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REVIEW ARTICLE

Obstructive sleep apnea in obesity: traditional and emerging treatment strategies

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Abstract

Obstructive sleep apnea (OSA) is highly prevalent and strongly associated with obesity. Mechanistic pathways include parapharyngeal fat deposition, reduced lung volumes, impaired upper-airway muscle responsiveness, and metabolic dysregulation, alongside a bidirectional relationship in which OSA can also contribute to weight gain. Weight-loss interventions consistently attenuate OSA severity: lifestyle programs yield meaningful reductions in apnea–hypopnea index and continuous positive airway pressure (CPAP) dependency; pharmacotherapies, particularly incretin-based agents, produce substantial improvements in respiratory and cardiometabolic outcomes; and surgical approaches achieve durable weight loss but variable effects on OSA remission. Despite these advances, most evidence derives from middle-aged males with obesity, limiting applicability to females, older adults, and individuals whose OSA is driven by non-obesity-related pathophysiology. Recognition of OSA heterogeneity underscores the need for integrated, phenotype-guided treatment strategies. This review aims to synthesize current evidence linking obesity and OSA and to critically evaluate weight-loss interventions alone and in combination with CPAP.

Keywords: *Obstructive sleep apnea. Obesity. CPAP. Pharmacotherapy. Lifestyle modification.*

Introduction

Obstructive sleep apnea (OSA) is a highly prevalent disorder associated with a wide range of adverse health outcomes. Obesity is one of the most well-established risk factors for OSA, and its global prevalence has continued to rise over recent decades. In the adult population, the prevalence of OSA is estimated to be around 25%; however, it increases among obese adults, reaching approximately 45%[1]. An individual participant data meta-analysis[2] reported that 31.5% of OSA patients were obese, whereas 44% were overweight. Notably, among obese adults, 74% had OSA (apnea-hypopnea index [AHI] ≥ 5) and 41% had clinically significant disease (AHI ≥ 15).

Longitudinal data indicate that a 10% increase in body weight predicts a six-fold increase in the odds of developing moderate-to-severe sleep-disordered breathing[3], suggesting a strong dose-response relationship between body weight and OSA severity. This suggests that OSA severity is highly responsive to changes in body weight. Despite this, the relationship between OSA and obesity remains insufficiently explored in certain populations, particularly in females, in whom postmenopausal physiological changes have been associated with increased OSA prevalence. Similarly, the relationship between OSA and obesity is insufficiently explored in older adults, and the aging process is also associated with increased prevalence of OSA. It is projected that by 2050, many regions

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will have doubled the proportion of individuals aged 65 years and older[4]. These demographic shifts raise important questions about the epidemiological evolution of both obesity and OSA, as well as about the impact of weight-loss interventions, on OSA outcomes.

In this chapter, we synthesize current evidence on the key aspects linking obesity and OSA and critically appraise lifestyle, pharmacological, and surgical weight-loss interventions, individually and in combination with the gold standard therapy with continuous positive airway pressure (CPAP).

OSA and obesity: key aspects

OSA is highly heterogeneous, with multiple factors contributing to the disease. Among them, obesity rises as one of the main risk factors for OSA, as well as its contribution to its pathogenesis. Consistent with this notion, a longitudinal population-based cohort study by Peppard et al. demonstrated that weight gain is associated with proportional increases in the AHI, whereas weight loss is linked to substantial reductions in AHI, underscoring the sensitivity of OSA severity to changes in body weight[3].

Several mechanisms have been proposed to explain the strong association between obesity and OSA. Obesity exacerbates OSA primarily through the accumulation of fat in specific body regions[1]. Both animal and human studies have demonstrated that parapharyngeal fat deposition can directly compress the upper airway, promoting narrowing and collapsibility. Abdominal adiposity reduces lung volumes and tracheal traction, further increasing airway collapsibility. Fat accumulation in the tongue has also been associated with higher AHI values in OSA patients, providing an additional explanation for the link between obesity and airway obstruction.

Conversely, OSA itself may contribute to the development or persistence of obesity. Sleep fragmentation and/or sleep deprivation reinforce behavioral and metabolic dysregulation, which collectively alter energy balance parameters and promote weight gain or hinder weight loss by increasing appetite or reducing physical activity, among others[5,6].

Treatment of OSA in patients with obesity

CPAP therapy

CPAP therapy is the standard treatment for patients with severe or symptomatic OSA. CPAP delivers constant positive airway pressure to the nose and/or mouth, maintaining the upper airway open during sleep. Its use is associated with a reduced risk or attenuation of numerous OSA-related consequences, including excessive daytime sleepiness (EDS), reduced quality of life, traffic accidents, hypertension, cognitive impairments, Alzheimer's disease, and cardiovascular events[7,8].

CPAP therapy is not without challenges. Side effects such as discomfort, skin irritation, claustrophobia, and dryness of the mouth and nose often contribute to low adherence, reducing its overall effectiveness. The discomfort and challenges associated with CPAP therapy, coupled with a lack of proven benefit in asymptomatic populations, often lead clinicians to prioritize treatment for patients who are willing and able to adhere to therapy. This underscores the urgent need for further research to explore alternative treatment options. In addition, while CPAP therapy effectively alleviates symptoms and mitigates the long-term risks associated with OSA, it does not cure the condition or address one of its frequent root causes, obesity.

Lifestyle interventions

Weight management should be considered a primary therapeutic approach in patients with OSA, particularly those who are overweight or obese. This strategy is most effective when integrated into a comprehensive treatment plan alongside other therapeutic modalities. Lifestyle medicine is a rapidly growing discipline that emphasizes the role of lifestyle factors, such as diet and physical activity, in preventing, managing, and even reversing chronic diseases[9,10].

Previous systematic reviews and meta-analyses consistently show that weight reduction through lifestyle interventions, including hypocaloric dietary strategies and structured exercise programs, leads to significant reductions in OSA severity and improvements in related outcomes such as daytime sleepiness. The meta-analysis by Carneiro-Barrera et al.[11] examined 12 randomized controlled trials (RCTs) and 23 uncontrolled studies (n = 1420), mostly middle-aged males with obesity and moderate-to-severe OSA, who underwent combined diet and exercise interventions. The results revealed significant reductions in AHI, oxygen desaturation index, and EDS. Although full remission of OSA was uncommon, these reductions were clinically meaningful. Notably, none of the included studies directly addressed smoking or alcohol consumption, despite their established role in worsening OSA.

Recent RCTs provide further evidence supporting lifestyle and weight-loss interventions in OSA[12-14]. One trial evaluated the effects of an interdisciplinary weight-loss and lifestyle program in adults

with moderate-to-severe OSA and overweight or obesity. Participants were middle-aged males (mean age 54.1 ± 8.0 years). Compared with controls, the intervention group showed a greater reduction in AHI at the study endpoint (–21.2 events/h; 51% reduction), with a between-group difference of –23.6 events/h (95% confidence interval [CI], –28.7 to –18.5 events/h). At 6 months post-intervention, the AHI reduction reached 57%. Clinically relevant outcomes included a marked reduction in CPAP dependency: at the intervention endpoint, 45.0% of participants no longer required CPAP, increasing to 61.8% at six months. Complete remission of OSA was achieved in 15.0% of participants at endpoint and in 29.4% at 6 months.

Lifestyle and behavioral interventions have also been evaluated through RCTs in patients with varying degrees of OSA severity. Tuomilehto et al.[15] focused on adults with mild OSA, testing a 12-week program centered on dietary modification, physical activity, and broader lifestyle change. Participants in the intervention group demonstrated a 40% reduction in AHI, and two out of three achieved an AHI < 5 at follow-up. In contrast, two out of three participants in the control group remained at an AHI ≥ 5, and the mean AHI did not change from baseline. These findings indicate that lifestyle modification can yield clinically meaningful improvements even in the context of mild OSA.

In an RCT evaluating patients with moderate-to-severe OSA, a Mediterranean diet-based weight-loss program combined with CPAP therapy[16] produced larger reductions in respiratory events, daytime sleepiness, and insomnia symptoms over 1 year compared with standard care alone. Structured guidance on healthy diet, physical activity, and optimal sleep habits was associated with additional improvements in sleep architecture and daytime alertness, suggesting that lifestyle modification can enhance the physiological and symptomatic effects of CPAP across a broader clinical spectrum.

Evidence of durability is provided by long-term follow-up data. In the 10-year study by Kuna et al.[17], participants in an intensive lifestyle intervention maintained significant improvements, with mean AHI values of 9.7, 8.0, and 7.9 events/h at years 1, 2, and 4, respectively, and a decline to 4.0 events/h at year 10. AHI trajectories were associated both with the amount of weight loss and with participation in the intervention itself, independent of weight change. OSA remission at 10 years occurred in 34.4% of individuals in the lifestyle intervention group compared with 22.2% in the control group, highlighting the potential for long-term clinically meaningful reductions in OSA severity through sustained behavioral strategies.

Pharmacological treatment

In individuals with both OSA and obesity, treatment includes lifestyle modifications such as dietary changes and increased physical activity. While these interventions can be effective, patient adherence is often limited, underscoring the need for additional therapeutic strategies[18,19]. When lifestyle interventions do not achieve adequate therapeutic outcomes, pharmacological treatment is recommended. To date, the U.S. Food and Drug Administration (FDA) has approved seven medications for weight loss (Table 1); however, only one (tirzepatide) has recently received specific approval for the treatment of OSA in individuals with obesity[20].

In this context, Malhotra et al.[21] conducted the SURMOUNT-OSA phase 3 RCT to evaluate the efficacy and safety of tirzepatide in individuals with moderate-to-severe OSA (AHI ≥ 15 events/h) and obesity (body mass index [BMI] ≥ 30 kg/m^2). A total of 469 participants were randomized (1:1) to receive tirzepatide (10 or 15 mg) or placebo for 52 weeks. Two parallel trials were performed: Trial 1 included participants who were not receiving CPAP therapy, either due to intolerance or unwillingness to use the device (n = 234), and Trial 2 included those on stable CPAP therapy (n = 235). In Trial 1, the tirzepatide group experienced a mean AHI reduction of –25.3 events/h (95% CI, –29.3 to –21.2) compared to –5.3 events/h (95% CI, –9.4 to –1.1) in the placebo group. In Trial 2, the reduction was –29.3 events/h (95% CI, –33.2 to –25.4) versus –5.5 events/h (95% CI, –9.9 to –1.2), respectively. Tirzepatide also led to significant reductions in BMI (–17.7% vs. –1.6% in Trial 1; –19.6% vs. –2.3% in Trial 2), along with improvements in hypoxic burden, high-sensitivity C-reactive protein, and systolic blood pressure. Gastrointestinal adverse events were the most common but were generally mild-to-moderate in severity.

Other weight-loss medications have also demonstrated efficacy in the management of OSA. In a 28-week RCT, Winslow et al.[22] evaluated the combination of phentermine and topiramate in 45 patients with OSA, randomized to either placebo (n = 23) or active treatment (n = 22). The treatment group experienced a significantly greater reduction in AHI, with a mean decrease of –31.5 events/h (95% CI, –40.0 to –22.9), compared to –16.6 events/h (95% CI, –25.0 to –8.2) in the placebo group. Similarly, weight loss was more substantial in the treatment arm (–10.8 kg; 95% CI, –13.5 to –8.0) than in the placebo group (–4.7 kg; 95% CI, –7.2 to –2.2).

Other glucagon-like peptide-1 receptor agonists have also been investigated in the context of

Table 1. FDA-approved drugs for weight loss

Weight-loss medication	Pharmacological class	Route of administration	Recommended/approved dose
Orlistat	Lipase inhibitor	Oral	120 mg 3 times per day with meals[33]
Phentermine-topiramate complex	A combination of a noradrenergic agonist and a GABA A receptor agonist	Oral	15-37.5 mg once daily[34]
Naltrexone-bupropion complex	A combination of an opioid receptor antagonist and a moderately weak inhibitor of neuronal reuptake of dopamine and noradrenaline	Oral	Start with one tablet daily (90 mg ER bupropion), and increase weekly to 2 tablets twice daily (32 mg ER naltrexone/360 mg ER bupropion)[35]
Liraglutide	GLP-1 agonist	Subcutaneous	Start with 0.6 mg daily. Increase by 0.6 mg weekly until a maintenance dose of 3 mg daily[36,37]
Semaglutide	GLP-1 agonist	Subcutaneous	Start with 0.25 mg weekly for 4 weeks. Increase gradually to a maintenance dose of 2.4 mg weekly[38]
Setmelanotide*	MC4R agonist	Subcutaneous	Start with 2 mg daily, adjust between 1 and 3 mg based on tolerance[39,40]
Tirzepatide	GIP and GLP-1 agonist	Subcutaneous	Start with 2.5 mg. Escalate 2.5 mg every 4 weeks up to 15 mg weekly[41,42]

Prescription medications to treat overweight and obesity. U.S. Department of Health and Human Services.
*Specific to rare genetic disorders that cause obesity.
GABA: gamma-aminobutyric acid; GIP: glucose-dependent insulinotropic polypeptide; GLP-1: glucagon-like peptide-1; MC4R: melanocortin 4 receptor.
Adapted from the National Institute of Diabetes and Digestive and Kidney Diseases[23].

OSA. The SCALE Sleep Apnea trial, conducted by Blackman et al.[24] assessed the effects of liraglutide in patients with moderate OSA (AHI 15-30 events/h) and obesity (BMI ≥ 30 kg/m^2). A total of 359 participants (n = 180 liraglutide; n = 179 placebo) were enrolled, all adhering to a hypocaloric diet and physical activity program. After 32 weeks, AHI decreased by –12.2 ± 1.8 events/h in the liraglutide group compared to –6.1 ± 2.0 events/h in the placebo group, with an estimated treatment difference of –6.1 events/h (95% CI, –11.0 to –1.2). Weight loss was also greater in the liraglutide group (–5.7 ± 0.4%) versus placebo (–1.6 ± 0.3%). Further supporting evidence comes from Jiang et al.[25], who evaluated liraglutide effects in patients with type 2 diabetes and moderate-to-severe OSA (AHI > 15 events/h). In this study, all participants received CPAP and standard antidiabetic therapy. Over the 3-month intervention, the liraglutide group showed a statistically significant reduction in AHI, decreasing from 31.0 ± 7.3 to 26.1 ± 7.1 events/h. In contrast, the control group exhibited no significant change, with AHI moving from 30.1 ± 6.2 to 31.6 ± 6.9 events/h. Consequently, post-treatment AHI values differed significantly between groups. The liraglutide group also demonstrated improvements in minimum oxygen saturation, whereas no change was observed in controls. These respiratory benefits occurred alongside reductions in BMI and favorable changes in low-density lipoprotein cholesterol, glycated hemoglobin, and blood pressure.

In addition, O'Donnell et al.[26] conducted an RCT comparing liraglutide, CPAP, and their combination in 30 patients with severe-to-moderate OSA (AHI > 15 events/h) and obesity (BMI 30-40 kg/m^2), randomized in a 1:1:1 ratio. After 24 weeks, significant weight loss was observed in both the liraglutide group (–6.17 kg; 95% CI, –8.73 to –3.61) and the combination group (–3.67 kg; 95% CI, –6.91 to –0.43), but not in the CPAP-only group. AHI improved across all groups, with CPAP showing the greatest effect, although reductions were also seen with liraglutide and combination groups.

Collectively, these findings underscore the substantial benefits of weight-loss pharmacotherapy in patients with OSA and obesity, including reductions in disease severity and improvements in cardiovascular and metabolic risk profiles. However, the potential for adverse effects must be considered, and it is important to recognize that a proportion of OSA patients are not obese, making this approach unsuitable for all individuals.

Surgical approaches

Bariatric surgery constitutes an invasive therapeutic option indicated for patients with obesity when less aggressive interventions such as lifestyle modification, dietary changes, physical exercise, or positional

therapy have proven ineffective. Current clinical guidelines recommend the procedure for individuals with a BMI > 40 kg/m², or > 35 kg/m² when obesity-related comorbidities, such as hypertension, diabetes mellitus, heart failure, or OSA, are present[27]. The most commonly performed metabolic/bariatric procedures include sleeve gastrectomy, Roux-en-Y gastric bypass (RYGB), biliopancreatic diversion, and the placement of adjustable gastric bands[28].

Given its capacity to promote substantial weight reduction in patients with obesity, metabolic/bariatric surgery has been proposed as a potential therapeutic strategy for OSA[27]. Consequently, several RCTs have evaluated the effects of bariatric surgery on OSA severity in obese individuals.

Furlan et al.[29] conducted a sub-analysis of the GATEWAY trial to assess the impact of RYGB on OSA severity among patients with class I or II obesity. Thirty-seven participants with mild, moderate, or severe OSA (AHI > 5 events/h) were randomly assigned to undergo RYGB (n = 24) or to a control group receiving standard care (n = 13). After 3 years of follow-up, BMI decreased by –10.6 kg/m² (p25; p75: –12.7; –9.2) in the RYGB group, whereas it increased by 1.7 kg/m² (p25; p75: –1.9; 2.7) in the control group. Similarly, AHI decreased by –13.2 events/h (p25; p75: –22.7; –7) following surgery, compared to an increase of 5 events/h (p25; p75: –4.2; 12.7) in controls. Notably, 70.8% of patients in the surgical group achieved complete remission of OSA (AHI < 5 events/h) at the end of follow-up.

In contrast, Bakker et al.[30] conducted an RCT comparing CPAP therapy with laparoscopic adjustable gastric banding (LGB) in patients with severe OSA (AHI ≥ 30 events/h) and obesity (BMI 35-45 kg/m²). Forty-nine participants were randomized: 28 to the LGB group and 21 to CPAP. The LGB group experienced a significantly greater BMI reduction (from a baseline mean of 39.1 ± 2.9 kg/m² to 35.9 ± 3.5 kg/m² at 9 months and 35.7 ± 3.9 kg/m² at 18 months), whereas BMI remained unchanged in the CPAP group (38.7 ± 3.1, 37.4 ± 3.7, and 37.4 ± 4.5 kg/m², respectively). Regarding AHI, mean values were 29.5 ± 23.4 events/h (LGB) and 20.0 ± 25.3 events/h (CPAP) at 9 months, and 20.9 ± 16.0 vs. 21.4 ± 17.6 events/h at 18 months. No significant differences were found in Epworth Sleepiness Scale scores between groups. The authors concluded that CPAP was superior to LGB for OSA control, although surgical intervention achieved greater weight loss.

Dixon et al.[31] conducted another RCT comparing bariatric surgery with conventional weight-loss therapy over a 2-year follow-up period. Sixty patients with OSA (AHI > 20 events/h), obesity (BMI 35-55 kg/m²), and prior CPAP treatment were randomized to LGB (n = 30) or to a conventional weight-loss program (n = 30). Mean weight loss was –27.8 kg (95% CI, 20.9-34.7) in the LGB group and –5.1 kg (95% CI, 0.8-9.3) in the conventional group. AHI decreased by –25.5 events/h (95% CI, 14.2-36.7) and –14 events/h (95% CI, 3.3-24.6), respectively, with a between-group difference of –11.5 events/h (95% CI: –28.3-5.3), which did not reach statistical significance. CPAP adherence remained unchanged. By the end of the study, 27% of surgical patients and 7% of controls had mild OSA (AHI < 15 events/h), whereas complete remission (AHI < 5 events/h) occurred in only one participant from the conventional weight-loss program group.

Finally, Feigel-Guiller et al.[32] compared weaning rates from non-invasive ventilation (NIV) between patients undergoing LGB and those participating in an intensive nutritional care program. Participants were individuals with obesity (BMI > 35 kg/m²) and either OSA (AHI > 30 events/h) or obesity hypoventilation syndrome (OHS), defined by a partial pressure of carbon dioxide ($PaCO_2$) > 6.5 kPa, requiring NIV. Weaning criteria were defined as AHI < 20 events/h for OSA or sufficient $PaCO_2$ improvement for OHS. Thirty-three patients were assigned to nutritional care and 30 to surgery, with follow-up assessments at 1, 3, and 10 years. Weight loss was consistently greater in the surgical group (15% vs. 6% at 1 year; 14% vs. 3% at 3 years). However, no significant differences were observed in weaning rates at any time point. After 10 years, weaning rates remained low and comparable between groups, and a substantial proportion of patients required additional bariatric interventions.

Collectively, while bariatric surgery has proven effective for achieving substantial and sustained weight loss, its impact on OSA improvement appears variable. The relationship between the extent of weight reduction and the degree of AHI improvement remains uncertain, warranting further investigation.

Conclusions

Taken together, the current evidence indicates that weight-loss interventions, especially lifestyle-based and pharmacological, can substantially attenuate OSA severity in adults with obesity, and can also potentiate the benefits of CPAP. However, most interventional studies, including those evaluating CPAP, adjunctive lifestyle programs, and surgical or pharmacologic approaches, have predominantly enrolled middle-aged males, with limited inclusion of older adults (> 65 years) and females. This underrepresentation restricts the external validity of existing findings and hampers our ability to determine whether the magnitude and

durability of benefit observed in these trials can be extrapolated to populations with different hormonal profiles, body composition, multimorbidity, and adherence patterns.

Furthermore, while obesity is a major and modifiable risk factor, OSA is clearly not confined to individuals with obesity. Craniofacial anatomy, ventilatory control instability, arousal threshold, and upper airway muscle responsiveness contribute to disease expression in ways that are only partially captured by BMI. The growing recognition of OSA phenotypes and endotypes underscores the need for broader diagnostic strategies and more nuanced treatment algorithms that integrate CPAP, weight management, and other targeted interventions according to individual pathophysiology. Future trials should therefore be designed to deliberately include females, older adults, and other underrepresented populations, incorporate multidimensional outcomes beyond AHI, and test stratified or personalized treatment strategies aligned with the biological and clinical heterogeneity described throughout this chapter.

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Conflicts of interest

The authors have no competing interests to disclose.

Ethical considerations

Protection of human subjects and animals. The authors declare that no experiments on humans or animals were performed for this research.

Confidentiality, informed consent, and ethical approval. This study does not involve personal patient data, medical records, or biological samples, and does not require ethical approval. SAGER guidelines do not apply.

Declaration on the use of artificial intelligence. The authors declare that no generative artificial intelligence was used in the writing or creation of the content of this manuscript.

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