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Review

Evidence-based strategies for the treatment of sleep apnea: a systematic literature review

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Abstract: Obstructive sleep apnea is a common sleep disorder characterized by repeated upper airway obstruction during sleep, causing intermittent oxygen desaturation, sleep fragmentation, and reduced quality of life. Among the main treatments, continuous positive airway pressure and nocturnal oxygen therapy are widely used. Despite their efficacy, both have limitations, including poor long-term adherence, device-induced discomfort, and the need for regular monitoring. The pharmacist can play a key role in improving therapeutic support and adherence. This review aims to evaluate the effectiveness and safety of continuous positive airway pressure and nocturnal oxygen therapy in the treatment of obstructive sleep apnea and to identify pharmaceutical interventions to optimize adherence and manage adverse effects. This systematic review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses guidelines and included studies from PubMed, Cochrane Library, and ScienceDirect involving adults treated with these therapies. Thirty-one studies were included. Across the included studies, continuous positive airway pressure therapy consistently reduced the apnea-hypopnea index by approximately 15 to 30 events per hour, often corresponding to a relative reduction exceeding 50% from baseline. Improvements in nocturnal oxygenation were also observed, with increases in mean SpO_2 ranging from 2 to 9 percentage points. In cardiovascular outcomes, the use of continuous positive airway pressure was associated with reductions in nocturnal systolic blood pressure typically ranging from 3 to 7 mmHg among adherent patients. Oxygen therapy improved oxygenation but showed variable effects on respiratory events and adherence. In conclusion, continuous positive airway pressure remains the reference treatment, while pharmacist interventions may strengthen adherence and optimize management.

Keywords: obstructive sleep apnea; continuous positive airway pressure; oxygen therapy; pharmaceutical interventions; adherence

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Introduction

Obstructive sleep apnea (OSA) is a respiratory disorder characterized by repeated interruptions in breathing during sleep, caused by partial or complete obstruction of the upper airway [1]. These episodes, known as apneas or hypopneas, disrupt normal ventilation and fragment sleep, resulting in intermittent hypoxemia and repeated activation of the sympathetic nervous system [2]. Beyond its clinical manifestations, OSA is increasingly recognized for its complex pathophysiological consequences. Intermittent hypoxia triggers oxidative stress, promotes systemic inflammation, and contributes to endothelial dysfunction [3]. Mechanisms believed to underlie the strong association between OSA and cardiovascular and metabolic complications include sympathetic overactivation during sleep, which may persist into wakefulness, exacerbating blood pressure (BP) variability and insulin resistance [4]. OSA was

also associated with an increased risk of hypertension (HTN), depression, excessive daytime sleepiness, and a marked decline in quality of life, so it has emerged as a major public health concern. OSA is highly prevalent among overweight adults, especially those with cardiovascular and/or metabolic comorbidities. Despite potentially severe clinical consequences, OSA remains significantly underdiagnosed [4]. Its impact on patients' cognitive function, professional activity, and social well-being is considerable. Epidemiologically, the prevalence of moderate to severe OSA is estimated to range between 10% and 20% in middle-aged adults, with a clear male predominance [5]. Moderate OSA is usually defined by an apnea-hypopnea index (AHI) of 15–30 events per hour, while severe OSA corresponds to an AHI greater than 30 events per hour [6]. Given the substantial burden of this condition, the development and implementation of effective, tolerable, and personalized therapeutic strategies are critical. Understanding the underlying processes is essential for evaluating the true impact of therapeutic interventions [7]. Continuous positive airway pressure (CPAP) is widely recognized as the gold-standard treatment for moderate-to-severe OSA, as it maintains upper airway patency during sleep by delivering constant positive pressure [8,9]. While CPAP has demonstrated efficacy in reducing the AHI and improving symptoms, its long-term use is often limited by side effects, discomfort, and suboptimal adherence [10]. As an alternative or adjunct therapy, nocturnal oxygen supplementation (NOS) has been investigated, particularly in patients with significant oxygen desaturation or CPAP intolerance [11–13]. However, its utility remains controversial due to heterogeneous study outcomes and concerns about potential adverse effects, especially in patients at risk of hypoventilation or carbon dioxide retention (hypercapnia) [11–13]. To date, the evidence supporting the use of oxygen therapy in OSA is limited and fragmented.

Other therapeutic approaches for OSA also exist, including oral appliances [8,9], weight loss and lifestyle interventions [14–16], positional therapy [17], upper airway surgery [16], and, more recently, hypoglossal nerve stimulation [18]. These alternatives may be considered depending on disease severity, patient preferences, and treatment tolerance. Clinical trials vary widely in terms of patient selection, intervention protocols, and outcome measures, and many include small sample sizes, lack long-term follow-up, or fail to control for key confounding factors. Moreover, few high-quality trials directly compare oxygen therapy to CPAP or assess their combined use [12].

Given these uncertainties, it is essential to clarify the benefits, limitations, and risks associated with the use of medical oxygen and positive airway pressure (PAP) ventilation in the treatment of OSA. A rigorous analysis of the existing literature would not only help define their respective efficacy but also assess their safety, tolerability, and overall clinical impact.

The objective of this study is to conduct a systematic literature review, according to Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines, on the use of oxygen therapy and PAP ventilation in the treatment of patients with OSA and compare the clinical efficacy, safety profile, and adverse effects of these interventions, whether used independently or in combination. Additionally, this work aims to integrate a pharmaceutical care perspective, emphasizing the pharmacist's role in promoting adherence, ensuring safety, and managing comorbidities among patients undergoing treatment for OSA. Through this approach, this study seeks to support a better understanding of available therapeutic options, promote their integration into pharmaceutical practice, and guide clinical decisions toward optimized, evidence-based strategies.

Despite the abundance of studies evaluating CPAP and nocturnal oxygen therapy in OSA, existing reviews primarily focus on clinical efficacy or physiological outcomes, with limited attention to treatment safety, real-world tolerability, and the role of pharmaceutical care. To our knowledge, no previous systematic review has simultaneously compared CPAP and oxygen therapy while explicitly integrating the pharmacist's contribution to adherence optimization, adverse effect management, and patient safety. By addressing this gap, the present review provides an original, practice-oriented synthesis that bridges clinical evidence with pharmaceutical care perspectives.

This work also aims to explore the pharmacist's role in supporting patients with OSA. Pharmacists can contribute to early detection [19,20], improve treatment adherence and provide therapeutic education [19], manage comorbidities, and monitor adverse effects related to oxygen therapy and CPAP [21]. Through patient-centered counselling and coordination with healthcare teams, their involvement may enhance both the safety and effectiveness of care [20].

Materials and Methods

Study Design

This systematic review was conducted in accordance with the PRISMA guidelines and registered in PROSPERO (ID 1165397). The primary objective was to identify and synthesize original studies evaluating the clinical efficacy and safety of PAP ventilation (CPAP, bilevel positive airway pressure (BiPAP)) and nocturnal oxygen therapy in the treatment of OSA in adults.

The research strategy was guided by the following PICO framework:

P (Population): Adult patients diagnosed with OSA, with or without associated comorbidities (obesity, cardiovascular diseases, chronic obstructive pulmonary disease (COPD)), requiring respiratory support.

I (Intervention): Oxygen therapy (medical oxygen) and/or PAP ventilation (CPAP, BiPAP), used either alone or in combination. This includes pressure or flow adjustments, titration protocols, and interventions aimed at monitoring and improving treatment adherence.
C (Comparison): Patients receiving standard treatment (CPAP alone), another form of non-invasive ventilation, oxygen therapy alone, or no intervention (placebo or absence of treatment), depending on the control groups described in the included studies.
O (Outcomes): Reduction of the AHI, improvement in SpO_2, decrease in cardiovascular risk or all-cause mortality, occurrence of adverse events or toxicity related to the interventions, and overall efficacy and tolerability of the treatments.

PICO Question

Are oxygen therapy and PAP ventilation effective strategies for the treatment of sleep apnea? How can pharmacists intervene in order to improve the efficacy and safety of these therapeutic strategies?

Search Strategy

A comprehensive literature search was performed using two main electronic databases: PubMed and ScienceDirect. The Cochrane Library was also screened in the early stages to identify existing reviews and guide the search strategy.
The search period covered January 2015 to July 2025.
Predefined filters were applied to:

- Restrict the search to original research articles (excluding reviews, meta-analyses, editorials, and letters);
- Include studies conducted exclusively on adult human subjects; animal studies were not considered.

The search terms combined keywords related to OSA, CPAP, and oxygen therapy (Table 1).

Table 1. Bibliographic research strategy.

Data base	Research strategy
Pubmed/Cochrane	(oxygen [MeSH Terms]) AND (sleep apnea [MeSH Terms]); (positive pressure ventilation [MeSH Terms]) AND (sleep apnea [MeSH Terms])
ScienceDirect	oxygen sleep apnea AND positive pressure ventilation OR oxygen therapy

Eligibility Criteria

Inclusion criteria:

- Original clinical studies (randomized controlled trials, observational studies, interventional studies) involving adult patients with OSA;
- Articles assessing the efficacy, tolerability, or adverse effects of CPAP, oxygen therapy, or their combination;
- Publications between January 2015 and July 2025, written in English, French, Spanish, or Portuguese.

Exclusion criteria:

- Narrative reviews, systematic reviews, or meta-analyses (excluded from the Results section but consulted for background information in the Introduction);
- Animal or pediatric studies;
- Editorials, commentaries, letters to the editor, or articles without original data.

Results

A total of 710 references were identified (694 from PubMed and 16 from ScienceDirect) after applying the initial filters (research articles, 10-year range, exclusion of animal studies). Titles and abstracts were screened for relevance, and when there was uncertainty, the full text was reviewed. The initial selection of titles and abstracts was carried out independently by all authors. Full texts of potentially eligible studies were retrieved and assessed based on the inclusion and exclusion criteria. Discrepancies were resolved through discussion and consensus. In the end, 31 studies were included, 30 from PubMed and 1 from ScienceDirect (Figure 1). The main characteristics of the 31 included studies, focusing on adult patients with OSA, are summarized in Table 2. These studies evaluate the efficacy and tolerability of PAP ventilation (CPAP, BiPAP) and nocturnal oxygen therapy, either alone or in combination, using various methodological approaches.

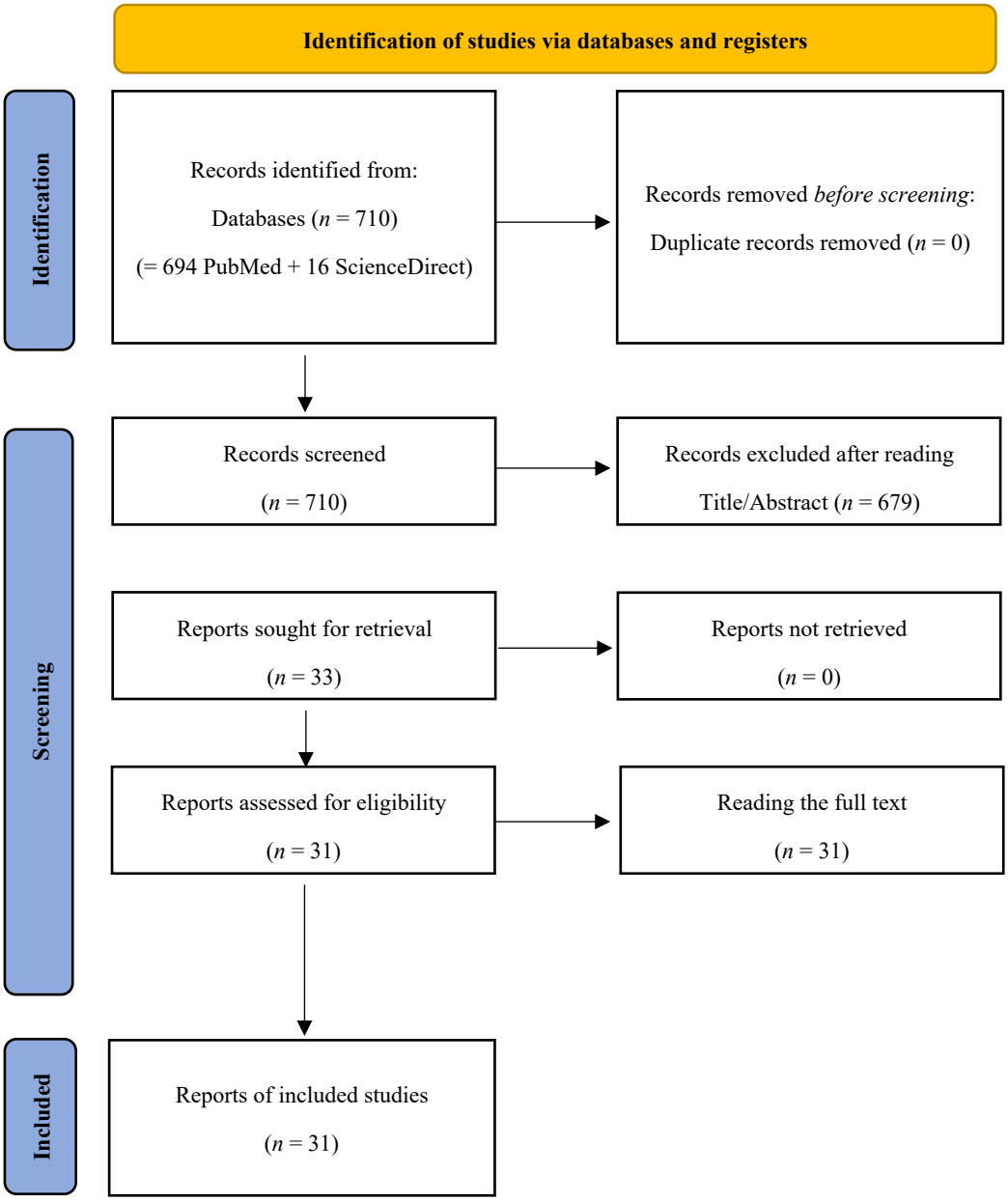

Figure 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) flow diagram.

Table 2. Results of the study.

Author(s), Year	Objective	Study Type	Population	Intervention /Comparison	Effectiveness	Safety	Limitations
Tan L *et al.*, 2023 [22]	To explore the clinical, polysomnographic, and HRV characteristics of highland OSA patients receiving one night NOS and identify predictors of treatment response	Post-hoc analysis of a randomized, placebo controlled, crossover trial	34 highland OSA patients (Tibetan men, 30–60 years old, living at 3200 m altitude, Shangri-La, China)	NOS / sham oxygen	NOS significantly reduced AHI (median −23.2/h in responders), improved ODI, SpO_2, sleep efficiency, reduced arousals and daytime BP in responders. Non-responders showed smaller improvements	No major adverse events reported. Study focused on efficacy and physiological responses; safety profile acceptable	Small sample size; post-hoc design; short-term intervention (one night only); limited generalizability to non-highland populations; no long-term outcomes
Ni YN *et al.*, 2024 [23]	To investigate whether the AHI, a measure of residual sleep apnea burden during therapy, predicts BP response to CPAP and oxygen treatment	Secondary analysis of a randomized controlled trial (HeartBEAT study)	169 adults with moderate to severe OSA and cardiovascular risk factors (mean age ~63 years; 71% male)	CPAP therapy / oxygen therapy (2 L/min)	Greater reduction in nighttime systolic BP when $\Delta AHI \geq 8/h$; CPAP showed stronger BP reduction than oxygen. Effective AHI also correlated with BNP (negative association) and troponin changes (positive)	No major new safety concerns were identified; adverse effects were not the focus of this secondary analysis	Single-night follow-up sleep assessment; relatively short duration (12 weeks); small sample size; limited generalizability, and reliance on residual AHI metrics
Tan L *et al.*, 2020 [24]	To evaluate whether nocturnal oxygen therapy prevents nocturnal hypoxemia, sleep apnea, and altitude-related adverse health effects in COPD patients traveling to 2048 m	Randomized, placebo-controlled crossover clinical trial	32 lowlanders with moderate-to-severe COPD (mean age 65.6 years, FEV_1 30–80% predicted), living below 800 m, not on home oxygen or CPAP	Nocturnal oxygen therapy (3 L/min by nasal cannula) during stays at 2048 m / Placebo (ambient room air via nasal cannula) at 2048 m	Nocturnal oxygen therapy increased SpO_2 by ~9%, reduced AHI by ~20 events/h, improved subjective sleep quality, decreased periodic breathing, and reduced incidence of altitude-related adverse health effects by 85%	Some patients experienced hypoxemia requiring withdrawal in the placebo group; overall, nocturnal oxygen therapy was well tolerated with no major safety concerns reported	Small sample size; short exposure (2 nights at altitude); single altitude level (2048 m); findings may not generalize to higher altitudes, very severe COPD, or longer exposures

Joosten SA *et al.*, 2021 [25]	To determine whether patients with OSA who fail to respond to upper airway surgery can be successfully treated with supplemental oxygen and whether baseline physiologic endotypes predict response	Randomized, double-blind, placebo-controlled crossover trial (single-night intervention)	20 adults with OSA who had persistent disease after upper airway surgery (85% male, mean age ~52 years, body mass index (BMI) ~31 kg/m^2)	Supplemental oxygen 4 L/min during sleep / Placebo (medical air 4 L/min)	Significant reduction in AHI (42.4 → 30.5 events/h, $p = 0.008$); reduction in flow-based AHI and arousal index (41.1 → 33.0 events/h, $p = 0.006$); no significant change in morning BP or ESS; 35% of patients were responders (≥ 50% reduction in AHI)	No major adverse events reported; short-term oxygen therapy considered safe	Single night intervention only (no long-term data); small sample size ($n = 20$); inability to assess mechanistic endotypes (loop gain, etc.) accurately due to signal limitations; limited generalizability beyond surgical non-responders
Turnbull CD *et al.*, 2019 [26]	To determine whether intermittent hypoxia is the dominant mechanism of daytime BP rise in OSA by testing the effect of supplemental oxygen during CPAP withdrawal	Randomized, double-blind, crossover trial	38 patients with moderate-to-severe OSA, long-term CPAP users (> 1 year, ≥ 4 h/night), screened for OSA recurrence after CPAP withdrawal	Supplemental oxygen (5 L/min via concentrator, nasal cannula or mask) for 2 weeks during CPAP withdrawal / Sham air (identical concentrator) for 2 weeks during CPAP withdrawal. Crossover after 2-week CPAP washout	Oxygen abolished the rise in morning systolic (+26.6 mmHg vs. air; $p = 0.008$) and diastolic (+4.6 mmHg; $p = 0.006$) BP; significant reduction in intermittent hypoxia: ODI −23.8/h ($p < 0.001$), time SpO_2 < 90% −9.8% ($p < 0.001$); minimal effect on AHI, subjective or objective sleepiness	Oxygen increased venous base excess (+3.1 mM; $p < 0.001$), indicating risk of hypercapnia	Short duration (2 weeks); small sample size
dos Santos Neto JM *et al.*, 2021 [27]	To evaluate whether CPAP ventilation and passive CPAP oxygenation could delay oxygen desaturation (SpO_2 dropping to 95%) after apnea onset in	Double-blind, parallel, randomized controlled trial	68 children aged 2–6 years, ASA physical status I–II, undergoing elective surgery under general anesthesia	CPAP ventilation and passive CPAP oxygenation at 10 cm H_2O using an anesthesia	Children in the CPAP group had significantly longer time to reach SpO_2 of 95% after apnea onset (median 278 s vs. 124 s in controls, $p = 0.002$). Hazard ratio	No severe adverse events such as laryngospasm, bronchospasm, bradycardia, cardiac arrest, or death were reported in either	Small single-center study with a narrow age range; possible compromise of blinding as researchers observed apnea time and saturation; absence

	children during anesthesia induction compared with the standard technique without positive airway pressure			workstation / Standard technique with no positive airway pressure (0 cm H_2O)	for desaturation was 0.26, indicating a 74% risk reduction. CPAP also maintained higher SpO_2 values between 60–210 s after apnea	group. CPAP was well tolerated and feasible to apply	of subgroup analyses (e.g., OSA prevalence, BMI); limited power for secondary outcomes such as recovery time of SpO_2
Batool-Anwar S *et al.*, 2016 [28]	To investigate whether CPAP improves quality of life in patients with OSA, and whether these benefits are sustained across different severities of OSA	Randomized controlled trial (subset analysis of APPLES trial)	845 adults with OSA (randomized to CPAP, $n = 443$; or sham CPAP, $n = 402$), stratified into mild, moderate, and severe OSA	CPAP therapy / Sham CPAP	CPAP improved quality of life (measured by SAQLI) primarily in patients with moderate to severe OSA and those adherent to CPAP use (≥ 4 h/night). No significant quality of life improvement was observed in mild OSA patients. CPAP also significantly reduced daytime sleepiness (ESS)	No major safety concerns reported	Excluded very severe OSA (baseline $SpO_2 <$ 75% for > 10% of the night); results limited to CPAP vs. sham CPAP, not generalizable to other treatments (oral devices, surgery); follow-up limited to 6 months; possible placebo effects cannot be excluded even with sham CPAP
Peker Y *et al.*, 2016 [29]	To assess whether CPAP reduces long-term adverse cardiovascular outcomes in patients with coronary artery disease and non-sleepy OSA	Randomized controlled trial, single-center, prospective, open-label with blinded evaluation	244 patients with recently revascularized coronary artery disease and OSA (AHI > 15/h) but without daytime sleepiness (ESS < 10)	CPAP therapy ($n = 122$) / no CPAP ($n = 122$)	Intention-to-treat analysis showed no significant difference in cardiovascular outcomes (18.1% vs. 22.1%). However, on-treatment analysis revealed a significant reduction in cardiovascular events among patients using CPAP ≥ 4h/night (HR 0.29, 95% CI 0.10–0.86)	CPAP was generally well tolerated; side effects included nasal dryness, mask discomfort, and one case of nasal bleeding	Single-center trial, relatively low CPAP adherence
McEvoy RD *et al.*, 2016	To determine whether CPAP	Multicenter, randomized	2,717 adults aged 45–75	CPAP therapy plus	CPAP reduced AHI from 29.0/h to 3.7/h,	No increase in serious adverse	Suboptimal adherence (average

[30]	therapy prevents cardiovascular events in patients with moderate-to-severe OSA and established cardiovascular disease	controlled trial (SAVE study)	with moderate-to-severe OSA and coronary or cerebrovascular disease	usual care / usual care alone	improved sleepiness, quality of life, and mood, but did not reduce cardiovascular events compared to usual care (HR 1.10, $p = 0.34$)	events; CPAP was safe, with similar rates of accidents and adverse outcomes compared to usual care	3.3 h/night); exclusion of patients with excessive sleepiness; reliance on a simplified diagnostic tool, and limited generalizability
O'Donnell C *et al.*, 2024 [31]	To assess the effect of CPAP on early atherosclerotic processes in OSA and compare it with GLP-1 mediated weight loss (liraglutide) and their combination	Randomized, proof-of-concept trial	30 adults with moderate-to-severe OSA (AHI > 15/h, BMI 30–40 kg/m^2, no diabetes, no unstable CVD)	CPAP alone / liraglutide based weight loss vs. combination therapy	CPAP significantly reduced AHI, vascular inflammation, unstable coronary plaque volume, improved endothelial function, and decreased CRP. Liraglutide led to weight loss and modest BP reduction but no vascular inflammation or plaque improvement. Combination therapy showed mixed benefits	CPAP was well tolerated, with good adherence. Liraglutide caused mild gastrointestinal side effects in a few participants, leading to crossover in 2 patients. No severe adverse events reported	Small sample size; short follow-up (24 weeks); proof-of-concept design, exclusion of patients with severe daytime sleepiness, and ethical limits on repeat imaging reduced generalizability
Xu H *et al.*, 2025 [32]	To determine whether CPAP therapy over 12 months improves neuroimaging biomarkers and cognitive performance in middle-aged OSA patients with normal baseline cognition	Multicenter, randomized clinical trial	148 adults with OSA (AHI ≥ 15/h) and normal cognition, recruited across five hospitals between 2017 and 2021	CPAP therapy plus best supportive care / best supportive care alone	After 6 months, CPAP did not improve global cognitive function (MoCA score: difference −0.04, 95% CI −0.72 to 0.65; $p = 0.91$), but significantly improved functional connectivity of the default mode network (difference −13.73; 95% CI −23.40 to −4.06; $p = 0.01$) and cortical thickness (difference −0.06 mm; 95% CI −0.10 to	No serious adverse events were reported	Based on the abstract alone, long term cognitive outcomes and broader neurocognitive function remain unassessed

					−0.01 mm; $p = 0.02$), compared with controls		
Facco FL *et al.*, 2023 [33]	To evaluate the effect of autotitrating PAP on OSA during pregnancy, specifically its impact on a composite cardiometabolic risk profile	Randomized controlled trial	Pregnant women ≥ 18 years, BMI ≥ 30 kg/m^2, gestational age 14–20 weeks, diagnosed with OSA (AHI ≥ 5 but < 50). Total: 89 participants randomized	Autotitrating PAP / Sham PAP (phase 1) or sleep hygiene advice (phase 2)	The trial did not find significant differences in the composite cardiometabolic risk profile between groups. However, secondary analyses showed a lower uterine artery Doppler pulsatility index in autotitrating PAP users compared with sleep hygiene controls, and greater autotitrating PAP adherence was linked to lower fasting glucose	No major safety concerns reported. Adherence to autotitrating PAP was low overall but improved when sham PAP was replaced by sleep hygiene control	Low adherence to autotitrating PAP therapy; relatively small sample size, and changes in control arm design (sham PAP replaced by sleep hygiene), which may have influenced outcomes
Fox H *et al.*, 2021 [34]	To evaluate the impact of automatic PAP on exercise capacity, cardiac function, quality of life, and nocturnal hypoxemia in patients with heart failure with reduced ejection fraction and moderate to severe OSA	Randomized controlled pilot trial, single center	76 patients with chronic, stable heart failure with reduced ejection fraction (left ventricular ejection fraction ≤ 45%), NYHA class ≥ II, and moderate-to-severe OSA	Automatic PAP / Nasal strips (placebo)	The AHI dropped from 34/h to 9/h in the automatic PAP group, while remaining unchanged in controls	No major adverse events were reported. High adherence to automatic PAP (average 6 ± 1.6 h/night) contributed to positive outcomes	Single center; small sample size; 6-month follow-up; high adherence that may not generalize to broader populations
Xiao S *et al.*, 2016 [35]	To test whether CPAP levels used during sleep increase neural respiratory drive and breathlessness in	Observational physiological cohort	15 obese patients with confirmed OSA (mean age 48 ± 10 years, mostly male)	Daytime titration with stepwise CPAP (4–20 cm H_2O) while awake /	CPAP initially reduced neural respiratory drive at moderate pressures (~10 cm H_2O), but higher CPAP	No severe adverse events reported; main safety concern was discomfort and breathlessness at	Small pilot sample; short follow-up (6 weeks); results may not generalize to non-obese OSA patients or real-

	obese OSA patients, potentially discouraging compliance			neural respiratory drive and perceived breathlessness with therapeutic CPAP levels determined during sleep	increased neural respiratory drive and breathlessness. Compliance at 6 weeks was negatively correlated with breathlessness	higher CPAP pressures	world long-term adherence
Sánchez-de-la-Torre M *et al.*, 2022 [36]	To assess the long-term effect of OSA and CPAP treatment on BP in patients with acute coronary syndrome	Multicenter randomized controlled trial	1,803 patients admitted for acute coronary syndrome; 596 without OSA and 1,207 with OSA (AHI > 15/h). Among OSA patients, 229 had good CPAP adherence (> 4 h/night), while others had poor adherence or usual care	CPAP therapy with good adherence (> 4 h/night) / Usual care or poor CPAP adherence; patients without OSA served as a reference	Severe OSA (AHI > 40/h) was linked to increased BP over time. Good adherence to CPAP significantly reduced mean, systolic, and diastolic BP, particularly in severe OSA, with reductions of ~4–7 mmHg after 18–60 months	No major adverse events specifically attributed to CPAP were reported. Low adherence limited the overall effectiveness of CPAP in the general OSA group	Excluded sleepy OSA patients (Epworth > 10), so results apply only to non-sleepy OSA; low mean adherence (2.78 h/night) may underestimate CPAP's effect; limited to patients with acute coronary syndrome; results may not apply broadly
Sterling KL *et al.*, 2022 [37]	To investigate the effects of PAP adherence on health outcomes, healthcare resource usage, and costs in patients with overlap syndrome (OSA and COPD)	Retrospective observational study	6,810 adults in the United States with overlap syndrome (OSA and COPD), mean age 60.8 years, 56% female	Adherent PAP therapy / Nonadherent PAP users	Adherent PAP use was associated with significantly fewer all-cause hospitalizations, emergency room visits, and severe acute exacerbations over 2 years compared to nonadherent patients	No specific safety issues reported	Missing data on lifestyle factors (e.g., smoking, alcohol); possible residual confounding despite propensity matching
Cukierman DS *et al.*, 2023 [38]	To determine whether a nasal CPAP mask decreases	Single-center prospective randomized controlled trial	109 adult patients with obesity and/or OSA	Nasal CPAP mask delivering oxygen at 10	Nasal CPAP did not reduce the incidence of hypoxemia (SpO_2 < 90%) compared to	Both devices had similar rates of adverse events. Some minor	Single-center design; low overall incidence of hypoxemic events,

	hypoxemic events compared with a simple face mask in obese and OSA patients undergoing colonoscopy under propofol anesthesia		undergoing colonoscopy under propofol-based general anesthesia without tracheal intubation	L/min/simple face mask oxygenation at 10 L/min	face mask. However, it significantly reduced the need for airway maneuvers such as chin lift and oral airway insertion	device-related issues were reported (eye pain, skin erythema)	underpowered to detect differences in secondary outcomes
Hoyos CM *et al.*, 2022 [39]	To evaluate whether CPAP treatment improves cognition in older adults with MCI and OSA	Pilot randomized controlled crossover clinical trial	29 adults aged 50–80 years with MCI and moderate-to-severe OSA (AHI > 15/h)	CPAP therapy for 12 weeks / no treatment	CPAP did not significantly improve primary outcomes of processing speed or executive functioning but showed moderate improvements in verbal learning and memory retention	Three serious adverse events occurred during the CPAP arm (urinary infection, fainting with coccyx fracture, septicemia), though these were not clearly attributable to CPAP. Minor adverse events included a car accident. Overall, CPAP was considered generally safe, but adherence was modest (3.2 h/night)	Small sample size; pilot design without sham CPAP control; low adherence; no power calculation; and lack of generalizability to broader OSA populations
Gentina T *et al.*, 2019 [40]	To assess the impact of marital quality and partner engagement on adherence to CPAP treatment in patients with OSA	Multicenter prospective observational study	290 newly diagnosed OSA patients, median age 53 years	Standard CPAP initiation with structured education / comparison across levels of partner engagement and marital quality	Higher partner encouragement and long-term stable relationships (> 30 years) were associated with better CPAP adherence	No safety issues reported	Follow-up limited to 120 days; mostly male participants; psychosocial factors may not generalize; no randomization

Khan NNS *et al.*, 2022 [41]	To evaluate whether a multidimensional patient-centered program involving caregiver engagement improves CPAP adherence in newly diagnosed OSA patients	Randomized controlled trial	60 adults with newly diagnosed OSA eligible for CPAP therapy and living with a caregiver (intervention $n = 28$; control $n = 32$)	28 patients participated with their caregiver in 4 structured sessions (interactive education, peer coaching, hands-on CPAP training, motivational interview), plus follow-up phone calls and text messages / 32 patients and caregivers received unrelated education on physical activity and lifestyle	The intervention group significantly increased mean daily CPAP use by ~1.2 hours between 3 and 6 months compared to controls ($p = 0.008$). However, there was no significant difference in the percentage of patients meeting standard adherence criteria	No adverse effects from the intervention were reported	Small sample size; limited generalizability (mostly recruited from private clinics); variable timing of group visits
Khadadah S *et al.*, 2021 [42]	To evaluate the effect of CPAP treatment on fatigue sleep quality, somnolence, pain, disability, and quality of life in patients with multiple sclerosis and OSA	Randomized, double-blind, placebo-controlled trial	49 multiple sclerosis patients with fatigue, poor sleep quality, and OSA (AHI ≥ 15/hour but not severe OSA). 34 patients completed the 6-month protocol	Active CPAP for 6 months / Sham CPAP (placebo) for 6 months	CPAP improved objective sleep parameters at 3 months, patients on CPAP showed reduced daytime sleepiness (ESS, $p = 0.03$) and a tendency toward improved fatigue ($p = 0.09$). However, after 6 months, CPAP did not significantly reduce fatigue compared to placebo	Adverse effects were mild and related mainly to mask discomfort, nasal irritation, and air leaks	Small sample size; high dropout rate and suboptimal CPAP adherence; possible unblinding of participants reduced internal validity

Chalkiadaki E *et al.*, 2021 [43]	To assess how better oxygenation during sleep, resulting from increased CPAP adherence, influences corneal endothelial morphology	Prospective observational study	Patients with OSA treated with CPAP	CPAP treatment with improved adherence / lower adherence	Improved sleep oxygenation via CPAP was associated with reduced corneal endothelial polymegathism and pleomorphism	No safety issues reported	Small sample size; observational design; variable follow-up duration; limited generalizability
Gupta A *et al.*, 2018 [44]	To evaluate the effect of CPAP therapy on preventing new vascular events and improving stroke outcomes in patients with OSA following a first ever stroke	Randomized controlled trial	70 stroke patients with moderate to severe OSA (AHI > 15), randomized into CPAP (n = 30) and non-CPAP (n = 40) groups	CPAP therapy, titrated and prescribed with auto-adjusting devices / non-CPAP group received best medical treatment	CPAP users had fewer new vascular events (3.3% vs. 15%, not statistically significant) and significantly better stroke recovery, with more patients improving ≥ 1 point on the Modified Rankin Scale at 6 and 12 months	No major safety concerns reported; some adherence issues due to disability, cognitive impairment, or discomfort with CPAP	Small sample size; single-center; relatively young and male-dominated cohort (limiting generalizability)
Zhao YY *et al.*, 2022 [45]	To determine the long-term effect of CPAP treatment on 24-hour ambulatory BP in patients with moderate-to-severe OSA at high cardiovascular risk but without severe sleepiness	Randomized, controlled, parallel-group clinical trial	169 adults with moderate-to-severe OSA and either established CVD or multiple cardiovascular risk factors	CPAP therapy / Control group (sham CPAP or no CPAP, with conservative medical therapy)	CPAP did not significantly reduce mean 24-hour systolic BP compared to control (treatment effect –2.7 mmHg; p = 0.105). However, CPAP significantly reduced nighttime systolic BP (–5.9 mmHg; p = 0.004) and improved the day-night BP ratio. These results suggest CPAP may be more effective for nocturnal BP control	No major safety concerns were reported	The study was underpowered to detect modest BP changes, and adherence to CPAP was relatively low; some baseline BP differences existed between groups despite randomization, and follow-up was limited to 6–12 months for most participants

Lorenzi-Filho G *et al.*, 2025 [46]	To determine whether CPAP therapy reduces central and peripheral BP in patients with moderate to severe OSA and uncontrolled HTN, despite existing antihypertensive treatment	Multicenter, randomized controlled trial	123 patients with moderate-to-severe OSA and uncontrolled HTN	CPAP therapy (≥ 4 h/night for 6 months) / sham placebo (nasal dilator strips)	CPAP significantly reduced office systolic BP compared with nasal dilator strips (both $p < 0.001$)	No major safety concerns reported	While effective in lowering BP, results may not apply to patients with controlled HTN or poor CPAP adherence
Hoyos CM *et al.*, 2015 [47]	To determine whether CPAP reduces both central and peripheral BP and if those effects are consistent throughout the day	Randomized, controlled, crossover trial	38 patients with OSA	Therapeutic CPAP / Sham CPAP	CPAP significantly reduced central systolic and diastolic blood pressure (mean decreases of approximately –4.1 mm Hg and –3.9 mm Hg, respectively), as well as peripheral systolic and diastolic blood pressure (around –4.1 mm Hg and –3.8 mm Hg), regardless of the time of day	Not explicitly reported	Small sample size; short duration (8 weeks per arm)
Shirahama R *et al.*, 2021 [48]	To evaluate the long-term effect of CPAP adherence on BP and body weight over 24 months in OSA patients	Retrospective observational cohort study	918 adults with moderate to severe OSA, aged 20–80 years, from a Japanese sleep clinic	Patients receiving CPAP therapy / Comparison was made between those with good adherence (≥ 4h/night on ≥ 70% of nights) and	Good CPAP adherence was associated with significant reductions in diastolic BP over 24 months, independent of weight change. Systolic BP effects were less consistent. Normotensive patients showed clearer benefits than hypertensive patients	No specific adverse effects of CPAP were reported	Retrospective design; lack of untreated control group; reliance on office BP rather than ambulatory monitoring

				poor adherence			
Hu S.-T *et al.*, 2017 [49]	To evaluate whether a structured nurse-led educational program improves quality of life and reduces CPAP-related discomfort in patients with OSA	Randomized controlled trial	Patients with OSA treated with CPAP	Structured nurse-led educational program / usual care	The intervention significantly reduced CPAP-related discomfort (β = −1.83, p = 0.040) and improved quality of life (SAQLI total: β = 1.669, p = 0.014), with improvements also noted in subdomains (β = 5.69, p = 0.007)	Not mentioned	Sample size; follow-up duration
Lisan Q. *et al.*, 2019 [50]	To investigate whether prescription of PAP is associated with lower all-cause mortality in patients with obesity and severe OSA	Observational cohort	392 participants with obesity and severe OSA; 81 with PAP prescribed vs. 311 without; matched by age, sex, and AHI	PAP prescription / no PAP prescription	PAP prescription was associated with lower all-cause mortality	Not assessed	PAP self-reported and date of initiation unknown; potential residual confounding despite matching / adjustment
Zhao YY *et al.*, 2017 [51]	To assess the long-term effect of CPAP on health-related quality of life and daytime sleepiness in patients with moderate-to-severe OSA at high cardiovascular risk but without severe sleepiness	Randomized, controlled, parallel group trial	169 participants, aged 45–75 years, with moderate-to-severe OSA and either established CVD	CPAP therapy / Conservative medical therapy or with sham CPAP	CPAP improved multiple health-related quality of life domains (bodily pain, vitality, general health, physical functioning, physical health summary score) and reduced daytime sleepiness (ESS −1 point)	No major safety concerns were reported	Average CPAP use was low; limiting effect size; only partial follow-up to 12 months; limited power to assess adherence subgroups
Azarbarzin A *et al.*, 2022 [52]	To investigate whether the cardiovascular benefits of CPAP in	Post-hoc analysis of a randomized controlled trial	226 adults with angiography-verified coronary artery	CPAP therapy / Usual care	CPAP reduced cardiovascular risk significantly in patients with high-rate	No major adverse safety issues reported	Secondary analysis with relatively small sample size; underrepresentation

patients with coronary artery disease and OSA without excessive sleepiness depend on the heart rate response to respiratory events	disease and OSA (AHI > 15/h), without excessive daytime sleepiness (ESS ≤ 10)	response to respiratory events (≥ 10 bpm, ~59% reduction), but not in those with average or low-rate response to respiratory events	of women and minorities

ASA = American Society of Anesthesiologists; AHI = Apnea–Hypopnea Index; APPLES = Apnea Positive Pressure Long-term Efficacy Study; BMI = Body Mass Index; BNP = B-type Natriuretic Peptide; BP = Blood Pressure; CI = Confidence Interval; COPD = Chronic Obstructive Pulmonary Disease; CPAP = Continuous Positive Airway Pressure; CRP = C-reactive Protein; CVD = Cardiovascular Disease; ESS = Epworth Sleepiness Scale; FEV_1 = Forced Expiratory Volume in 1 second; GLP-1 = Glucagon-Like Peptide 1; HR = Hazard Ratio; HRV = Heart Rate Variability; HTN = Hypertension; MCI = Mild Cognitive Impairment; MoCA = Montreal Cognitive Assessment; NOS = Nocturnal Oxygen Supplementation; NYHA = New York Heart Association; ODI = Oxygen Desaturation Index; OSA = Obstructive Sleep Apnea; SAQLI = Sleep Apnea Quality of Life Index; SAVE = Sleep Apnea Cardiovascular Endpoints study; SpO_2 = Peripheral Capillary Oxygen Saturation.

To assess the quality of the studies and the risk of bias, the following protocol was followed: first, the included studies were grouped according to their study design (Table 3); second, the appropriate tool was selected based on the study design according to the Java Business Integration (JBI) guidelines [53].

Table 3. Summary of different types of studies.

Types of studies	Number (*n*)
Randomized controlled trials (including crossover, multicenter, pilot, proof-of-concept, double-blind)	21
Cohort studies (prospective or retrospective, observational)	4
Quasi-experimental studies (post-hoc, secondary analyses, physiological)	6

Overall, the methodological quality of the included studies was satisfactory, with most assessments showing a low risk of bias. This strengthens the reliability of the evidence presented in this review. As the JBI critical appraisal tools do not provide official cut-off values to classify the risk of bias, studies were categorized using a pragmatic threshold commonly applied in the literature: ≥ 70% of "Yes" responses = low risk of bias, 50–69% = moderate risk, and < 50% = high risk [54]. To provide a clear overview, the following tables (Tables 4-6) summarize the risk of bias across different study designs.

Table 4. Risk of bias in the randomized controlled trials.

Authors	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	Q12	Q13	% answer yes	Risk of bias
Tan L *et al.* [24]	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Joosten SA *et al.* [25]	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Turnbull CD *et al.* [26]	Y	Y	Y	Y	Y	Y	Y	Y	Y	N	Y	Y	Y	92	Low
dos Santos Neto JM *et al.* [27]	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Batool-Anwar S *et al.* [28]	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Peker Y *et al.* [29]	Y	Y	Y	N	N	Y	Y	Y	Y	Y	Y	Y	Y	85	Low
McEvoy RD *et al.* [30]	Y	Y	Y	N	N	Y	Y	Y	Y	Y	Y	Y	Y	85	Low
Xu H *et al.* [32]	Y	U	Y	U	U	Y	U	Y	Y	Y	Y	U	Y	62	Moderate
Facco FL *et al.* [33]	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Fox H *et al.*	Y	Y	Y	N	N	Y	Y	Y	Y	N	Y	Y	Y	77	Low

[34]															
Sánchez-de-la-Torre M *et al.* [36]	Y	Y	Y	N	N	Y	Y	Y	Y	Y	Y	Y	Y	85	Low
Cukierman DS *et al.* [38]	Y	Y	Y	N	N	Y	Y	Y	Y	Y	Y	Y	Y	85	Low
Hoyos CM *et al.* [39]	Y	U	Y	U	U	Y	U	Y	Y	U	Y	U	Y	54	Moderate
Khan NNS *et al.* [41]	Y	Y	Y	N	N	Y	Y	Y	Y	N	Y	Y	Y	77	Low
Khadadah S *et al.* [42]	Y	Y	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	85	Low
Gupta A *et al.* [44]	Y	Y	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	85	Low
Zhao YY *et al.* [45]	Y	Y	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	85	Low
Lorenzi-Filho G *et al.* [46]	Y	U	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	92	Low
Hoyos CM *et al.* [47]	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Hu S.-T *et al.* [49]	Y	U	Y	N	N	Y	U	Y	Y	Y	Y	Y	Y	69	Moderate

Zhao YY *et al.* [51]	Y	Y	Y	Y	N	Y	Y	Y	Y	N	Y	Y	Y	85	Low

Q1 = Were the two groups similar and recruited from the same population?; Q2 = Were the exposures measured similarly to assign people to both exposed and unexposed groups?; Q3 = Was the exposure measured validly and reliably?; Q4 = Were confounding factors identified?; Q5 = Were strategies to deal with confounding factors stated?; Q6 = Were the participants free of the outcome at the start of the study (or at the moment of exposure)?; Q7 = Were the outcomes measured validly and reliably?; Q8 = Was the follow-up time reported and long enough for outcomes to occur?; Q9 = Was follow-up complete, and if not, were the reasons described and analyzed?; Q10 = Were strategies to address incomplete follow-up utilized?; Q11 = Was an appropriate statistical analysis used? Q12 = Was appropriate statistical analysis used?; Q13 = Was the trial design appropriate, and any deviations from the standard RCT design accounted for in the conduct and analysis of the trial?
N = No; U = Unclear; Y = Yes

Table 5. Risk of bias in the cohort studies.

Authors	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	Q10	Q11	% answer yes	Risk of bias
Sterling KL, Pépin JL *et al.* [37]	Y	Y	Y	Y	Y	Y	Y	Y	Y	NA	Y	91	Low
Gentina T, Bailly S *et al.* [40]	Y	Y	Y	Y	Y	Y	Y	Y	U	U	Y	82	Low
Shirahama R., Tanigawa T *et al.* [48]	Y	Y	Y	Y	Y	Y	Y	Y	U	N	Y	82	Low
Lisan Q., Van Sloten T *et al.* [50]	Y	Y	Y	Y	Y	Y	Y	Y	Y	NA	Y	91	Low

Q1 = Were the two groups similar and recruited from the same population?; Q2 = Were the exposures measured similarly to assign people to both exposed and unexposed groups?; Q3 = Was the exposure measured validly and reliably?; Q4 = Were confounding factors identified?; Q5 = Were strategies to deal with confounding factors stated?; Q6 = Were the participants free of the outcome at the start of the study (or at the moment of exposure)?; Q7 = Were the outcomes measured validly and reliably?; Q8 = Was the follow-up time reported and long enough for outcomes to occur?; Q9 = Was follow-up complete, and if not, were the reasons described and analyzed?; Q10 = Were strategies to address incomplete follow-up utilized?; Q11 = Was an appropriate statistical analysis used?
N = No; NA = Not Applicable; U = Unclear; Y = Yes

Table 6. Risk of bias in the quasi-experimental studies.

Authors	Q1	Q2	Q3	Q4	Q5	Q6	Q7	Q8	Q9	% answer yes	Risk of bias
Tan L, Li T *et al.* [22]	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Ni YN, Lei F *et al.* [23]	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
O'Donnell C., Ryan S *et al.* [31]	Y	Y	Y	Y	Y	Y	Y	Y	Y	100	Low
Xiao S, Bastianpillai J *et al.* [35]	Y	Y	Y	N	Y	Y	Y	Y	Y	89	Low
Chalkiadaki E, Andreanos K *et al.* [43]	Y	Y	Y	Y	Y	Y	Y	U	Y	89	Low
Azarbarzin A., Zinchuk A *et al.* [52]	Y	Y	Y	Y	N	Y	Y	Y	Y	89	Low

Q1 = Is it clear in the study what the 'cause' is and what the 'effect' is?; Q2 = Were the participants included in any comparisons similar?; Q3 = Were the participants included in any comparisons receiving similar treatment/care, other than the exposure/intervention of interest?; Q4 = Was there a control group?; Q5 = Were there multiple measurements of the outcome pre- and post-intervention?; Q6 = Was follow-up complete, and if not, were differences between groups adequately described and analyzed?; Q7 = Were the outcomes of participants included in any comparisons measured in the same way?; Q8 = Were outcomes measured reliably?; Q9 = Was an appropriate statistical analysis used?
N = No; U = Unclear; Y = Yes

Discussion

The present review confirms that CPAP remains the most effective treatment for moderate-to-severe OSA. Across the 31 included studies, CPAP consistently produced substantial reductions in the apnea–hypopnea index, frequently exceeding 50% of baseline values and corresponding to absolute decreases of approximately 15–30 events per hour in moderate-to-severe OSA [11]. These improvements were associated with clinically meaningful increases in nocturnal oxygenation, with mean SpO_2 gains generally ranging from 2% to 9%, as well as reductions in daytime sleepiness and improvements in quality of life and cognitive function. The efficacy of CPAP has been consistently reported in the literature [7,8,10,11]. From a cardiovascular perspective, several studies reported modest but clinically relevant reductions in nocturnal BP and improvements in endothelial function, supporting the role of CPAP as a first-line therapy with potential long-term cardiovascular benefits [7,10].

Nocturnal oxygen therapy, although less extensively studied as a standalone intervention, consistently improved oxygen saturation and may be beneficial in selected patients with severe nocturnal desaturation or intolerance to CPAP [55]. However, these findings should be interpreted with caution. The number of robust randomized controlled trials evaluating oxygen therapy in OSA remains limited, sample sizes are often small, and study protocols are heterogeneous, which restricts the generalizability of results. While oxygen corrects hypoxemia, it does not prevent upper airway collapse, and absolute reductions in AHI were generally modest and substantially lower than those observed with CPAP. Moreover, evidence regarding cardiovascular outcomes remains inconsistent. Safety concerns, particularly the risk of hypoventilation and CO_2 retention in patients with comorbid conditions such as COPD or obesity hypoventilation syndrome, further limit its use [12,13,55–57]. Overall, the clinical benefits of oxygen therapy alone and of combined CPAP + oxygen strategies should be interpreted with caution due to the limited number of studies and small sample sizes. Consequently, oxygen therapy should be considered a complementary or alternative option in well-defined clinical contexts rather than an equivalent substitute for CPAP.

Adverse effects associated with CPAP and oxygen therapy are common and represent a major barrier to long-term adherence [58–60]. For CPAP, nasal dryness or congestion, mask discomfort, air leaks, aerophagia, and claustrophobia frequently occur during the early treatment phase and may lead to premature discontinuation [58–60]. Oxygen therapy is mainly associated with mucosal dryness and, in susceptible patients, hypercapnia [55,57,61]. Because therapeutic efficacy is closely dependent on actual device use, generally requiring at least 4 hours per night [62], effective management of these adverse effects is essential to ensure sustained clinical benefit.

It should be acknowledged that high-quality randomized controlled trials specifically evaluating pharmacist-led interventions in OSA are limited. Much of the available evidence is derived from observational studies, real-world experience, and extrapolation from other chronic diseases, as well as expert consensus. Pharmacist-led interventions may improve treatment safety, tolerability, and adherence, which are recognized determinants of clinical effectiveness in OSA. Their interventions include patient education at treatment initiation, early identification and management of adverse effects, optimization of device settings and accessories, and reinforcement of motivation [58,63,64]. Pharmacists can also contribute to the safe use of oxygen therapy by verifying prescription parameters, identifying risk factors for hypercapnia, and educating patients and caregivers on fire prevention and device maintenance [55–57]. Through regular follow-up, pharmacists are well positioned to detect inadequate use, poor tolerance, or lack of clinical benefit and to coordinate timely medical reassessment [65,66].

Telemonitoring further strengthens pharmacists' clinical contribution [67–69]. Modern CPAP devices provide objective data on usage, residual AHI, leaks, and mask fit, enabling early identification of suboptimal patterns and rapid intervention [65,70–72]. Monitoring adherence and symptom evolution over time also allows pharmacists to document clinical outcomes, support interdisciplinary collaboration, and contribute to structured follow-up protocols [65,71,73,74]. Such approaches facilitate personalized care, reduce early dropouts, and maximize the long-term effectiveness of therapy. While direct evidence linking pharmacist interventions to hard clinical outcomes in OSA remains limited, expert opinion and indirect evidence strongly support their role in optimizing adherence, managing adverse effects, and enhancing patient education [58,64,66].

Overall, this review highlights the central role of CPAP in the management of obstructive sleep apnea, while clarifying the limited, context-specific role of nocturnal oxygen therapy. It also underscores the value of pharmacist-led interventions and structured follow-up strategies in improving adherence, safety, and clinical outcomes, thereby bridging clinical evidence and real-world practice.

To enhance the clinical applicability of the pharmacist's role, key interventions, expected outcomes, and monitoring parameters are summarized in Table 7. This structured approach translates existing evidence into actionable pharmaceutical care strategies, facilitating real-world implementation and interdisciplinary collaboration.

Table 7. Pharmacist-led interventions in the management of obstructive sleep apnea.

Pharmacist intervention	Targeted problem	Expected clinical outcome	Follow-up
Patient education at treatment initiation	Poor understanding of device use, low motivation	Improved adherence, reduced early treatment discontinuation	CPAP usage hours/night, patient-reported comfort, ESS score
Device and mask optimization	Mask leaks, discomfort, nasal dryness, claustrophobia	Improved comfort and sustained CPAP use	Leak rate, residual AHI, patient-reported adverse effects
Management of CPAP-related adverse effects	Treatment intolerance	Increased tolerability and persistence of therapy	Symptom resolution, continuation of CPAP ≥ 4h/night
Oxygen therapy safety assessment	Hypercapnia risk, misuse of oxygen	Safer oxygen use, prevention of complications	Symptoms of hypoventilation, SpO_2 trends, medical reassessment if needed
Adherence monitoring and motivational support	Suboptimal CPAP use (< 4h/night)	Improved long-term adherence and clinical benefit	CPAP telemonitoring data, adherence at 1-3-6 months
Coordination of care and referral	Persistent symptoms or inefficacy	Timely therapy adjustment	Residual AHI, uncontrolled BP, referral to sleep specialist
Management of comorbidities and medication review	Drugs impairing ventilation, poor control of HTN or diabetes	Optimized global clinical outcomes	BP control, medication adherence, safety alerts
Telemonitoring-based follow-up	Early drop out, unrecognized technical issues	Early intervention and sustained effectiveness	Remote CPAP data, adherence dashboards, ESS improvement

AHI = Apnea-Hypopnea Index; BP = Blood Pressure; CPAP = Continuous Positive Airway Pressure; ESS = Epworth Sleepiness Scale; HTN = Hypertension; SpO_2 = Peripheral Capillary Oxygen Saturation.

CPAP remains the gold standard treatment for OSA, with proven efficacy in reducing respiratory events, improving nocturnal oxygenation, and decreasing daytime sleepiness. Oxygen therapy, while effective in correcting hypoxemia, shows heterogeneous results on clinical and cardiovascular outcomes and should therefore be reserved for specific indications or considered as an adjunct to CPAP.

One of the major challenges in the management of OSA is long-term adherence. Common adverse effects, equipment comfort, and psychological factors strongly influence treatment persistence. In this context, pharmacists play a strategic role: they act as educators, supporters in managing side effects, coordinators in caring for comorbidities, and guarantors of the safe use of devices, particularly in oxygen therapy. Modern tools such as telemonitoring and structured follow-up protocols further enhance their contribution to treatment continuity and effectiveness.

Regarding the impact of pharmaceutical interventions on adherence and clinical outcomes of the treatment of OSA, scientific evidence remains limited. Randomized controlled trials and real-world implementation studies are required to validate and formalize the integration of pharmacists into OSA care pathways. Finally, the emergence of novel therapeutic options (including metabolic agents, oral devices, and hypoglossal nerve stimulation) will require pharmacists to continuously adapt their role to technological advances and evolving patient profiles. Optimizing the management of OSA relies on a multidisciplinary approach, in which CPAP remains central but must be reinforced by personalized, sustained support. By virtue of their accessibility and expertise, pharmacists are well-placed to play a key role in this strategy.

They are capable of improving adherence, safety, and clinical outcomes, and can contribute to the integration of future therapeutic innovations.

Despite growing recognition of pharmacists' roles in the management of OSA, scientific evidence remains limited, which hinders the consolidation of structured practices based on robust data. Clinical trials evaluating the use of oxygen in OSA demonstrate significant methodological variability, including differences in inclusion criteria, small sample sizes, inconsistent protocols, and heterogeneous outcomes. The lack of direct comparisons with CPAP or studies on therapeutic combinations makes it difficult to formulate clear recommendations, also affecting evidence-based pharmaceutical practice.

Studies that systematically evaluate the impact of pharmacist intervention on CPAP adherence, sleepiness reduction, or improvement of clinical markers are rare. The lack of randomized trials with structured follow-up programs limits the scientific validation of practices that have already been successfully adopted in the field.

To integrate pharmacists into formal care pathways in AOS, collaborative models in real-world settings need to be investigated. Implementation studies should address not only clinical outcomes, but also adherence, safety, patient satisfaction, and cost-effectiveness. The use of digital tools (telemonitoring, dashboards, shared software) will be crucial in this process.

With the introduction of glucagon-like peptide-1 (GLP-1) receptor agonists [75,76], oral devices [8,9], hypoglossal nerve stimulation [77,78], and future pharmacological options, the role of the pharmacist will have to evolve. The ability to adapt to new technologies, drugs, and patient profiles will be critical to ensuring effective, integrated, and patient-centered intervention.

Future research should aim to overcome the current methodological weaknesses by conducting large, well-designed randomized controlled trials that directly compare CPAP, oxygen therapy, and combined strategies, while systematically including pharmacist-led interventions on adherence, symptom control, and cardiovascular outcomes in patients with OSA. Pragmatic studies in real-world settings are also needed to evaluate the impact of structured pharmaceutical protocols on adherence, safety, and long-term clinical outcomes. Digital health solutions, such as telemonitoring platforms and shared care dashboards, should be further explored to strengthen collaboration between pharmacists, physicians, and other healthcare providers. Finally, as novel therapies such as GLP-1 receptor agonists, hypoglossal nerve stimulation, and innovative oral devices expand the therapeutic landscape, research must investigate how pharmacists can best adapt to these evolving modalities, ensuring that their role remains patient-centered, integrated, and future-proof.

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Author Contributions

AT and MT conceived and designed the study protocol. AT and DL conducted the literature search and drafted the initial manuscript, contributed to data analysis, interpretation of results, and manuscript editing. All the authors participated in study selection, data extraction, and critical revision of the manuscript. All authors read and approved the final manuscript.

Conflicts of interest

The authors declare no competing interests.

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