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THE EFFECT OF CPAP ON HYPERTENSION IN PATIENTS WITH OBSTRUCTIVE SLEEP APNEA: A LITERATURE REVIEW

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ABSTRACT

Obstructive sleep apnea (OSA) is a chronic condition characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia, sympathetic nervous system activation, and blood pressure dysregulation. Numerous studies have confirmed the frequent coexistence of OSA and hypertension, making this association a significant clinical and public health concern. The aim of this review was to assess the effect of CPAP (Continuous Positive Airway Pressure) therapy on blood pressure in different groups of patients with obstructive sleep apnea, based on current scientific data. The review included publications from 2015 to 2025, available in the PubMed database, regarding randomized controlled trials and meta-analyses assessing the effect of CPAP on blood pressure. A total of 15 randomized controlled trials and meta-analyses were included in the analysis. CPAP therapy remains the gold standard for OSA treatment and can be an effective adjunct to blood pressure control, particularly in patients with resistant hypertension. However, treatment effectiveness depends on adherence to recommendations and appropriate patient selection. Further research should focus on assessing the long-term effects of CPAP, identifying patient phenotypes that best respond to therapy, and developing treatment strategies in combination with antihypertensive pharmacotherapy.

KEYWORDS

OSA, CPAP, Hypertension, Obstructive Sleep Apnea, Continuous Positive Airway Pressure, Hypertension, Ambulatory Blood Pressure Monitoring

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1. Introduction and Aims

1.1. Introduction

Obstructive sleep apnea (OSA) is a sleep-related breathing disorder characterized by recurrent episodes of partial (hypopnea) or complete (apnea) obstruction of the upper airway, leading to decreased oxygen saturation and/or awakenings. Diagnosis is made based on polysomnography or home sleep apnea testing (HSAT) and the apnea-hypopnea index (AHI). According to the American Academy of Sleep Medicine (AASM) guidelines, OSA is diagnosed when the AHI is ≥ 5/h with clinical symptoms or ≥ 15/h regardless of symptoms (Kapur et al., 2017). The latest 2023 AASM recommendations emphasize that hypopnea should be defined as a decrease in respiratory flow of ≥ 30% with desaturation ≥ 3% or arousal (American Academy of Sleep Medicine, 2023).

Epidemiologically, OSA is a common condition of increasing health importance. Global estimates indicate that approximately 936 million (95% CI [903-970] million) adults worldwide have OSA (AHI ≥ 5), and approximately 425 million (95% CI [399-450] million) have moderate to severe OSA (AHI ≥ 15) (Benjafield et al., 2019). The HypnoLaus population-based study found moderate to severe sleep-disordered breathing in 23.4% of women and 49.7% of men (Heinzer et al., 2015). Literature reviews suggest that the prevalence of OSA in adults ranges from 9 to 38% depending on diagnostic criteria and assessment methods (Senaratna et al., 2017).

Obstructive sleep apnea (OSA) is of significant clinical importance due to its wide range of health consequences. Numerous studies have shown that untreated OSA is associated with an increased risk of cardiovascular disease, including hypertension, heart failure, and arrhythmia (Stansbury & Strollo, 2015). OSA is also associated with metabolic disorders such as insulin resistance and type 2 diabetes, as well as with cognitive decline and an increased risk of traffic and occupational accidents (Stansbury & Strollo, 2015). Furthermore, recent analyses have also indicated an association between OSA (especially with nocturnal hypoxemia) and higher cancer morbidity and mortality—in one meta-analysis, individuals with OSA had

approximately a 1.5-fold increased risk of developing cancer compared to individuals without OSA (Tan et al., 2022). Early diagnosis and effective treatment of OSA can therefore not only improve the quality of life of patients but also potentially limit the development of long-term complications.

The mechanisms leading to the development of hypertension in patients with obstructive sleep apnea (OSA) are complex and involve interconnected neurohumoral, oxidative, and vascular processes. In patients with OSA, alternating hypoxia and reoxygenation enhance carotid body excitability and chemoreflex response, which perpetuates increased sympathetic activation, increases nocturnal and daytime blood pressure, and promotes non-dipping (Prabhakar, 2016; Sforza & Roche, 2016; Turnbull, 2018). Intermittent hypoxia and sleep fragmentation also generate oxidative stress and an inflammatory response, accompanied by a decrease in nitric oxide bioavailability and an increase in asymmetric dimethylarginine (ADMA) concentration, leading to endothelial dysfunction and increased arterial stiffness (Orrù et al., 2020; Peracaula et al., 2022). In parallel, the renin–angiotensin–aldosterone system (RAAS) is activated: patients with OSA are characterized by increased plasma renin activity, elevated angiotensin II and aldosterone, which leads to sodium retention, vascular remodeling, and perpetuation of hypertension (Jin & Wei, 2016; Loh et al., 2025). Recent reviews emphasize that these three pathways—chemoreflex-sympathetic, oxidative-endothelial, and RAAS—act synergistically, explaining the high incidence and resistance of hypertension in OSA (Bangash et al., 2020; Shiina, 2024).

The comorbidity of obstructive sleep apnea (OSA) and hypertension is very common and has significant clinical and population implications. Epidemiological studies indicate that patients with OSA have a significantly higher risk of developing hypertension, and the severity of respiratory disorders is closely related to blood pressure values (Kwon et al., 2024; Lombardi et al., 2018). In a nationwide cross-sectional study in Korea (KNHANES) in individuals aged 40–59 years, diagnosed hypertension was more common in the group with OSA than in the group without OSA: 48.1% vs. 10.5%, and uncontrolled hypertension despite treatment was more common (45.5% vs. 32.6%) (Lee & Kim, 2025). In turn, in individuals with hypertension, data suggest a high prevalence of OSA, especially in clinical populations and in resistant hypertension (Khamsai et al., 2021). In the subgroup of resistant hypertension, the co-occurrence of OSA is particularly common (usually ~70–80%) (Ahmed et al., 2023; Shiina, 2024). The population-level problem is large: the global burden of OSA is estimated at nearly 1 billion adults (AHI ≥ 5), which—given its high comorbidity with hypertension—translates into a significant burden on healthcare systems (Benjafield et al., 2019). Integrated literature confirms that the non-dipping, nocturnal, and resistant hypertension phenotypes are overrepresented in patients with OSA, which clinically justifies an active search for OSA in the hypertensive population (Kwon et al., 2024; Lombardi et al., 2018).

CPAP remains the gold standard for the treatment of obstructive sleep apnea (OSA) because it most effectively and comprehensively normalizes breathing during sleep, reduces daytime sleepiness, and improves quality of life compared with no treatment or alternatives (Bratton et al., 2015; Patil et al., 2019). The 2019 AASM recommendations clearly recommend PAP therapy (including CPAP/auto-CPAP) as the first-line treatment for adults with OSA, emphasizing its favourable efficacy and safety profile (Patil et al., 2019). In the SAVE study, including patients with moderate/severe OSA and concomitant cardiovascular disease, CPAP did not reduce the combined risk of major cardiovascular events, but significantly improved symptoms (sleepiness, snoring) and quality of life, and lowered blood pressure—especially with improved adherence (McEvoy et al., 2016). Meta-analyses indicate that the antihypertensive effect of CPAP is greatest in patients with resistant hypertension, particularly at night and at 24-hour mean blood pressure (Labarca et al., 2021), and critical reviews emphasize the dependence of benefits (including cardiovascular benefits) on the severity of OSA and duration of use (Kwon et al., 2024). In indirect comparisons, CPAP achieves greater improvement in sleepiness (ESS) and respiratory outcomes than mandibular advancement devices (MADs, although the effect on blood pressure may be similar with good adherence (Bratton et al., 2015). Overall, CPAP is the preferred, best-documented treatment method for OSA, bringing clear symptomatic and metabolic-hemodynamic benefits, especially in people with initially uncontrolled or resistant hypertension (Labarca et al., 2021; McEvoy et al., 2016; Patil et al., 2019).

1.2. Aims

The aim of this review is to assess the effect of CPAP (Continuous Positive Airway Pressure) treatment on blood pressure values in different groups of patients with obstructive sleep apnea (OSA).

2. Materials and methods

This literature review of the effects of CPAP (Continuous Positive Airway Pressure) on blood pressure in patients with obstructive sleep apnea (OSA) included publications from 2015 to 2025, retrieved from the PubMed database. Studies assessing the effects of CPAP on blood pressure in various patient groups, including those with primary and resistant hypertension, were analyzed. A total of 15 randomized controlled trials and meta-analyses were analyzed, providing a synthetic overview of the current state of knowledge regarding the effects of CPAP on blood pressure regulation in patients with OSA.

3. Results

3.1. Randomized controlled trial confirming the effectiveness of CPAP therapy

Several well-designed randomized clinical trials have shown that the use of CPAP therapy leads to a significant reduction in blood pressure in patients with obstructive sleep apnea (OSA), especially in 24-hour measurements (ABPM) and among people with initially uncontrolled or resistant hypertension.

One of the most frequently cited studies confirming this effect is Hoyos et al. (2015), conducted in Australia, which included 56 men aged 30–65 years with moderate to severe OSA (mean AHI: 33 ± 18 events/hour). The diagnosis of OSA was made based on full nocturnal polysomnography (PSG) according to the American Academy of Sleep Medicine criteria. The study excluded individuals with severe cardiovascular disease, type 2 diabetes, or previously diagnosed pharmacologically treated hypertension. Participants were randomized to an active CPAP treatment group or a control group (sham-CPAP) with a 12-week follow-up period. Blood pressure was assessed by 24-hour ABPM monitoring and office measurements. Mean baseline systolic blood pressure was approximately 131 ± 10 mmHg and diastolic blood pressure was 83 ± 7 mmHg. After 12 weeks of CPAP use, significant reductions in central and peripheral blood pressure were achieved, averaging 3–5 mmHg, with the effect being more pronounced at night. In participants who used CPAP for more than 4 hours per day, the BP reduction was twice as great as in those with low adherence. A strength of the study is the use of sham-CPAP as a placebo and the assessment of central aortic pressure, which allowed for a precise analysis of the treatment's impact on the circulatory system. The small sample size and short follow-up period remain limitations (Hoyos et al., 2015).

Similar conclusions were drawn from the study by Pépin et al. (2016), which assessed the effects of fixed positive airway pressure (CPAP) and automatically adjusted CPAP (auto-CPAP) on blood pressure. The study included 322 adult patients with moderate to severe OSA (mean AHI: 35 events/hour) and without other significant respiratory disorders. The diagnosis of OSA was confirmed by full polysomnography, and hypertension was defined according to the ESH/ESC criteria as SBP ≥ 140 mmHg or DBP ≥ 90 mmHg, or antihypertensive treatment. The follow-up period was 3 months, and the effects were assessed using ABPM. Although no differences in office blood pressure changes were found between groups, patients using fixed positive airway pressure (CPAP) had a significant reduction in 24-hour diastolic blood pressure by 2.6 mmHg compared with auto-CPAP. In subgroup analyses, a particularly beneficial effect was observed in individuals with high adherence (≥5 hours/night) and uncontrolled nocturnal blood pressure at baseline. This study confirms the importance of device mode and individualized therapy in achieving hemodynamic effects. Strengths include the large sample size and blinded BP measurements; limitations include the lack of a placebo group and short follow-up time (Pépin et al., 2016).

Another important study is Zhao et al. (2022), which included 213 patients with moderate to severe OSA, recruited from the general population, without significant daytime sleepiness symptoms (Epworth Sleepiness Scale < 10). The diagnosis was made based on nocturnal polysomnography, and the mean AHI was 36 ± 14 events/hour. Participants were randomized to CPAP or a 12-month control group. The primary endpoint was change in blood pressure assessed by ABPM and office monitoring. After one year of therapy, a reduction in mean systolic blood pressure of 3.1 mmHg and diastolic blood pressure of 2.5 mmHg ($p < 0.05$) was observed, particularly in nocturnal measurements. In individuals using CPAP for more than 4 hours per day, these reductions reached as much as 5–6 mmHg. The authors emphasize that despite the lack of sleepiness symptoms, the antihypertensive effect of CPAP is evident with appropriate duration of use. The strengths of the study include the long observation period and ABPM measurement, while the limitations include the lack of pharmacotherapy control and low average adherence in the entire population (Zhao et al., 2022).

3.2. Randomized controlled trials that did not confirm the effectiveness of CPAP therapy

Despite ample evidence supporting the benefits of CPAP therapy for blood pressure control, some randomized trials have failed to demonstrate significant reductions in blood pressure following this treatment. Analysis of these studies indicates that the reasons for these discrepant results are complex and include differences in study designs, patient populations, blood pressure measurement methods, and, above all, adherence to CPAP treatment.

One of the most prominent studies that failed to confirm a significant effect of CPAP on blood pressure is the study by Muxfeldt et al. (2015). This was a randomized, controlled trial involving 117 patients with resistant hypertension and moderate or severe OSA, defined as an AHI of ≥ 15/h on polysomnography. Participants were randomized to CPAP or continued standard pharmacological therapy. The follow-up period was 6 months, and the primary endpoint was the change in mean 24-hour systolic and diastolic blood pressure on ABPM. Baseline blood pressure values averaged 142 ± 16 / 90 ± 10 mmHg, despite the use of ≥ 3 antihypertensive medications. After six months of treatment, no significant differences were observed between groups – the mean change in blood pressure was only –1.8 mmHg for SBP and –0.8 mmHg for DBP ($p > 0.05$). Only a post-hoc analysis revealed a slight improvement in nighttime dipping among individuals with initially uncontrolled nocturnal blood pressure. The authors suggest that this modest effect may be due to low adherence (average 3.3 hours/night) and the relatively short duration of therapy, which does not allow for stable neurohormonal changes (Muxfeldt et al., 2015). Additional limitations included heterogeneous pharmacotherapy, lack of control over medication use, and the fact that most participants did not experience significant sleepiness symptoms, which could have negatively impacted their motivation to regularly use the device.

Similarly, Chen et al. (2020) conducted a randomized, 12-week trial of 94 patients with moderate to severe OSA and nocturnal hypertension. Diagnosis of OSA was based on full polysomnography (mean AHI 36 ± 11 events/h), and blood pressure was measured using 24-hour ABPM. After three months of CPAP treatment, no significant differences were found in systolic and diastolic blood pressure between the treatment and control groups. In the CPAP group, nocturnal blood pressure decreased nonsignificantly by 1.9 mmHg and daytime blood pressure by 0.7 mmHg. The authors indicated that the main factors limiting efficacy were insufficient adherence (mean 3.1 ± 1.5 h/night) and the absence of clinical symptoms of sleepiness, which typically increase subjective benefit and motivation for therapy. Furthermore, participants did not have significantly elevated BP values at baseline, which may have limited the potential for further reduction. Another limitation was the relatively short follow-up and the lack of data on changes in the circadian BP rhythm, which could better capture the therapeutic effect (Chen et al., 2020).

McEvoy et al. (2016), in the multicenter SAVE (Sleep Apnea Cardiovascular Endpoints) study of 2,717 patients with moderate to severe OSA and concomitant cardiovascular disease, found no effect of CPAP on the composite endpoint of myocardial infarction, stroke, and cardiovascular death. Secondary analyses revealed a modest reduction in systolic blood pressure of 2.5 mmHg and diastolic blood pressure of 2.0 mmHg, but this did not reach statistical significance. In this study, mean adherence to CPAP was only 3.3 hours per day, and most patients were on pharmacological therapy and had well-controlled blood pressure at baseline, further limiting the possibility of observing a hypotensive effect. The authors emphasized that the lack of benefit in "hard" endpoints does not rule out a positive effect of CPAP on blood pressure, especially in well-selected populations (McEvoy et al., 2016).

3.3. Meta-analyses and systematic reviews confirming the effectiveness of CPAP therapy

The most consistent evidence for the beneficial effect of CPAP therapy on blood pressure comes from meta-analyses combining data from numerous randomized clinical trials. Over the past decade (2015–2025), a number of analyses have consistently shown that CPAP, despite its modest antihypertensive effect, produces statistically and clinically significant reductions in blood pressure, particularly in 24-hour blood pressure monitoring (ABPM) and among patients with resistant hypertension.

One of the first large meta-analyses of this period was the work by Liu et al. (2016), which included 10 randomized clinical trials with a total of 572 patients with resistant hypertension (RH) and concomitant moderate or severe OSA (AHI ≥ 15/h). The analysis included only studies with 24-hour ABPM, which allowed for a precise assessment of diurnal blood pressure fluctuations. The authors showed that CPAP treatment led to a reduction in mean SBP by 4.8 mmHg (95% CI [–7.2 to –2.4]) and DBP by 2.9 mmHg (95% CI [–4.2 to –1.7]). This effect was more pronounced in the group with initially uncontrolled BP, but disappeared in the population with well-controlled BP. The Cochrane Risk of Bias method confirmed a moderate risk of bias, and

the heterogeneity of the analyses was limited ($I^2 < 40\%$). The authors emphasized that even a small reduction in BP is clinically important, as it translates into a 10–15% lower risk of stroke and coronary heart disease (Liu et al., 2016).

A meta-analysis by Lei et al. (2017) followed a similar trend, analyzing 12 randomized trials in patients with resistant hypertension. CPAP therapy was shown to reduce 24-hour SBP by 5.4 mmHg and DBP by 3.2 mmHg, with the most significant effect observed during the night. Importantly, in studies with follow-up longer than 3 months, the effect was more pronounced (–6.5/–3.7 mmHg), confirming the importance of duration of therapy. The authors also noted that the antihypertensive effect correlated linearly with the number of hours of CPAP use – each additional 1 hour per night translated into a decrease in SBP by approximately 1 mmHg. These findings indicate that adherence is one of the key factors determining therapy effectiveness (Lei et al., 2017).

In the systematic review and meta-analysis by Labarca et al. (2021) (Sleep Medicine Reviews), focusing exclusively on patients with resistant hypertension, the authors analyzed seven randomized trials (n = 555). A random-effects model was used, analyzing both ABPM and office blood pressure. A significant reduction in mean 24-hour SBP by 3.1 mmHg ($p < 0.01$) and DBP by 2.9 mmHg ($p < 0.05$) was found in the CPAP group. Importantly, in studies in which patients used CPAP ≥4 hours/night, the reduction in SBP reached 5–6 mmHg. The authors also emphasized that this effect was independent of BMI and age, and was most pronounced in individuals with high levels of nocturnal desaturations and elevated baseline BP. The strength of this meta-analysis was the selection of only studies with full ABPM and a detailed analysis of the impact of adherence, while its limitations included a small number of included trials and a short follow-up time in some of them (Labarca et al., 2021).

A meta-analysis by Shang et al. (2022), including 24 studies (including 16 RCTs, with a total of 1889 patients), confirmed a significant antihypertensive effect of CPAP in people with OSA and concomitant hypertension. The mean reduction in SBP was –2.6 mmHg (95% CI: –4.0 to –1.2) and in DBP –2.0 mmHg (95% CI: –3.2 to –0.8). The effect was more pronounced in groups with uncontrolled BP and with a follow-up period of ≥3 months. In studies shorter than 12 weeks, the differences were non-significant. The authors noted that, despite small absolute values, regular CPAP use in this population can significantly reduce long-term cardiovascular risk. Limitations included heterogeneity in BP measurement methods and a lack of standardization in reporting adherence (Shang et al., 2022).

Another valuable study is the network meta-analysis by Kou et al. (2022), which compared various interventions used in patients with OSA and hypertension, including CPAP, mandibular advancement devices (MADs), pharmacotherapy, and combination therapy. An analysis of 32 studies (n = 4560) demonstrated that CPAP led to a moderate reduction in BP (mean –3.3/–2.1 mmHg), with an effect comparable to MADs but less than that of pharmacological treatment. The authors emphasized that CPAP works synergistically with drug therapy, making it an important component of comprehensive treatment for patients with OSA and hypertension (Kou et al., 2022).

The most up-to-date data was provided by Pengo et al.'s (2025) individual meta-analysis, which included data from 12 RCTs and over 2,700 participants. Analysis at the individual patient data (IPD) level allowed for the assessment of factors modifying the response to CPAP. The study found that the greatest benefit was observed in patients with uncontrolled blood pressure at baseline (ΔSBP –5.5 mmHg) and those who used CPAP for at least 4 hours per day. This effect was observed regardless of gender and sleepiness level, but was more pronounced in younger individuals (<60 years of age). The authors noted that previous studies may have underestimated the effectiveness of CPAP due to short follow-up and low adherence (Pengo et al., 2025).

3.4. Meta-analyses with mixed results and limited effectiveness of CPAP therapy

Some meta-analyses from 2015–2025 indicate a small or inconsistent effect of CPAP therapy on blood pressure, particularly in general populations (all-comers) and in studies characterized by short follow-up periods and low treatment adherence. A classic network meta-analysis by Bratton et al. (2015), comparing the effectiveness of CPAP, mandibular advancement devices (MADs), and standard care, demonstrated a reduction of several mmHg after both interventions, but no significant difference between CPAP and MADs in terms of antihypertensive effect. The authors noted that the mean effect was small, and its magnitude significantly depended on factors such as treatment duration, adherence (less than 4 hours per night, the effect disappeared), type of blood pressure measurement (ABPM vs. office blood pressure), and population selection (patients with well-controlled blood pressure showed minimal changes). The limitations of the study included

analysis based on aggregated data, heterogeneity of protocols, and lack of standardization of antihypertensive pharmacotherapy (Bratton et al., 2015).

Elbadawi et al. (2021) presented similar conclusions in a systematic literature review of 25 randomized and observational studies examining the effects of CPAP on blood pressure. They found that although most analyses indicated a small decrease in mean SBP and DBP values, only a few reached statistical significance. The antihypertensive effect of CPAP was most noticeable among patients with resistant hypertension and good adherence, but was not observed in patients with mild OSA or well-controlled blood pressure. The authors emphasized that including studies with short follow-up (<3 months) and low adherence to therapy may have significantly underestimated the overall effect (Elbadawi et al., 2021).

In contrast, umbrella reviews, which combined data from studies including both OSA populations and mixed groups with primary and resistant hypertension, yielded statistically insignificant or borderline effects (Bratton et al., 2015; Elbadawi et al., 2021). The main reasons for this phenomenon included: 1) the predominance of short-term studies (≤12 weeks), 2) low average CPAP use, 3) lack of stratification of results according to the degree of baseline blood pressure control, 4) lack of division into daytime and nighttime measurements, which underestimated the nocturnal effect of ABPM. Nevertheless, even in these analyses, a moderate reduction in SBP of approximately 2–3 mmHg was observed, which may be of population significance given the large number of patients (Elbadawi et al., 2021).

4. Discussion

Analysis of available studies from 2015–2025 indicates that the effectiveness of CPAP therapy in reducing blood pressure in patients with obstructive sleep apnea (OSA) is variable and dependent on a number of clinical and methodological factors. Most studies confirming the beneficial effects of CPAP observed moderate reductions in systolic and diastolic blood pressure, particularly in 24-hour ABPM measurements and in populations with initially uncontrolled or resistant hypertension. Studies in which the antihypertensive effect was absent or statistically insignificant had common features such as short follow-up (less than 3–6 months), insufficient treatment adherence (average <4 hours/night), and baseline normal blood pressure values, which limited the potential for further reduction.

Differences between studies may also result from different methods of diagnosing OSA and measuring blood pressure. Studies using polysomnography and 24-hour ABPM monitoring were more likely to demonstrate a therapeutic effect than those based on simplified diagnostics and office measurements. Furthermore, the effectiveness of CPAP may be modulated by the individual patient's phenotype, including the degree of nocturnal hypoxemia, the AHI index, the coexistence of obesity and insulin resistance, and the activity of the sympathetic nervous system. These factors influence both the pathogenesis of hypertension in OSA and the response to treatment.

Meta-analyses confirm that although the mean blood pressure reduction following CPAP is relatively small (typically 2–6 mmHg), it is clinically significant, particularly in the context of long-term cardiovascular risk. According to epidemiological data, even such a reduction can reduce the risk of stroke by approximately 10% and coronary heart disease by 5–15%. However, it should be noted that the effect of CPAP is largely dependent on consistent use of the device; lack of regularity in therapy means that potential benefits remain untapped.

Clinically, CPAP should be considered an adjunct therapy to, and not a replacement for, pharmacological treatment of hypertension. Its role is to reduce nocturnal sympathetic activation, improve endothelial function, and normalize the circadian blood pressure rhythm. In clinical practice, patients with severe OSA, high cardiovascular risk, and uncontrolled blood pressure despite antihypertensive treatment benefit the most.

However, from a methodological perspective, most available studies have certain limitations, including small sample sizes, heterogeneous inclusion criteria, variable follow-up times, and lack of standardization in adherence reporting. Further research should focus on identifying patient phenotypes that best respond to CPAP, analyzing long-term treatment effects, and assessing the efficacy of combined treatment with antihypertensive pharmacotherapy.

5. Conclusions

Based on the analysis of current research, it can be concluded that CPAP (Continuous Positive Airway Pressure) therapy is an effective method for supporting blood pressure control in patients with obstructive sleep apnea (OSA), especially in populations with uncontrolled or resistant hypertension. The antihypertensive effect of CPAP is usually moderate but clinically significant, with the greatest benefits observed in 24-hour ABPM measurements and in relation to nocturnal blood pressure. Treatment effectiveness depends largely on adherence to therapy – regular use of the device for at least 4–5 hours per day is required to achieve measurable hemodynamic effects. The lack of significant effects in some studies is primarily due to short follow-up periods, low CPAP use rates, and well-controlled baseline blood pressure, which limits potential improvement. CPAP should be considered an adjunct to pharmacological treatment because it improves endothelial function, reduces sympathetic nervous system activity, and reduces cardiovascular risk. Further research should focus on identifying the phenotypes of patients who respond best to therapy, assessing the long-term effects of CPAP, and determining optimal combination treatment strategies in patients with various forms of hypertension.

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