Approach to obstructive sleep apnea

Interdisciplinary considerations for optimal management

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Abstract

Objective To provide primary care physicians with up-to-date information on obstructive sleep apnea (OSA) and current management options.

Sources of information Reviews of relevant literature retrieved via the PubMed and MEDLINE databases with no date limitations. Searches for the impact on OSA of dentures with implant oral restoration.

Main message A narrowing of the upper airway axial surface due to fat deposition or hypertrophic tonsils and upper airway neuromuscular pathology can cause OSA. OSA can be asymptomatic, but most patients develop a variety of symptoms, including nocturnal awakenings, nocturia, unrefreshing sleep, daytime sleepiness and fatigue, impaired concentration, and chronic morning headaches. Several screening tools exist, including the Berlin Questionnaire. Laboratory polysomnography (level 1) is the accepted standard for the evaluation of sleep-disordered breathing. Addressing modifiable risk factors can help control OSA. Obesity and overweight status in the adult population, which can be addressed with behavioural interventions, are associated with severity of OSA. Medical options include positive airway pressure (PAP) therapy. Mandibular advancement devices are an alternative for patients with mild or moderate OSA or those who cannot tolerate PAP therapy. Surgical options can address both the biomechanical aspects of OSA and patients' overweight status.

Conclusion Careful history-taking and understanding of the underlying biomechanical pathophysiology can orient physicians to consider OSA. Personalized and interdisciplinary approaches should be considered when prescribing OSA treatment and during follow-up assessments.

Editor's key points

- Obstructive sleep apnea (OSA) is a common pathology in the primary care setting, and patients can present with various symptoms.
- Laboratory polysomnography (level 1) is the accepted standard for evaluating OSA.
- Addressing modifiable risk factors, including overweight status, can help control OSA.
- Medical options include positive airway pressure therapy. Surgical options can address both the biomechanical aspects of OSA and patients' overweight status.

Nonspecific symptoms such as fatigue are frequently reported in medical practice.[1,2] Some are associated with other clinical cues that can help narrow the differential diagnosis. Sleep-related breathing disorders such as sleep apnea can be contributing factors, but can be difficult to detect because of the variety of clinical presentations.[3-5] Obstructive sleep apnea (OSA), a complete or transient breathing cessation lasting 10 seconds or longer, is caused by obstruction of the upper respiratory tract.[6] Although estimates vary, OSA affects up to 40% of men aged 50 years and older and one-fifth of the postmenopausal population.[7,8] Family physicians should recognize OSA in a timely manner because the condition increases the risk of many complications and affects quality of life.[9,10]

Case description

A 77-year-old man presented to the family medicine clinic with recurring diurnal fatigue, dizziness, nocturia, and frequent awakenings for the past 3 weeks. His medical history included OSA syndrome treated with the use of a continuous positive airway pressure (CPAP) machine. His symptoms had

resolved, according to self-assessment, and he had not used his CPAP for the past year. One week before symptom onset, he underwent dental surgery consisting of multiple dental extractions and placement of dual jaw-implant–supported dentures.

He did not describe irritative urinary symptoms. Reviews of cardiovascular, respiratory, and neurology systems had negative findings. Physical examination findings were normal. Orthostatic hypotension was ruled out. Blood count and dysthyroidism panel results were negative. Prostate palpation findings were normal.

Sources of information

Literature searches of the PubMed and MEDLINE databases were performed with no date limitations on March 30, 2025. The medical subject headings *sleep apnea syndromes* and *clinical/practice guidelines* were searched independently as were free-word searches for *sleep apnea* and similar terms. We also searched for descriptions of the uses of dentures with implant oral restoration.

Main message

While sleep apnea and hypopnea can be categorized as central and obstructive to describe the underlying cause, this review focuses on the latter.

Pathophysiology. The pathophysiology of OSA is biomechanical, with the negative pressure during inspiration causing a partial or complete collapse of the upper airway soft tissues, which results in partial or complete obstruction of airflow despite respiratory effort. During inspiration, the genioglossus muscle and associated upper airway dilator muscles contract to prevent posterior collapse of the tongue.[4,11] Narrowing of the upper airway axial surface due to fat deposition or hypertrophic tonsils and upper airway neuromuscular pathology can lead to OSA. Individual phenotypes or morphologic traits (**Box 1**),[4] including a narrow upper airway and low respiratory arousal threshold, can also contribute to OSA.[12]

Signs and symptoms. The signs and symptoms of OSA are diverse and demonstrate the overall impact of sleep on physiology. The various manifestations are both patient- and severity-dependent. While some patients may be asymptomatic, most develop a variety of symptoms, including nocturnal awakenings, nocturia, unrefreshing sleep, daytime sleepiness and fatigue, impaired concentration, and chronic morning headaches.[3,6,13,14] While nonspecific to OSA, loud snoring is a common concern among patients' partners and others who sleep nearby.[15] Episodes of apnea are sometimes followed by abrupt awakenings with gasping.[15]

Analyses and reviews describe hypertension,[5] type 2 diabetes,[16] atrial fibrillation,[5] heart failure,[4] cardiovascular disease,[4] anxiety disorders,[5] depression,[17] and others[4,5] as potential complications of OSA.

Evaluation. Patients who express concern about related symptoms should be offered evaluation of suspected OSA. Evaluation tools include the Berlin Questionnaire[18] (**Box 2**), which was developed to identify sleep apnea in the primary care setting,[4] and the STOP-Bang (snoring, tiredness, observed apnea, high blood pressure, body mass index [BMI], age, neck circumference, and male gender)

Box 1. Risk factors for obstructive sleep apnea

Modifiable
- BMI
- Neck circumference
- Volumetric enlargement of tongue
- Smoking

Nonmodifiable
- Age
- Male sex
- Menopause
- Retrognathia
- Deep palate

BMI—body mass index.
Data from Gottlieb et al.[4]

Box 2. Summary of the Berlin Questionnaire and STOP-Bang questionnaire

Berlin Questionnaire[18]

Category 1—snoring
- Snoring
- Quantify the level of snoring
- Frequency of snoring
- Impact on others
- Apnea during snoring

Category 2—fatigue and tiredness
- Frequency of morning fatigue or tiredness
- Frequency of daytime fatigue or tiredness
- Falling asleep while driving
- Frequency of falling asleep while driving

Category 3—systemic symptoms
- Hypertension

STOP-Bang questionnaire[19]

History
- Snoring
- Tiredness or fatigue during the day
- Apnea during sleep
- Hypertension (pressure)

Physical examination—objective data
- BMI
- Age
- Neck circumference
- Gender

BMI—body mass index; STOP-Bang—snoring, tiredness, observed apnea, high blood pressure, BMI, age, neck circumference, and male gender.

questionnaire, which is more widely used in the clinical setting to screen for OSA despite being developed and validated for preoperative surgical patients.[19] The Epworth Sleepiness Scale[20] is not specific for OSA and should not be used in the evaluation process in this population.

Many items on the Berlin Questionnaire are based on the epidemiologic distribution of risk factors associated with OSA. Other items, such as about snoring and fatigue, may be part of a patient's history. The prevalence of OSA is greater in men than in women and increases with BMI and with age, especially after age 50 years.[9] Clinical suspicion of OSA increases as the number of risk factors increases. A higher clinical probability of OSA is seen for patients with OSA symptoms and with 2 or more criteria of the STOP-Bang questionnaire.[19]

No specific physical examination can confirm OSA, but an examination may reveal anatomic variants such as tonsillar hypertrophy, retrognathia, and macroglossia.[4] Attention should also be paid to the fit of dentures. A nasal examination should be performed as several treatable anatomic variants or pathologies, including rhinitis, nasal polyps, or septal deviation, can obstruct airflow.

Diagnosis. Polysomnography (level 1) is the accepted standard for the evaluation of sleep-disordered breathing, including OSA. The presence and severity of OSA are determined by the apnea-hypopnea index (AHI), which expresses the number of events per hour. However, the AHI does not accurately account for ventilatory deficits. Both the Canadian Thoracic Society[6,14] and the American Academy of Sleep Medicine (AASM) have proposed criteria and definitions for sleep apnea or hypopnea, although physicians use the AASM[21,22] criteria in the clinical setting (**Table 1**). Such criteria are not sufficient to assess health risks, and a complete assessment of the patient's quality of life, occupation, and cardiovascular and metabolic risks is warranted.

Portable monitors, with high sensitivity (79%) and specificity (79%), allow for in-home sleep testing,[4] which is frequently used for first-line diagnostic testing. Level 1 is reserved for severe cases or for patients with critical comorbidities. For patients with a high pretest probability, a negative home sleep test result should be followed by level 1 polysomnography, if available, or repeated in-home testing.[4,23] In the case of a negative result on subsequent or repeat testing, another diagnosis should be considered. In the case of a marginally positive test result (or inconclusive evaluation), a CPAP trial should be considered to guide next steps based on symptom response. Sleep tests may need to be repeated, particularly if symptoms change, which can happen with age, development of new comorbidities or worsening of existing ones, and changes in weight.

Management. Modifying risk factors can help control OSA (**Table 2**).[24-29] Obesity and overweight status in the adult population have been associated with OSA severity.[24,25] Weight control is of utmost importance in the patient-centred approach to OSA management. Multimodal strategies including dietary approaches, exercise, bariatric surgery, and treatment with glucagon-like peptide-1 (GLP-1) agonists may help achieve weight control most effectively, although GLP-1 agonist treatment has not been approved for OSA management in Canada. Clinicians should inform patients that achieving optimal weight and using a CPAP or mandibular advancement device (MAD) can contribute to reducing their health risk and improving their quality of life and of sleep. However, mild to moderate residual apnea and hypopnea may persist.

Behavioural intervention management. Behavioural interventions include weight loss, aerobic exercise, and alcohol abstinence.[4] Weight loss, which the American Thoracic Society recommends as part of its clinical practice guideline, is associated with improvements in OSA severity, cardiometabolic comorbidities, and quality of life.[30] Physical activity independent of weight loss is also associated with improvements in OSA.[5,26]

Whether patients should wear their dentures at night is a common and important question in the context of OSA. The current literature tends to support that such a decision should be made on an individual basis, with some recent

Table 1. Characteristics of obstructive sleep apnea

CHARACTERISTIC	DETAILS
Definition	Complete cessation of breathing or transient reduction in breathing with possibly greater respiratory effort[14]
Time	≥10 s[14]
Respiratory pattern	CTS definition[6,14] • >50% decrease in nasal pressure or respiratory inductance • Decrease in respiratory amplitude AASM definition[21,22] • ≥50% decrease in airflow resistance
Saturation	CTS definition[6,14] • ≥4% oxygen desaturation or arousal from sleep AASM definition[21,22] • ≥3% oxygen desaturation or arousal from sleep
Severity—empirical criteria (based on apnea-hypopnea index)	Normal: <5 Mild: 5-14.9 Moderate: 15-29.9 Severe: ≥30

AASM—American Academy of Sleep Medicine, CTS—Canadian Thoracic Society. Data from Fleetham et al,[6,14] Ruehland et al,[21] and Kapur et al.[22]

data showing no major overall difference.[31] However, sleeping with dentures was associated with greater daytime sleepiness for some, while for others, sleeping without dentures was associated with better breathing, probably due to a different anatomic airway configuration and length, as shown by 3-dimensional imaging.[31,32] More research is needed to reach a conclusion on this issue. In addition, the ability to properly use CPAP or MAD in an aging population is a crucial factor to consider.[33]

Medical and noninvasive options. Positive airway pressure (PAP) therapy is recommended as first-line therapy for patients with OSA symptoms and polysomnograph findings showing an AHI greater than 5.[27,34] The AASM suggests using CPAP or autoadjusting PAP over bilevel PAP in the treatment of OSA.[27] The use of nasal devices, compared to oral interfaces, resulted in greater adherence to PAP therapies.[34] One study reported a statistically significant reduction in mean hourly use of CPAP compared to MAD at 30 days.[35]

Using a MAD is the second choice after PAP therapy for patients with mild or moderate OSA or for those who cannot tolerate PAP therapy. Several randomized controlled trials have compared the efficacy of MAD to CPAP (as standard of care). A meta-analysis showed that both treatments decreased the AHI and the oxygen desaturation index compared to controls, although MAD was less effective than CPAP.[26] Nevertheless, using a MAD may be a reasonable alternative to PAP therapy because the higher adherence rates help achieve similar patient-oriented outcomes over time.[36] In the CHOICE (Adherence and Preference of Continuous Positive Airway Pressure vs Mandibular Advancement Splints in Obstructive Sleep Apnea Patients: A Randomized Trial) randomized clinical trial, 35 of the 64 participants (55%) preferred alternating between CPAP and MAD versus using only 1 or the other intervention, which suggests that a multimodal treatment improves patient-centred outcomes and therefore OSA management.[28]

A review of prescribed drugs and adequate withdrawal of sleep-inducing medication, such as muscle relaxants and benzodiazepines, could help with excessive daytime sleepiness.

Surgical options and considerations. Otorhinolaryngologic evaluations to assess for nasal or pharyngeal obstruction are recommended. Surgical alternatives to PAP therapy are possible if adherence to or efficacy of CPAP is low. These include uvulopalatoplasty, nasal septoplasty, and treatment of nasal polyps. Other proposed surgical procedures include maxillary expansion, with the goal of reducing nasal resistance and improving nasomaxillary airway flow. The American Academy of Dental Sleep Medicine supports surgical approaches to maxillary expansion if orthodontic transverse discrepancy is present, but otherwise concluded there is insufficient evidence supporting the use of ablative procedures to prevent, manage, or cure OSA.[29]

While behavioural interventions and lifestyle changes are preferable for controlling weight, options such as bariatric surgeries have shown promising results.[37] Three systematic reviews and meta-analyses reported that bariatric surgery did not resolve OSA.[38-40] While weight loss occurred after the intervention, the authors highlighted the absence of linear correlation between the decrease in both weight and AHI.[38,39]

Prognosis. The prognosis of OSA depends on the severity of the condition and adherence to treatment. PAP therapy or MAD do not cure patients and only address the underlying biomechanical airway obstruction. Lifelong treatment is required unless sufficient weight loss or surgery results in disease remission.[4] Treatment is important

Table 2. Options for managing obstructive sleep apnea

THERAPEUTIC CATEGORY	THERAPY	ASSOCIATED PATHOLOGY
Behaviour modifications	Dietary approaches	Weight control
	Exercise	Weight control
Medical options	GLP-1 agonists (not yet approved for OSA treatment alone in Canada) Other pharmacologic options are undergoing testing	Weight control
	CPAP, MAD, or multimodality treatments	Airway obstruction
	Intranasal corticosteroids	Rhinosinusitis or obstructive nasal polyps
Surgical options	Otolaryngologic	Anatomic obstruction of the upper airway
	Bariatric	Weight control
	Orthognathic	Retrognathia or reduced upper airway volume
	Hypoglossal nerve stimulation (not yet approved in Canada)	Airway obstruction

CPAP—continuous positive airway pressure, GLP-1—glucagonlike peptide-1, MAD—mandibular advancement device, OSA—obstructive sleep apnea.
Data from Newman et al,[24] Young et al,[25] Iftikhar et al,[26] Patil et al,[27] Hamoda et al,[28] and Sheats et al.[29]

as patients who do not receive treatment or who experience nonadherence barriers are at increased risk of developing cardiovascular and cerebrovascular disease—especially those with more severe OSA, for whom treatment benefits are likely greater.[6,14] The daytime sleepiness associated with OSA also puts people at increased risk of motor vehicle accidents and work-related challenges.

Case resolution

The patient's history showed a temporal association between the recurrence of his symptoms and the recent dental procedure. Investigations ruling out anemia and dysthyroidism suggested that decompensated OSA was likely. Considering the biomechanical impacts of the patient's recent dental surgery, it was suspected that the new permanent implant-supported dentures reduced the overall volume of the oral cavity, probably causing the tongue to project posteriorly and in a downward position in the oropharyngeal space, thus exacerbating a previously compensated sleep apnea.

Following 10 days of CPAP use, the patient reported a return to baseline sleep and nocturia and a marked decrease in dizziness and daytime sleepiness. This clinically significant improvement strongly supports the biomechanical hypothesis of decompensated sleep apnea.

Conclusion

OSA has a range of signs and symptoms that affect quality of life and risk of additional comorbidities. While considering differential diagnosis, careful history-taking and understanding of the underlying biomechanical pathophysiology can orient physicians to consider OSA. Appropriate counselling should include regular use of CPAP or the use of removable dentures rather than a dental implant–supported solution.

Family physicians should motivate patients to regularly use CPAP, as treatment adherence is essential to obtain benefits. Personalized and interdisciplinary approaches adapted to the motor and cognitive changes related to aging are additional factors to consider when prescribing OSA treatment and at follow-up.

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Contributors

All authors contributed to conducting the literature review and to preparing the manuscript for submission.

Competing interests

Drs Gilles Lavigne and **Pierre Mayer** receive financial contribution from Panthera Dental (Québec, Que) for research projects in sleep medicine. **Drs Philippe Harris** and **René Wittmer** do not have conflicts of interest.

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References

1. Galland-Decker C, Marques-Vidal P, Vollenweider P. Prevalence and factors associated with fatigue in the Lausanne middle-aged population: a population-based, cross-sectional survey. BMJ Open. 2019 Aug 24;9(8):e027070. **doi: 10.1136/bmjopen-2018-027070**. Epub 2019 Aug 24.
2. Cullen W, Kearney Y, Bury G. Prevalence of fatigue in general practice. Ir J Med Sci. 2002 Jan;171(1):10-2. **doi: 10.1007/BF03168931**.
3. Maspero C, Giannini L, Galbiati G, Rosso G, et al. Obstructive sleep apnea syndrome: a literature review. Minerva Stomatol. 2015 Apr 1;64(2):97-109.
4. Gottlieb DJ, Punjabi NM. Diagnosis and management of obstructive sleep apnea: a review. JAMA. 2020 Apr 14;323(14):1389-400. **doi: 10.1001/jama.2020.3514**. Epub 2020 Apr 14.
5. Choi BY, Kim JK, Cho JH. A review of a recent meta-analysis study on obstructive sleep apnea. J Rhinol. 2022 Nov 30;29(3):134-40. **doi: 10.18787/jr.2022.00422**. Epub 2022 Nov 30.
6. Fleetham J, Ayas N, Bradley D, Fitzpatrick M, et al. Canadian Thoracic Society 2011 guideline update: diagnosis and treatment of sleep disordered breathing. Can Respir J. 2011 Jan 1;18(1):25-47. **doi: org/10.1155/2011/506189**.
7. Huang T, Lin BM, Redline S, Curhan GC, et al. Type of menopause, age at menopause, and risk of developing obstructive sleep apnea in postmenopausal women. Am J Epidemiol. 2018 Jul 1;187(7):1370-9. **doi: 10.1093/aje/kwy011**. Epub 2018 Jan 22.
8. McArdle N, Reynolds AC, Hillman D, Moses E, et al. Prevalence of common sleep disorders in a middle-aged community sample. J Clin Sleep Med. 2022 Jun 1;18(6):1503-14. **doi: 10.5664/jcsm.9886**. Epub 2022 Jun 1.
9. Peppard PE, Young T, Barnet JH, Palta M, et al. Increased prevalence of sleep-disordered breathing in adults. Am J Epidemiol. 2013 Apr 14;177(9):1006-14. **doi: 10.1093/aje/kws342**.
10. Canadian Thoracic Society [Internet]. Canadian respiratory guidelines: recommendations for the diagnosis and treatment of sleep apnea: 2011 update. Canadian Thoracic Society; 2011 [cited 2025 Oct 10]. Available from: **https://cts-sct.ca/wp-content/uploads/2018/08/5339_THOR-Sleep-apnea-slimjim_0718_en-web.pdf**.
11. Dempsey JA, Veasey SC, Morgan BJ, O'Donnell CP. Pathophysiology of sleep apnea. Physiol Rev. 2010 Jan 1;90(1):47-112. **doi: 10.1152/physrev.00043.2008**. Epub 2010 Jan 1.
12. Carberry JC, Amatoury J, Eckert DJ. Personalized management approach for OSA. Chest. 2018 Mar;153(3):744-55. **doi: 10.1016/j.chest.2017.06.011**. Epub 2017 Jun 16.
13. Ayas N, Skomro R, Blackman A, Curren K, et al. Obstructive sleep apnea and driving: a Canadian Thoracic Society and Canadian Sleep Society position paper. Can Respir J. 2014 Mar-Apr;21(2):114-23. **doi: org/10.1155/2014/357327**. Epub 2014 Jan 1.
14. Fleetham J, Ayas N, Bradley D, Ferguson K, et al. Canadian Thoracic Society guidelines: diagnosis and treatment of sleep disordered breathing in adults. Can Respir J. 2006 Oct;13(7):387-92. **doi: 10.1155/2006/627096**. Epub 2006 Jan 1.
15. Myers KA, Mrkobrada M, Simel DL. Does this patient have obstructive sleep apnea?: The Rational Clinical Examination systematic review. JAMA. 2013 Aug 21;310(7):731-41. **doi: 10.1001/jama.2013.276185**. Epub 2013 Aug 21.
16. Nagayoshi M, Punjabi NM, Selvin E, Pankow JS, et al. Obstructive sleep apnea and incident type 2 diabetes. Sleep Med. 2016 Nov 4;25:156-61. **doi: 10.1016/j.sleep.2016.05.009**. Epub 2016 Sep 29.
17. Asghari A, Mohammadi F, Kamrava SK, Tavakoli S, et al. Severity of depression and anxiety in obstructive sleep apnea syndrome. Eur Arch Otorhinolaryngol. 2012 Feb 2;269(12):2549-53. **doi: 10.1007/s00405-012-1942-6**.
18. Netzer NC, Stoohs RA, Netzer CM, Clark K, et al. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. Ann Intern Med. 1999 Oct 5;131(7):485-91. **doi: 10.7326/0003-4819-131-7-199910050-00002**. Epub 2000 Aug 15.
19. Chung F, Yegneswaran B, Liao P, Chung SA, et al. STOP questionnaire: a tool to screen patients for obstructive sleep apnea. Anesthesiology. 2008 May;108(5):812-21. **doi: 10.1097/ALN.0b013e31816d83e4**.
20. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. Sleep. 1991 Nov 1;14(6):540-5. **doi: 10.1093/sleep/14.6.540**.
21. Ruehland WR, Rochford PD, O'Donoghue FJ, Pierce RJ, et al. The new AASM criteria for scoring hypopneas: impact on the apnea hypopnea index. Sleep. 2009 Feb 1;32(2):150-7. **doi: org/10.1093/sleep/32.2.150**.
22. Kapur VK, Auckley DH, Chowdhuri S, Kuhlmann DC, et al. Clinical practice guideline for diagnostic testing for adult obstructive sleep apnea: an American Academy of Sleep Medicine clinical practice guideline. J Clin Sleep Med. 2017 Mar 15;13(3):479-504. **doi: 10.5664/jcsm.6506**. Epub 2017 Mar 15.
23. Jonas DE, Amick HR, Feltner C, Weber RP, et al. Screening for obstructive sleep apnea in adults: evidence report and systematic review for the US Preventive Services Task Force. JAMA. 2017 Jan 24;317(4):415-33. **doi: 10.1001/jama.2016.19635**. Epub 2017 Mar 28.
24. Newman AB, Foster G, Givelber R, Nieto FJ, et al. Progression and regression of sleep-disordered breathing with changes in weight: the Sleep Heart Health Study. Arch Intern Med. 2005 Nov 14;165(20):2408-13. **doi: 10.1001/archinte.165.20.2408**. Epub 2005 Nov 14.
25. Young T, Peppard PE, Taheri S. Excess weight and sleep-disordered breathing. J Appl Physiol (1985). 2005 Oct 1;99(4):1592-9. **doi: 10.1152/japplphysiol.00587.2005**. Epub 2005 Oct 1.
26. Iftikhar IH, Bittencourt L, Youngstedt SD, Ayas N, et al. Comparative efficacy of CPAP, MADs, exercise-training, and dietary weight loss for sleep apnea: a network meta-analysis. Sleep Med. 2017 Feb;30:7-14. **doi: 10.1016/j.sleep.2016.06.001**. Epub 2016 Jun 28.
27. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, et al. Treatment of adult obstructive sleep apnea with positive airway pressure: an American Academy of Sleep Medicine clinical practice guideline. J Clin Sleep Med. 2019 Feb 15;15(2):335-43. **doi: 10.5664/jcsm.7640**. Epub 2019 Feb 15.
28. Hamoda MM, Hyunh N, Ayas NT, Rompré P, et al. Continuous positive airway pressure and mandibular advancement splints: the CHOICE multicentre open-label randomised clinical trial. Eur Respir J. 2025 Apr 3;65(4):2401100. **doi: 10.1183/13993003.01100-2024**. Epub 2025 Apr 3.

29. Sheats R, Masse JF, Levine M, Aarab G, et al. Novel therapies for preventing, managing and treating obstructive sleep apnea and snoring in pediatric and adult patients. J Dent Sleep Med. 2024 Apr;11(2). **doi: 10.15331/jdsm.7332**.
30. Hudgel DW, Patel SR, Ahasic AM, Bartlett SJ, et al. The role of weight management in the treatment of adult obstructive sleep apnea. An official American Thoracic Society clinical practice guideline. Am J Respir Crit Care Med. 2018 Sep 15;198(6):e70-87. **doi: 10.1164/rccm.201807-1326ST**.
31. Emami E, Salah MH, Rompré P, Huynh N, et al. The nocturnal use of complete dentures and sleep stability in edentulous elders. J Dent. 2013 Aug;41(8):703-9. **doi: 10.1016/j.jdent.2013.05.017**. Epub 2013 Jun 8.
32. Chen H, Emami E, Kauffmann C, Rompré P, et al. Airway phenotypes and nocturnal wearing of dentures in elders with sleep apnea. J Dent Res. 2023 Mar;102(3):263-9. **doi: 10.1177/00220345221133278**. Epub 2022 Nov 4.
33. Okuno K, Wang L, Almeida FR. Focus of dental sleep medicine on obstructive sleep apnea in older adults: a narrative review. J Prosthodont Res. 2024 Apr 8;68(2):227-36. **doi: 10.2186/jpr.JPR_D_23_00047**. Epub 2023 Aug 30.
34. Patil SP, Ayappa IA, Caples SM, Kimoff RJ, et al. Treatment of adult obstructive sleep apnea with positive airway pressure: an American Academy of Sleep Medicine systematic review, meta-analysis, and GRADE assessment. J Clin Sleep Med. 2019 Feb 15;15(2):301-34. **doi: 10.5664/jcsm.7638**. Epub 2019 Feb 15.
35. Guralnick AS, Balachandran JS, Szutenbach S, Adley K, et al. Educational video to improve CPAP use in patients with obstructive sleep apnoea at risk for poor adherence: a randomised controlled trial. Thorax. 2017 Dec 30;72(12):1132-39. **doi: 10.1136/thoraxjnl-2017-210106**. Epub 2017 Jun 30.
36. Barnes H, Edwards BA, Joosten SA, Naughton MT, et al. Positional modification techniques for supine obstructive sleep apnea: a systematic review and meta-analysis. Sleep Med Rev. 2017 Dec;36:107-15. **doi: 10.1016/j.smrv.2016.11.004**. Epub 2016 Nov 18.
37. Qin H, Wang Y, Chen X, Steenbergen N, et al. The efficacy of bariatric surgery on pulmonary function and sleep architecture of patients with obstructive sleep apnea and co-morbid obesity: a systematic review and meta-analysis. Surg Obes Relat Dis. 2023 Dec;19(12):1444-57. **doi: 10.1016/j.soard.2023.07.007**. Epub 2023 Jul 31.
38. Wong AM, Barnes HN, Joosten SA, Landry SA, et al. The effect of surgical weight loss on obstructive sleep apnoea: a systematic review and meta-analysis. Sleep Med Rev. 2018 Nov 3;42:85-99. **doi: 10.1016/j.smrv.2018.06.001**. Epub 2018 Jun 19.
39. Greenburg DL, Lettieri CJ, Eliasson AH. Effects of surgical weight loss on measures of obstructive sleep apnea: a meta-analysis. Am J Med. 2009 Jun;122(6):535-42. **doi: 10.1016/j.amjmed.2008.10.037**.
40. Al Oweidat K, Toubasi AA, Tawileh RB, Tawileh HB, et al. Bariatric surgery and obstructive sleep apnea: a systematic review and meta-analysis. Sleep Breath. 2023 Dec;27(6):2283-94. **doi: 10.1007/s11325-023-02840-1**. Epub 2023 May 5.

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