

Environmental Factors That Can Affect Sleep and Breathing: Allergies



David T. Kent, MD^a, Ryan J. Soose, MD^{b,*}

KEYWORDS

• Allergic rhinitis • Nasal obstruction • Snoring • Sleep apnea • Sleep • Environment

KEY POINTS

- Sleep disorders, including insomnia and sleep-disordered breathing, are common in patients with allergic rhinitis and contribute greatly to the disease morbidity and effects on daytime function and quality of life.
- The degree of sleep disturbance is directly related to the severity of the allergic disease.
- The negative impact of allergic rhinitis on sleep is complex and multifactorial but is primarily mediated through nasal obstruction, which frequently has a diurnal variation and characteristic worsening in the overnight period and/or the supine position.
- Nasal obstruction increases the likelihood of snoring, obstructive sleep apnea, and intolerance to medical device therapies for sleep apnea.
- Although the impact of allergy and sinonasal treatment on polysomnography measures remains unclear and variable, medical and surgical therapy to lower nasal resistance has been shown to improve snoring, subjective sleep quality, daytime function, as well as disease-specific and general-health quality-of-life measures.

EPIDEMIOLOGIC CONSIDERATIONS

Environmental exposures have a significant role in sleep-disordered breathing. Sleep-related symptoms are common in patients with allergic rhinitis and have been observed for centuries. Hippocrates reported an association between poor sleep and nasal polyposis.¹ Almost 500 years ago, the Dutch physician Levinus Lemnius² reported that mouth breathing in the supine position causes restless sleep. Sleep impairment is likely a major contributor to the overall disease morbidity, direct and indirect health care costs, and the loss of work and academic productivity associated

with allergic rhinitis. Allergic disease and sleep disturbance are linked via a complex constellation of pathophysiologic, environmental, and patient-related factors (**Fig. 1**).³

Epidemiologic studies have estimated that allergic rhinitis afflicts 9% to 42% of the US population, equating to approximately 58 million people as of 2007.⁴ Its impact on disease-specific and general-health quality-of-life measures is well documented in large epidemiologic studies as well as controlled clinical trials.^{5,6} In a survey of patients with allergies, 48% with seasonal allergic rhinitis (SAR) and 68% with perennial allergic rhinitis reported that their condition interfered

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^a Department of Otolaryngology, University of Pittsburgh School of Medicine, UPMC, Pittsburgh, PA, USA;

^b Division of Sleep Surgery, Department of Otolaryngology, University of Pittsburgh School of Medicine, UPMC Mercy Building B, Suite 11500, 1400 Locust Street, Pittsburgh, PA 15219, USA

* Corresponding author.

E-mail address: sooserj@upmc.edu

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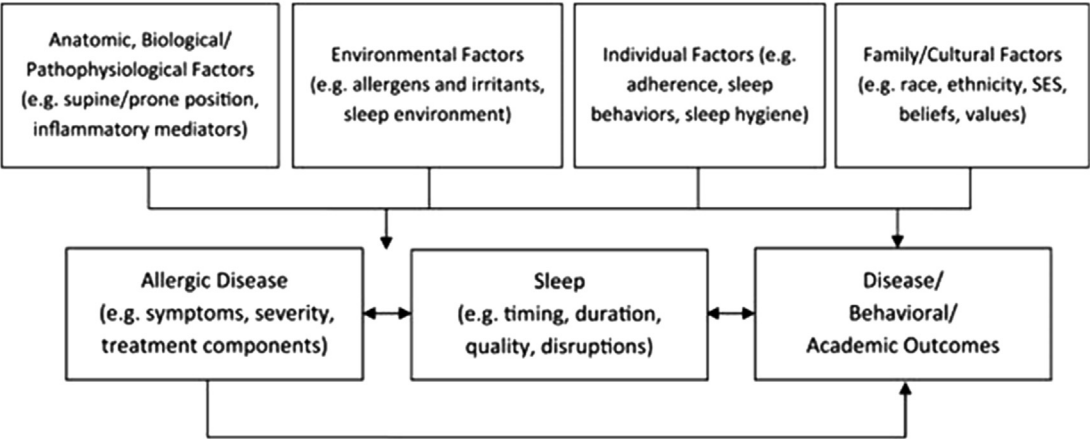


Fig. 1. Conceptual model of the association between allergic disease and sleep. SES, socioeconomic status. (From Koinis-Mitchell D, Craig T, Esteban CA. Sleep and allergic disease: a summary of the literature and future directions for research. *J Allergy Clin Immunol* 2012;130(6):1277; with permission from Elsevier Health Sciences.)

with their sleep.⁷ In the Wisconsin Sleep Cohort (a prospective population study of almost 5000 patients), self-reported nocturnal congestion was associated with a 3-fold increased incidence of snoring and daytime sleepiness.⁸ Craig and colleagues⁹ surveyed more than 2000 patients with allergies and approximately half reported difficulty falling asleep or staying asleep as a result of their allergic condition (Fig. 2).

Leger and colleagues⁵ showed that allergic rhinitis can affect multiple aspects of sleep with

numerous consequences on daytime function. Sleep-related breathing disorders, insomnia, as well as daytime somnolence, morning headaches, and cognitive dysfunction, were all more common in patients with allergic rhinitis compared with controls. In the Wisconsin Sleep Cohort, patients with allergic rhinitis were 1.8 times more likely to have moderate to severe sleep-disordered breathing than those without allergic rhinitis.⁸ Furthermore, patients with obstructive sleep apnea (OSA) with allergic rhinitis reported increased levels of general

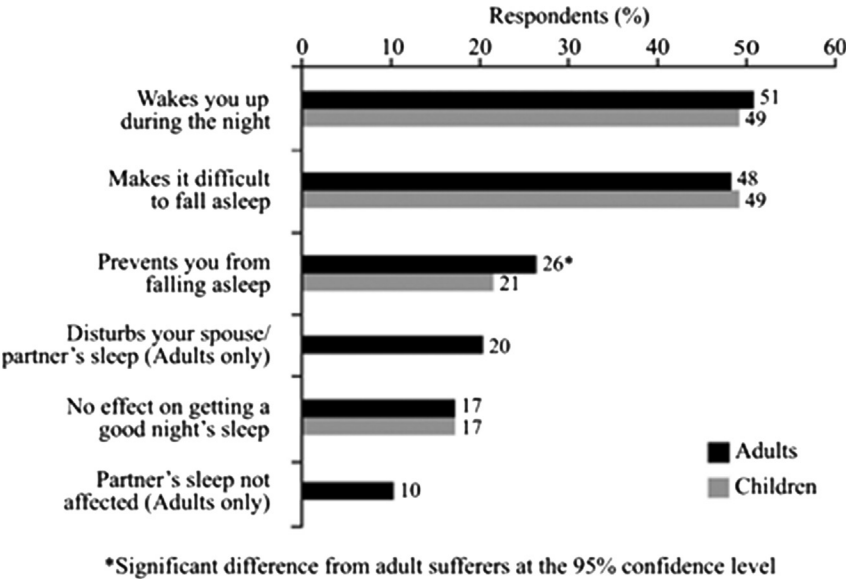


Fig. 2. Results of a survey of 2355 patients with allergic rhinitis and the subjective impact of nasal congestion on sleep. Note that approximately half of the respondents reported that their allergy-related nasal congestion makes it difficult to both fall and stay asleep. (From Craig TJ, Ferguson BJ, Krouse JH. Sleep impairment in allergic rhinitis, rhinosinusitis, and nasal polyposis. *Am J Otolaryngol* 2008;29:211; with permission.)

stress and fatigue compared with matched patients with OSA without allergic rhinitis.¹⁰

In patients with SAR, seasonal variation in sleep-related symptoms has been shown with worsening of subjective sleep quality and daytime sleepiness during the allergy season.⁵ Other research has found that subjective and objective sleep measures in patients with allergic rhinitis correlate with the severity of sinonasal disease.⁶ As such, sleep disturbance is one of the key factors in the Allergic Rhinitis and its Impact on Asthma (ARIA) guidelines that distinguishes between mild and moderate-severe disease.¹¹ The degree of sleep impairment, the frequency and duration of respiratory events, and associated daytime dysfunction all seem to be directly related to the severity of the allergic rhinitis (Figs. 3 and 4).^{6,12,13}

In addition to rhinitis caused by allergic disease, clinicians should recognize that symptomatic nasal obstruction and associated sleep disturbance may result from a multitude of other inflammatory and structural nasal disorders, often in combination. A recent study found that both patients with allergic rhinitis and patients with nonallergic have impaired sleep quality, with up to 83% of patients with nonallergic rhinitis reporting sleep disturbances.¹⁴ McNicholas¹⁵ noted that studies assessing sleep-disordered breathing were more likely to be associated with reversible nasal obstructive disease than chronic obstruction, implying that allergic rhinitis may have an even greater impact on sleep-disordered breathing than static structural problems such as a deviated septum. However, other large epidemiologic studies have confirmed that nasal obstruction of either type is an independent risk factor for both snoring and OSA.^{16–18}

PATHOPHYSIOLOGY

The mechanisms by which allergic rhinitis affects sleep are likely multifactorial. Upper airway resistance, fraction of oral breathing, ventilatory effort, obstructive respiratory events, the confounding effects of allergy medication and sedative drugs, anxiety, depression, cytokine production, and other biochemical and hormonal effects all may play a role in the negative impact on sleep quality. Multiple inflammatory mediators have been linked to sleep dysregulation (including interferon gamma, tumor necrosis factor alpha, interleukin [IL]-1 β , IL-4, and IL-10).^{19,20} Some of these show a circadian rhythm with progressive worsening after midnight in patients with allergic rhinitis, peaking in the early morning hours (Fig. 5).^{3,9} Proposed mechanisms for the observed diurnal variation in nasal congestion include the gravity effect of the dependent sleeping position, normal nocturnal decline in serum cortisol levels, and upregulation of inflammatory cytokines overnight. Simple elevation of the head of bed may counteract the gravity effect and significantly lower nasal resistance.²¹ The increase in nasal congestion and inflammation in the second half of the night may be further compounded by the increased proportion of rapid eye movement (REM) sleep, which is already characterized by autonomic instability and reduced pharyngeal muscle activity.

Medical therapy for allergic rhinitis and/or sleep-disordered breathing may further contribute to the sleep disturbance. Oral antihistamines, frequently used to manage allergy symptoms, are also commonly used by the public as an over-the-counter insomnia treatment despite scant data on improvement in objective sleep measures.

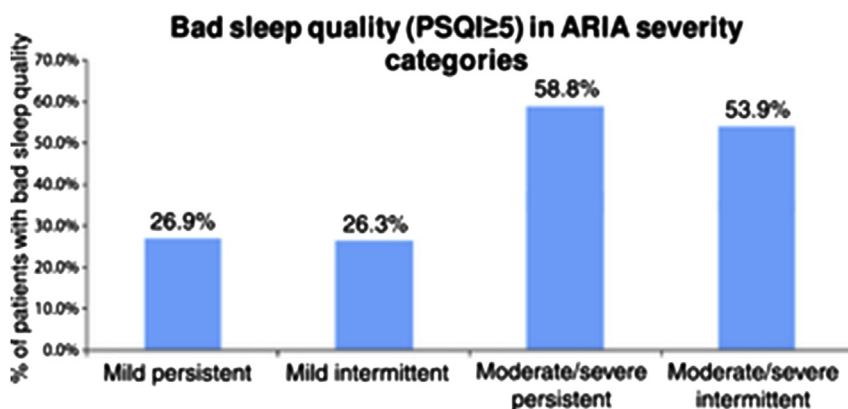


Fig. 3. Pittsburgh Sleep Quality Index (PSQI) scores in relation to ARIA severity categories in a survey of 2275 patients with allergic rhinitis. PSQI scores of greater than or equal to 5 define bad sleepers. (From Colás C, Galera H, Añibarro B, et al. Disease severity impairs sleep quality in allergic rhinitis (The SOMNIAAR study). *Clin Exp Allergy* 2012;42(7):1084; with permission.)

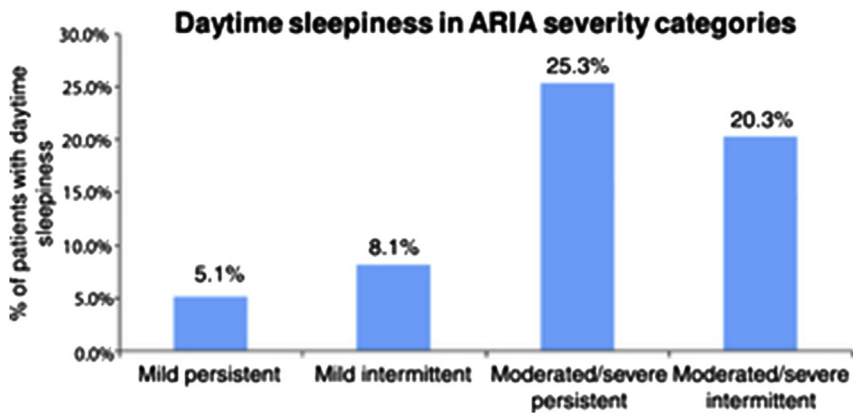


Fig. 4. Excessive daytime sleepiness in relation to ARIA severity categories in a survey of 2275 patients with allergic rhinitis. (Reprinted from Colás C, Galera H, Añibarro B, et al. Disease severity impairs sleep quality in allergic rhinitis (The SOMNIAAR study). Clin Exp Allergy 2012;42(7):1084; with permission.)

Oral antihistamines may cause side effects of drowsiness and fatigue as well as dryness of the mucous membranes caused by the anticholinergic effects. Decongestant medications and beta agonist inhalers may cause sleep-onset and sleep-maintenance insomnia. The impact of positive pressure therapy on the nose is still unclear and likely varies from patient to patient. In some

patients with OSA, continuous positive airway pressure (CPAP) therapy with heated humidification lowers nasal resistance and improves symptoms. However, for other patients, CPAP causes or exacerbates sinonasal symptoms, which may be related to pressure requirements, mask type, and humidity level. A 2012 study by Alahmari and colleagues²² found that CPAP therapy resulted in the dose-dependent upregulation of inflammatory markers associated with nasal and systemic inflammation as well as reduced mucociliary clearance.

Although the pathophysiology linking allergic rhinitis and sleep disturbance is complex and multifactorial, nasal obstruction seems to be the primary factor. Several airway models and experiments have suggested that nasal obstruction plays a role in sleep-disordered breathing through a variety of mechanisms, regardless of the cause of the nasal resistance (Table 1).²³

Nasal breathing normally accounts for 92% and 96% of inhaled ventilation in awake and sleep states, respectively.²⁴ The Starling resistor model of the airway explains that increasing the resistance of airflow through the nose (which in normal individuals already accounts for 50% to 66% of total airway resistance²⁵) may potentiate the risk of pharyngeal collapse because upstream nasal resistance of airflow generates greater negative intraluminal pressure within the oropharynx during inspiration.^{26,27} Many individuals with inadequate nasal airflow also convert to maladaptive oral breathing, which may be associated with a 2.5-fold greater airway resistance¹⁶ and subsequently a narrower and more collapsible pharyngeal airway.²⁸

Multiple studies have shown that experimentally induced nasal obstruction leads to abnormal sleep

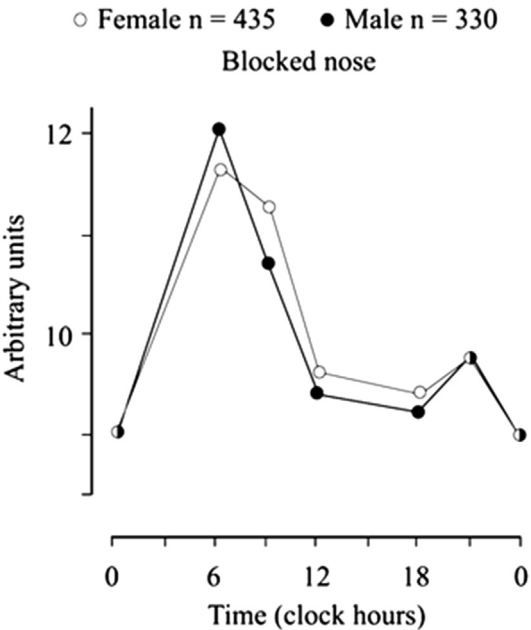


Fig. 5. Circadian variation of nasal congestion shows progressive worsening overnight with an average peak in nasal resistance at approximately 6 AM. (From Craig TJ, Ferguson BJ, Krouse JH. Sleep impairment in allergic rhinitis, rhinosinusitis, and nasal polypsis. Am J Otolaryngol 2008;29:211; with permission.)

Table 1
Proposed pathophysiologic mechanisms
linking nasal obstruction and sleep-disordered
breathing

Starling resistor model	Increased nasal resistance upstream results in increased negative intraluminal pressure (suction force) in the oropharyngeal airway downstream
Unstable oral breathing	Increased nasal resistance results in compensatory increase in oral breathing fraction and shift to breathing through an unstable oral airway, resulting in an increase in total airway resistance
Nasal ventilatory reflex	Decreased nasal airflow results in decreased activation of nasal neurologic receptors with subsequent reduction in pharyngeal muscle activity and central control of breathing
Nitric oxide	Decreased nasal airflow causes reduced lung nitric oxide with resultant potential perfusion ventilation mismatch as well as other central and pharyngeal effects

Adapted from Georgalas C. The role of the nose in snoring and obstructive sleep apnoea: an update. Eur Arch Otorhinolaryngol 2011;268(9):1366; with permission.

continuity, abnormal sleep architecture, and increased obstructive respiratory events in normal patients.^{29–34} In contrast, the nasal route of breathing additionally has been shown to improve minute ventilation and increase pharyngeal muscle activity.^{35,36} In a randomized, single-blind, placebo-controlled, and sham-controlled crossover study, McLean and colleagues³⁷ compared the impact of the fraction of oral breathing on objective sleep measures. With polysomnography, they showed that patients with lower nasal resistance and a normal nasal route of breathing had decreased light sleep (stage 1), increased deep sleep (slow wave), increased REM sleep, and improved sleep efficiency. However, nasal obstruction, caused by postoperative nasal packing for example, significantly worsens the apnea-hypopnea index (AHI) in patients with and without OSA.³⁸

In addition to physical nasal obstruction, sensorineural feedback from the nose also seems to play

a key role in control of breathing during sleep. Even in the absence of anatomic obstruction, abolishment of nasal receptor reflexes with topical lidocaine seems to increase the frequency of obstructive events and depress central respiratory drive.³⁹ In a controlled crossover study, patients treated with nasal lidocaine (4%) with decongestant had a mean AHI increase from 6.4 to 25.8, compared with the same patients treated with nasal saline with decongestant. An increase in both central and obstructive respiratory events was observed, leading the investigators to conclude that nasal receptor stimulation is critical for central respiratory control and maintenance of pharyngeal muscle activity. Decreased stimulation of these receptors has been found to increase the frequency of apneic episodes as well as their severity.^{39,40} In contrast, their activation during normal breathing increases resting respiratory rate and minute ventilation.⁴¹ This pathway may be mediated in part by nitric oxide, which is produced in large quantities in the nose.⁴² Nitric oxide acts as a pulmonary vasculature vasodilator, affects pharyngeal muscle activity, and also has central effects on breathing and sleep regulation that are not yet well understood.^{42,43}

EVALUATION OF ALLERGIC RHINITIS AND NASAL OBSTRUCTION

A thorough sinonasal history and an upper airway examination are integral components of successful management of patients with sleep-disordered breathing. Symptomatic nasal obstruction and associated sleep disturbance may be caused by a wide variety of medical and structural disorders (Table 2). Although considerable overlap exists, in general terms, the inflammatory/mucosal conditions are commonly treated with medical therapy, whereas the structural problems are often modifiable with surgical therapy.

Initial evaluation should focus on a detailed clinical history of allergic and nasal obstructive symptoms as well as a thorough examination to evaluate for both inflammatory and anatomic abnormalities. A thorough nasal evaluation can be brief and inexpensive in the routine patient, and does not necessarily require sophisticated rhinomanometry, imaging, or endoscopic techniques. In-office examination of the external nasal dorsum and anterior rhinoscopy with a nasal speculum and light source is often adequate and informative for most patients and can distinguish between structural and inflammatory causes of nasal obstruction.

History is critical in the evaluation of environmental factors contributing to nasal obstruction. A history of SAR is easy to elicit because most

Table 2
Examples of causes of symptomatic nasal obstruction and associated sleep disturbance

Medical	Structural
Allergic rhinitis	Deviated nasal septum
Vasomotor rhinitis	Inferior turbinate hypertrophy
Atrophic rhinitis	Acquired nasal deformity
Rhinitis medicamentosa	Nasal valve collapse
NARES	Concha bullosa
Medications (eg, antihypertensives, aspirin, oral contraceptives)	Nasal polyps
Infection (eg, common cold, chronic rhinosinusitis)	Adenoid hypertrophy
Hormonal (eg, pregnancy, hypothyroidism)	Tonsillar hypertrophy
Autoimmune (eg, lupus, Wegener, rheumatoid arthritis)	Narrow pyriform aperture/hard palate
Occupational (eg, chemicals, paint fumes, chalk dust, sawdust, perfumes)	Inverting papilloma
Smoking	Neoplasm
	Foreign body
	Synechiae
	Meningocele
	Granulomatous disease

Abbreviation: NARES, nonallergic rhinitis with eosinophilia syndrome.

patients can recall the recurrent onset of symptoms in relation to changing seasons or locations. A history of perennial allergic rhinitis may be more difficult to assess, because patients may not recall a clear association between exposure and onset of symptoms. Questions targeting symptoms during a change in environment (such as recent travel) may be helpful in identifying these exposures. Patients may also recall acute exposure events, such as a moldy basement, or work-related exposures, such as sawdust or coal dust. A sinonasal history should also include home or occupational exposures as well as previously attempted successful and unsuccessful medical therapies. Patients may have acclimated to a lifetime of chronic nasal obstruction and may not initially realize the significance of their symptoms. It is also important to assess the temporality of nasal obstruction, because many patients with allergic rhinitis and sleep-disordered breathing primarily experience nasal symptoms when supine and/or overnight. A clinician may miss or underestimate sinonasal disorders if relying only on daytime questioning and examination.

Reliable, validated questionnaires such as the Nasal Obstruction Symptom Evaluation (NOSE) scale or the Sinonasal Questionnaire are quick and easy to administer and may be useful for further quantifying nasal obstruction (Fig. 6, Table 3).^{44,45} Inhalant and food allergy testing, either via skin prick sensitivity or immunoglobulin E-specific blood work, can also provide useful information to implement environmental controls and avoidance strategies. Allergy testing results can also serve as the basis for subcutaneous or sublingual immunotherapy treatment options.

EVIDENCE FOR MEDICAL THERAPY

Treatment of nasal obstruction may be classified broadly into either medical or surgical management strategies, although in practice many patients undertake individualized therapy tailored to their specific symptoms and anatomy. Many allergy exposures can be modulated with behavioral changes including allergen avoidance, dietary modification, and smoking cessation. Medical therapy may be approached through symptomatic management using decongestants, antihistamines, and topical steroid therapy. In patients with more severe allergic rhinitis or persistent symptoms despite environmental and medical treatment options, subcutaneous or sublingual immunotherapy may be beneficial.

Nasal steroids are the most extensively studied medical treatment of allergic rhinitis as it relates to sleep disturbance, and several randomized controlled trials have evaluated their efficacy along with other pharmacologic therapy (Table 4).

The largest trial examining the use of intranasal corticosteroids in patients with OSA was recently published by Lavigne and colleagues,⁴⁶ who reported statistically significant improvement in supine AHI as well as baseline O₂ saturation in 34 patients with OSA and allergic rhinitis compared with 21 patients without allergic rhinitis. However, the patient group with allergic rhinitis had statistically significant worse AHI at enrollment. Another study, completed by Craig and colleagues^{47–50} in 2005, examined the results of 3 previously completed double-blind, placebo-controlled crossover studies on patients with non-OSA allergic rhinitis with sleep-related symptoms. All 3 trials excluded patients with OSA, and varied only in the nasal steroid applied in each trial (budesonide, flunisolide, or fluticasone). Pooled results described a significant decrease in nasal congestion and daytime somnolence, as well as a significant negative correlation between nasal congestion and improved sleep quality as measured using a nonvalidated, self-reported measurement scale.

Over the last 3 months how often, on average, did you have the following symptoms?

	Never	1 - 4 times per month	2 - 6 times per week	Daily
Runny Nose	☐	☐	☐	☐
Post nasal drip	☐	☐	☐	☐
Need to blow your nose	☐	☐	☐	☐
Facial pain/pressure	☐	☐	☐	☐
Nasal obstruction	☐	☐	☐	☐

Scoring: Never (0), 1-4 times per month (1), 2- 6 times per week (2), and daily (3).

Score reported as average of 5 items: range of possible scores 0 - 3.

Fig. 6. The Sinonasal Questionnaire. (From Dixon AE, Sugar EA, Zinreich SJ, et al. Criteria to screen for chronic sinonasal disease. *Chest* 2009;136(5):1330; with permission.)

Keily and colleagues⁵¹ reported on the use of fluticasone in 24 consecutive snoring patients with associated rhinitis using a randomized, placebo-controlled crossover design in 2004. Patients were divided into apneic and nonapneic groups based on an AHI cutoff of 10. The investigators reported a decrease in AHI from 30.3 to 23.3 in the apneic group despite no subjective symptom improvements. Daytime alertness was improved in nonapneic patients without any significant improvement in AHI.

A double-blind parallel study of mometasone versus saline published by Meltzer and colleagues⁵² in 2010 found an improvement in sleep

quality and quality of life as measured using the Rhinoconjunctivitis Quality of Life Questionnaire–Standardized score (RQLQ), Epworth Sleepiness Scale score (ESS), and Work Productivity and Activities Impairment–Allergy Specific (WPAI-AS) questionnaire score. None of the 30 patients had an AHI greater than 20, and no significant change in AHI or snoring was noted.

The benefit of topical antihistamine therapy on sleep is less clear. In a randomized, double-blinded crossover trial by Golden and colleagues,⁵³ subjective outcome measures were assessed in patients with allergic rhinitis with sleep-disordered breathing. Twenty-four patients

Table 3
The NOSE scale

	Not a Problem	Very Mild Problem	Moderate Problem	Fairly Bad Problem	Severe Problem
Nasal congestion or stuffiness	0	1	2	3	4
Nasal blockage or obstruction	0	1	2	3	4
Trouble breathing through my nose	0	1	2	3	4
Trouble sleeping	0	1	2	3	4
Unable to get enough air through my nose during exercise or exertion	0	1	2	3	4

From Stewart MG, Witsell DL, Smith TL, et al. Development and validation of the Nasal Obstruction Symptom Evaluation (NOSE) scale. *Otolaryngol Head Neck Surg* 2004;130:162; with permission.

Table 4
Randomized controlled trials of pharmacologic therapy for nasal obstruction and sleep-disordered breathing

Investigators	Study Design	Patient Population	Objective Outcomes	Subjective Outcomes
Lavigne et al, ⁴⁶ 2013	Prospective cohort study	21 patients with OSA and AR vs 34 patients with OSA and no AR	Improved O ₂ nadir and supine AHI in AR group	Significant improvement in daytime somnolence in AR group
Craig et al, ⁴⁷ 2005	Pooled data from 3 randomized, double-blind, placebo-controlled crossover studies	69 patients with AR but no OSA	No significant change in AHI (only measured in 1 study)	Decreased daytime somnolence and nasal congestion
Meltzer et al, ⁵² 2010	Double-blind evaluation of mometasone vs saline	30 patients with AR	No significant change in AHI	Improved QOL; decreased nasal congestion and daytime somnolence
Kiely et al, ⁵¹ 2004	Double-blind crossover study of fluticasone vs saline	10 patients without OSA (AHI<10); 13 patients with OSA (AHI>10)	AHI decrease from 30.3 to 23.3 in patients with OSA treated with fluticasone	Improved daytime somnolence in patients without OSA
Golden et al, ⁵³ 2000	Randomized, double-blind crossover trial of azelastine vs saline	24 patients with perennial AR (seasonal AR excluded)	Not measured	Improved rhinorrhea and sleep quality; no improvement in congestion or daytime somnolence
Santos et al, ⁵⁴ 2008	Randomized, double-blind crossover trial of montelukast vs placebo	31 patients with perennial AR (seasonal AR excluded)	Not measured	Significant improvement in daytime somnolence and fatigue
Kerr et al, ⁵⁷ 1992	Single-blind crossover study of oxymetazoline and nasal dilator vs placebo	10 patients with OSA	No change in AHI	Improved sleep quality
McLean et al, ³⁷ 2005	Single-blind crossover study of oxymetazoline and nasal dilator vs placebo	10 patients with OSA	Improved AHI, nasal resistance, and sleep architecture	No change in daytime somnolence
Clarenbach et al, ⁵⁸ 2008	Double-blind crossover study of oxymetazoline vs placebo	12 patients with OSA	No change in AHI	No change in daytime somnolence or sleep quality

Abbreviations: AHI, apnea-hypopnea index; AR, allergic rhinitis; QOL, quality of life.

were randomized to either treatment with azelastine or saline spray. Subjects reported that sleep was subjectively improved with azelastine; however, neither daytime sleepiness nor congestion was significantly improved.

Subjective response to montelukast therapy was examined in a randomized, double-blinded controlled trial by Santos and colleagues⁵⁴ in patients with allergic rhinitis. Significant improvement in daytime somnolence and daytime fatigue were noted with montelukast use compared with the placebo group.

Two studies have examined the role of nasal dilators in patients with nasal obstruction.^{55,56} A double-blind, crossover study by Pevernagie and colleagues⁵⁵ examining an external nasal valve dilator found a decrease in snoring frequency but otherwise no significant improvements in AHI or sleep architecture. Djupesland and colleagues⁵⁶ reported worsened AHI with an external valve dilator and no improvement in subjective or objective symptoms. Other available studies examined the use of sympathomimetic decongestant agents that are not likely to be suitable for long-term therapy because of concern for rebound effects, limiting their usefulness for treatment.^{37,57,58}

Summarizing the available literature, in patients with sleep-disordered breathing and rhinitis, intranasal steroids may improve both subjective and objective outcome measures, are associated with minimal risk/morbidity, and may provide a useful adjunct to CPAP and other more definitive forms of OSA therapy. Nevertheless, the consistency of the results remains unclear and adherence to intranasal steroids may be suboptimal and difficult to monitor. Further research is needed to determine the role of intranasal steroids, as well as nasal decongestants and other nasal medical therapy, in patients with sleep-disordered breathing and allergic rhinitis.

SURGICAL THERAPY FOR NASAL OBSTRUCTION

In many patients with allergic rhinitis, the symptomatic nasal obstruction and sleep-disordered breathing may be attributable to a combination of both inflammatory and structural disorders. A recent study by de Aguiar Vidigal and colleagues⁵⁹ found that patients with moderate to severe sleep apnea were significantly more likely to have significant nasal septal deviation or inferior turbinate hypertrophy.

The management approach to these patients generally involves first identifying and medically treating any underlying inflammatory process that may be present. For structural anatomic problems,

surgical therapy can be effective but the specific procedures indicated vary from patient to patient based on the specific anatomic and clinical problem. Surgical therapy also has a role in improving adherence to CPAP.

Structural causes of increased nasal resistance may not be confined to the nasal cavity alone. For example, Benninger and colleagues⁶⁰ reported that subjective sleep quality improved significantly following endoscopic sinus surgery in patients with chronic rhinosinusitis. It is well known, particularly in pediatric patients, that adenoid hypertrophy contributes significantly to nasal obstruction and sleep-disordered breathing, and that adenoidectomy can dramatically improve sleep and breathing. However, the impact of tonsillar hypertrophy on nasal resistance may be greatly underestimated. In 2007, Nakata and colleagues⁶¹ showed that patients with OSA with 3 to 4+ tonsils had significantly higher nasal resistance. Furthermore, simple tonsillectomy alone not only improved subjective and objective sleep measures but also greatly lowered nasal resistance.

Although adenotonsillectomy alone often successfully treats most children with OSA, many pediatric patients with allergic rhinitis require concurrent treatment of turbinate hypertrophy. In a large retrospective study, Sullivan and colleagues⁶² showed that pediatric patients with enlarged inferior turbinates often had both subjective and polysomnographic evidence of persistent sleep-disordered breathing after adenotonsillectomy alone. These residual symptoms and AHI increase were subsequently improved further with the addition of radiofrequency inferior turbinate reduction. Thus, overlooking treatment of nasal obstruction in children may lead to inadequate sleep apnea management.

Several recent studies have reported on the effect of surgically lowering nasal resistance on both subjective and objective measures of sleep-disordered breathing, although cautious interpretation is needed because of the heterogeneity of the reports and variable outcome measures. A meta-analysis of 13 studies completed by Li and colleagues⁶³ in 2011 found multiple reports of improved nasal resistance and subjective sino-nasal and sleep-related symptoms, despite a lack of significant change in the overall AHI (35.3–33.5; $P = .69$). Reports of improvement in other objective measures of success such as oxygen saturation nadir and improved sleep architecture were more variable. Two additional case series published in 2011 and 2012 additionally failed to show an improvement in AHI, although both reported improved sleep architecture.^{64,65}

These reports are consistent with a recent publication by Victores and Takashima,⁶⁶ which showed that nasal surgery did not significantly alter patterns of pharyngeal collapse after surgery as assessed by drug-induced sleep endoscopy, nor did it significantly improve the AHI in 24 patients.

It is again important to note that consistent improvement in objective measures of sleep and snoring may be difficult to identify in previous studies because of heterogeneous patient populations and definitions of surgical success. Despite these difficulties, there are significant data regarding subjective outcomes with regard to treatment of nasal obstruction and sleep symptoms. Significant improvement in subjective symptoms were reported in 9 of the 11 aforementioned studies that examined them, with large gains being made in measures of sleepiness and quality of life using tools such as the Epworth Sleepiness Scale and the Short Form Health Survey.^{64,67-74} Although AHI provides an important gauge of OSA severity in many patients, it remains only 1 metric of the complex relationship between sleep and breathing. A critical opportunity to improve patients' sleep may be lost by focusing only on the AHI rather than evaluating and managing the patients' subjective symptoms.

Although the current literature suggests that, across a heterogeneous population, lowering nasal resistance has little overall effect on the AHI, select patients, with otherwise good pharyngeal and craniofacial anatomy, in whom the nasal obstruction is a primary contributor to the OSA pathophysiology, may achieve meaningful improvement in AHI with nasal surgery. Findings that have been attributable to successful lowering of the AHI with nasal surgery alone include small tonsils, low modified Mallampati score, and a normal mandibular plane to hyoid distance on cephalometry. In contrast, large tonsils, modified Mallampati III/IV, and/or a low hyoid position suggest an unfavorable improvement in the AHI with nasal surgery alone.^{37,67,75}

In addition, treatment of nasal obstruction in patients with OSA can significantly improve adherence and effectiveness of CPAP and oral appliance therapy. Mounting evidence suggests that increased nasal resistance negatively affects success rates and tolerance of medical therapy devices for OSA, which critically depends on regular usage to be effective. High nasal resistance was one of only 2 independent predictors (in addition to body mass index) of inadequate treatment response to oral appliance therapy.⁷⁶ Suguira and colleagues⁷⁷ concluded that increased nasal resistance was also associated with nonacceptance of CPAP. Further, in patients with poor

CPAP compliance and nasal obstruction, lowering nasal resistance with surgical therapy has been shown to lower mean CPAP pressures and improve adherence.⁶⁹ Poirier and colleagues⁷⁸ recently showed that with careful selection of patients intolerant of CPAP therapy because of nasal obstruction, corrective nasal surgery could dramatically decrease obstructive nasal symptoms, increase CPAP adherence (0.5 hours per night before surgery to 5 hours per night after surgery; $P < .05$), and convert CPAP failures into successes.

SUMMARY

Nasal obstruction is common in both allergic rhinitis and sleep-disordered breathing patient populations. Allergic rhinitis and associated symptomatic nasal obstruction negatively affect sleep through a variety of mechanisms and may contribute to persistent symptoms and poor adherence with medical device therapy for sleep apnea. A history of sinonasal symptoms, particularly those that occur at night or in the supine position, is the cornerstone of the medical evaluation. Validated questionnaires are available to facilitate this process. A brief nasal examination with anterior rhinoscopy provides important information on the inflammatory and structural contributions to the nasal obstruction and helps to guide treatment. More advanced or refractory cases may benefit from otolaryngology consultation and additional radiographic or endoscopic evaluation.

Of the available medical therapy options, intranasal steroids currently have the most data supporting improvement in both sinonasal and sleep-related symptoms, although further investigation is needed. In patients with allergic rhinitis with anatomic contributions to nasal obstruction (particularly those who have failed medical therapy), surgical therapy to lower nasal resistance may improve snoring, subjective sleep quality, daytime alertness, disease-specific and general quality-of-life measures, as well as objective measures of sleep continuity and sleep architecture. Further research efforts would benefit from standardized measurement and outcomes variables as well as the power to carefully elucidate small subgroups of patients who would derive benefit from targeted therapies.

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