suggests that custom-made MADs are more efficacious in reducing OSA severity than the prefabricated MADs [79]. Due to the reduced efficacy of thermoplastic prefabricated MADs, these devices should not be used as a screening tool for the success of treatment OSA using OAT [79]. MADs vary from "monobloc," where the upper and lower devices are attached, to "duobloc," where the upper and lower devices are separated. The benefit of the newer duobloc designs is that they allow for titration of the advancement of the mandible and lead to less discomfort of the TMJ [78]. There are several types of duobloc designs summarized below in **Figure 8** that allow for titration in a variety of ways [78].

7.3 Titration of oral appliances

The amount of protrusion of the mandible is a key factor in the efficacy of MAD therapy [80]. However, more protrusion does not necessarily mean a better result for the patient [80]. Since the optimal protrusion of the mandible depends on the individual patient, the device must be titrated to determine the greatest efficacy versus tolerability [80]. It has been suggested by literature that titration of the patient in the range of 50–75% of the patient's MMP is the most efficacious, with evidence supporting no clinical difference between 50 and 75% MMP [81]. An increase in the protrusion can lead to heavier forces applied to the oral/maxillofacial system and lead to more side effects, including TMD and dental changes [81]. Therefore, a titration target of 50% of MMP is most beneficial in the decrease in airway collapsibility, increase in airway space, and limitation of potential side effects [81].

Evaluating the titration can be accomplished using a variety of methods. The patient should return to the dentist within 30 days to evaluate the calibration of the device The device can be titrated using a standardized stepwise protocol, advancing the protrusion if subjective criteria (such as daytime sleepiness or perceived reduction in apneic

events) do not show signs of improvement and a reduction of the protrusion if there are side effects reported by the patient [82]. Evaluation of the device can also be completed using an at home monitoring device, such as a high-resolution pulse oximeter or home sleep apnea test (HSAT) [83]. The results of these objective tests can be sent to the patient's physician, who can then discuss the titration of the device with the oral healthcare team. The HSAT must be evaluated by the patient's physician [82]. Once the dentist has determined that the patient has achieved final calibration, the patient is referred back to the treating physician for evaluation of the success of the OAT [83].

7.4 Side effects/potential complications of oral appliance therapy

It is important that the dentist/oral healthcare team discuss fully with the patient the potential for complications and the importance of routine follow-ups to evaluate for both efficacy and possible side effects. During the initial stages of OAT, patients may report soreness or tenderness of the TMJ, teeth, and gums, along with either excessive salivation or dryness of the mouth [84]. These side effects are typically transient and can be related to the strain in the musculature of the due to protrusion or an increase in vertical dimension while the patient is wearing the appliance [84]. The first line treatment for soreness of the TMJ or muscles of mastication, joint sounds, excess salivation, and dry mouth associated with the appliance is watchful waiting [85]. During this time, the dentist will carefully observe and document the patient's symptoms and discuss follow-up, depending on the persistence of the side effect [85]. Fortunately, long-term discomfort or impact that is detrimental to the TMJ do not seem to be long-term side effects [84]. Pain tends to decrease after 1–2 years, likely due to the adaptability of the temporomandibular complex [84].

A key part of the OAT would be the fabrication of a morning occlusal guide, also known as an AM aligner [85]. These guides function by engaging the patient's biting force to help maintain the patient's normal occlusal relationship in the morning following the use of the OAT at night [85]. These guides may also help in the stretching of the muscles of mastication [85]. This morning occlusal guide is fabricated by the oral healthcare team/dental laboratory and is made in the patient's normal occlusion [85]. If the patient notices that he/she is unable to maintain the proper occlusion following wear of the oral appliance, the patient should immediately follow up with his/her oral healthcare provider to prevent permanent changes to the dentition or joint [85].

If the patient is experiencing soft-tissue irritation, gagging, or appliance breakage, modification of the appliance is necessary [85]. Passive jaw stretching exercises and palliative care with the use of intermittent application of ice, a soft diet, and use of anti-inflammatory medication are the first line therapy for tenderness in the muscles of mastication or persistent TMJ pain throughout the day [85]. Changes to the dentition, decreased overbite/overjet, and tooth mobility all require thorough evaluation by the dentist. In these cases, decreasing the titration of the appliance, changing the design of the appliance, or ceasing oral appliance therapy may be required [85]. To help avoid these changes, it is important for the patient to routinely use the morning occlusal guide.

Skeletal changes caused by the downward rotation of the mandible and an increase in the lower facial height are common with mandibular advancement OAT [84]. There is also a tendency for the development of a bilateral crossbite in the posterior dentition after long-term oral appliance therapy use [84]. Using a stepwise approach to the titration and calibration of the oral appliance helps lower the risk of mandibular advancement beyond the optimal position and therefore lessens the side effects experienced by

• Transient morning jaw pain
• Persistent temporomandibular joint pain
• Tenderness in muscles of mastication
• Joint sounds
• Intraoral tissue-related side effects
• Soft tissue and tongue irritation
• Gingival irritation
• Excessive salivation/drooling
• Dry mouth
• Occlusal changes
• Altered occlusal contacts/bite changes
• Incisor changes
• Decreased overjet and overbite
• Alterations in position of mandibular canines and molars
• Interproximal gaps
• Damage to teeth or restorations
• Tooth mobility
• Tooth fractures or damage to dental restorations
• Appliance issues
• Appliance breakage
• Allergies to appliance material
• Gagging
• Anxiety

Table 3.
Oral appliance therapy related side effects [85].

the patient [84]. Due to the potential side effects and changes, it is important that the patient seek routine follow-up with the oral healthcare provider to prevent long-term changes that are detrimental to the patient's health or function [85]. In some cases, it is prudent to cease OAT and consult with the patient's physician (**Table 3**) [85].

7.5 Benefits of oral appliance therapy

Although there are potential complications with OAT, there are also numerous benefits that make OAT an appropriate first line therapy for patients with mild to moderate OSA or who are not able to tolerate PAP therapy in severe OSA. OAT and PAP have equivalent health outcomes [63]. This is due to the fact that PAP therapy is highly efficacious but has moderate usage/compliance, while OAT therapy is moderately efficacious with higher usage [63, 86]. Although PAP has a higher efficacy, it has a lower rate of compliance when compared to OAT and therefore has a similar effectiveness [86]. Furthermore, studies show patient preference toward OAT over PAP [86]. OAT has shown to be either equal or superior to PAP therapy when evaluating

quality of life and subjective sleepiness scores [86]. Along with better patient compliance, OAT also shows an improvement over PAP in social factors such as perceptions of the partner, which would lead to further success of the OAT through better adherence [87]. Although PAP has been shown to be a highly efficacious treatment for OSA, OAT should also be considered for patients due to the improvements over PAP in adherence to OAT.

7.6 Combination therapy

Oral appliance therapy can be used in conjunction with other treatment modalities to improve adherence and patient outcomes. Using oral appliance therapy in conjunction with PAP can reduce the PAP pressure requirement [63, 88]. When used in combination, PAP and OAT together have been shown to decrease both AHI and oxygen desaturation of the patient [88]. Furthermore, patients undergoing combination PAP and OAT had lowered therapeutic pressures, which helps with patient compliance in patients who are PAP intolerant [88]. OAT has also been shown to be beneficial and an increase in efficacy when used in combination with uvulopalatopharyngoplasty (UPPP) vs. UPPP alone [89]. Along with the benefits of combining OAT with other treatments, combination therapy can allow for patients to use the treatments interchangeably depending on the patient's preference [63]. For example, when a patient is traveling, the patient can use the oral appliance as a short term treatment [90]. Oral appliance therapy can be used to help improve outcomes when paired with other therapies or can provide a short term therapy for patients when compliance is difficult.

8. Professional organizations

With the increase in the use of oral appliance therapy, there has been development in the number and specificity of organizations focused on the treatment of obstructive sleep apnea with oral appliance therapies. In the United States, the American Academy of Dental Sleep Medicine (AADSM) was founded in 1991 as the Sleep Disorders Dental Society and is the only non-profit national professional society dedicated solely to the practice of dental sleep medicine [91]. In 2004, the American Board of Dental Sleep Medicine was established. To support the training of qualified dentists, the AADSM has Mastery Programs that certify a dentist as a "Qualified Dentist" to practice dental sleep medicine. The AADSM has worked with the American Academy of Sleep Medicine (AASM) to establish guidelines for interdisciplinary treatment of OSA between physicians and dentists [92]. Furthermore, the American Dental Association has released an evidence brief [93] for the use of oral appliances for sleep-related breathing disorders and a policy statement in 2019 outlining the Role of Dentistry in Sleep Related Breathing Disorders [94]. In this policy statement, dentists are encouraged to screen for SRBDs and establishes that OAT is an appropriate treatment for patients with mild and moderate OSA or for severe OSA when CPAP is not tolerated by the patient [94]. In Europe, the European Academy of Dental Sleep Medicine promotes best practices to support the dental treatment of sleep related breathing disorders [95]. The growth of dental sleep medicine has been supported by the development of numerous organizations to help promote and guide the interdisciplinary treatment of OSA between dentists and physicians.

9. Conclusion

OSA is a common sleep-related breathing disorder which predisposes to life-threatening disease and can affect the quality and well-being of life. Early screening is an effective approach which could lead to referral, diagnosis and treatment. The dental team can play a vital role in this process by incorporating an in-office screening program and making referrals to the medical team. Reciprocating referrals from the medical team for fabrication of oral appliances is another way the dental team can help to improve the quality of life and well-being of patients who are diagnosed with OSA.

Acknowledgements

The authors acknowledge Ms. Maud Mundava for her assistance in conducting the literature search and compiling the references. The authors acknowledge Ms. Alyssa Timmer for her assistance in formatting the chapter. The authors would also like to acknowledge Ms. Danielle Williams for her assistance in obtaining permissions for the images and figures in this chapter. The authors would like to thank Dr. Hanan Omar for help in obtaining funding for this chapter and the ATSU Division of Research, Grants and Scholarly Innovations for their financial support in publishing this chapter.

Conflict of interest

The authors have no conflicts of interest to disclose.

Author details

Michael Greaves*, Dwight McLeod and Ignacio Christian Marquez
Missouri School of Dentistry and Oral Health, A.T. Still University,
St. Louis, Missouri, United States

*Address all correspondence to: michaelgreaves@atsu.edu

References

[1] Sakamoto Y. Gross anatomical observations of attachments of the middle pharyngeal constrictor. Clinical Anatomy. 2014;**27**(4):603-609

[2] Heyd C, Yellon R. Anatomy, Head and Neck, Pharynx Muscles. Treasure Island (FL): StatPearls Publishing; 2022

[3] Ball M, Hossain M, Padalia D. Anatomy, Airway. Treasure Island (FL): StatPearls Publishing; 2022

[4] Blausen.com Staff. Medical gallery of Blausen medical 2014. WikiJournal of Medicine. 2014;**1**(2). DOI: 10.15347/wjm/2014.010. ISSN 2002-4436

[5] Kagaya H, Yokoyama M, Saitoh E, et al. Isolated pharyngeal swallow exists during normal human feeding. The Tohoku Journal of Experimental Medicine. 2015;**236**(1):39-43

[6] Shaw SM, Martino R. The normal swallow: Muscular and neurophysiological conrol. Otolaryngologic Clinics of North America. 2013;**46**(6):937-956

[7] Casale J, Shumway KR, Hatcher JD. Physiology, Eustachian Tube Function. Treasure Island (FL): StatPearls Publishing; 2023

[8] Eckert DJ, Atul M. Pathophysiology of adult obstructive sleep apnea. Proceedings of the American Thoracic Society. 2008;**5**(2):144-153

[9] Chan AS, Lee RW, Srinivasan VK, et al. Nasopharyngoscopic evaluation of oral appliance therapy for obstructive sleep Apnoea. The European Respiratory Journal. 2010;**35**:836-842

[10] Chan AS, Sutherland K, Schwab RJ, Zeng B, Petocz P, Lee RW, et al. The effect of mandibular advancement on upper airway structure in obstructive sleep apnoea. Thorax. 2010;**65**(8):726-732. DOI: 10.1136/thx.2009.131094

[11] Brown EC, Cheng S, McKenzie DK, Butler JE, Gandevia SC, Bilston LE. Tongue and lateral upper airway movement with mandibular advancement. Sleep [Internet]. 2013;**36**(3):397-404. DOI: 10.5665/sleep.2458

[12] Adult Obstructive Sleep Apnea Task Force of the American Academy of Sleep Medicine. Clinical guidelines for the evaluation, management, and long-time care of obstructive sleep apnea in adults. Journal of Clinical Sleep Medicine. 2009;**5**:263-276

[13] Patil PS, Schneider H, Schwartz AR, et al. Adult obstructive sleep apnea pathophysiology and diagnosis. Chest. 2007;**132**:325-337

[14] Pahkala R, Puustinen R, Tuomilehto H, et al. Risk factors for sleep-disordered breathing: The role of craniofacial structure. Acta Odontologica Scandinavica. 2011;**69**:137-143

[15] Rodríguez-Lozano FJ, Sáez-Yuguero Mdel R, Linares Tovar E, et al. Sleep apnea and mandibular advancement device. Revision of the literature. Medicina Oral, Patología Oral y Cirugía Bucal. 2008;**13**(9):E549-E554

[16] Mayo Foundation for Medical Education and Research. Obstructive Sleep Apnea [Internet]. Mayo Foundation for Medical Education and Research; Available from: https://www.mayoclinic.org/diseases-conditions/obstructive-sleep-apnea/symptoms-causes/syc-20352090

[17] Ruangsri S, Jorns TP, Puasiri S, et al. Which oropharyngeal factors are significant risk factors for obstructive sleep apnea? An age-matched study and dentist perspectives. Nature and Science of Sleep. 2016;**8**:215-219 [Online]

[18] Vuorjoki-Ranta TR, Lobbezoo F, Vehkalahti M, et al. Treatment of obstructive sleep apnoea patients in community dental care: Knowledge and attitudes among general dental practitioners and specialist dentists. Journal of Oral Rehabilitation. 2016;**43**(12):937-942

[19] Chung F, Subramanyam R, Liao P, et al. High STOP-bang score indicates a high probability of obstructive sleep apnoea. British Journal of Anaesthesia. 2012;**108**(5):768-775

[20] Chung F, Yegneswaran B, Liao P, et al. STOP questionnaire: A tool to screen patients for obstructive sleep apnea. Anesthesiology: The Journal of the American Society of Anesthesiologists. 2008;**108**(5):812-821

[21] Mallampati S, Gatt S, Gugino L, et al. A clinical sign to predict difficult tracheal intubation: A prospective study. Canadian Anaesthetists' Society Journal. 1985;**32**(4):429-434

[22] Jauhar S, Orchardson R, Jauhar I, et al. The role of the dentist in sleep disorders. Dental Update. 2010;**37**(10):674-679

[23] Glick M, Williams DM, Kleinman DV, et al. A new definition for oral health developed by the FDI world dental federation opens the door to a universal definition of oral health. The Journal of the American Dental Association. 2016;**147**(12):915-917

[24] Kale SS, Kakodkar P, Shetiya SH. Assessment of oral findings of dental patients who screen high and no risk for obstructive sleep apnea (OSA) reporting to a dental college—a cross sectional study. Sleep Science. 2018;**11**(2):112-117

[25] Franklin KA, Lindberg E. Obstructive sleep apnea is a common disorder in the population-a review on the epidemiology of sleep apnea. Journal of Thoracic Disease. 2015;**7**(8):1311

[26] Garvey JF, Pengo MF, Drakatos P, Kent BD. Epidemiological aspects of obstructive sleep apnea. Journal of Thoracic Disease. 2015;**7**(5):920-929

[27] Morsy NE, Farrag NS, Zaki NFW, Badawy AY, Abdelhafez SA, El-Gilany A-H, et al. Obstructive sleep apnea: Personal, societal, public health, and legal implications. Reviews on Environmental Health (Berlin/Boston). 2019;**34**(2):153-169

[28] Stepnowsky C, Sarmiento KF, Bujanover S, Villa KF, Li VW, Flores NM. Comorbidities, health-related quality of life, and work productivity among people with obstructive sleep apnea with excessive sleepiness: Findings from the 2016 US National Health and wellness survey. Journal of Clinical Sleep Medicine. 2019;**15**(2):235-243

[29] Iacono Isidoro S, Salvaggio A, Lo Bue A, Romano S, Marrone O, Insalaco G. Quality of life in patients at first time visit for sleep disorders of breathing at a sleep centre. Health and Quality of Life Outcomes. 2013;**11**:207

[30] D'Ambrosio C, Bowman T, Mohsenin V. Quality of life in patients with obstructive sleep apnea: Effect of nasal continuous positive airway pressure—A prospective study. Chest. 1999;**115**(1):123-129

[31] Lacasse Y, Godbout C, Sériès F. Health-related quality of life in

obstructive sleep apnoea. The European Respiratory Journal. 2002;**19**(3):499-503

[32] Diamanti C, Manali E, Ginieri-Coccossis M, et al. Depression, physical activity, energy consumption, and quality of life in OSA patients before and after CPAP treatment. Sleep & Breathing. 2013;**17**(4):1159-1168

[33] Punjabi NM. The epidemiology of adult obstructive sleep apnea. Proceedings of the American Thoracic Society. 2008;**5**(2):136-143

[34] Young T, Peppard PE, Gottlieb DJ. Epidemiology of obstructive sleep apnea: A population health perspective. American Journal of Respiratory and Critical Care Medicine. 2002;**165**(9):1217-1239

[35] Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP. The spread of the obesity epidemic in the United States, 1991-1998. JAMA. 1999;**282**:1519-1522

[36] Sunitha C, Kumar SA. Obstructive sleep apnea and its management. Indian Journal of Dental Research. 2010;**21**(1):119-124

[37] Jordan AS, McSharry DG, Malhotra A. Adult obstructive sleep apnoea. Lancet. 2014;**383**(9918):736-747

[38] Hudgel DW, Thanakitcharu S. Pharmacologic treatment of sleep-disordered breathing. American Journal of Respiratory and Critical Care Medicine. 1998;**158**:691-699

[39] Chung F, Yegneswaran B, Liao P, Chung SA, Vairavanathan S, Islam S, et al. STOP questionnaire. Anesthesiology [Internet]. 2008;**108**(5):812-821. DOI: 10.1097/aln.0b013e31816d83e4

[40] Tranfić Duplančić M, Pecotić R, Lušić Kalcina L, Pavlinac Dodig I, Valić M, Roguljić M, et al. Salivary parameters and periodontal inflammation in obstructive sleep apnoea patients. Scientific Reports. 2022;**12**(1):19387

[41] Makeeva IM, Budina TV, Turkina AY, Poluektov MG, Kondratiev SA, Arakelyan MG, et al. Xerostomia and hyposalivation in patients with obstructive sleep apnoea. Clinical Otolaryngology. 2021;**46**(4):782-787

[42] Malhotra A, White DP. Obstructive sleep apnoea. Lancet. 2002;**360**:237-245

[43] Chervin RD. Sleepiness, fatigue, tiredness, and lack of energy in obstructive sleep apnea. Chest. 2000;**118**:372-379

[44] Sanders AE, Essick GK, Fillingim R, et al. Sleep apnea symptoms and risk for temporomandibular disorder: OPPERA cohort. Journal of Dental Research. 2013;**92**:70S-77S

[45] Al-Jewair T, Shibeika D, Ohrbach R. Temporomandibular disorders and their association with sleep disorders in adults: A systematic review. Journal of Oral & Facial Pain and Headache. 2021;**35**(1):41-53. DOI: 10.11607/ofph.2780

[46] Wetselaar P, Manfredini D, Ahlberg J, Johansson A, Aarab G, Papagianni CE, et al. Associations between tooth wear and dental sleep disorders: A narrative overview. Journal of Oral Rehabilitation. 2019;**46**(8):765-775

[47] Marko Vujicic PD, Hilton Israelson DDS, James Antoon DMD, Roger Kiesling DDS, DDS TP, DDS MZ. A profession in transition. The Journal of the American Dental Association (JADA). 2014;**145**(2):118-121

[48] Demko BG. Screening for obstructive sleep apnea in the dental office setting. Journal of the Massachusetts Dental Society. 2008;**57**(1):18-20

[49] Berggren K, Broström A, Firestone A, Wright B, Josefsson E, Lindmark U. Oral health problems linked to obstructive sleep apnea are not always recognized within dental care-as described by dental professionals. Cllinical and Experimental Dental Research. 2022;**8**(1):84-95

[50] Lyons-Coleman M, Bates C, Barber S. Obstructive sleep apnoea and the role of the dental team. British Dental Journal. 2020;**228**(9):681-685. DOI: 10.1038/s41415-020-1523-9

[51] Parmenter D, Millar BJ. How can general dental practitioners help in the management of sleep apnoea? British Dental Journal. 2023;**234**(7):505-509. DOI: 10.1038/s41415-023-5684-1

[52] Mehra P, Wolford LM. Surgical management of obstructive sleep apnea. Proc (Bayl Univ Med Cent). 2000;**13**:338-342

[53] Ikematsu T. Study of snoring, 4th report: Therapy. Journal of Otolaryngology of Japan. 1964;**64**:434-435

[54] Fujita S, Conway W, Zorick F, Roth T. Surgical correction of anatomic abnormalities in obstructive sleep apnea syndrome: Uvulopalatopharyngoplasty. Otolaryngology–Head and Neck Surgery. 1981;**89**:923-934

[55] Ferguson KA, Heighway K, Ruby RR. A randomized trial of laser-assisted uvulopalatoplasty in the treatment of mild obstructive sleep apnea. American Journal of Respiratory and Critical Care Medicine. 2003;**167**:15-191

[56] Rw R, Powell NB, Guilleminault C. Obstructive sleep apnea syndrome: A surgical protocol for dynamic airway reconstruction. Journal of Oral and Maxillofacial Surgery. 1993;**51**:742-747

[57] Reiche-Fischel O, Wolford LM. Posterior airway space changes after double jaw surgery with counter-clockwise rotation. Journal of Oral and Maxillofacial Surgery. 1996;**54**:96-99

[58] Cartwright R, Venkatesan TK, Caldarelli D, Diaz F. Treatments for snoring: A comparison of somnoplasty and an oral appliance. Laryngoscope. 1999;**110**:1680-1683

[59] Tiner BD. Surgical management of obstructive sleep apnea. Journal of Oral and Maxillofacial Surgery. 1996;**54**:1109-1114

[60] Tanna N, Smith BD, Zapanta P, et al. Surgical management of obstructive sleep apnea. Plastic and Reconstructive Surgery. 2016;**137**:1263-1272

[61] Motamedi KK, McClary AC, Amedee RG. Obstructive sleep apnea: A growing problem. The Ochsner Journal. 2009;**9**:149-153

[62] Simmons M. What your Dentist Looks for In Diagnosing Sleep Apnea [Internet]. Dear Doctor, Inc.; 2010. Available from: https://www.deardoctor.com/articles/sleep-disorders-and-dentistry/page2.php

[63] Sutherland K, Cistulli PA. Oral appliance therapy for obstructive sleep apnoea: State of the art. Journal of Clinical Medicine. 2019;**8**(12):2121. DOI: 10.3390/jcm8122121

[64] American Academy of Sleep Medicine. Obstructive Sleep Apnea. Illinois: American Academy of Sleep Medicine; 2008

[65] Isono S, Tanaka A, Tagaito Y, Ishikawa T, Nishino T. Influences of head

positions and bite opening on collapsibility of the passive pharynx. Journal of Applied Physiology. 2004;**97**(1):339-346, Policy Statement 2019

[66] Ng AT, Gotsopoulos H, Qian J, Cistulli PA. Effect of oral appliance therapy on upper airway collapsibility in obstructive sleep apnea. American Journal of Respiratory and Critical Care Medicine. 2003;**168**(2):238-241. DOI: 10.1164/rccm.200211-1275OC. Epub 2003 Apr 30

[67] Issa FG, Sullivan CE. Upper airway closing pressures in obstructive sleep apnea. Journal of Applied Physiology: Respiratory, Environmental and Exercise Physiology. 1984;**57**(2):520-527. DOI: 10.1152/jappl.1984.57.2.520

[68] Goyal M, Johnson J. Obstructive sleep apnea diagnosis and management. Missouri Medicine. 2017;**114**(2):120-124

[69] Marklund M. Update on oral appliance therapy for OSA. Current Sleep Medicine Reports. 2017;**3**(3):143-151. DOI: 10.1007/s40675-017-0080-5. Epub 2017 Jul 10

[70] Sutherland K, Takaya H, Qian J, Petocz P, Ng AT, Cistulli PA. Oral appliance treatment response and polysomnographic phenotypes of obstructive sleep apnea. Journal of Clinical Sleep Medicine. 2015;**11**(8):861-868. DOI: 10.5664/jcsm.4934

[71] Labarca G, Sands SA, Cohn V, Demko G, Vena D, Messineo L, et al. Mouth closing to improve the efficacy of mandibular advancement devices in sleep apnea. Annals of the American Thoracic Society. 2022;**19**(7):1185-1192. DOI: 10.1513/AnnalsATS.202109-1050OC

[72] Bachour A, Maasilta P. Mouth breathing compromises adherence to nasal continuous positive airway pressure therapy. Chest. 2004;**126**:1248-1254

[73] Medical Advisory Secretariat. Oral appliances for obstructive sleep apnea: An evidence-based analysis. Ontario Health Technology Assessment Series. 2009;**9**(5):1-51. Epub 2009 Sep 1

[74] Chang ET, Fernandez-Salvador C, Giambo J, Nesbitt B, Liu SY, Capasso R, et al. Tongue retaining devices for obstructive sleep apnea: A systematic review and meta-analysis. American Journal of Otolaryngology. 2017;**38**(3):272-278. DOI: 10.1016/j.amjoto.2017.01.006. Epub 2017 Jan 18

[75] Alshhrani WM, Hamoda MM, Okuno K, et al. The efficacy of a titrated tongue-stabilizing device on obstructive sleep apnea: A quasi-experimental study. Journal of Clinical Sleep Medicine. 2021;**17**(8):1607-1618

[76] Mandibular repositioning device [Internet]. Healthwise, Incorporated. 2022. Available from: https://www.cigna.com/knowledge-center/hw/mandibular-repositioning-device-zm6313 [Accessed: September 1, 2023]

[77] Ramar K, Dort LC, Katz SG, Lettieri CJ, Harrod CG, Thomas SM, et al. Clinical practice guideline for the treatment of obstructive sleep apnea and snoring with oral appliance therapy: An update for 2015 an American Academy of sleep medicine and American Academy of dental sleep medicine clinical practice guideline. Journal of Clinical Sleep Medicine. 2015;**11**(7):773-827

[78] Dieltjens M, Vanderveken O. Oral appliances in obstructive sleep apnea. Healthcare (Basel). 2019;**7**(4):141. DOI: 10.3390/healthcare7040141

[79] Vanderveken OM, Devolder A, Marklund M, Boudewyns AN, Braem MJ,

Okkerse W, et al. Comparison of a custom-made and a thermoplastic oral appliance for the treatment of mild sleep apnea. American Journal of Respiratory and Critical Care Medicine. 2008;**178**(2):197-202. DOI: 10.1164/rccm.200701-114OC. Epub 2007 Aug 2

[80] Kato J, Isono S, Tanaka A, Watanabe T, Araki D, Tanzawa H, et al. Dose-dependent effects of mandibular advancement on pharyngeal mechanics and nocturnal oxygenation in patients with sleep-disordered breathing. Chest. 2000;**117**(4):1065-1072. DOI: 10.1378/chest.117.4.1065

[81] Ma Y, Yu M, Gao X. The effect of gradually increased mandibular advancement on the efficacy of an oral appliance in the treatment of obstructive sleep apnea. Journal of Clinical Sleep Medicine. 2020;**16**(8):1369-1376

[82] de Ruiter MHT, Aarab G, de Vries N, Lobbezoo F, de Lange J. A stepwise titration protocol for oral appliance therapy in positional obstructive sleep apnea patients: Proof of concept. Sleep & Breathing. 2020;**24**(3):1229-1236. DOI: 10.1007/s11325-020-02045-w. Epub 2020 Mar 11

[83] Levine M, Bennett K, Cantwell M, Postol K, Schwartz D. Dental sleep medicine standards for screening, treating, and managing adults with sleep-related breathing disorders. Journal of Dental Sleep Medicine. 2018;**5**(3):61-68

[84] Martins OFM, Chaves Junior CM, Rossi RRP, Cunali PA, Dal-Fabbro C, Bittencourt L. Side effects of mandibular advancement splints for the treatment of snoring and obstructive sleep apnea: A systematic review. Dental Press Journal of Orthodontics. 2018;**23**(4):45-54. DOI: 10.1590/2177-6709.23.4.045-054.oar

[85] Sheats RD, Schell TG, Blanton AO, et al. Management of side effects of oral appliance therapy for sleep-disordered breathing. Journal of Dental Sleep Medicine. 2017;**4**(4):111-125

[86] Phillips CL, Grunstein RR, Darendeliler MA, Mihailidou AS, Srinivasan VK, Yee BJ, et al. Health outcomes of continuous positive airway pressure versus oral appliance treatment for obstructive sleep apnea: A randomized controlled trial. American Journal of Respiratory and Critical Care Medicine. 2013;**187**(8):879-887. DOI: 10.1164/rccm.201212-2223OC

[87] Gjerde K, Lehmann S, Bjorvatn B, Berge M, Thuen F, Berge T, et al. Partner perceptions are associated with objective sensor-measured adherence to oral appliance therapy in obstructive sleep apnea. Journal of Sleep Research. 2022;**31**:e13462

[88] Liu HW, Chen YJ, Lai YC, Huang CY, Huang YL, Lin MT, et al. Combining MAD and CPAP as an effective strategy for treating patients with severe sleep apnea intolerant to high-pressure PAP and unresponsive to MAD. PLoS One. 2017;**12**(10):e0187032. DOI: 10.1371/journal.pone.0187032. Erratum in: PLoS One. 2018;13(4):e0196319

[89] Yang D, Zhou HF, Xie Y. Efficacy of uvulopalatopharyngoplasty combined with oral appliance in treatment of obstructive sleep apnea-hypopnea syndrome. Irish Journal of Medical Science. 2015;**184**(2):329-334. DOI: 10.1007/s11845-014-1112-5. Epub 2014 Apr 16

[90] Almeida FR, Mulgrew A, Ayas N, Tsuda H, Lowe AA, Fox N, et al. Mandibular advancement splint as short-term alternative treatment in patients with obstructive sleep apnea already effectively treated with continuous positive airway pressure. Journal of Clinical Sleep Medicine.

2013;**9**(4):319-324. DOI: 10.5664/jcsm.2576

[91] About - American academy of dental sleep medicine [Internet]. Aadsm.org. Available from: https://www.aadsm.org/about.php [Accessed: September 1, 2023]

[92] American Academy of Sleep Medicine – Association for Sleep Clinicians and Researchers [Internet]. AASM and AADSM Issue New Joint Clinical Practice Guideline for Oral Appliance Therapy. American Academy of Sleep Medicine – Association for Sleep Clinicians and Researchers; 2017. Available from: https://aasm.org/aasm-and-aadsm-issue-new-joint-clinical-practice-guideline-for-oral-appliance-therapy/ [Accessed: July 6, 2019]

[93] American Dental Association. Evidence Brief: Oral Appliances for Sleep-Related Breathing Disorders [Internet]. American Dental Association. Available from: https://www.ada.org/-/media/project/ada-organization/ada/ada-org/files/resources/research/ada_sci_oralappl_srbd_brief_final_15.pdf; [Accessed: September 1, 2023]

[94] American Dental Association. Policy Statement on the Role of Dentistry in the Treatment of Sleep Related Breathing Disorders [Internet]. Darien, IL: American Dental Association; 2017. Available from: https://www.ada.org/-/media/project/ada-organization/ada/ada-org/files/resources/research/oral-health-topics/ada_2019_policy_role_of_dentistry_sleep_related_breathing_disorders.pdf; [Accessed: September 1, 2023]

[95] About [Internet]. EADSM. European Academy of Dental Sleep Medicine'. Bad Reichenhall, Germany; 2014. Available from: https://eadsm.academy/about/ [Accessed: September 2, 2023]

Chapter 5

The Role of Midline Glossectomy with Coblation in Obstructive Sleep Apnea

Gabriel Santos De Freitas

Abstract

The aim of this study is to determine the effectiveness of COBLATION midline glossectomy for obstructive sleep apnea (OSA) when used as an isolated or with palatal procedure. We also aim to compare the effect of this surgical procedure. The combination of palatal procedures and tongue-based surgery in a single stage led to improvement in excessive daytime sleepiness, snoring, respiratory parameters of polysomnography (PSG), an overall improvement in quality of life, we can show how de tongue base procedures with COBLATION can give better results to all sleep apnea surgeries. Currently, there is a wide variety of surgical approaches to tongue-base modification in OSA patients, including radiofrequency ablation, COBLATION or laser assisted resection, tongue base suspension, genioglossus advancement, and robot-assisted resection. COBLATION midline glossectomy is an effective surgical procedure.

Keywords: coblation, midline glossectomy, OSA, tongue base surgery, snore

1. Introduction

Obstructive sleep apnea (OSA), is a disease that leads to recurrent episodes of upper airway obstruction, which can lead to partial or complete obstruction of the upper airway, it is a chronic and progressive condition [1]. OSA causes excessive daytime sleepiness, and brings many adverse consequences, such as hypertension, obesity, diabetes mellitus, cardiac and encephalic alterations, depression e anxiety, irritability, diminuiçao da libido is a public health problem and studies show that today it affects approximately 30% of the world's population [2].

Continuous positive airway pressure (CPAP) devices are traditionally used as the standard treatment for OSA; however, patient compliance with the treatment is low, and non-compliant patients are commonly considered candidates for surgical treatment [3, 4]. Usually the levels of obstruction are usually different from one patient to the next; Therefore, the appropriate surgical procedure should be evaluated for each case, in this the topographic diagnosis is fundamental for surgical success. The surgery will only be successful if the diagnosis and the sites of obstruction have been well established, only then we can compare the success of each surgical technique and choose the one that best suits the patient, according to the particularity of each patient.

There are numerous, procedures directed to specific sites of the airway and widely disseminated to treat obstructive sleep apnea (OSA), one of them and what we cover extensively in this chapter is the glossectomy of the midline, a procedure that acts directly by amplifying the hypopharyngeal airspace, Fujita in 1991, described the tongue base procedure using the carbon dioxide laser and stars a new era in tongue base surgery access [5].

As previously stated, several surgeries aim to tackle different sites of upper airway obstruction, each with its particularity and always taking into account the treatment of obstructive sleep apnea. We can consider that the palate and the base of the tongue are the most common targets of OSAS surgery, and the resection of the base of the tongue with COBLATION has been proposed and has become the target of numerous studies due to the great efficacy that has been presented; The positive results and the low level of complications speak in favor of this type of approach. Given this idea, we observed that the approach of several surgical sites in the same step and surgical moment is the golden surgery for the treatment of sleep apnea.

The tongue base surgery with COBLATION, can be associated with any procedure related to the treatment of OSA, the maxillomandibular advancement for example, can have immense benefit when we use the association of COBLATION, since we can perform a smaller advance, reducing craniofacial changes, postoperative morbidities, dysfunctions of the temporo-mandibular joint and reducing the index of sleep apnea and hypopnea drastically, with improvement in the patient's quality of life.

2. COBLATION what is that?

COBLATION means 'Controlled Ablation'. COBLATION Technology uses a plasma field to precisely dissolve targeted tissue while maintaining the integrity of surrounding tissue. Unlike radiofrequency-based devices that use high temperatures to cut and cauterize, COBLATION Technology uses lower temperatures causing minimal damage to surrounding tissue [6].

A range of COBLATION Wands are designed for both tonsil and adenoid procedures, with integrated saline delivery and suction, and malleable shafts. In our procedures we use the tip PROCISE MAX, designed for surgeons who prefer rapid tissue removal in his procedures with low thermal effect and easily during hemostasis, since the device itself allows us to use the ablation and coagulation function, the system is integrated with suction and with distillation of saline solution, that reduces thermal effect and assists suction, is an all-in-one instrument designed to help decrease surgical time and avoid some complications.

3. Access to the tongue base

In fact, there is a wide variety of surgical approaches for partial reduction of the base of the tongue for patients with sleep apnea, including radiofrequency ablation, COBLATION or laser assisted resection, tongue base suspension, genioglossus advancement, and transoral robotic surgery (TORS) [7]. We have not yet found the ideal treatment technique, even with the evidence of high success rates, on the other hand, there are studies that show high variability of these rates, which makes it even more difficult to adhere to the ideal technique for this complex and challenging disease. We always provide surgery in associated with palatal techniques because we

believe that in this way in addition to reducing the rate of sleep apnea and hypopnea above the expected, we will also have the reduction of snoring.

We began the procedure by performing dexmedetomidine induced sonoendoscopy, where we evaluate airway collapse at different levels improving surgical accuracy and definition, drug induced sleep endoscopy (DISE), usually takes 15 min [8] reaching only N2 sleep. However, it is during this phase that most events occur, we believe that this short period of time can already show us a large part of what happens during the patient's night, in the ideal world we should spend the night next to this patient and perform the nasofibroscopy during natural sleep. Therefore, DISE, despite not providing all obstructive events that occur in the patient, is an extremely effective tool, easy to apply and which often leads us to realize that we have missed some point of obstruction during the assessment in the office, where we perform the muller maneuver, which today we know how limited it is. After careful visualization of the obstructive surgical sites, we performed traction of the tongue and suture it in the surgical field, to improve exposure of the base of the tongue.

A 30° optics is used to assist in the visualization of the base of the tongue, we use the COBLATION in ablation mode, where there is a rapid tissue removal while maintaining the hemostatic and minimal thermal effect benefits (**Figure 1A** and **B**), so without deepening the ablation by the risk of injury of noble structures of the base of the tongue such as the lingual artery, the ideal would be to evaluate the distance from the surface of the tongue to the hypoglossal/lingual artery neurovascular bundle to constant surface landmarks in the base of tongue during surgically simulated retraction versus resting anatomic position (**Figure 2**), so that we can with tranquility carry out the dissection of this tissue without produce injurys [9, 10].

Soon after the end of the ablation we perform the coagulation of bleeding points of the base of the tongue with the COBLATION itself, which has this option during the procedure, the coagulations should be punctual and should not extend to the lateral redundant tissue of the base of the tongue, we also know that there is no correct amount of lingual tissue to be removed, This depends on the previous characteristics evaluated preoperatively and in sonoendoscopy, which show how bulky this tongue base is and how much it occupies the anteroposterior airspace.

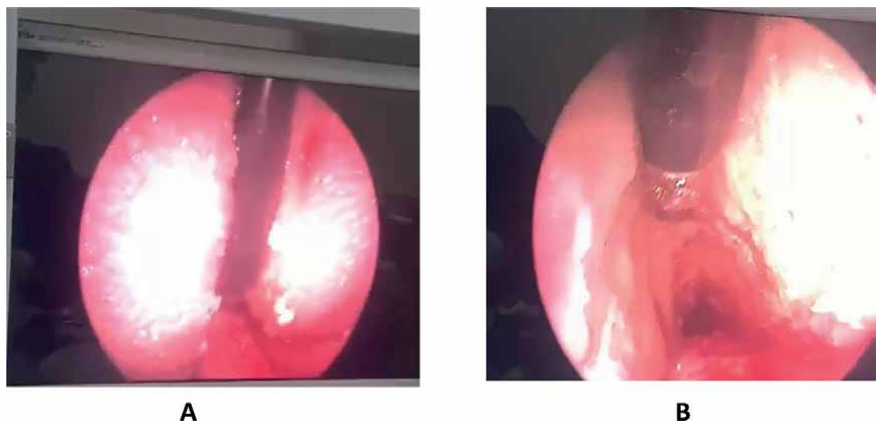

Figure 1.
A. Note the work of coblation wands, at the base of the tongue, always in the midline, B. then already during the partial ablation of the base of the tongue we see much of the tongue tissue removed showing an expressive reduction of the base of the tongue.

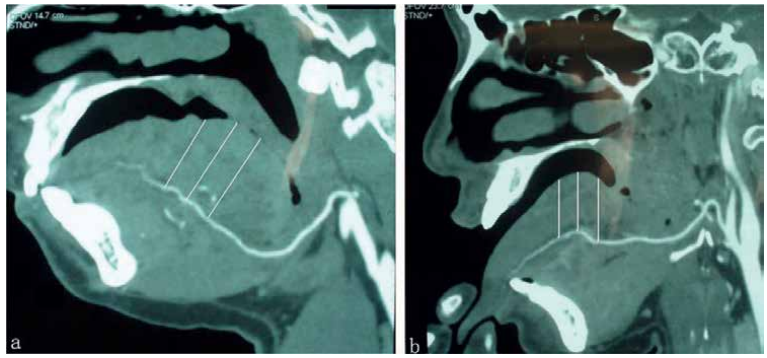

Figure 2.
Sagital views of computed tomography scan of a patient with obstructive sleep apnea syndrome the lingual artery in the resting position (a) and during the extension position of the way we perform the surgical procedure (b). The length of the white line indicates the depths of the lingual artery beneath the lingual surface based on three reference marks.

After the procedure of the base of the tongue is finished, we continue the multilevel surgery, using palatal techniques and nasal surgeries in most cases, and at the end of the whole procedure we performed the review of coagulation and intraoperative edema that can be caused, usually insignificant in our point of view, patients are extubated and taken to the recovery room without the need for intensive care, but with hospitalization of at least 1 day for evaluation of ventilatory parameters and prevention of possible bleeding.

4. Complications

Postoperative pain and submandibular edema were 2 most common postoperative complications which can be easily controlled by antibiotics, [11] the most common intraoperative complication is bleeding, already during the postoperative period we have hematoma, edema, abscess, taste changes, reduced mobility of the base of the tongue, temporary dysphagia, changes during swallowing, paresthesia, usually all reversible after a few months of the procedure and with easy management in the office, This patient should be followed for at least 1 year with careful evaluations and nasofibrolaryngoscopy.

The most feared complication of surgery is velopharyngeal stenosis, which occurs due to excessive removal of the base of the tongue outside the midline when associated with palatal surgeries, a complication that leads to reduced quality of life of the patient as well as respiratory disorders (**Figure 3**).

Just to know, this patient was treated with a new surgery of expansion and lateralization of the palate with important improvement, but with maintenance of fibrotic tissue, in the future probably should be submitted to new palatal expansion, and application of local anti-inflammatory medication.

5. Role of the base of the tongue in sleep apnea

Glossectomy significantly improves sleep outcomes as part of multilevel surgery in adult patients with OSA [12]. The studies have already shown the surgical efficacy

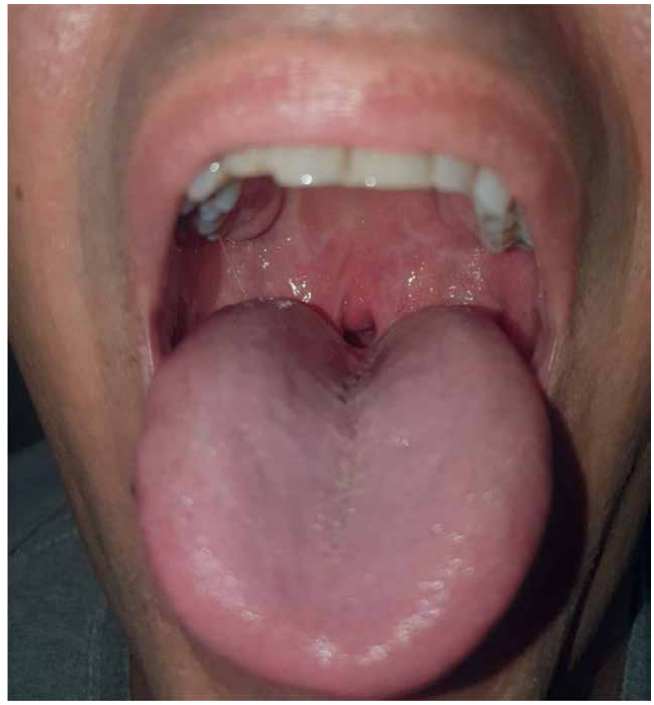

Figure 3.
Patient in 4 months after surgery of lateral expansion pharyngoplasty, midline glossectomy with COBLATION, turbinectomy and septoplasty, we see fibrous scar tissue stenosing the airway and maintaining small respiratory pertuito through the oropharynx.

both alone and in multilevel surgeries, when there is a careful selection of patients and a good indication the surgery has an important response in the treatment of sleep apnea.

In our sample of 28 operated patients, all in surgeries combined with midline glossectomy with COBLATION, expansive lateral pharyngoplasty, septoplasty and turbinectomy, we verified a significant improvement in the evaluation of the epworth sleep scale from 13 ± 9.4 preoperatively to 3.1 ± 2.4 postoperatively, the visual score analogue mean score of preoperative snoring ranged from 7.8 ± 4.1 to 3.1 ± 1.5 postoperatively.

And when we talk about the sleep apnea and hypopnea index (AHI), we show a significant reduction from 26.3 ± 28.2 to 10.4 ± 16.2 in the postoperative period.

The technique of partial glossectomy for the treatment of OSA has been described for some time, today we can present numerous techniques with varying degrees of efficacy, all these procedures are based on the attempt to partially remove the base of the tongue or its redundant tissue. Sleep surgeries that fit this criterion include midline glossectomy (MLG), submucosal minimally invasive lingual excision (SMILE), and lingualplasty (LP). Fujita was the first to describe removal of tongue tissue for OSA with the MLG [5].

We believe that tongue-based surgery, isolated is effective, brings benefit of sleep apnea, but we know that for us to have a better reduction of AHI, the surgery combines multilevel is certainly more effective.

A recent review about the new treatments for OSA, they try to affirm that midline glossectomy is not a good option and cases of moderate sleep apnea, especially in

obese people, obviously believe that when there is a body mass index above 40 kg/m2, no surgical alternative is coherent, and the first step should be weight loss [13]. The evidence we present does not disagree with this statement, but when there is a correct surgical indication seen the patient as a whole, as well as the topographic evaluation evidencing that the base of the tongue is the problem in question, glossectomy alone can become a strong ally and can be effective in the treatment of OSA.

6. What we can expect from tongue-base surgeries

Surgeries that access the base of the tongue have evolved significantly in recent years, with the advent of robotic surgery transoral robotic surgery (TORS), and hypoglossal nerve stimulator.

Vicini et al. demonstrated, the condition presented in the hypertrophy of the base of the tongue, is a difficult management, the surgery to remove the accentuated hypertrophy of the base of the tongue, is a challenge for both the patient and the surgeon, so the studies are increasingly in search of the perfect surgery for this structure [14].

The robotic setup, which permits multiplanar tissue transection at any angle, enhances the surgeon's dissecting skills in such a complex anatomical region, and, as opposed to traditional open approaches, tongue base resection by means of TORS seems to be very well tolerated.

On the other hand, and talking about another type of surgery, one of the most recent treatments for OSA and that has shown efficacy is the hypoglossal nerve stimulator, is a device that generates electrical impulses through a generator that is implanted in the upper right chest (under the skin). The electrical impulse is transmitted directly to the hypoglossal nerve. Today we have in the market three devices being tested in clinical trials, with publications in the most important otorhinolaryngology journals in the world. The Apnex Medical Inc. (St. Paul, MN, USA) device was promising in a phase II trial but failed at phase III because it did not meet efficacy standards and the company does not exist now (**Figure 4**). The second device is the ImThera aura 6000 (San Diego, CA, USA). It places six electrodes around the trunk of the hypoglossal nerve. It is still in phase III clinical trial (**Figure 5**). The third device, which is the only one approved by the FDA, is Inspire Medical Systems (Maple Grove, MN, USA) (**Figure 6**). Since it is the only one approved by the FDA, so we believe that future medical literature papers about hypoglossal neurostimulators will be based directly on Inspire medical Systems [15].

7. Final considerations

Snoring is extremely common in the general population and when irregular may indicate a predisposing to sleep apnea [16].

The combination of palatal procedures with tongue-based surgery in a single stage, lead to improvement of excessive daytime sleepiness, snoring, respiratory parameters of polysomnography, an overall improvement in quality of life. The results

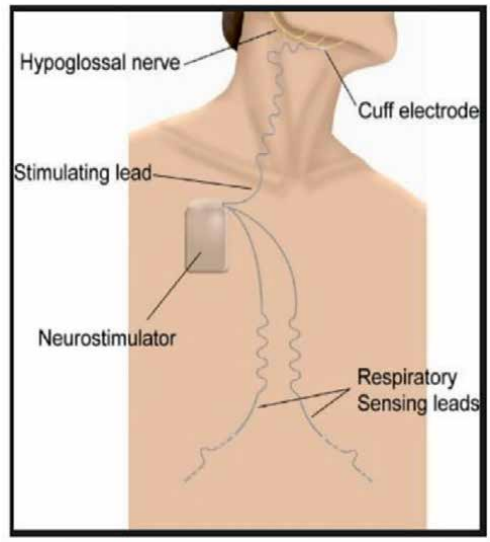

Figure 4.
Apnex Medical Inc. (St. Paul, MN, USA).

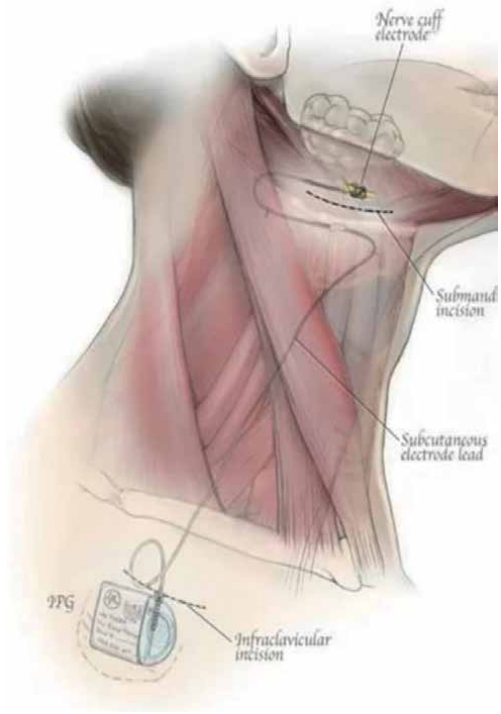

Figure 5.
ImThera aura6000 (San Diego, CA, USA).

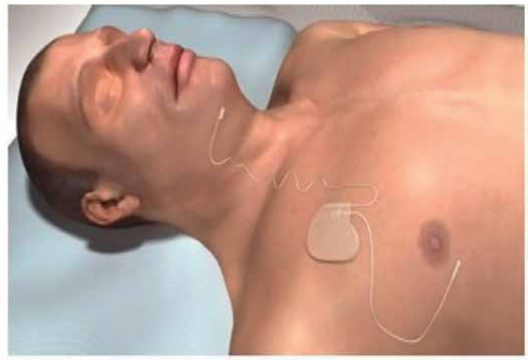

Figure 6.
The Inspire system (Maple Grove, MN, USA).

presented in the literature favor the application of the combined procedure, even knowing that the tongue base treated alone is effective, thus contributing as another therapeutic option in obstructive sleep apnea.

It is important to emphasize that the more we study the neurophysiology of the complex syndrome of obstructive sleep apnea, the more we come across studies indicating the basis of the tongue as a problem and the surgery of the same as a resolution even if temporary of the disorders related to sleep apnea, I believe that the evolution within the tongue-based surgeries as well as the understanding of the functionality of the same within this wonderful world that is the knowledge of the syndrome itself brings us easier and easier paths with less morbidity and with increasingly satisfactory results.

During these years of experience and in my view the surgery performed with the COBLATION has easy applicability and extremely favorable results compared to other types of accesses to the base of the tongue. Evolution is imminent and we will work so that the future shows us more and more conditions to cure this complex disease that is obstructive sleep apnea.

Author details

Gabriel Santos De Freitas
Núcleo de Oftalmologia – Otorrinolaringologia e Medicina Do Sono de Ribeirão Preto, Ribeirão, Preto SP, Brazil

*Address all correspondence to: dr.gabrielfreitas@gmail.com

IntechOpen

References

[1] Park JG, Ramar K, Olson EJ. Updates on definition, consequences, and management of obstructive sleep apnea. Mayo Clinic Proceedings. Jun 2011;**86**(6):549-554; quiz 554-5. DOI: 10.4065/mcp.2010.0810. PMID: 21628617; PMCID: PMC3104914

[2] Pinto JA, Ribeiro DK, Cavallini AF, Duarte C, Freitas GS. Comorbidities associated with obstructive sleep apnea: A retrospective study. International Achieves of Otorhinolaryngology. 2016;**20**(2):145-150. DOI: 10.1055/s-0036-1579546

[3] Weaver TE, Maislin G, Dinges DF, Bloxham T, George CFP, Greenberg H. Relationship between hours of CPAP use and achieving normal levels of sleepiness and daily functioning. Sleep. 2007;**30**:711-719

[4] Engleman HM, Martin SE, Douglas NJ. Compliance with CPAP therapy in patients with the sleep apnea/hypopnoea syndrome. Thorax. 1994;**49**:263-266

[5] Fujita S, Woodson BT, Clark JL, Wittig R. Laser midline glossectomy as a treatment for obstructive sleep apnea. The Laryngoscope. 1991;**101**:805-809

[6] Stadler KR. Electrosurgical plasmas. Journal of Physics D: Applied Physics. 2005;**38**:1728-1738

[7] Kezirian EJ, Goldberg AN. Hypopharyngeal surgery in obstructive sleep apnea: An evidence-based medicine review. Archives of Otolaryngology – Head & Neck Surgery. 2006;**132**(2):206-213

[8] Carrasco Llatas M, Agostini Porras G, Cuesta González MT, Rodrigo Sanbartolomé A, Giner Bayarri P, Gómez-Pajares F, et al. Drug-induced sleep endoscopy: A two drug comparison and simultaneous polysomnography. European Archives of Oto-Rhino-Laryngology. 2014;**271**(1):181-187. DOI: 10.1007/s00405-013-2548-3

[9] Cohen DS, Low GMI, Melkane AE, Mutchnick SA, Waxman JA, Patel S, et al. Establishing a danger zone: An anatomic study of the lingual artery in base of tongue surgery. The Laryngoscope. 2017;**127**:110-115. DOI: 10.1002/lary.26048

[10] Wu D, Qin J, Guo X, Li S. Analysis of the difference in the course of the lingual arteries caused by tongue position change. The Laryngoscope. 2015;**125**:762-766. DOI: 10.1002/lary.24959

[11] Chen JH, Luo ZH, Xu HX, Yang XL, Zhu MW, Tao ZZ. Complications of tongue base reduction with radiofrequency tissue ablation on obstructive sleep apnea hypopnea syndrome. Zhonghua Er Bi Yan Hou Tou Jing Wai Ke Za Zhi. 2010;**45**(7):574-577

[12] Murphey AW, Kandl JA, Nguyen SA, Weber AC, Gillespie MB. The effect of Glossectomy for obstructive sleep Apnea: A systematic review and Meta-analysis. Otolaryngology and Head and Neck Surgery. 2015;**153**(3):334-342. DOI: 10.1177/0194599815594347

[13] Keymel S, Kelm M, Randerath WJ. Non-CPAP therapies in obstructive sleep apnoea: An overview. Pneumologie. 2013;**67**:50-57

[14] Vicini C, Dallan I, Canzi P, Frassineti S, La Pietra MG, Montevecchi F. Transoral robotic tongue base resection in obstructive sleep apnoea-hypopnoea

syndrome: A preliminary report. ORL: Journal for Otorhinolaryngology and Its Related Specialties. 2010;**72**(1):22-27. DOI: 10.1159/000284352

[15] Mashaqi S, Patel SI, Combs D, Estep L, Helmick S, Machamer J, et al. The hypoglossal nerve stimulation as a novel therapy for treating obstructive sleep Apnea—A literature review. International Journal of Environmental Research and Public Health. 2021;**18**:1642. DOI: 10.3390/ijerph18041642

[16] Alencar AM, Vaz DG, da Silva C, Oliveira B, Vieira AP, Moriya HT, et al. Dynamics of snoring sounds and its connection with obstructive sleep apnea. Physica A: Statistical Mechanics and its Applications. 2013;**392**(1):271-277

Chapter 6

Changes in the Level of Anxiety and Depression in the Couples of Patients with Obstructive Sleep Apnea after One Year of CPAP Therapy

Isabel Portela Ferreño

Abstract

Sleeping next to someone with sleep apnea can have a significant impact on the quality of sleep and the health of both partners. The objective was to assess the emotional changes in the partners of patients with OSA. It is a comparative longitudinal study of 102 pairs of patients with a possible OSA diagnosis from January 2018 to October 2019. Female couples 85.7%. The mean age was 48.60 ± 8.99 for the couples. Emotional variables were analyzed through the HAD questionnaire (depression and anxiety), and we also included a Likert scale of the impact of OSA in couples created by the research team. The quality of life was evaluated with the SF-36 questionnaire. For the statistical processing and analysis of the data, the spss program was used. The conclusion was that couples after one year of CPAP treatment improvement in their mood. The results obtained suggest that treating the patient improves the quality of sleep for both.

Keywords: OSA, bed couple, quality of life, anxiety, depression, sleep quality, CPAP treatment, sleep deprivation

1. Introduction

Obstructive sleep apnea (OSA) is a major public health problem that, in its most serious forms, affects 3–6% of men and 2–5% of women [1, 2]. It causes arterial hypertension, increased risk of cardiovascular diseases, and deterioration of the quality of life of those who suffer from it and of those who live with the affected person [3, 4].

This pathology is characterized by the appearance of recurrent episodes of limitation of the passage of air during sleep, followed by awakening from subclinical sleep to return to normal breathing, leading to fragmented and poor-quality sleep, excessive daytime sleepiness, observed breathing interruptions, or awakenings due to gasping or choking in the presence of at least five obstructive respiratory events

(apneas, hypopneas, or arousals related to respiratory effort) per hour of sleep. The presence of 15 or more obstructive respiratory events per hour of sleep in the absence of sleep-related symptoms is also sufficient for the diagnosis of OSA due to the greater association of this severity of obstruction with important consequences, such as increased risk of cardiovascular disease [5–7].

Daytime sleepiness is the most common manifestation of OSA. However, other common daytime effects include irritability, decreased concentration, memory impairment, decreased energy, and depressive symptoms [8]. Nocturnal symptoms include restlessness, diaphoresis, awakening with a sensation of suffocation or dyspnea, esophageal reflux, heartburn, laryngospasm, frequent nycturia, dry mouth, etc. Many studies have indicated an association between sleep apnea and cardiovascular/cerebrovascular disease-related morbidity and mortality. It has been associated with hypertension, coronary artery disease, congestive heart failure, arrhythmias, and stroke [9–13]. It has also been associated with increased mortality, with the most catastrophic result of daytime sleepiness being falling asleep behind the wheel and causing fatal car accidents [14].

The symptoms reported by the patient at night during their sleeping hours are normally witnessed by the partner as snoring, apnea, micro-arousals, and nycturia [15, 16]. Thus, breaking not only his own circadian rhythm but also that of his companion. Specifically, snoring, present in 35–45% of men and close to a quarter of women, often motivates the couple themselves to request a medical visit for the patient.

Recently, studies have confirmed that the impact of OSA on patients' perceived quality of life (HRQoL) is widespread, affecting physical health outcomes (negative health perceptions, increased bodily pain, and poor physical functioning) and psychosocial functioning (e.g., mood disturbance, poor academic performance) [17]. Indeed, there are many domains of life that remain unexplored in the sleep laboratory. For such cases, Lacasse et al., have defined four key domains of HRQoL: somatic sensation, physical function, emotional state, and social interaction [18]. Since physiological measures alone cannot be taken as surrogate markers of HRQoL, this emphasizes the need to measure it directly [19].

The diagnosis of this disease is made by polysomnography (PSG), which includes neurophysiological and cardiorespiratory variables or, failing that, given its high cost, respiratory polygraphy (RP) to identify respiratory events through O2 saturation, nasobuccal flow, and respiratory effort in order to classify the severity of the disease [20, 21].

General measures to curb OSA include weight loss and reeducation of body position. Several studies have shown that initial weight loss decreases the number of apneas-hypopneas and can even make the symptoms disappear by reducing lung volume [22, 23]. A total of 50% of patients have longer and more frequent apneas when sleeping in the supine position; these decrease the collapsibility of the nasopharynx and improve significantly in lateral decubitus [24].

Continuous positive pressure (CPAP) is the first line of treatment, that is, the gold standard, indicated mainly in patients with moderate to severe OSA. It consists of a turbine that transmits a predetermined pressure through a nasal or facial mask adapted to the subject's face and fixed with a harness, thus closing the circuit. In this way, an authentic "pneumatic splint" is produced that transmits positive pressure to the entire upper airway (UPA) and prevents its collapse, both static (apneas) and dynamic (hypopneas) during sleep. The adjustment of the CPAP level must be individualized in each patient through a PSG or through a validated auto-CPAP system

[25, 26]. This treatment can reduce respiratory disorders and daytime sleepiness and improve quality of life [27]; it also reduces the risk of traffic accidents [28] and seems to normalize blood pressure (BP) figures in a relevant percentage of hypertensive subjects with OSA.

Among the effects of sleeping next to someone who suffers from OSA, the following three stand out:

Sleep disturbances: The snoring and breathing pauses associated with sleep apnea can disturb a partner's sleep, leading to difficulty falling asleep and staying asleep.

Emotional exhaustion: Lack of sleep can lead to irritability and emotional exhaustion in the bed partner, which can affect the relationship.

Worry and anxiety: Sleep apnea can be a worrisome condition for a partner, who may feel anxious about their loved one's health and well-being. The levels of anxiety experienced by someone sleeping next to a person with sleep apnea can vary depending on individual circumstances and the severity of the sleep disorder [29].

2. Objectives

2.1 Main objective

To assess the emotional changes in the partners of patients with OSA after one year of CPAP use.

2.2 Secondary objective

Analyze the impact that OSA has on the quality of life of those who suffer from it and their partner; considering variables such as the impact on sleep, daytime sleepiness, and the level of satisfaction in the intimate sphere. Likewise, we want to know if the use of CPAP by the patient improves their symptoms and that of their partner in the short, medium, and long term.

3. Methodology

Comparative longitudinal study, n = 104, study with 52 patients with obstructive sleep apnea and their partners at the Álvaro Cunqueiro Hospital in Vigo, Spain. Patients received CPAP therapy for one year (2018–2019). We analyzed physical, mental, and sexual changes with validated questionnaires: SF-36 (for quality of life), HAD (for depression and anxiety), and CSFQ (for sexual variables). We also use a Likert scale created by our research team to assess the impact on their partners.

3.1 Subjects of study

The recruitment was carried out in the Pneumology Service of the Integrated Management Organizational Structure (EOXI) of Vigo, which covers an area of 600,000 inhabitants. In this area is the largest hospital in Galicia, Hospital Álvaro Cunqueiro, which, in turn, is the headquarters of the Galicia Sur Health Research Institute (IISGS), with which we collaborate to develop this project (http://novohospitalvigo.sergas.es/Paxinas/Portada.aspx) and which combines assistance, teaching, and translational research in the health area of Vigo.

To carry out this study, it will be necessary to recruit patients diagnosed with SAHS and their partners. The set of participants will be recruited from patients who attend the sleep respiratory disorders consultations of the Pneumology Service of the Álvaro Cunqueiro Hospital in Vigo.

The criteria for the inclusion of patients are: (1) age > 18 years and > 65 years, (2) diagnosis of OSA, (3) share rest with your partner, (4) not present other neurological or psychiatric pathologies or a history of head trauma, (5) in women, not being pregnant or lactating, (6) not consume toxins that can affect the results, (7) sign the Informed Consent and, (8) patients with complete consciousness (full authority to participate in the study and grant the informed consent).

The criteria for excluding patients are: (1) comorbidity with another serious diagnosis or concomitant disease that could interfere and (2) the presence of neuromuscular disorders that affect the respiratory pattern or cycle.

The patients' partners will be invited to participate when they accompany their partners to the consultation. If they agree to participate, they will cover, date, and sign the informed consent.

As criteria for the inclusion of patients in the second phase after starting positive pressure therapy, it was necessary to use it for no less than 4 hours per day on average.

The sample size has been determined considering previous studies referring to the study of quality of life in patients with OSA. For OSA patients, the mean Epworth sleepiness scale (ESS) decreased from 12.9 +/− 4.4 to 7.3 +/− 4.0 after CPAP treatment. For bed partners, mean SES decreased from 7.4 +/− 6.1 to 5.8 +/− 4.7. Mean SF-36 scores were significantly reduced in both patients and bed partners. Significant improvements were observed in the subjects in the role domains: physical, vitality, social functioning, emotional role, and mental health. In bed partners, significant changes were observed on the SF-36 in the domains of physical function, vitality, social functioning, and mental health. If we consider these standard deviations to detect differences in mean HRQoL scores of four points, assuming a confidence level of 95% and a statistical power of 80%, we obtain a sample size of 51 patients in each group for a total of 102 subjects.

3.2 Data analysis

The different variables collected in the questionnaires will be studied, looking for possible associations between the clinical variables and sociodemographic variables. In this way, we consider intervariable interactions that may determine the interpretation of the results. The possible correlations existing between the scores of the AHI, snore and quality of life.

For data analysis, the statistical program SPSS for Windows version 20.0 will be used. This part will be carried out with advice from the IBI Statistics Unit. The Kolmogorov-Smirnov test (Lilliefors modification) will be used to verify the normality of the variables, the student's t-test and the Mann-Whitney U statistic to compare the differences between means with independent data, and the Spearman correlation index and regression line. Categorical variables will be expressed as percentages and will be compared using the chi-square test.

3.3 Benefits, applicability, validity, and limitations

With this project, we are interested in knowing the perception of our patients with OSA and that of their partners before and after receiving treatment with CPAP,

regarding the changes they perceive in the quality of life in order to better understand the educational and psychological needs that both have. In this way, we can provide them with the best comprehensive assistance possible. Normally, we focus only on the person who suffers from this disease, but according to the reviewed bibliography, its consequences can have a very negative effect on the health of the roommate, although there are still few scientific studies on the subject.

It is expected to demonstrate that in parallel to the improvement in the patient's symptoms, the quality of life of their partner will also improve, reducing anxiety, and problems in intimate relationships.

Various self-report instruments have been used to assess the impact of OSA on HRQoL: the SF-36 health questionnaire, the life satisfaction scale, the Nottingham health profile, and the 28-item general health questionnaire. Since these instruments are generic, their ability to detect subtle effects of the disease on the quality of life and the effects caused by various treatment modalities led to the development of specific questionnaires for the OSA, such as the sleep apnea quality of life index (SAQLI), the functional sleep impact questionnaire (FOSQ), the obstructive sleep apnea severity index, and the Epworth sleepiness scale. The American Thoracic Society and the American Sleep Disorders Association have made reviews of the use and properties of these instruments; however, none of the mentioned organizations has issued recommendations on the use of specific CVRS instruments due to the lack of comparative data. In a study, the SAQLI was more sensitive to the changes in the quality of life in patients. For the OSA that the SF-36 has a strong content and a constructive validity, but it has to be administered by an interview. Sexual function is addressed only in the FOSQ and OSAPOSI. Aspects related to work are evaluated specifically and in detail mainly by the OSAPOSI, while leisure activities are evaluated in depth by the FOSQ. On the other hand, all the instruments evaluate various aspects of interpersonal interactions and relationships with the exception of the OSAPOSI, which includes only three items limited to marital and sexual relationships.

We developed a study to investigate the impact of OSA and its treatment with CPAP on the quality of life of the patient and their partner, where it affects so that a complete therapy can be planned that meets the specific needs of the patient. Any treatment modality chosen on the basis of disturbances in physiological parameters is unlikely to be complete as these parameters may not be the true representative of the magnitude of suffering in SAHS patients. Therefore, there is a need for a comprehensive holistic treatment that considers the physiological, emotional, and social impairment of the individual patient.

As limitations, the lack of a control group to avoid observation bias stands out.

Variables and questionnaires used.

Variables of the sociodemographic questionnaire:

- Age.
- Sex.
- Civil status.
- Country of birth.
- Education level.
- Socioeconomic level.

- Employment situation (Shifts).
- Toxic consumption.

Variables of the clinical questionnaire:

- BP
- Weight and height
- BMI
- Neck perimeter
- AHI
- Presence of ACVA, ischemic cardiopathy, heart failure, COPD, rhinitis, psychiatric disorders, and other diseases.
- Symptoms of respiratory disorder during sleep.
- Questionnaire on sleep quality during the nocturnal PLG study.
- Assessment of sleepiness: excessive daytime sleepiness (Epworth sleepiness scale).
- Functional impact of sleep questionnaire (FOSQ).
- SF-36 health questionnaire.
- Scale of anxiety and depression (HAD).

The **Epworth sleepiness scale** is a short Likert-type questionnaire that attempts to determine cministered test in which the investigated subject is asked about the frequency (or probability) of falling asleep on an increasing scale that goes from 0 to 3, for eight different everyday situations, in which most people may be involved, in their daily life, although not necessarily every day. The score of the eight situations is added to obtain a total number. A result between 0 and 9 is considered normal, while one between 10 and 24 indicates that the patient should be referred to a specialist. For example, a score between 11 and 15 indicates the possibility of mild to moderate sleep apnea, while a score of 16 and above indicates the possibility of severe sleep apnea or narcolepsy.

The **FOSQ questionnaire** was specifically designed to measure the impact that primary or secondary excessive sleepiness disorders have on daily functioning. The instrument is based on the concept of functional status, that is, the daily behavioral performance in the physical, psychological, and social areas. The FOSQ is made up of 30 items that make up five domains: (a) activity level, (b) surveillance, (c) intimacy and partner relationships, (d) general productivity, and (e) socialization level. You have four response options: 0 (I do not do this activity for other reasons), 1 (yes, extremely), 2 (yes, moderately), 3 (yes, little), and 4 (no).

The **SF-36 health questionnaire** is made up of 36 questions (items) that assess both positive and negative states of health. The questionnaire covers eight scales,

which represent the health concepts most frequently used in the main health questionnaires, as well as the aspects most related to the disease and treatment. The 36 items of the instrument cover the following scales: Physical function, physical role, bodily pain, general health, vitality, social function, emotional role, and mental health. Additionally, the SF-36 includes a transition item that asks about the change in the general state of health compared to the previous year. This item is not used for the calculation of any of the scales but provides useful information on the perceived change in health status during the year prior to the administration of the SF-36.

The **HAD scale** has 14 items and was designed for the evaluation of anxiety and depression in nonpsychiatric outpatient hospital services. It is a state measure with two scales, one for anxiety and one for depression. One of its main virtues is the suppression of somatic symptoms so that they can be evaluated independently of the underlying somatic disease.

4. Results

In the baseline sample, 85.7% of the patients were male. Mean age in patients is 47.6 +/− 4.2 and in couples 48.6 +/− 8.9. AHI: 41.3 +/− 27.6. (52% severe).

In the social function (SF), role emotional (RE), and mental health (MH) dimensions of the SF-36, the average score was lower in couples than in patients ($p < 0.03$, $p < 0.05$, and $p < 0.03$, respectively).

In the physical function (PF), physical role (PR), general health (GH), and vitality (V) dimensions, the average score was lower in patients than in their partners ($p < 0.05$, $p < 0.01$, $p < 0.02$, and $p < 0.02$, respectively) (**Figure 1**).

The total score in the questionnaire for changes in sexual function (SF) was lower in the couple ($p < 0.02$).

Quality of life is as affected in patients with OSA as in their partners, although in different dimensions. The couples show a greater alteration in the emotional, social, and sexual sphere, while the patients perceive greater affectation in the physical plane.

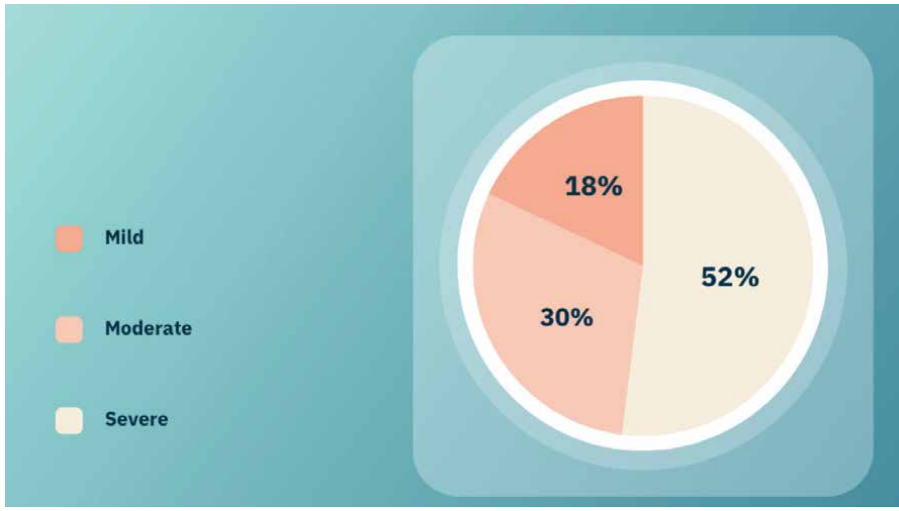

Figure 1.
Classification of patients according to severity of AHI.

	Variable	HAD	SF-36
Patients	AHI	P < 0,05 R = 0,237	P < 0,02 R = −0,309
Couples	Snore	P < 0,03 R = 0,298	P < 0,01 R = −0,396

Table 1.
Pearson correlation analysis for the HAD and SF-36 questionnaires of OSA patients and bed partners.

Group of patients: The AHI showed a statistically significant correlation, positive for the HAD questionnaire, and negative for the SF-36 questionnaires.

Group of couples: The correlation is found in snoring, positive for HAD and negative for the SF-36 questionnaires. In addition to a positive (moderate) correlation with the level of symptoms reported by the patient ($r = 0.424$; $p < 0.01$) (**Table 1**).

After one year of CPAP therapy by the patients, the partners showed lower levels of anxiety and depression compared to the previous year ($P < 0.001$). Items related to quality of life (physical functioning, limitation due to physical problems, discomfort, social role, mental health, limitation due to emotional problems, vitality, and general perception of health) showed significant improvement.

Ninety percent of members reported that CPAP has significantly improved the health of patients and 85% stated that the use of CPAP has had a beneficial impact on their own health status.

5. Discussion

An adequate understanding and comprehension on the part of the patient and partner about the type of disease they are facing can be a great support to ensure correct adherence to treatment with CPAP, the studies carried out by Cartwright reflected this [30]. It is also of great importance to maintain a healthy relationship to have an open dialog about rest, even if it means doing it separately on certain occasions so as not to interfere with the other's sleep [31].

Fatigue-inducing conditions, such as sleep loss, compromise these factors, leading to decline in decision performance. Aidman et al., using a 40-hour sleep deprivation protocol, examined these factors and the resulting decision performance. Thirteen Australian Army male volunteers (aged 20–30 years) were tested at multiple time points on psychomotor vigilance, inhibitory control, task switching, working memory, short-term memory, fluid intelligence, and decision accuracy and confidence in a medical diagnosis-making test. Assessment took place in the morning and night over two consecutive days, during which participants were kept awake. Consistent with previous work, cognitive performance declined after a night without sleep. Extending previous findings, self-regulation and self-monitoring suffered significantly greater declines immediately after the sleepless night. These results indicate that the known decline in complex decision-making performance under fatigue-inducing conditions might be facilitated by metacognitive rather than cognitive mechanisms [32].

The Meta-analysis conducted by Natan et al. indicates that sleep deprivation, whether total or not, leads to a significant increase in state anxiety levels, but sleep restriction does not. Regarding the effect of the length of the period of sleep deprivation, no significant results were observed, but there was a notable tendency for an

increase in anxiety in longer sleep deprivations. With regard to tools, the state-trait anxiety inventory (STAI) seems to be the best one to measure sleep-induced anxiogenesis, while the profile of mood states (POMS) presented inconclusive results [33].

In the pediatric field Smirni et al. found interesting results on how this pathology affects the quality of life of mothers, the authors concluded that "the child respiratory disease, with its sudden and unpredictable features, appeared as a significant source of stress for the mother". Such stress condition may have an impact on mothers' personality traits (self-esteem, locus of control) and on their memory performances [34]. On the other hand, Operto FF et al. concluded that children with sleep apnea have fewer emotional intelligence skills than children who do not suffer from this clinical condition, which considerably affects the response to stress and decision-making [35].

Adherence to treatment is an essential condition for proper compliance. As CPAP is a chronic treatment, and due to its special characteristics, the percentage of patients who abandon the therapy or who decide not to use it from the beginning is not negligible. According to some studies, from 5 to 50% reject treatment or interrupt it on the first night/week of its use, while abandonment at 5 years stands at 23% (although we can find figures as diverse as 4–46%, most in the first year) [36].

6. Conclusions

It is essential to understand that anxiety levels can vary from person to person, and sleep apnea is a medical disorder that requires proper care and treatment. If the bed partner is experiencing elevated levels of anxiety due to sleep apnea, it is important to seek medical support and consider options to improve the situation, such as seeking sleep apnea treatment or considering temporarily separate bedrooms. Emotional support and open communication are also critical in addressing any worries or concerns related to shared sleep disorder.

The impact on the quality of life of patients with OSA without CPAP treatment depends on the AHI, while in their partners their snoring is the determining factor.

After one year of treatment, the couples showed a decrease in anxiety and depression variables, as well as an increase in all areas of quality of life, including libido. It can be affirmed that CPAP treatment improves the general state of health of both the patient and the couple, that is the reason why we can conclude that treating one improves the sleep quality of both.

Acknowledgements

I thank Dr. Alberto Fernandez Villar for his support in presenting this work to the Ethics Committee. Thanks to Dr. Mar Mosteiro for always believing in me. Thanks to Tania Baltanas for her support with the statistical analysis.

Thank you to each couple who participated in the project.

Conflict of interest

During my career, I have received fees related to various training collaborations: Philips, GSK, Boehringer, Chiesi, and VitalAire.

Acronyms and abbreviations

OSA	obstructive sleep apnea
CPAP	continuous positive airway pressure
UPA	upper airway
PSG	polysomnography
BP	blood pressure
RP	respiratory polygraphy
STAI	state-trait anxiety inventory
POMS	profile of mood states
SAQLI	sleep apnea quality of life index
FOSQ	sleep impact questionnaire
SF	social function
ER	emotional role
MH	mental health
PF	physical function
PR	physical role
GH	general health
V	vitality

Author details

Isabel Portela Ferreño[1,2]

1 Air Liquide Healthcare, Madrid, Spain

2 Spanish Society of Pneumology and Thoracic Surgery (SEPAR), Madrid, Spain

*Address all correspondence to: isabelportelafer@hotmail.com

IntechOpen

References

[1] Peppard PE, Young T, Barnet JH, et al. Increased prevalence of sleep-disordered breathing in adults. American Journal of Epidemiology. 2013;**177**(9):1006-1014

[2] Lam JC, Sharma SK, Lam B. Obstructive sleep apnoea: Definitionsepidemiology, natural history. Indian Journal of Medical Research. 2010;**131**:165-170

[3] Budhiraja R, Quan SF. Sleep-disordered breathing and cardiovascular health. Current Opinion in Pulmonary Medicine. 2005;**11**(6):501-506. DOI: 10.1097/01.mcp.0000183058.52924.70

[4] Baldwing Baldwin CM, Griffith KA, Nieto FJ, O'Connor GT, Walsleben JA, Redline S. The association of sleep-disordered breathing and sleep symptoms with quality of life in the sleep heart health study. Sleep. 2001;**24**:96-105

[5] Montserrat JM, Amilibia J, Barbé F, Capote F, Durán J, Mangado NG, et al. Tratamiento del síndrome de las apneas-hipopneas durante el sueño. Archivos de Bronconeumología. 1998;**34**:204-206

[6] Roest AM, Carney RM. Obstructive sleep apnea/hypoapnea síndrome and por response to sertraline in patients with coronary heart disease. The Journal of Clinical Psychiatry. 2012;**73**:31-36

[7] American Academy of Sleep Medicine. International Classification of Sleep Disorders: Diagnostic and Coding Manual. 2nd ed. Westchester, IL: American Academy of Sleep Medicine; 2005

[8] Cheshire K, Engleman H, Deary I, Shapiro C, Douglas NJ. Factors impairing daytime performance in patients with sleep apnea/hypopnea syndrome. Archives of Internal Medicine. 1992;**152**:538-541

[9] Shahar E, Whitney CW, Redline S, Lee ET, Newman AB, Nieto FJ, et al. Sleep-disordered breathing and cardiovascular disease: Cross-sectional results of the sleep heart health study. American Journal of Respiratory and Critical Care Medicine. 2001;**163**:19-25

[10] Franklin KA, Nilsson JB, Sahlin C, Näslund U. Sleep apnoea and nocturnal angina. Lancet. 1995;**345**:1085-1087

[11] Mehra R, Benjamin EJ, Shahar E, Gottlieb DJ, Nawabit R, Kirchner HL, et al. Association of nocturnal arrhythmias with sleep-disordered breathing: The sleep heart health study. American Journal of Respiratory and Critical Care Medicine. 2006;**173**:910-916

[12] Khayat RN, Jarjoura D, Patt B, Yamokoski T, Abraham WT. In-hospital testing for sleep-disordered breathing in hospitalized patients with decompensated heart failure: Report of prevalence and patient characteristics. Journal of Cardiac Failure. 2009;**5**(9):739-746

[13] Dyken ME, Im KB. Obstructive sleep apnea and stroke. Chest. 2009;**136**:1668-1677

[14] Young T, Finn L, Peppard PE, Szklo-Coxe M, Austin D, Nieto FJ, et al. Sleep disordered breathing and mortality: Eighteen-year follow-up of the Wisconsin sleep cohort. Sleep. 2008;**31**:1071-1078

[15] Walther A-S et al. Apnea obstructiva del sueño. Del Sistema Nacional de Salud. Vol 23. N° 5-1999

[16] Alva JL, Perez. Consecuencias metabólicas y cardiovasculares del síndrome de apneaobstructiva del sueño. Medicina Interna de México. 2009;**25**(2):116-128

[17] Baldassari CM, Mitchell RB, Schubert C, Rudnick EF. Pediatric obstructive sleep apnea and quality of life: A meta-analysis. Otolaryngology—Head and Neck Surgery. 2008;**138**:265-273

[18] Lacasse Y, Godbout C, Sériès F. Health-related quality of life in obstructive sleep apnoea. The European Respiratory Journal. 2002;**19**:499-503

[19] Lacasse Y, Godbout C, Sériès F. Independent validation of the sleep apnoea quality of life index. Thorax. 2002;**57**:483-488

[20] Manarino MR, Di Filippo F, Pirro M. Obstructive sleep apnea síndrome. European Journal of Internal Medicine. 2012;**23**:586-593

[21] Alonso Fernández F. Claves de la depresión. Madrid: Editorial Ars Vivendi; 2001

[22] Harman EM, Wynne JW, Block AJ. The effect of weight los son sleep disorder breathing and oxigen desaturation in morbidly obese men. Chest. 1982;**82**:291-294

[23] Surta PM, McTier RF, et al. Changes in breathing and pharynx after weight loss in obstructive sleep apnea. Chest. 1987;**92**:631-637

[24] Surta PM, McTier RF, et al. Changes in breathing and pharynx after weight loss in obstructive sleep apnea. Chest. 1987;**92**:638-639

[25] Lloberes P, Ballester E, Montserrat JM, et al. Comparison of manual and automatic CPAP titration in patients with sleep apnea/hipopnea syndrome. American Journal of Respiratory and Critical Care Medicine. 1996;**154**:755-758

[26] Aurora RN, Collop NA, Jacobowitz O, et al. Quality measures for the care of adult patients with obstructive sleep apnea. Journal of Clinical Sleep Medicine. 2015;**11**(3):357-383

[27] Antic NA, Catcheside P, Buchan C, et al. The effect of CPAP in normalizing daytime sleepiness, quality of life, and neurocognitive function in patients with moderate to severe OSA. Sleep. 2011;**34**(1):111-119

[28] Muñoz L, Findley L, Antó JN, et al. Impact of CPAP on automovile accidents in patients with sleep apnea syndrome (SAS). European Respiratory Journal. 2001;**18**(Suppl 33):16

[29] Kolotkin RL, Binks M, et al. Obesity and sexual quality of life. Obesity. 2006;**14**:472-479

[30] Sucena M, Liistro G, Aubert G, Rodenstein DO, Pieters T. Continuous positive airway pressure treatment for sleep apnoea: Compliance increases with time in continuing users. The European Respiratory Journal. 2006;**27**:761-766

[31] Troxel WN, Obles TF, Hall M, et al. Marital quality and the marital bed: Examining the covariation between relationship quality and sleep. Sleep Medicine Reviews. 2007;**11**:389-404

[32] Aidman E, Jackson SA, Kleitman S. Effects of sleep deprivation on executive functioning, cognitive abilities, metacognitive confidence, and decision making. Applied Cognitive Psychology. 2019;**33**(2):188-200

[33] Pires GN, Bezerra AG, Tufik S, Andersen ML. Effects of acute sleep

deprivation on state anxiety levels: A systematic review and meta-analysis. Sleep Medicine. 2016;**24**:109-118

[34] Smirni D, Carotenuto M, Precenzano F, Smirni P, Operto FF, Marotta R, et al. Memory performances and personality traits in mothers of children with obstructive sleep apnea syndrome. Psychology Research and Behavior Management. 2019;**12**:481-487

[35] Operto FF, Precenzano F, Bitetti I, Lanzara V, Fontana ML, Pastorino GMG, et al. Emotional intelligence in children with severe sleep-related breathing disorders. Behavioural Neurology. 5 Sep 2019;**2019**:6530539. DOI: 10.1155/2019/6530539

[36] Márquez-Baez C, Paniagua-Soto J, Castilla-Garrido JM. Treatment of sleep apnea syndrome with CPAP: Compliance with treatment, its efficacy and secondary effects. Revista de Neurologia. 1998;**26**:375-380

Edited by Marco Carotenuto

This edited volume, *Obstructive Sleep Apnea - New Insights in the 21st Century*, is a collection of reviewed and relevant research chapters offering a comprehensive overview of recent developments in obstructive sleep apnea (OSA). It provides a thorough overview of the latest research efforts by international authors on OSA and opens new possible research paths for further novel developments.

Published in London, UK

IntechOpen

ISBN 978-0-85466-363-7