



Guideline for the Management of Obesity in Adult Patients With Obstructive Sleep Apnea

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Objectives. Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder that is strongly influenced by obesity, the most important modifiable risk factor. Effective weight management is therefore essential for optimal OSA care. This guideline provides updated, evidence-based recommendations for the evaluation and management of obesity in adults with OSA, incorporating recent advances in lifestyle, pharmacologic, and surgical interventions.

Methods. The Korean Society of Sleep and Breathing convened a multidisciplinary panel. A systematic literature search was performed across major databases through 2025. Evidence was appraised using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach, and recommendations were formulated by consensus using the Evidence-to-Decision framework.

Results. Eight statements were developed. Key recommendations include recognizing obesity as a major risk factor; routinely assessing overweight and obesity; and implementing structured weight reduction through diet, exercise, and behavioral therapy. Glucagon-like peptide-1 receptor agonists are recommended when lifestyle interventions are insufficient, and bariatric/metabolic surgery is advised for severe obesity unresponsive to nonsurgical treatment. Weight loss improves the apnea-hypopnea index, symptoms, and cardiometabolic markers and may reduce positive airway pressure needs. Continued follow-up is recommended even after clinical improvement.

Conclusion. Weight management is a core component of OSA treatment. This guideline offers practical, evidence-based strategies to support clinicians in delivering effective, obesity-focused care for adults with OSA.

Keywords. Obstructive Sleep Apnea; Obesity; Weight Loss; Glucagon-Like Peptide-1 Receptor Agonist; Guideline

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INTRODUCTION

Obstructive sleep apnea (OSA) is a common sleep-related breathing disorder characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia and sleep fragmentation [1-3]. When left untreated, OSA can substantially diminish quality of life, exacerbate cardiovascular and metabolic comorbidities, and increase mortality risk [1-4]. Although prevalence estimates vary across studies, moderate OSA—defined as an apnea-hypopnea index (AHI) ≥ 15 —is thought to affect approximately 6%–17% of adults [5-7]. Given its clinical significance and high prevalence, OSA warrants substantial public health attention.

Obesity is the most important modifiable risk factor for the development and progression of OSA. Numerous population-based and cohort studies have demonstrated a dose-dependent relationship between body mass index (BMI) and both the prevalence and severity of OSA [8-10]. A 10% increase in body weight is associated with an approximately 32% increase in AHI, whereas a 10% weight loss predicts an approximately 26% reduction in AHI [11]. Proposed mechanisms include upper airway narrowing due to parapharyngeal fat deposition; reduced lung volume and functional residual capacity due to abdominal obesity; and impaired central respiratory control and neuromuscular responsiveness related to obesity-associated inflammation and leptin resistance [12-14].

Accordingly, effective weight management is a cornerstone of comprehensive OSA care. Although prior OSA treatment guidelines have emphasized weight control, most have lacked specific, evidence-based recommendations on implementation. In 2018, the American Thoracic Society published the first comprehensive guideline addressing reduced-calorie diets, physical activity, lifestyle modification, pharmacotherapy, and bariatric surgery for weight management in OSA [15]. However, since its publication, new evidence and treatment options—particularly glucagon-like peptide-1 receptor agonists (GLP-1 RAs)—have transformed the therapeutic landscape, highlighting the need for an updated guideline that reflects current scientific and clinical advances [16].

HIGHLIGHTS

- Obesity is the most important modifiable risk factor for obstructive sleep apnea (OSA), and weight management should be a core component of OSA care.
- Structured weight-loss strategies—including lifestyle intervention, pharmacotherapy (e.g., glucagon-like peptide-1 receptor agonists), and bariatric surgery—significantly improve OSA severity, symptoms, and cardiometabolic outcomes.
- Even after weight loss and clinical improvement, regular follow-up is essential because residual or recurrent OSA is common and requires objective reassessment.

Guideline scope and purpose

This guideline aims to provide evidence-based recommendations to help clinicians evaluate and manage obesity in adults with OSA.

MATERIALS AND METHODS

Organization of the panel and working group for guideline development

This guideline was developed under the direction of the President of the Korean Society of Sleep and Breathing (KSSB), SHL, who initiated the process and appointed five panel members (CSP, JHC [Ji Ho Choi], JHC [Jae Hoon Cho], SJH, and himself). These panelists—selected from the KSSB Board of Directors—were chosen for their expertise in OSA and weight-related clinical management. Each panelist independently drafted candidate statements aligned with the predefined aims and scope of the guideline. Through repeated rounds of discussion and refinement, a total of eight statements were agreed upon based on clinical importance and relevance. To manage operational aspects of guideline preparation, a working group was assembled, consisting of a chair (JHC [Jae Hoon Cho]) and three additional members (JHC [Ji Ho Choi], SKP, and YJK). Each stage of development, from statement generation to manuscript drafting, was reviewed and approved by the full panel. All members of the panel and working group declared no conflicts of interest. The guideline was produced with full organizational and financial support from the KSSB.

Literature search

A systematic literature search was conducted across three major databases—PubMed, Embase, and Google Scholar. Search terms combined controlled vocabulary (e.g., MeSH and Emtree terms) with free-text keywords and related expressions. The search strategy focused on two central themes: the relationship between obesity and OSA, and the role of weight reduction or weight-loss therapies in OSA management. When needed, filters for study type or methodological rigor were applied to improve specificity. No limits were imposed on publication year or language. All databases were searched from inception through the latest updates, conducted in June and August 2025.

Classification of evidence-based statements

Clinical practice guidelines are designed to promote optimal patient outcomes, limit potential harms, and reduce unwarranted variability in clinical practice. An evidence-based framework for guideline development involves systematic identification, critical appraisal, and synthesis of available evidence, as well as establishing a transparent relationship between the evidence base and each corresponding statement [17,18]. Evidence-based statements therefore incorporate both the overall quality of support-

ing evidence and the anticipated balance of benefits and harms associated with adherence to each statement.

The terminology used to categorize evidence-based statements is defined in Tables 1 and 2. These guidelines are not intended to replace clinical expertise or professional judgment; rather, they provide a structured framework that may guide clinical decision-making in specific circumstances. Statements classified as “strong recommendations” are expected to result in less variability in clinical practice than those categorized as “recommendations,” whereas “options” allow the greatest degree of flexibility and clinician discretion (Table 3).

Table 1. Levels of evidence

Term	Definition
High	RCTs without important limitations or overwhelming evidence from observational studies
Moderate	RCTs with important limitations or strong evidence from observational studies
Low	Observational studies/case studies/expert opinions

RCT, randomized controlled trial.

Table 2. Aggregate grades of evidence

Grade	Treatment	Harm
A	Systematic review of randomized trials	Systematic review of randomized trials, nested case-control studies, or observational studies with dramatic effect
B	Randomized trials or observational studies with dramatic effects or highly consistent evidence	Randomized trials or observational studies with dramatic effects or highly consistent evidence
C	Non-randomized or historically controlled studies, including case-control and observational studies	Non-randomized controlled cohort or follow-up study with sufficient numbers to rule out a common harm, case series, case-control, or historically controlled studies
D	Case reports, mechanism-based reasoning, or reasoning from first principles	
X	Exceptional situations where validating studies cannot be performed and there is a clear preponderance of benefits over harms	

Table 3. Guideline definitions for evidence-based statements

Statement	Definition	Implied obligation
Strong recommendation	A strong recommendation means that the benefits of the recommended approach clearly exceed the harms (or that the harms clearly exceed the benefits in the case of a strong negative recommendation) and that the quality of the supporting evidence is excellent (grade A or B). In some clearly identified circumstances, strong recommendations may be made on the basis of lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits strongly outweigh the harms.	Clinicians should follow a strong recommendation unless a clear and compelling rationale for an alternative approach is present.
Recommendation	A recommendation means that the benefits exceed the harms (or that the harms exceed the benefits in the case of a negative recommendation), but the quality of evidence is not as strong (grade B or C). In some clearly identified circumstances, recommendations may be made on the basis of lesser evidence when high-quality evidence is impossible to obtain and the anticipated benefits outweigh the harms.	Clinicians should also generally follow a recommendation but should remain alert to new information and sensitive to patient preferences.
Option	An option means that either the quality of evidence that exists is suspect (grade D) or those well-done studies (grade A, B, or C) show little clear advantage for a certain approach versus another.	Clinicians should be flexible in their decision-making regarding appropriate practice, although they may set bounds on alternatives. Patient preference should have a substantial influencing role.

Regardless of guideline recommendations, clinicians retain responsibility for acting in the best interests of their patients while practicing within the limits of their training and professional scope. Guidelines reflect the collective judgment of multidisciplinary panels composed of experienced clinicians and methodologists who evaluate and interpret the scientific evidence relevant to a given topic. Importantly, formulating clinical recommendations inherently involves value-based decisions regarding the relative desirability of potential outcomes associated with different management strategies. A key objective of the guideline development process was to ensure transparency in how these values were applied and to clearly document the rationale underlying each recommendation.

Manuscript preparation

The working group prepared the first complete draft of the guideline, with each member responsible for assigned sections. The full panel subsequently reviewed the entire document, and the chair consolidated comments and incorporated necessary revisions. The revised manuscript was redistributed to all panelists

for a second comprehensive review. This process of revision and panel-wide feedback was repeated twice before the guideline was finalized. Expanded evidence summaries and all supporting data tables are provided in the online supplementary materials.

Statement 1. Clinicians should recognize obesity as a major risk factor for OSA and consistently consider it during the evaluation and management of patients in clinical practice.

Action statement profile

- Level of confidence in evidence: High
- Aggregate evidence quality: Grade B. Multiple large-scale prospective cohort studies and meta-analyses have consistently demonstrated a strong, dose-dependent association between obesity and both the prevalence and severity of OSA. These findings are supported by interventional studies showing clinically meaningful improvements in OSA severity following weight reduction.
- Benefits: Systematic assessment of obesity-related parameters enables improved risk stratification and phenotyping of patients with OSA. Incorporating individualized weight management strategies can reduce the AHI, improve symptoms, enhance the effectiveness of adjunctive OSA therapies, and improve cardiometabolic outcomes.
- Risks, harms, and costs: Potential risks and harms are minimal. Weight assessment is noninvasive and low cost. Lifestyle-based weight management interventions carry negligible risk. Pharmacologic or surgical obesity treatments may be associated with adverse effects and financial costs; however, these are generally acceptable and manageable when appropriately selected and monitored.
- Benefit-harm assessment: The anticipated benefits substantially outweigh the potential harms and costs.
- Exclusions: This statement does not apply to patients with conditions in which weight reduction is contraindicated or clinically inappropriate, such as advanced malignancy, severe frailty, or acute medical instability.
- Policy level: Strong recommendation

Supporting text

Obesity is one of the strongest and most extensively validated risk factors for OSA. Numerous population-based and clinical studies have demonstrated a clear, dose-dependent relationship between excess body weight and both the prevalence and severity of OSA. In large epidemiologic cohorts, increases in BMI, waist circumference, visceral adiposity, and neck circumference are each independently associated with higher AHI values. A 10% increase in body weight has been shown to increase the risk of developing moderate-to-severe OSA by up to sixfold, whereas equivalent weight loss predicts an approximately 26% reduction in AHI [11,19]. These findings have been replicated across multiple clinical trials and longitudinal studies.

Physiologically, obesity contributes to upper airway obstruction through several mechanisms. Excess adipose tissue in the neck and pharyngeal structures increases extraluminal pressure and promotes upper-airway collapsibility. Central and visceral obesity reduce functional residual capacity and impair ventilatory control stability, thereby exacerbating nocturnal hypoxemia and increasing respiratory load. Obesity-associated systemic inflammation, hormonal changes, and metabolic dysregulation may further increase susceptibility to airway collapse [12,20]. Together, these mechanisms operate synergistically and help explain the strong, causal association between elevated BMI and OSA severity.

Interventional evidence reinforces the clinical importance of obesity in OSA. Randomized controlled trials demonstrate that structured weight-loss interventions—including intensive lifestyle modification, anti-obesity pharmacotherapy, and bariatric surgery—produce meaningful reductions in AHI and, in some cases, complete remission of OSA. Gastric bypass and other bariatric surgery cohorts typically show substantial reductions in AHI relative to baseline, while lifestyle-based interventions consistently yield proportional improvements in severity according to the degree of weight loss achieved [21,22]. Recent evidence further quantifies this dose-response relationship between weight reduction and AHI improvement [23].

Given this evidence, clinicians should systematically assess body weight, BMI, waist circumference, and fat distribution during the diagnostic evaluation of patients with suspected or confirmed OSA. Weight-related risk should be discussed routinely with patients, and management plans should incorporate individualized weight-reduction strategies when appropriate. Clinicians should also recognize that weight management complements—but does not replace—other treatments such as positive airway pressure (PAP) therapy, oral appliances, or surgical interventions [24-26]. Integrating structured weight-loss counseling and evidence-based obesity treatments into the care pathway is essential for improving both respiratory outcomes and broader cardiometabolic health.

Statement 2. Clinicians should assess overweight and obesity in patients suspected of OSA (strong recommendation).

Action statement profile

- Level of confidence in evidence: High
- Aggregate evidence quality: Grade A. Multiple large-scale prospective cohort studies, randomized controlled trials, and meta-analyses have consistently demonstrated the association between obesity and OSA, as well as the beneficial effects of weight reduction. The benefits clearly outweigh the harms.
- Benefits: Assessing obesity indices and comorbidities in patients with OSA enables early diagnosis and optimization of treatment pathways, contributing to prediction and preven-

tion of cardiovascular risk.

- Risks, harms, and costs: Minimal harm and negligible cost burden.
- Benefit–harm assessment: The benefits clearly outweigh the harms.
- Exclusions: None (except in special circumstances where anthropometric measurement is not feasible)
- Policy level: Strong recommendation

Supporting text

Obesity is the most important risk factor for OSA. Longitudinal data from the Wisconsin Sleep Cohort Study demonstrated a close association between changes in body weight and changes in AHI [11]. These findings underscore the importance of assessing weight and obesity in OSA management. Diagnostic criteria for obesity vary by ethnicity and region. The World Health Organization defines obesity as a BMI ≥ 30 kg/m², whereas in the Asia–Pacific region, obesity is defined as a BMI ≥ 25 kg/m² [27,28], reflecting differences in body composition and adiposity among Asian populations (Table 4). The selection and use of obesity indices are important in OSA management. Western cohort studies and pathophysiological reviews have shown strong associations between BMI, waist circumference, waist-to-hip ratio, neck circumference, and the presence and severity of OSA [29]. Similarly, studies in Korean patients with OSA have demonstrated that BMI, neck circumference, and waist circumference are significantly correlated with AHI, supporting the importance of abdominal and upper-airway fat distribution in Asian populations [30]. Taken together, these findings support the use of multiple anthropometric indices, rather than reliance on BMI alone, when assessing obesity-related risk in patients with OSA.

Comorbid conditions such as type 2 diabetes mellitus, hypertension, dyslipidemia, and cardiovascular disease have important implications for prognosis and therapeutic planning in patients with obesity and OSA. Because metabolic syndrome and OSA interact to increase cardiovascular event risk, these comorbidities must be assessed at the initial evaluation [31,32].

The efficacy of obesity interventions has been demonstrated in randomized controlled trials. The Interdisciplinary Weight Loss and Lifestyle Intervention for Obstructive Sleep Apnea (INTERAPNEA) trial reported that, among patients with obesi-

ty and OSA, an 8-week multidisciplinary lifestyle intervention reduced body weight by a mean of 7 kg and decreased AHI by more than 23 events per hour [33]. Georgoulis et al. [34] also reported a linear association between $\geq 5\%$ body weight reduction and improvement in AHI. More recently, Malhotra et al. [35] reported that tirzepatide improved AHI, body weight, blood pressure, and daytime sleepiness (Epworth Sleepiness Scale [ESS]) in adults with obesity and OSA. A meta-analysis of GLP-1 RAs also reported consistent improvements in both AHI and weight [36].

Bariatric (metabolic) surgery can lead to substantial weight loss and reductions in AHI in patients with severe obesity, and some long-term studies have also reported reduced cardiovascular risk. However, residual OSA may persist after surgery, emphasizing the need for postoperative polysomnography (PSG) follow-up [21,37]. Therefore, assessing obesity indices and metabolic comorbidities at the initial visit is essential for optimizing diagnosis, treatment, and prognostication in OSA.

Statement 3. Clinicians should ensure that a structured weight-loss strategy is an integral component of the evaluation and management of patients with OSA accompanied by obesity (strong recommendation).

Action statement profile

- Level of confidence in evidence: High
- Aggregate evidence quality: Grade A. Numerous randomized controlled trials, long-term cohort studies, and systematic reviews have consistently reported that weight control through lifestyle modification, pharmacotherapy, and bariatric/metabolic surgery improves OSA severity (AHI), symptoms, and metabolic and cardiovascular indices in patients with obesity and OSA.
- Benefits: Implementing structured weight management in patients with obesity and OSA improves AHI and daytime sleepiness (ESS) and enhances metabolic parameters such as blood pressure, glucose, and lipid levels, thereby reducing cardiovascular events and mortality.
- Risks, harms, and costs: Lifestyle intervention has minimal risks; the main limitation is difficulty with long-term adherence. Pharmacotherapy may cause gastrointestinal adverse effects (e.g., nausea, vomiting, or diarrhea) and impose ongoing costs. Surgery is invasive and entails a risk of complications and higher expenses.
- Benefit–harm assessment: Clear preponderance of benefits over harms.
- Exclusions: Patients with contraindications to anti-obesity medications, high-risk surgical candidates, or conditions in which weight loss is not recommended.
- Policy level: Strong recommendation

Table 4. BMI classification: WHO Western criteria vs. Asia-Pacific criteria

Classification	WHO Western (kg/m ²)	Asia-Pacific (kg/m ²)
Underweight	<18.5	<18.5
Normal range	18.5–24.9	18.5–22.9
Overweight	25.0–29.9	23.0–24.9
Obesity class I	30.0–34.9	25.0–29.9
Obesity class II	35.0–39.9	30.0–34.9
Obesity class III	≥ 40.0	≥ 35.0

BMI, body mass index; WHO, World Health Organization.

Supporting text

Obesity is a major risk factor for OSA; therefore, a structured, stepwise weight-management strategy is essential [15,28]. Lifestyle interventions encompass a multidisciplinary approach that includes dietary modification, exercise, and behavioral therapy. Dietary therapy aims to achieve an energy deficit of 500–1,000 kcal/day and may include approaches such as low-carbohydrate, low-fat, Mediterranean, or intermittent fasting plans, with nutritional balance and sustainability as key goals [38,39]. Exercise therapy should combine ≥ 150 minutes of aerobic training per week with 2–3 sessions of resistance training; this approach not only reduces body weight and fat mass but also improves AHI and blood pressure [33,38,39]. Behavioral therapy—including self-monitoring, goal setting, motivational interviewing, and group-based programs—supports long-term weight maintenance [35].

Tuomilehto et al. [38] and the Sleep Apnea in Look AHEAD (Action for Health in Diabetes; Sleep AHEAD) trial demonstrated that lifestyle interventions significantly improved AHI and metabolic indices, including body weight, glycemic control, blood pressure, and lipid profiles, in patients with overweight or obesity and OSA [39]. The INTERAPNEA randomized trial further showed that even a short-term, intensive multidisciplinary program reduced both body weight and AHI [33]. The primary goal of these interventions is a 5%–10% weight reduction within 6 months, followed by maintenance or additional weight loss [28].

Pharmacotherapy has recently emerged as a major therapeutic pillar. Liraglutide 3.0 mg improved both body weight and AHI in the Satiety and Clinical Adiposity Liraglutide Evidence (SCALE) Sleep Apnea trial [16]. In the 2024 NEJM randomized controlled trial, tirzepatide improved body weight, AHI, blood pressure, and daytime sleepiness in adults with obesity and moderate-to-severe OSA, leading to U.S. Food and Drug Administration approval [35]. Although semaglutide has not been directly studied in OSA, it produced a mean 15% weight loss in the Semaglutide Treatment Effect in People with Obesity (STEP) trial, suggesting potential benefit for OSA [40]. Meta-analyses have also reported that GLP-1 RAs improve both body weight and AHI [36,41].

Bariatric/metabolic surgery has been shown to achieve sustained weight loss and reduce AHI in patients with severe obesity. A meta-analysis by Greenburg et al. [37] reported a significant decrease in AHI after surgery, and a systematic review by Al Oweidat et al. [21] emphasized the need for PSG follow-up because residual OSA is common. The Swedish Obese Subjects study reported reductions in cardiovascular events and mortality and improved survival with long-term follow-up [42–44]. Recent international consensus statements and guidelines have also highlighted the evidence supporting metabolic surgery for weight loss and cardiovascular risk reduction [45,46].

The relative roles of pharmacotherapy and surgery remain under discussion. GLP-1 RAs have produced clinically meaningful weight loss and reductions in AHI, in some cases approaching surgical outcomes [40,41]. However, long-term data suggest that

bariatric surgery yields more durable weight loss and cardiovascular benefits [42–45]. Accordingly, this guideline recommends a stepwise, individualized strategy based on patient characteristics and available resources.

Statement 4. Clinicians should prioritize dietary modification and exercise therapy as first-line strategies for weight reduction in patients with OSA and obesity (recommendation).

Action statement profile

- Level of confidence in evidence: Moderate
- Aggregate evidence quality: Grade B. Numerous randomized controlled trials and systematic reviews have shown that dietary and exercise interventions reduce body weight and improve AHI, ESS, blood pressure, and metabolic profiles. However, heterogeneity in intervention type and duration across studies contributes to variability in long-term adherence and outcomes.
- Benefits: Dietary and exercise therapy promote weight loss and improve AHI and ESS, while improving blood pressure and metabolic indices, thereby reducing cardiovascular risk. These interventions are noninvasive, accessible, and suitable as first-line treatments.
- Risks, harms, and costs: Minimal risk. The main limitation is difficulty maintaining long-term adherence to lifestyle modification. The cost burden is negligible.
- Benefit–harm assessment: Benefits outweigh harms.
- Exclusions: Patients with unstable cardiovascular disease, uncontrolled diabetes or endocrine disorders, acute musculoskeletal injuries, or contraindications to exercise.
- Policy level: Recommendation

Supporting text

Dietary modification and exercise therapy are recommended as primary, noninvasive strategies to reduce body weight and improve AHI in patients with obesity and OSA. A low-calorie diet (energy deficit of 500–1,000 kcal/day) consistently produces clinically meaningful weight loss and improvements in AHI. Tuomilehto et al. [38] conducted a 1-year randomized controlled trial of lifestyle intervention in patients with obesity and OSA and reported significant reductions in body weight and AHI. The Sleep AHEAD trial reported that intensive dietary intervention in patients with OSA, obesity, and type 2 diabetes improved AHI, blood pressure, and metabolic indices [39].

Various dietary patterns—including low-carbohydrate, low-fat, high-protein, and intermittent fasting approaches—may be used, with nutritional balance and sustainability as key principles. A Mediterranean diet-based intervention not only improved body weight and blood pressure but also increased PAP adherence [47]. However, the relative superiority of specific dietary strategies remains uncertain in OSA populations because some evidence is extrapolated from studies of general obesity rather

than from trials conducted specifically in OSA.

Exercise interventions contribute not only to reductions in body weight and fat mass but also to improvements in AHI and ESS. The INTERAPNEA randomized controlled trial reported that an 8-week multidisciplinary program reduced body weight by approximately 7 kg and decreased AHI by more than 23 events per hour [33]. In the NEJM trial by Chirinos et al. [48], combining weight reduction with PAP produced greater improvements in cardiovascular and metabolic markers than PAP alone.

Systematic reviews have reached similar conclusions. In a systematic review, Peng et al. [49] reported that exercise improves AHI and sleep architecture in patients with OSA. Aiello et al. [50] also reported, in a meta-analysis, that exercise interventions in patients with obesity and OSA improve AHI, symptoms, and metabolic indices.

Exercise may influence OSA pathophysiology through dual mechanisms: reducing visceral and cervical fat while preserving and strengthening upper airway muscle tone. Combining aerobic exercise for fat reduction with resistance training and/or oropharyngeal myofunctional therapy to strengthen upper airway dilator muscles may maximize improvements in AHI and metabolic markers. Accordingly, individualized exercise prescriptions that target fat reduction and muscle preservation, rather than weight per se, are recommended [51-54].

Although the superiority of specific dietary or exercise regimens remains unclear, consistent evidence indicates that weight loss itself is the key determinant of improvement in AHI, ESS, blood pressure, and metabolic parameters in patients with OSA [33,38,39,48-50]. Accordingly, tailored strategies that reflect patient preferences and long-term sustainability are recommended. Lifestyle interventions are generally safe but may be limited by challenges in long-term adherence; incorporating behavioral techniques such as goal setting, self-monitoring, and motivational interviewing may enhance effectiveness. In summary, dietary modification and exercise therapy safely and cost-effectively improve body weight, AHI, ESS, blood pressure, and metabolic profiles, while potentially improving PAP adherence in patients with obesity and OSA.

Statement 5. Clinicians should consider pharmacological treatment with a GLP-1 RA when diet control and exercise therapy for weight loss have failed in the management of obese patients with OSA (recommendation).

Action statement profile

- Level of confidence in evidence: High
- Aggregate evidence quality: Grade A. Multiple systematic reviews, meta-analyses, and well-designed clinical trials.
- Benefits: Reductions in AHI in patients with obesity and OSA, which may mitigate symptoms and associated morbidities.
- Risks, harms, and costs: Common risks include gastrointestinal adverse effects (e.g., nausea or vomiting), and rare but

serious risks include pancreatitis and gallbladder disease. A key concern is the high ongoing medication cost, which can create substantial financial burden for patients requiring long-term therapy, particularly in the absence of adequate insurance coverage. In Korea, most GLP-1 RAs prescribed for obesity management are not covered by national health insurance, which may limit feasibility in clinical practice.

- Benefit-harm assessment: Preponderance of benefits over harms.
- Exclusions: GLP-1 RA therapy is contraindicated in patients with a personal or family history of medullary thyroid carcinoma or multiple endocrine neoplasia type 2. Caution is warranted in patients with a history of acute pancreatitis, cholecystitis, or cholelithiasis due to the risk of recurrence or exacerbation. Use also requires caution in patients with New York Heart Association class IV heart failure, active inflammatory bowel disease, or gastroparesis. In addition, severe renal impairment (e.g., estimated glomerular filtration rate <30 mL/min/1.73 m²) is a contraindication for certain agents such as exenatide. Because of hypoglycemia risk, careful management is required in patients who are already at risk.
- Policy level: Recommendation

Supporting text

Numerous original studies, reviews, and meta-analyses have evaluated GLP-1 RAs (e.g., liraglutide and tirzepatide) for improving both weight loss and sleep-disordered breathing in patients with obesity and OSA [16,35,41,36,55-63]. Across most relevant studies, changes in body weight and AHI before and after GLP-1 RA treatment indicate that GLP-1 RA therapy leads to reductions in AHI concomitant with weight loss.

Blackman et al. [16] investigated liraglutide 3.0 mg combined with diet and exercise for OSA severity in patients with obesity. After 32 weeks, the liraglutide group achieved greater weight loss (−5.7%) than the placebo group (−1.6%). The reduction in AHI was also greater with liraglutide (−12.2 events/hr) than with placebo (−6.1 events/hr). Post hoc analysis reported a significant correlation between weight loss and improvement in OSA indices. Liraglutide also demonstrated beneficial effects on hemoglobin A1c and systolic blood pressure, with a safety profile consistent with that observed in lower-dose studies. The authors concluded that liraglutide 3.0 mg improves AHI, body weight, blood pressure, and glycemic control in patients with moderate-to-severe OSA. Malhotra et al. [35] conducted two 52-week phase 3 trials evaluating tirzepatide in adults with obesity and moderate-to-severe OSA, including both non-PAP users (Trial 1) and PAP users (Trial 2). In both trials, tirzepatide was more effective than placebo, with mean reductions in AHI and body weight of −25.3 events per hour and −17.7% in Trial 1 and −29.3 events per hour and −19.6% in Trial 2, respectively. Tirzepatide also improved secondary endpoints, including hypoxia, sleep quality,

high-sensitivity C-reactive protein (hsCRP) levels, and systolic blood pressure. Gastrointestinal adverse events were common but were predominantly mild to moderate. The authors concluded that tirzepatide reduces AHI and body weight and improves cardiovascular risk factors and sleep-related indices. Despite these clinical benefits, the high cost of GLP-1 RA therapy remains a major barrier to widespread use. In Korea, GLP-1 RAs are large-ly classified as non-reimbursable medications for obesity treatment. Clinicians should therefore discuss the economic burden and current insurance coverage with patients to support long-term adherence and real-world feasibility.

Following publication of the tirzepatide efficacy study in 2024, several meta-analyses evaluating the effect of GLP-1 RAs on sleep-disordered breathing alongside weight loss have been published [36,60-63]. Bardóczy et al. [60] conducted a systematic review and meta-analysis of five randomized controlled trials (n=1,024) assessing the impact of incretin-based therapies, including GLP-1 agonists and dual GLP-1/glucose-dependent insulinotropic polypeptide agonists, on AHI in patients with OSA. Over treatment periods of at least 12 weeks, all included studies reported significant reductions in body weight and BMI. The pooled results showed a mean reduction in AHI of -14.45 events/hr. Pairwise meta-analysis also showed that incretin-based therapy produced greater improvement in AHI than usual care (mean difference: -11.61 events/hr). The authors concluded that weight loss achieved through these therapies improves AHI, highlighting their therapeutic potential for treating sleep-disordered breathing in patients with overweight and OSA.

Aside from GLP-1 RA medications, the only randomized controlled trial evaluating a non-GLP-1 RA pharmacologic agent for OSA in adults with obesity investigated a phentermine/topiramate combination [64]. Winslow et al. [64] reported that 28 weeks of phentermine/topiramate improved moderate-to-severe OSA in adults with obesity. Compared with placebo, combination therapy produced greater reductions in AHI (-31.5 vs. -16.6 events/hr) and weight loss (-10.2%), and these outcomes were significantly correlated. Although this regimen may be an alternative option, adverse effects and high costs remain notable barriers. Given limited evidence from a single randomized trial, this therapy is not currently included in formal guideline recommendations.

Statement 6. Clinicians may consider that, in patients with obesity and OSA who are using PAP therapy, weight loss potentially improves OSA severity and enhances adherence to PAP treatment (optional).

Action statement profile

- Level of confidence in evidence: Low. Reductions in pressure requirements and PAP need are consistently supported by randomized trials, prospective cohorts, and systematic reviews (Grade B). Direct, short-term increases in nightly

PAP use (adherence) are not consistently demonstrated (Grade C).

- Aggregate evidence quality: Grade B for reductions in PAP need/pressure and cardiometabolic benefits; Grade C for short-term adherence improvements. Evidence that weight loss directly increases short-term PAP adherence (hours/night) is inconsistent, with randomized data showing no between-group difference; therefore, certainty is Grade C, and the recommendation strength reflects this uncertainty.
- Benefits: Reductions in AHI and respiratory disturbance index and, in some cases, the possibility of PAP discontinuation; improvements in blood pressure, triglycerides, hsCRP, and health-related quality of life.
- Risks, harms, and costs: Costs and adverse effects associated with lifestyle intervention, pharmacotherapy (e.g., GLP-1 RAs), or bariatric surgery; the risks associated with evaluation and follow-up are low.
- Benefit-harm assessment: Clear preponderance of benefits.
- Exclusions: Contraindications to short-term weight reduction (e.g., acute severe illness or active eating disorder).
- Policy level: Optional recommendation

Supporting text

Weight loss reduces OSA severity and can diminish the clinical need for PAP. In a Spanish randomized clinical trial, an 8-week interdisciplinary intervention reduced AHI by 51%; 45% of participants no longer required PAP at 8 weeks, and 61.8% no longer required PAP at 6 months. Blood pressure, body weight, and quality of life also improved [33]. Weight loss also reduces the therapeutic pressure required for PAP. In a prospective study of laparoscopic adjustable gastric banding (LAGB), follow-up polysomnography showed lower prescribed pressures among continuing PAP users and a reduction in the proportion requiring nasal PAP [65].

Whether weight loss directly increases PAP adherence (hours/night) should be interpreted cautiously. In a randomized trial comparing PAP alone with PAP plus weight loss, mean PAP use was approximately 4.0 hours/night, with no significant between-group difference, indicating that short-term adherence gains are not assured [48]. Because few short-duration randomized controlled trials have evaluated adherence as a primary endpoint, longer multicenter trials are needed. Accordingly, adherence-related claims should be made cautiously, and future multicenter trials powered for adherence endpoints are needed.

Importantly, combined management addresses cardiometabolic risk domains that are not reliably corrected by PAP alone. A meta-analysis reported that adding weight reduction to PAP resulted in additional systolic blood pressure lowering (approximately 8–9 mmHg) and reductions in triglycerides compared with PAP alone [66]. Weight loss combined with PAP improved hsCRP and insulin sensitivity more than PAP monotherapy, whereas changes in the PAP-only arm were inconsistent [67]. Clinical-

ly meaningful blood pressure reductions commonly occur with approximately 10% loss of baseline body weight [66].

For adults with obesity-related OSA who use PAP, PAP remains the foundational therapy; however, clinicians should co-initiate structured weight-loss interventions (lifestyle, GLP-1 RAs, and/or surgery, as indicated) to improve blood pressure, triglycerides, inflammation, and insulin sensitivity and to reduce required PAP pressure [66,67].

Weight loss does not automatically justify stopping PAP. Instead, clinicians should de-escalate pressure stepwise and determine maintenance or discontinuation based on objective data, including PSG, home sleep apnea testing (HSAT), and PAP device reports, with a prespecified re-evaluation plan [33]. In adults with obesity-related OSA who use PAP, pragmatic follow-up during weight loss should include repeat PSG or HSAT approximately 3–6 months after achieving $\geq 10\%$ body-weight reduction (or earlier if device downloads show residual AHI ≥ 5 , symptoms recur, or leak becomes significant), with reassessment again at 6–12 months. PAP should be retitrated, including stepwise pressure reduction, based on objective results rather than symptoms alone.

Clinicians should recognize that PAP use itself has been associated with weight gain in meta-analyses, underscoring the need for concurrent weight management during PAP therapy. A meta-analysis of 25 randomized trials ($n=3,181$) reported significant increases in BMI and body weight with PAP [9]. A subsequent systematic review and meta-analysis reported increases in BMI after PAP, particularly with ≤ 5 hours/night use and in participants without cardiovascular disease and/or with dysglycemia [68]. Routine BMI monitoring and concurrent interventions (GLP-1 RAs, nutrition, and physical activity) are therefore recommended to mitigate PAP-associated weight gain.

Evidence remains limited regarding whether the mode of weight loss (lifestyle vs. GLP-1 RAs vs. surgery), magnitude, and durability of weight loss differentially affect long-term PAP pressure requirements and adherence; this is an important area for future research.

Statement 7. Clinicians should consider surgical weight-loss interventions for patients with OSA and severe obesity when all nonsurgical weight-loss treatments have failed (recommendation).

Action statement profile

- Level of confidence in evidence: Moderate. Although randomized trials are limited, multiple systematic reviews/meta-analyses and prospective cohorts show consistent direction of effect, with follow-up extending to 5 years in some studies; however, overall certainty is tempered by risk of bias and heterogeneity.
- Aggregate evidence quality: Grade B. Prospective cohorts and systematic reviews/meta-analyses with some heteroge-

neity and publication bias.

- Benefits: Improvements in AHI, oxygen desaturation index (ODI), time with oxygen saturation below 90% (T-90), mean and nadir oxygen saturation (SpO_2), ESS, and sleep architecture (rapid eye movement and stage N3 sleep), and improved quality of life; in selected patients, PAP de-escalation or discontinuation may be possible.
- Risks, harms, and costs: Surgical complications (e.g., bleeding, leak, and reoperation), nutritional deficiencies, and long-term follow-up burden and costs; residual or recurrent OSA may occur.
- Benefit–harm assessment: The magnitude of benefit in AHI/desaturation and broader metabolic and quality-of-life outcomes exceeds procedural risks in appropriately selected patients.
- Exclusions: Standard surgical contraindications (e.g., uncorrected coagulopathy/active bleeding, severe uncontrolled psychiatric illness, active substance misuse); situations in which operative risk outweighs benefit; and OSA phenotypes not primarily obesity-driven (e.g., pronounced craniofacial structural abnormalities), for which alternative priorities may supersede bariatric surgery.
- Policy level: Recommendation for adults with OSA and BMI ≥ 35 kg/m² who have not achieved adequate weight loss with nonsurgical therapy.

Supporting text

Among adults with severe obesity and OSA who do not achieve adequate weight loss with lifestyle and/or pharmacotherapy (including GLP-1 RAs), bariatric/metabolic surgery is associated with clinically meaningful improvements in sleep-disordered breathing and related outcomes. Contemporary systematic reviews and meta-analyses report mean reductions in AHI of approximately 19–23 events per hour, reductions in respiratory disturbance index of approximately 34 events per hour, and remission rates of approximately 65%, typically accompanied by substantial reductions in BMI (approximately 11.6–11.9 kg/m²) and improvement in ESS [21,69]. These benefits extend to oxygenation metrics, including improvements in ODI, T-90, and mean and nadir SpO_2 [21,69].

Procedure-level differences are suggested; however, heterogeneity limits firm comparative conclusions. Available evidence suggests that procedures with combined malabsorptive and restrictive mechanisms, such as Roux-en-Y gastric bypass (RYGB), yield larger improvements in AHI than purely restrictive procedures such as LAGB [70]. Broad literature syntheses have also described a gradient of effect across procedures, with biliopancreatic diversion with duodenal switch, followed by RYGB, then sleeve gastrectomy, and finally LAGB showing decreasing average impact on OSA-related endpoints [71,72].

Durability of effect is supported by longer-term follow-up in selected cohorts. A 5-year prospective multicenter study report-

ed a reduction in mean AHI from 27.8 to 8.8 events per hour and remission in 55% of patients; however, approximately 20% had persistent moderate-to-severe OSA, indicating that remission is not universal and underscoring the need for follow-up testing [72]. In an Asian prospective cohort, AHI declined from 51.0 to 9.3 events per hour by 12 months; however, only approximately 50% achieved remission, and postoperative PSG was recommended to reassess residual disease [73]. With respect to PAP therapy, prospective data after LAGB showed fewer patients requiring PAP and lower prescribed pressures among those who continued PAP, supporting stepwise PAP de-escalation guided by objective reassessment [65].

Important limitations temper overall certainty. Several systematic reviews have rated the aggregate evidence as low because of study design, variable follow-up, heterogeneous inclusion criteria and diagnostic methods, and potential publication bias [69]. Reports also note that OSA recurrence risk may increase with longer follow-up in some series, whereas other studies document sustained benefit, underscoring heterogeneity in long-term trajectories [72,74,75]. Accordingly, postoperative pathways should incorporate long-term surveillance, structured weight-maintenance programs, and standardized sleep testing (PSG or HSAT) to guide PAP pressure adjustment, maintenance, or discontinuation. Preoperative planning should document sustained attempts at nonsurgical weight loss, include psychological evaluation, and set expectations for ongoing monitoring and nutritional follow-up [70,72].

For adults with OSA and class II/III obesity (Table 4) who have not achieved adequate weight loss with nonsurgical therapy, bariatric/metabolic surgery improves AHI, oxygen desaturation measures, sleep architecture, and patient-reported outcomes, with benefits that may persist for years in many patients. Given residual and recurrent disease in a substantial minority, objective reassessment and longitudinal care remain integral components of management [73,75,76]. Postoperative PSG (or HSAT, when appropriate) should be planned approximately 6–12 months after surgery and with any $\geq 10\%$ weight change, given that remission is not universal and moderate-to-severe OSA can persist.

Statement 8. Clinicians should ensure continued regular follow-up for patients with obesity and OSA, even when weight loss has led to improvement in OSA (strong recommendation).

Action statement profile

- Level of confidence in evidence: High for the persistence/recurrence signal; moderate regarding optimal testing intervals and modality selection.
- Aggregate evidence quality: Grade A. Consistency across multicenter cohorts, randomized trials, and surgical cohorts.
- Benefits: Early identification of residual/recurrent OSA; PAP retitration, including pressure reduction; reconciliation of symptom-objective discrepancies; and strengthened

management of cardiovascular and metabolic risk.

- Risks, harms, and costs: Costs and inconvenience associated with PSG, HSAT, and clinic follow-up; procedural risk is low.
- Benefit-harm assessment: The potential harm of missing residual OSA (daytime sleepiness, cardiovascular and metabolic deterioration) outweighs the modest burden of monitoring; overall benefit predominates.
- Exclusions: If short-term sleep testing is not feasible because of acute severe illness, defer testing and perform interim surveillance of symptoms and SpO₂.
- Policy level: Strong recommendation

Supporting text

Weight loss reduces OSA severity and improves symptoms; however, complete remission is not universal, and some patients may have residual disease or experience symptom recurrence over time. In a Korean prospective cohort, despite a marked reduction in mean AHI (51.0 to 9.3 events/hr) after bariatric surgery, residual OSA remained common, and postoperative PSG was recommended—particularly for patients with lower preoperative nadir SpO₂ and higher supine AHI [73].

A 1-year follow-up study reported that, despite substantial weight loss, only 1 of 24 patients (4%) achieved complete resolution of OSA, whereas most patients (71%) continued to exhibit moderate-to-severe disease [77]. Although the required PAP pressure decreased from 11.5 to 8.4 cm H₂O, many patients voluntarily discontinued PAP therapy because of subjective improvement in symptoms and reduced snoring. These findings suggest that relying on weight loss as a “cure” for OSA may lead to premature or inappropriate discontinuation of therapy. If persistent OSA is not recognized or properly treated after weight loss, patients may continue to experience poor health and reduced quality of life, and may also be at higher risk for weight regain and cardiovascular disease [77].

Long-term monitoring is also necessary after nonsurgical weight loss. In a randomized lifestyle trial with 4-year follow-up, improvements in AHI were sustained; however, remission occurred in only approximately 20%, and complete remission was less common among those with greater baseline severity. Accordingly, even with symptomatic and AHI improvements, periodic reassessment using PSG or HSAT, symptom measures, and PAP device data are clinically prudent measures [39].

In a prospective LAGB cohort comparing pre- and postoperative PSG, more patients no longer required PAP after surgery, and among those who continued PAP, prescribed pressures were lower in every case. After weight loss, clinicians should not simply maintain prior settings or stop PAP; instead, objective testing should be repeated, and therapy should be adjusted—via stepwise pressure reduction, maintenance, or supervised discontinuation—based on measured results [65].

Weight loss should not be equated with cure. Clinicians should maintain regular objective assessment (PSG or HSAT) to deter-

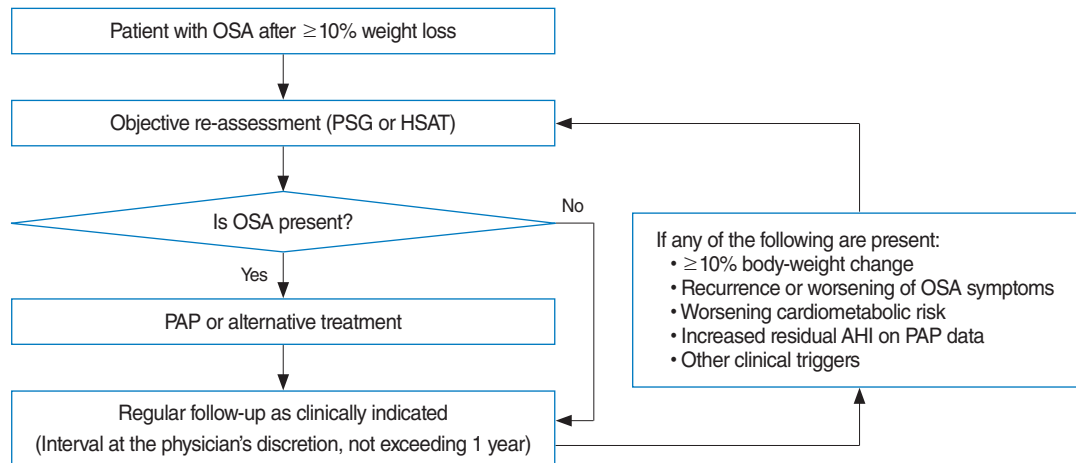


Fig. 1. Follow-up algorithm after weight loss in patients with obstructive sleep apnea. OSA, obstructive sleep apnea; PSG, polysomnography; HSAT, home sleep apnea testing; AHI, apnea-hypopnea index; PAP, positive airway pressure.

mine the presence and extent of residual or recurrent OSA and to detect deterioration early. PAP device data (usage hours, leak, and residual AHI) should be reviewed alongside symptoms (e.g., snoring and ESS), and PAP should be retitrated accordingly. Decisions regarding stepwise pressure reduction, maintenance, or supervised discontinuation should be based on objective findings. Clinicians should also establish and reinforce long-term plans for weight maintenance and metabolic care to sustain benefit. This longitudinal management reduces the risk of undertreating residual OSA and the risk of prematurely discontinuing PAP without adequate evidence [73]. PSG or HSAT should be performed 6–12 months after weight loss or bariatric surgery and after any $\geq 10\%$ weight change or symptom relapse. PAP downloads should be reviewed every 3–6 months, and PAP should be retitrated when residual AHI is ≥ 5 or when symptoms recur or leak persists (Fig. 1).

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

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