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IMPACT OF NASAL OBSTRUCTION ON SLEEP QUALITY AND OBSTRUCTIVE SLEEP APNEA: A NARRATIVE REVIEW

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ABSTRACT

Background: Obstructive sleep apnea (OSA) is a common and clinically significant sleep disorder involving recurrent episodes of upper airway collapse during sleep. While pharyngeal obstruction is the primary mechanism, nasal obstruction may also increase airway resistance, promote mouth breathing, and contribute to sleep disruption and OSA severity.
Objective: This narrative review aims to explore the impact of nasal obstruction on sleep quality and OSA severity, with a particular focus on pathophysiological mechanisms, clinical implications, and therapeutic relevance.
Methods: Relevant literature published between 2000 and 2025 was identified through PubMed and Google Scholar using keywords such as "nasal obstruction", "obstructive sleep apnea", "nasal breathing", "sleep disturbances", "nasal surgery", and "CPAP adherence". Included studies were review articles, meta-analyses, and original clinical research involving adult populations.
Results: Although nasal obstruction alone is rarely the primary cause of moderate to severe OSA, it may negatively influence sleep quality, promote mouth breathing, worsen upper airway collapsibility, and reduce adherence to CPAP therapy. Medical and surgical interventions aimed at improving nasal airflow have shown variable results in sleep-related outcomes.
Conclusion: Nasal obstruction plays a significant yet complex role in OSA and sleep quality. Improving nasal patency may enhance patient-reported outcomes and facilitate more effective management of sleep-disordered breathing.

KEYWORDS

Nasal Obstruction, Obstructive Sleep Apnea, Nasal Breathing, Sleep Disturbances, Nasal Surgery, CPAP Adherence

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Introduction

Obstructive sleep apnea (OSA) is a widespread disorder, estimated to affect nearly 936 million adults worldwide. Around 425 million of these individuals have moderate-to-severe disease (AHI ≥ 15), yet up to 80% remain undiagnosed. The likelihood of developing OSA increases with advancing age, male sex, obesity, specific anatomical features of the upper airway, and lower socioeconomic status. Without treatment, OSA can lead to a range of serious health problems, including cardiovascular disease, stroke, type 2 diabetes, cognitive decline, depression, a higher risk of road traffic accidents, and increased mortality [1–5].

The global burden of OSA is steadily increasing, driven by rising obesity rates and an aging population. Beyond its medical implications, OSA also contributes to substantial societal and economic costs by lowering quality of life, reducing work productivity, and increasing long-term morbidity [4; 6; 7].

While pharyngeal collapse is central to OSA pathogenesis, other structural and functional factors outside the pharyngeal segment can also shape disease severity. Nasal obstruction, in particular, is gaining recognition as a relevant cofactor that may aggravate symptoms and complicate management [8; 9]. Restricted nasal airflow can disturb normal sleep breathing patterns and make positive airway pressure (PAP) therapy less tolerable [10; 11]. Although nasal obstruction alone seldom causes moderate-to-severe OSA, it can significantly reduce treatment effectiveness, especially in patients with additional anatomical or functional vulnerabilities [9].

Patients with untreated nasal disorders often continue to experience nasal congestion, snoring, and daytime sleepiness, even when receiving other forms of OSA therapy [8]. Considering how common nasal conditions are in the general population, understanding their contribution to sleep-disordered breathing is essential for accurate diagnosis and optimal care.

This narrative review examines current evidence on how nasal obstruction influences OSA pathophysiology, symptom burden, and treatment outcomes, with particular attention to pharmacological and surgical strategies for improving nasal airflow. The role of routine nasal assessment in patients with persistent symptoms or inadequate response to OSA therapy is also discussed.

Methodology

This narrative review was based on literature retrieved from the PubMed and Google Scholar databases, covering publications from 2000 through 2025. The search strategy included combinations of terms such as”nasal obstruction”, ”obstructive sleep apnea”, ”nasal breathing”, ”sleep disturbances”, ”nasal surgery”, ”rhinitis”, and”CPAP adherence”.

Studies were selected based on their clinical relevance to the relationship between impaired nasal airflow, sleep quality, and symptoms of obstructive sleep apnea (OSA). Priority was given to review articles, meta-analyses, and original clinical studies published in English.

Animal studies, publications focused exclusively on pediatric populations, and papers without full-text availability were excluded.

The Role of the Nose in Sleep Physiology

The nose plays a pivotal role in normal respiratory physiology, particularly during sleep [12]. It is not only the primary pathway for airflow but also performs multiple functions essential for maintaining upper airway stability, efficient gas exchange, and sleep continuity. Nasal breathing facilitates humidification, warming, and filtration of inspired air, which protects the lower airways and optimizes pulmonary function [8]. In healthy individuals with normal nasal resistance, ventilation during sleep occurs almost exclusively via the nasal route (mean ~96%), regardless of body position or sleep stage, underscoring the mechanical advantage of this breathing pathway [11; 12].

Under physiological conditions, nasal airflow offers the lowest resistance, with the nasal passages contributing over 50% of total upper airway resistance. Mild resistance is not pathological; in fact, it helps maintain adequate inspiratory effort and pharyngeal muscle tone. However, a significant increase in resistance, whether due to structural or inflammatory causes, triggers compensatory mechanisms [8].

One such mechanism is the switch from nasal to oral breathing once a critical resistance threshold is exceeded. Oral breathing during sleep is physiologically disadvantageous—it is associated with up to 2.5-fold higher upper airway resistance in the supine NREM state (median 12.4 vs. 5.2 $cmH_2O\ L^{-1}\ s^{-1}$) [11], promotes retroglossal narrowing due to posterior tongue displacement, and increases pharyngeal collapsibility [8; 11]. Experimentally, exclusive oral breathing causes a marked increase in the frequency of obstructive apneas and hypopneas (AHI ~43 vs. 1.5 with nasal breathing), even in individuals without central events [11]. The mechanical advantage of nasal breathing is partly explained by the favorable positioning of the soft palate and tongue, which supports airway patency [11].

Increased nasal resistance also alters pressure dynamics described by the Starling resistor model—reducing upstream (nasal) pressure lowers retropalatal pressure, making it easier for pharyngeal transmural pressure to exceed the critical closing pressure (Pcrit), thus promoting flow limitation or collapse [9]. This effect is amplified in individuals with reduced pharyngeal tone, as in obstructive sleep apnea (OSA) [8; 9]. Nasal obstruction also impairs the nasal ventilatory reflex, decreasing dilator muscle activity and predisposing to microarousals and fragmented sleep [8].

An additional mechanism involves nitric oxide (NO) produced in the nasal passages. NO regulates vascular tone, gas exchange, and endothelial function, and may influence pharyngeal muscle tone and sleep regulation [8; 13]. Reduced nasal airflow decreases NO delivery to the lower airways, potentially worsening oxygenation and airway stability [13].

Population studies indicate that nocturnal nasal blockage is linked to a threefold higher likelihood of habitual snoring and excessive daytime sleepiness. Its impact appears greater in cases of mild or reversible obstruction, such as allergic rhinitis [8].

In summary, nasal breathing is essential for optimal sleep physiology—it minimizes airway resistance, stabilizes patency, supports NO-mediated regulatory mechanisms, and reduces the risk of pharyngeal collapse. Impaired nasal patency affects airway mechanics and reflex control, promoting sleep-disordered breathing and highlighting the importance of assessing nasal function in patients with sleep complaints or suspected OSA [8-13].

Nasal Obstruction as a Contributing Factor in OSA

Although upper airway collapse in OSA occurs primarily at the pharyngeal level, nasal obstruction can exacerbate this process by increasing airflow resistance and reducing airway stability. As outlined above, nasal breathing supports optimal upper airway mechanics, and its impairment destabilizes respiratory patterns during sleep [8; 14].

Experimental studies have demonstrated that artificially induced partial or complete nasal obstruction in healthy individuals increases the number of apneic events and microarousals, and can even trigger overt OSA episodes. While the correlation between obstruction severity and the apnea–hypopnea index (AHI) is generally weak—and nasal obstruction rarely serves as the sole cause of moderate-to-severe OSA—it can have a disproportionately greater clinical impact in mild disease or in cases of reversible obstruction, such as allergic rhinitis [8; 14].

Mechanistically, both structural abnormalities (e.g., septal deviation, turbinate hypertrophy) and inflammatory conditions (e.g., allergic rhinitis, chronic rhinosinusitis) increase nasal resistance, particularly during inspiration. This heightened resistance leads to more negative intrathoracic and intrapharyngeal pressures during sleep, lowering upstream segment pressure (PUS) towards the critical closing pressure (Pcrit) and promoting pharyngeal collapse [9].

In addition, nasal obstruction often shifts breathing toward the oral route, which—as discussed in the previous section—alters mandibular and tongue position and increases upper airway collapsibility. It also bypasses nasal mucosal reflexes and reduces the stabilizing influence of nitric oxide (NO) on airway patency [13]. Meta-analytic data indicate that OSA patients have significantly lower circulating NO metabolites compared with controls, with greater reductions in older and obese individuals; these decreases correlate with higher AHI values [13].

Clinical and observational evidence supports these mechanistic insights: greater nasal resistance correlates with more severe symptoms—such as louder snoring and increased daytime sleepiness—and, in certain populations, with higher AHI values [14]. Interventions aimed at restoring nasal patency, including septoplasty and turbinate reduction, consistently improve subjective sleep quality, reduce daytime symptoms, and, in some cases, lower therapeutic pressure requirements for positive airway pressure (PAP) therapy, although objective AHI improvements remain variable [8; 14].

To conclude, nasal obstruction contributes to OSA pathophysiology by increasing upper airway resistance, promoting oral breathing, reducing NO-mediated stability, and facilitating pharyngeal collapse. Recognition of these effects, and targeted treatment of nasal pathology, should be integral components of comprehensive OSA management strategies—particularly in patients with poor PAP tolerance, high pressure requirements, or persistent symptoms despite optimal therapy.

Impact on CPAP Therapy

Continuous positive airway pressure (CPAP) remains the standard approach for managing obstructive sleep apnea (OSA). The device delivers a steady stream of air through a mask worn over the nose or both the nose and mouth, creating a gentle pressure that keeps the upper airway open during sleep. This prevents the throat from collapsing and helps maintain uninterrupted breathing [1; 9].

By stabilizing breathing, CPAP reduces drops in blood oxygen levels and restores normal sleep architecture. Many patients notice better daytime alertness, sharper concentration, and improved overall well-being. Long-term adherence has been linked to cardiovascular benefits, including reduced blood pressure in those with hypertension, enhanced endothelial function, and lower oxidative stress and inflammation [1; 13; 17].

Still, sticking with CPAP can be difficult. Common barriers include discomfort from the mask, unintentional air leaks, nasal blockage or dryness, the need for high pressure settings, treatment costs, and a lack of thorough patient education and support [8; 16]. Nasal symptoms—reported by more than half of users [8; 14]—often trigger mouth breathing, which can worsen leaks and lower treatment effectiveness.

Nasal obstruction is a key factor in CPAP tolerance. When airflow through the nose is restricted, higher pressures are required to keep the airway open [9; 14]. This can worsen dryness, congestion, and pressure intolerance, ultimately reducing adherence. Treatments aimed at improving nasal airflow—such as intranasal corticosteroids, antihistamines, humidification, proper mask fitting, or surgical interventions like septoplasty or turbinate reduction—have been shown to lower the required pressure and make therapy more comfortable [8; 14; 15]. Some studies even report that patients who had abandoned CPAP were able to resume and extend nightly use by over two hours once nasal obstruction was corrected [15].

Current recommendations highlight the importance of assessing nasal patency before starting CPAP [16; 17]. This involves addressing reversible causes of obstruction, choosing the most suitable interface (ideally a nasal mask, if possible), and ensuring patients receive structured education and follow-up support. Early telemonitoring and prompt troubleshooting in the first weeks of therapy have proven particularly effective for individuals with pronounced nasal symptoms, high pressure needs, or a history of treatment intolerance [1; 16; 17].

Therapeutic Approaches Targeting Nasal Obstruction

This section synthesizes current evidence on how interventions designed to improve nasal airflow—whether pharmacological or surgical—affect sleep-related outcomes in patients with nasal obstruction. The review integrates findings from studies that use both objective indicators, such as the apnea–hypopnea index (AHI), oxygen desaturation index (ODI), minimum oxygen saturation ($MinSaO_2$), and respiratory disturbance index (RDI), and subjective measures, including the Epworth Sleepiness Scale (ESS), Pittsburgh Sleep Quality Index (PSQI), and patient-reported nasal symptom scores (e.g., TNSS, NOSE). In cases where research has examined continuous positive airway pressure (CPAP) therapy, emphasis is placed on whether nasal interventions influence titration pressure requirements, mask comfort, or adherence to treatment. Where available, additional disease-specific or functional endpoints are included to provide a comprehensive perspective on the therapeutic impact.

1. Pharmacological Treatment

Topical intranasal sprays—such as corticosteroids, antihistamines, and decongestants—are the mainstay of therapy for nasal obstruction. Current international guidelines recommend intranasal corticosteroids (INCS) as the first-line option, with fixed corticosteroid–antihistamine combinations preferred as a step-up strategy when symptoms persist despite monotherapy. Additional pharmacological options, including oral antihistamines, montelukast, and allergen immunotherapy, may be considered in selected cases to reduce nasal inflammation and congestion, thereby improving sleep quality [22; 23].

1.1 Topical treatment

Regular INCS use can reduce nasal congestion scores and improve CPAP tolerance, although the isolated effect on the AHI is typically minimal [19].

A systematic review and meta-analysis [20] found that topical nasal treatments may improve certain objective parameters of OSA—such as $MinSaO_2$, ODI, and RDI—as well as subjective measures of quality of life, particularly in patients with allergic rhinitis. However, pooled analysis did not demonstrate a statistically significant reduction in AHI in the general adult OSA population. Notably, allergic patients appeared to respond more favorably to topical corticosteroid therapy, with significant improvements in AHI and oxygenation compared to nonallergic patients [20].

In a randomized, double-blind, placebo-controlled trial [18] involving patients with moderate-to-severe OSA and coexisting chronic rhinitis, four weeks of INCS therapy significantly improved nonsupine RDI, TNSS, and PSQI. While between-group differences in AHI were not significant, within-group analysis in the INCS arm demonstrated reductions in AHI and improvements in both RDI and NREM RDI, highlighting a potential role for INCS in patients with concurrent nasal inflammation.

In another randomized, double-blind, placebo-controlled trial [21], a fixed intranasal antihistamine–corticosteroid spray (azelastine + fluticasone) administered for four weeks did not significantly improve overall CPAP adherence or AHI compared with placebo, though a modest benefit was observed in a prespecified subgroup using lower CPAP pressures (<15 cm H_2O). The treatment was well tolerated, with bitter taste being the most frequently reported side effect.

Overall, topical nasal therapy can relieve symptoms, improve nasal patency, and modestly enhance selected polysomnographic outcomes in specific subgroups—particularly in allergic patients—while its role may be most relevant in improving comfort and facilitating CPAP use, rather than substantially reducing AHI in unselected populations.

1.2 Oral treatment

Oral pharmacologic agents are generally used as adjunctive therapy for nasal obstruction secondary to allergic rhinitis or chronic rhinosinusitis and are not considered standalone treatments for OSA. Second-generation oral antihistamines reduce histamine-mediated symptoms—such as rhinorrhea, sneezing, and itching—thereby improving nasal patency and potentially supporting better sleep quality in allergic individuals [22; 23].

Allergen immunotherapy (AIT), particularly sublingual formulations, has been shown to improve sleep quality and nasal symptoms in patients with allergic rhinitis, with the greatest benefits observed in those with poor baseline sleep [22; 26].

These interventions are typically used alongside topical therapy, and their role in directly modifying OSA severity remains unproven.

1.3 Biological treatment

Biologic therapies, including omalizumab (anti-IgE) and dupilumab (anti–IL-4Rα), are approved for severe chronic rhinosinusitis with nasal polyps (CRSwNP) and can substantially improve nasal patency and disease-specific quality of life [23; 27; 28]. While improved airflow may theoretically reduce upper airway collapsibility and benefit sleep-disordered breathing, current evidence on their direct impact on OSA outcomes is limited [23]. Their use in this context should therefore be considered primarily for control of the underlying inflammatory disease rather than as a targeted treatment for OSA.

2. Surgical Treatment

Surgical interventions for nasal obstruction aim to restore or improve nasal airflow by correcting structural abnormalities or reducing mucosal hypertrophy. Common procedures include septoplasty, turbinate reduction, functional endoscopic sinus surgery (FESS), and polypectomy, often performed alone or in combination. In the context of obstructive sleep apnea (OSA), nasal surgery is rarely considered a stand-alone treatment; rather, it is typically used to alleviate obstruction that contributes to disease symptoms or impairs tolerance to positive airway pressure (PAP) therapy [33–40].

An analysis of available studies shows that the highest effectiveness in improving nasal patency is achieved after septoplasty, either performed alone or in combination with turbinoplasty. In Pedersen's study [29], both procedures provide comparable improvement in patency as measured by the VAS scale, with approximately 25% of patients achieving a normal score (<9) [29, 30]. The positive effect is particularly evident in patients with a high NOSE score (>30) and without coexisting nasal or sinus disease [30].

In contrast, turbinoplasty as a standalone procedure is significantly less effective, with only 8% of patients reaching a normal VAS score (<9), and is performed mainly in cases resistant to anti-inflammatory treatment, which may partly explain its lower efficacy.

Overall, the greatest postoperative improvement is observed in patients with severe nasal obstruction prior to surgery, with a ≥1-grade reduction in symptom severity in 90% of these cases [29].

The findings highlight that the choice of surgical method should be based on symptom severity, the results of standardized assessment tools (e.g., NOSE scale, VAS), and individual anatomical considerations [29; 30].

2.1 Impact of isolated nasal surgery on OSA severity (AHI and other objective parameters)

Meta-analyses indicate that isolated nasal surgery has only a limited impact on the apnea–hypopnea index (AHI), with changes generally small and clinically insignificant [31]. In two reviews, each including over 20 studies, findings were consistent—showing no clinically significant change in AHI or other polysomnographic measures, with more noticeable benefits only in mild OSA or selected subgroups [32; 33].

Randomized trials suggest that outcomes may depend on the patient's baseline breathing route. In a placebo-controlled study, improvement was seen in only 15% of patients, all of whom had a low proportion of nasal breathing during sleep before surgery (< ~62% of total sleep time). Patients who already breathed mainly through the nose showed no benefit [36]. These results indicate that increasing nasal patency rarely influences pharyngeal collapsibility when nasal breathing is already predominant.

2.2 Subjective Sleep-Related Outcomes

Nasal surgery has been shown to provide consistent and clinically relevant improvements in subjective sleep quality and daytime alertness, particularly when nasal obstruction is the main contributing factor to sleep-related symptoms [10]. Improvements are commonly quantified using the ESS and PSQI. Across multiple studies, reductions in ESS scores range from approximately 1.5 to 3 points, reflecting a noticeable decrease in excessive daytime sleepiness, while PSQI scores improve by 1.7 to 3.4 points, indicating better overall sleep quality. The magnitude of these benefits tends to be greater in patients with structural abnormalities—such as septal deviation—than in those with primarily inflammatory causes of obstruction [34].

Evidence from meta-analyses supports these observations. In one analysis [32], ESS scores decreased by 2–3 points postoperatively despite minimal changes in the AHI, suggesting that subjective improvements are not solely dependent on objective changes in respiratory events. Another review [33] reported parallel postoperative improvements across multiple domains, including ESS, PSQI, nasal resistance, and snoring frequency.

Prospective studies add further detail to these findings. For instance, septoplasty combined with or without inferior turbinate reduction has been shown to significantly improve objective measures of nasal airflow—such as minimal cross-sectional area (MCA2) and nasal cavity volume (VOL2) on acoustic rhinometry—accompanied by a statistically significant reduction in PSQI from 6.29 to 5.18 ($p = 0.047$) [37].

In another cohort of 61 patients, postoperative improvements were striking: the NOSE score dropped from 68.2 to 17.5, the Ease-of-Breathing score rose from 3.6 to 8.3, and both PSQI (7.8 → 4.6) and ESS (7.5 → 5.3) showed significant reductions (all $p < 0.0001$). Notably, in this study, the degree of PSQI improvement correlated more strongly with the reduction in nasal obstruction than did ESS, suggesting that sleep quality perception is particularly sensitive to nasal airflow restoration [38].

Finally, in a prospective study of 125 patients with nasal airway obstruction, functional septorhinoplasty significantly improved both nasal patency (NOSE score) and sleep-related quality of life (FOSQ-10), with the greatest gains observed in those with OSA or habitual snoring. These results suggest that the procedure can enhance both nasal airflow and subjective sleep quality, and highlight the importance of incorporating sleep-related symptom assessment into the evaluation and management of nasal obstruction [40].

Overall, the available evidence consistently demonstrates that nasal surgery can produce meaningful improvements in subjective sleep quality and daytime alertness, even in the absence of major changes in objective OSA severity, with the greatest benefits seen in patients with structural nasal obstruction.

2.3 Impact on CPAP therapy

The strongest evidence supports nasal surgery as an adjunct to continuous positive airway pressure (CPAP) therapy. Postoperative reductions in titration pressures of approximately 2–3 cm H_2O have been reported, with nearly 90% of previously intolerant patients initiating or resuming CPAP and increasing nightly use by more than two hours [31]. These benefits are most pronounced in patients whose sole barrier to CPAP use is nasal obstruction, particularly when preoperative nasal cavity volume is small and allergic inflammation is minimal. In selected patients, significant decreases in mean and peak therapeutic pressures have been observed postoperatively, whereas those with multiple concomitant barriers show no improvement despite resolution of nasal obstruction [39]. This evidence highlights the importance of appropriate patient selection for surgical intervention. Nasal evaluation with assessment of patency is recommended, and when indicated, surgery should be performed before initiating CPAP therapy—particularly in patients with poor tolerance or high pressure requirements—to enhance comfort, reduce therapeutic pressures, and lower the risk of treatment discontinuation [33].

2.4 Overall interpretation

Available evidence and clinical guidelines [35] confirm that isolated nasal surgery is not a stand-alone treatment for OSA. Its role is primarily adjunctive—recommended for patients with significant nasal obstruction to improve subjective sleep quality and facilitate CPAP use by lowering pressure requirements and enhancing adherence. Postoperative follow-up should include both objective sleep studies and patient-reported outcome measures.

Conclusions

Nasal obstruction significantly affects breathing mechanics during sleep, may exacerbate the symptoms of obstructive sleep apnea (OSA), and reduces both the tolerance and effectiveness of CPAP therapy. It is essential to tailor OSA management to address the underlying cause of nasal obstruction. Appropriate pharmacological or surgical treatment can improve sleep quality, reduce daytime sleepiness, and facilitate CPAP use by lowering the required therapeutic pressure.

Isolated nasal surgery rarely leads to a significant improvement in the apnea–hypopnea index (AHI); however, it provides notable subjective benefits, particularly in patients with severe structural obstruction. Optimizing nasal patency should be an integral component of comprehensive OSA management, especially in individuals with pronounced symptoms and difficulties tolerating CPAP therapy.

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