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NASAL CONGESTION AND ITS RELATIONSHIP WITH HYPOXIA: PATHOPHYSIOLOGICAL MECHANISMS, CLINICAL CORRELATES, AND THERAPEUTIC PERSPECTIVES

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SUMMARY

Nasal congestion is a common condition in which reduced nasal patency alters respiratory aerodynamics, increases upper airway resistance, and promotes a shift to mouth breathing, particularly during sleep. These changes impair ventilation–perfusion matching, decrease gas-exchange efficiency, and may lead to systemic or local hypoxia, especially in sleep-disordered breathing and in children who are obligate nasal breathers. Clinical evidence demonstrates that both chronic and acute nasal obstruction are associated with reduced SpO_2, increased intermittent hypoxemia, sleep disruption, and cognitive and behavioral consequences. Medical and surgical relief of obstruction improves nasal airflow, decreases the severity of hypoxic episodes, and enhances the effectiveness of sleep-disordered breathing treatment. Thus, maintaining nasal patency is a key component in the prevention and correction of hypoxia across diverse patient populations.

KEYWORDS: *nasal obstruction; nasal congestion; hypoxia; mouth breathing; obstructive sleep apnea.*

CONFLICT OF INTEREST. The authors declare no conflict of interest.

ЗАЛОЖЕННОСТЬ НОСА И ЕЕ СВЯЗЬ С ГИПОКСИЕЙ: ПАТОФИЗИОЛОГИЧЕСКИЕ МЕХАНИЗМЫ, КЛИНИЧЕСКИЕ КОРРЕЛЯТЫ И ТЕРАПЕВТИЧЕСКИЕ ПЕРСПЕКТИВЫ

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РЕЗЮМЕ

Заложенность носа – распространенное состояние, при котором снижение носовой проходимости нарушает аэродинамику дыхания, увеличивает сопротивление верхних дыхательных путей и способствует переходу на ротовое дыхание, особенно во сне. Эти изменения ведут к ухудшению вентиляционно-перфузионного соответствия, снижению эффективности газообмена и могут вызывать системную или локальную гипоксию, выраженную особенно при обструктивных нарушениях дыхания во сне и у детей, являющихся облигатными носовыми дыхателями. Клинические данные подтверждают, что как хроническая, так и острая носовая обструкция ассоциированы со снижением SpO_2, усилением интермиттирующей гипоксемии, нарушением сна, когнитивными и поведенческими эффектами. Медикаментозное и хирургическое устранение обструкции улучшает носовое дыхание, снижает выраженность гипоксических эпизодов и повышает эффективность лечения расстройств дыхания во сне. Таким образом, поддержание носовой проходимости является важным компонентом профилактики и коррекции гипоксии у различных категорий пациентов.

КЛЮЧЕВЫЕ СЛОВА: носовая обструкция, заложенность носа, гипоксия, ротовое дыхание, обструктивное апноэ сна.

КОНФЛИКТ ИНТЕРЕСОВ. *Авторы заявляют об отсутствии конфликта интересов.*

Introduction

Nasal congestion — a subjective sensation of "blocked" nasal passages with reduced airflow — is among the most frequent complaints that lead patients to seek care from otorhinolaryngologists and primary care physicians. It accompanies a wide range of conditions, from acute viral rhinitis to chronic allergic rhinitis and chronic rhinosinusitis with nasal polyps. Allergic rhinitis alone affects approximately 25% of the global population, and nasal congestion is often its most burdensome symptom. In addition to discomfort and reduced quality of life (e.g., impaired sleep and daytime performance), there is increasing awareness of the potential

systemic physiological consequences of nasal obstruction. In particular, there is justified concern that pronounced nasal congestion may reduce oxygen intake and cause tissue hypoxia in several clinical scenarios [1].

Hypoxia, defined as insufficient oxygen delivery to tissues, is a key pathophysiological factor in many diseases and may arise as a consequence of impaired breathing. Obstructive sleep apnea (OSA) illustrates how elevated upper airway resistance and airway collapse produce intermittent nocturnal hypoxemia, contributing to cardiovascular and cognitive comorbidities. Nasal obstruction is a modifiable factor that can exacerbate OSA severity in many patients. Epidemiological data show that habitual nighttime nasal congestion is associated with higher rates of snoring and OSA. In a population-based cohort study, individuals with persistent severe nighttime congestion had approximately a threefold higher likelihood of habitual snoring compared with those without congestion. OSA itself is highly prevalent (approximately 1 billion people worldwide), and its hallmark — recurrent hypoxemia — underlies many of its systemic consequences [2].

The purpose of this review is to analyze the relationship between nasal congestion and hypoxia. We examine the mechanisms through which nasal obstruction can reduce oxygenation — both systemically (hypoxemia) and locally (mucosal hypoxia) — along with clinical contexts in which this association is most significant. Particular attention is paid to the impact of nasal versus oral breathing on gas exchange, the role of nasal congestion in sleep-disordered breathing and intermittent hypoxia, the vulnerability of infants and children who rely on nasal respiration, and the potential benefits of relieving nasal obstruction for hypoxia-related outcomes. Synthesizing current evidence may help clinicians view nasal congestion not only as a local symptom but also as a factor with broader physiological implications.

Physiology of Nasal Breathing and Oxygenation

Under normal circumstances, humans predominantly breathe through the nose, and nasal airflow serves several essential physiological functions. The nasal passages warm, humidify, and filter inspired air, and they are a major source of nitric oxide (NO) produced in the paranasal sinuses. NO enters the lower airways during nasal inhalation and acts as an "aerocrine" mediator that enhances pulmonary oxygen uptake and reduces pulmonary vascular resistance. Experimental studies demonstrate that nasal breathing improves arterial oxygenation compared with oral breathing. Lundberg et al. showed that in healthy adults, transcutaneous oxygen tension ($tcPO_2$) was approximately 10% higher during nasal breathing, likely due to inhalation of endogenous NO and more favorable airflow distribution. In mechanically ventilated patients deprived of nasal airflow, reintroduction of NO-rich nasal air increased arterial oxygen tension (PaO_2) by 18% and reduced pulmonary vascular resistance by 11%, underscoring the direct contribution of nasal-derived NO to gas-exchange efficiency [3].

A second important aspect is airway resistance. While the nasal airway creates resistance, it supports physiologically optimal airflow patterns. During wakefulness, the difference in resistance between nasal and oral breathing may be modest, but the difference becomes pronounced during sleep. In the classical study by Fitzpatrick et al., healthy volunteers were evaluated during sleep under forced oral versus nasal breathing. Oral breathing produced substantially higher upper airway resistance (median ~12.4 cm $H_2O \cdot L^{-1} \cdot s^{-1}$) compared with nasal breathing (~5.2 cm $H_2O \cdot L^{-1} \cdot s^{-1}$) during stage 2 sleep. The greater collapsibility of the oropharynx with an open mouth accounted for most of this difference. The apnea–hypopnea index (AHI) was almost negligible during nasal breathing (~1.5 events/h) but reached pathological values during forced oral breathing (~43 events/h). This clearly demonstrated the mechanical advantage of nasal breathing in maintaining upper airway stability during sleep. Nasal breathing promotes physiologic jaw and tongue positioning (tongue against the palate), whereas mouth breathing causes posterior displacement of the tongue and airway narrowing. Thus, adequate nasal patency is crucial for stable nocturnal respiration without episodes of apnea and hypopnea [4].

In summary, the nasal passages play a central role in respiratory physiology by improving oxygenation (via NO-mediated effects on pulmonary hemodynamics) and stabilizing the upper airway. When nasal breathing is impaired, these advantages are lost. The next sections discuss how nasal congestion — by inducing mouth breathing or increasing resistance — can lead to measurable reductions in oxygenation.

Effects of Nasal Obstruction on Oxygenation and Breathing

Severe nasal obstruction (pronounced congestion or complete blockage) disrupts normal breathing mechanics. A frequent and immediate consequence is a shift to mouth breathing, which, as noted above, reduces ventilation efficiency and upper airway stability during sleep. Additionally, nasal obstruction may alter breathing parameters during wakefulness. Increased total airway resistance elevates the work of breathing, promoting hypoventilation: breathing becomes more shallow and less effective. As a result, blood oxygen levels (hypoxemia) may decrease, and $PaCO_2$ may rise (hypercapnia) if minute ventilation cannot be adequately maintained [5].

Clinical studies demonstrate the effect of acute and chronic nasal obstruction on gas exchange. In patients with long-standing nasal obstruction (septal deviation, polyposis, turbinate hypertrophy), baseline PaO_2 may be moderately reduced ("latent" hypoxemia) even in the absence of pulmonary disease. Sobh et al. evaluated 59 adults undergoing surgery for nasal obstruction (mostly septoplasty or polypectomy) and assessed arterial blood gases before and after surgery. Preoperatively, some patients exhibited low-normal PaO_2 and SpO_2. When postoperative nasal packing completely blocked nasal airflow, PaO_2 and SpO_2 decreased further, accompanied by a mild drop in pH (suggesting slight hypercapnia). After removal of the nasal packing (restored nasal breathing), oxygenation improved and exceeded preoperative levels. This reversible experiment directly demonstrates that nasal obstruction can induce hypoxemia, likely through hypoventilation and ventilation–perfusion mismatch.

Earlier studies reported similar findings. Öğretmenoğlu et al. showed that bilateral nasal packing in postoperative patients significantly reduced nocturnal oxygen saturation, whereas nasal airway tubes mitigated desaturation. Cassisi et al. (1971) showed that posterior nasal packing for epistaxis

significantly decreased PaO_2 in adults, with minimal changes in $PaCO_2$; hypoxemia resolved after removal of packing. Proposed mechanisms include reflex pathways (e.g., the "nasopulmonary reflex," reducing respiratory drive) and mechanical factors (mouth breathing with increased pharyngeal collapsibility). The resulting cascade is: nasal blockage → mouth breathing + reflex hypoventilation → ventilation–perfusion mismatch → hypoxemia [6, 7].

Local mucosal hypoxia of the nasal and sinus tissues is also significant. When sinus ventilation is impaired (as in chronic rhinosinusitis with polyps), oxygen tension in the sinus cavities decreases. Such hypoxia promotes inflammation by stabilizing hypoxia-inducible factor-1α (HIF-1α) and upregulating pro-inflammatory gene expression. The review by Zhong et al. (2022) highlights that hypoxic conditions in chronically inflamed sinonasal mucosa worsen epithelial dysfunction and polyp growth, creating a vicious cycle: obstruction → hypoxia → inflammation → further obstruction [8].

Individual variability should be noted. In a small study by Taasan et al., healthy volunteers underwent complete nasal occlusion during sleep assessment. Despite severe discomfort and forced oral breathing, some subjects did not exhibit significant desaturations. The authors suggested that young healthy adults can compensate effectively over short periods via oral breathing, and the small sample size (N=7) limits generalizability. Thus, short-term nasal obstruction in healthy subjects may be well tolerated, whereas patients with comorbidities or prolonged sleep are more prone to developing hypoxemia. Overall, the available evidence indicates that pronounced bilateral nasal obstruction adversely affects oxygenation, particularly during sleep or sedation, when compensatory mechanisms are diminished [9].

Nasal Congestion in Sleep-Disordered Breathing and Intermittent Hypoxia

The strongest link between nasal congestion and hypoxia is observed in sleep-disordered breathing — snoring and OSA. During sleep, muscle relaxation and the supine position predispose the airway to collapse; increased nasal resistance due to congestion further exacerbates this risk by promoting mouth breathing and upper airway instability.

Epidemiological and clinical studies confirm the association between reduced nasal patency and OSA. In the Wisconsin Sleep Cohort, individuals with chronic nighttime nasal congestion ("often" or "always") had a significantly higher risk of habitual snoring and probable undiagnosed OSA. A longitudinal analysis showed that subjects with severe nighttime congestion were nearly three times more likely to develop habitual snoring over five years. Allergic rhinitis — a common cause of chronic congestion — is associated with at least a twofold increase in OSA risk, primarily due to increased nasal resistance and mouth breathing during sleep. Chirakalwasan et al. showed that effective treatment of allergic rhinitis can moderately reduce OSA severity, underscoring the contributory role of nasal obstruction [10–12].

The mechanistic link is clear: nasal obstruction increases airway resistance and forces mouth breathing, which — as shown earlier — substantially increases the risk of obstructive events. Additionally, nasal obstruction reduces afferent stimulation from nasal airflow receptors that help maintain respiratory drive, potentially lowering ventilation during sleep transitions. Experiments with artificial nasal occlusion provide compelling evidence. Metes et al. demonstrated that nasal occlusion in healthy individuals induced snoring and apnea that were not present during normal nasal breathing. In patients with OSA, Lan et al. (2021) found that those with narrower or more resistant nasal passages spent more time in nocturnal hypoxemia, concluding that nasal obstruction is an "important factor" contributing to hypoxemia in moderate-to-severe OSA [11].

Intermittent hypoxia drives sympathetic activation, oxidative stress, inflammation, hypertension, cardiovascular disease, and cognitive decline. A newer metric — the hypoxic burden (area under the curve of oxygen desaturations during sleep) — has been shown to be a stronger predictor of cognitive impairment and cardiovascular outcomes than AHI alone. Huang et al. demonstrated that in OSA patients, higher hypoxic burden was associated with a significantly greater risk of mild cognitive impairment at comparable AHI values. This suggests that any factor worsening desaturation (including nasal congestion) may accelerate cognitive decline [13–16].

Improving nasal airflow can reduce OSA severity or improve disease control. Randomized trials show that intranasal corticosteroids in allergic rhinitis patients with OSA reduce AHI by 30–40% and improve subjective sleep quality and daytime alertness. Nasal surgery (septoplasty, turbinate reduction, polypectomy) often improves CPAP tolerance and may decrease apnea frequency in selected patients. Although nasal surgery rarely cures OSA, it provides clinically meaningful benefits, especially by improving CPAP adherence. McNicholas emphasized that variable obstruction (such as nighttime congestion or positional effects) may play a more important role in OSA physiology than fixed anatomical narrowing [13–16].

Oxidative stress is also relevant. Passali et al. (2025) reported elevated oxidative stress biomarkers in patients with isolated nasal obstruction (AHI <5) at levels comparable to those in OSA patients. This indicates that impaired nasal airflow alone may initiate systemic oxidative–inflammatory responses, supporting a continuum: isolated obstruction → snoring → OSA, united by hypoxia–reoxygenation cycles [15].

Thus, nasal congestion is both a risk factor for sleep-related hypoxemia and an exacerbating component in established OSA. Patients with chronic congestion and sleep symptoms should be evaluated for sleep-disordered breathing, and nasal pathology should be considered a treatment target to improve oxygenation, cognitive function, daytime alertness, and cardiovascular outcomes.

Special Populations: Pediatric Patients and the Impact of Nasal Obstruction

Infants and young children

Infants are particularly vulnerable to the consequences of nasal congestion. Newborns are predominantly nasal breathers in the first months of life. Due to anatomical and reflex factors, they have limited ability to switch effectively to oral breathing, especially during feeding. Even moderate

nasal obstruction can lead to respiratory distress, feeding difficulties, and hypoxic episodes. Trabalon and Schaal showed that forced oral breathing due to nasal obstruction significantly affects neonatal systemic adaptation and behavior. The clinical relevance is evident in bilateral choanal atresia — a congenital blockage of the posterior nasal airway. Affected infants are cyanotic and hypoxic at rest but improve during crying (temporary oral airflow), highlighting the critical role of nasal breathing for oxygenation in early life. Even less severe congestion (e.g., neonatal rhinitis or residual milk in the nasopharynx) may cause desaturation or apnea. Therefore, nasal hygiene (saline irrigation and secretion removal) is routinely recommended to prevent respiratory and hypoxic episodes in infants. Maintaining nasal patency during this period is essential to prevent hypoxemia and its consequences, including inadequate weight gain, impaired growth, and a theoretical contribution to sudden infant death risk in extreme cases [17].

Older children and adolescents

In preschool and school-aged children, chronic nasal obstruction is most commonly due to adenoid hypertrophy and/or allergic rhinitis. Such children often become chronic mouth breathers. Mouth breathing in childhood is associated with craniofacial abnormalities (long face, high-arched palate), sleep disturbances, and cognitive and behavioral impairments. Pediatric sleep-disordered breathing (often due to adenotonsillar hypertrophy) leads to intermittent hypoxia that adversely affects neurocognitive development and growth hormone secretion. Untreated pediatric OSA is associated with deficits in memory, attention, academic performance, and behavioral issues resembling ADHD. Even milder forms — primary snoring with mouth breathing — can negatively affect daytime function. In a cross-sectional study by Kuroishi et al., children with "mouth breathing syndrome" performed significantly worse on tests of working memory, reading comprehension, and arithmetic skills compared with nasal breathers [18].

Nocturnal hypoxemia is common in mouth-breathing children, even without full OSA. Allergic inflammation further disrupts sleep architecture. Parents frequently report snoring, noisy breathing, daytime sleepiness, and inattention. Many symptoms improve after adenotonsillectomy or effective allergy treatment [19].

Growth is another important aspect. Chronic hypoxemia from sleep-disordered breathing may suppress nocturnal growth hormone secretion. "Catch-up" growth is frequently observed after adenotonsillectomy. Similarly, restoring nasal patency may support adequate oxygenation required for optimal growth. Some observational studies show that treating allergic rhinitis improves not only sleep but also weight gain and growth metrics in children, likely by reducing breathing effort and hypoxic stress during sleep.

In adolescents, mouth breathing may persist and contribute to reduced tongue strength, impaired oral function, and lower cognitive performance. Masutomi et al. (2024) showed that adolescents who mouth breathe have reduced oral musculature performance and poorer cognitive scores compared with nasal breathers. Residual snoring or mild OSA is often present. Restoring nasal airflow — medically or orthodontically — remains important even at this age [19].

Thus, pediatric populations vividly demonstrate that nasal congestion can lead to hypoxia-associated problems, from acute respiratory compromise in infants to cognitive and growth impairments in older children. Early identification and treatment of nasal obstruction (medical, surgical, orthodontic) are essential to prevent these adverse outcomes.

Therapeutic Perspectives: Reducing Hypoxia by Relieving Nasal Obstruction

Given the evidence that nasal congestion contributes to hypoxemia and reduces ventilation efficiency, it is reasonable to assume that correcting obstruction may provide benefits beyond symptom relief — improving oxygenation and clinical outcomes. Therapeutic approaches include medical and surgical treatments, often combined.

Medical therapy targets reversible causes of congestion. In allergic rhinitis, intranasal corticosteroids are first-line agents; they reduce mucosal inflammation and edema. Improved nasal airway patency decreases mouth breathing and snoring. As noted above, randomized trials in children and adults with combined allergic rhinitis and OSA have shown that intranasal steroids produce moderate reductions in AHI and significant improvements in sleep quality and daytime alertness. Adjunctive therapies include antihistamines (for allergy), anticholinergic nasal sprays (for vasomotor rhinitis), and isotonic saline irrigations. Short-acting topical decongestants (oxymetazoline and others) provide rapid relief in acute rhinitis or during CPAP titration in congested patients but are limited by the risk of rhinitis medicamentosa. In OSA patients with pronounced nasal congestion, combining intranasal steroids with a decongestant improves CPAP tolerance and adherence — an important practical consideration, as optimizing nasal airflow helps disrupt the cycle of "nasal obstruction → CPAP intolerance → persistent hypoxia" [20].

Surgical interventions are indicated in structural abnormalities and chronic rhinosinusitis unresponsive to medical therapy. Septoplasty (often with turbinate reduction) significantly improves nasal airflow. Several studies report improvements in pulmonary function and oxygenation in patients with preexisting nasal obstruction following septoplasty. In one observational study, patients with septal deviation and snoring experienced improved minimum nocturnal oxygen saturation and reduced apnea frequency one month postoperatively. Endoscopic sinus surgery and polypectomy also relieve congestion and reduce local hypoxic inflammatory stimuli. Although nasal surgery alone rarely cures OSA, some patients — particularly those with mild disease where nasal obstruction is the major contributor — achieve clinically significant improvements. Most patients benefit indirectly through improved CPAP adherence.

New devices and technologies reflect the importance of nasal airflow. Nasal expiratory positive airway pressure (EPAP) devices are used for snoring and mild OSA and rely on nasal patency to generate positive pressure on exhalation. Their efficacy depends directly on unobstructed nasal passag-

es. High-flow nasal oxygen therapy, widely used in hospitals, also requires preserved nasal flow and can prevent hypoxemia in partial upper airway obstruction [20].

From a preventive standpoint, especially in pediatrics, early treatment of allergic rhinitis and timely adenoidectomy (when indicated) prevent long-term consequences of chronic hypoxia. Orthodontic interventions (e.g., rapid maxillary expansion) are used in children with high-arched palates and nasal narrowing to increase nasal cavity volume, which may improve nocturnal breathing and oxygenation.

Thus, targeted correction of nasal congestion should be considered part of the therapeutic strategy for patients with hypoxemia or sleep-disordered breathing secondary to nasal obstruction. In OSA, clinicians should maintain a low threshold for active nasal treatment: simple measures (daily intranasal steroids, allergen control) can reduce apnea frequency and increase minimum saturation levels. For structural obstruction, timely surgical correction is indicated — not only for subjective comfort, but also to reduce the contribution to systemic hypoxia. Relieving nasal congestion may improve cognitive function, daytime alertness, and blood pressure control by reducing intermittent hypoxia and sympathetic activation.

Conclusion

Nasal congestion and hypoxia are closely interconnected: reduced nasal patency increases airway resistance, promotes mouth breathing, and raises the likelihood of upper airway collapse during sleep, leading to intermittent hypoxia, sympathetic activation, and oxidative stress. Infants and children are especially vulnerable, as even moderate obstruction may cause desaturation, sleep disruption, cognitive impairment, and growth disturbances. Clinical evidence shows that medical and surgical correction of nasal obstruction improves oxygenation, reduces apnea frequency, and enhances sleep quality, although the effect depends on the severity of obstruction, anatomical factors, and comorbid conditions. Gaps remain in defining threshold levels of nasal resistance that produce clinically significant hypoxia and in evaluating the long-term impact of treating chronic nasal congestion on OSA risk and cardiovascular outcomes.

References

1. *Stewart MG, Ferguson BJ, Fromer L. Epidemiology and burden of nasal congestion. Int J Gen Med. 2010;3:37–45. https://doi.org/10.2147/ijgm.s8077.*
2. *Benjafield AV, Ayas NT, Eastwood PR, et al. Estimation of the global prevalence and burden of obstructive sleep apnoea: a literature-based analysis. Lancet Respir Med. 2019;7(8):687–698. https://doi.org/10.1016/S2213-2600(19)30198-5.*
3. *Lundberg JO, Settergren G, Gelinder S, et al. Inhalation of nasally derived nitric oxide modulates pulmonary function in humans. Acta Physiol Scand. 1996;158(4):343–347. https://doi.org/10.1046/j.1365-201X.1996.557321000.x.*
4. *Fitzpatrick MF, McLean H, Urton AM, et al. Effect of nasal or oral breathing route on upper airway resistance during sleep. Eur Respir J. 2003;22(5):827–832. https://doi.org/10.1183/09031936.03.00047903.*
5. *Sobh E, Elhussieny F, Ismail T. Elimination of nasal obstruction improves pulmonary functions and oxygenation. Egypt J Bronchol. 2021;15:32. https://doi.org/10.1186/s43168-021-00079-6.*
6. *Öğretmenoğlu O, Yılmaz T, Rahimi K, et al. The effect on arterial blood gases and heart rate of bilateral nasal packing. Eur Arch Otorhinolaryngol. 2002;259(2):63–66. https://doi.org/10.1007/s00405-001-0422-1.*
7. *Cassisi NJ, Biller HF, Ogura JH. Changes in arterial oxygen tension and pulmonary mechanics with the use of posterior packing in epistaxis: a preliminary report. Laryngoscope. 1971;81(8):1261–1266. https://doi.org/10.1288/00005537-197108000-00009.*
8. *Zhong B, Seah JJ, Liu F, et al. The role of hypoxia in the pathophysiology of chronic rhinosinusitis. Allergy. 2022;77(11):3217–3232. https://doi.org/10.1111/all.15384.*
9. *Taasan V, Wynne JW, Cassisi N, Block AJ. The effect of nasal packing on sleep-disordered breathing and nocturnal oxygen desaturation. Laryngoscope. 1981;91(7):1163–1172. https://doi.org/10.1288/00005537-197107000-00015*
10. *Young T, Finn L, Palta M. Chronic nasal congestion at night is a risk factor for snoring in a population-based cohort study. Arch Intern Med. 2001;161(12):1514–1519. https://doi.org/10.1001/archinte.161.12.1514.*
11. *Lan MC, Lan MY, Kuan EC, et al. Nasal obstruction as a potential factor contributing to hypoxemia in obstructive sleep apnea. Nat Sci Sleep. 2021;13:55–62. https://doi.org/10.2147/NSS.S288618.*
12. *Chirakalwasan N, Ruxrungtham K. The linkage of allergic rhinitis and obstructive sleep apnea. Asian Pac J Allergy Immunol. 2014;32(4):276–286. PMID: 25543037.*
13. *McNicholas WT. The nose and OSA: variable nasal obstruction may be more important in pathophysiology than fixed obstruction. Eur Respir J. 2008;32(1):3–8. https://doi.org/10.1183/09031936.00050208.*
14. *Metes A, Ohki M, Cole P, et al. Snoring, apnea and nasal occlusion. Chest. 1996;109(3):673–679. https://doi.org/10.1378/chest.109.3.673.*
15. *Passali D, Santantonio M, Passali GC, et al. Oxidative stress in patients with nasal respiratory impairment and OSAS. Sleep Breath. 2025;29(3):188. https://doi.org/10.1007/s11325-025-03361-9.*
16. *Huang X, Zhang Z, Lan X, et al. The association between hypoxic burden and the risk of cognitive impairment in patients with obstructive sleep apnea. Sleep. 2025;48(3):zsae269. https://doi.org/10.1093/sleep/zsae269.*
17. *Trabalon M, Schaal B. It takes a mouth to eat and a nose to breathe: abnormal oral respiration affects neonates' oral competence and systemic adaptation. Int J Pediatr. 2012;2012:207605. https://doi.org/10.1155/2012/207605.*
18. *Kuroishi RC, Garcia RB, Valera FC, et al. Deficits in working memory, reading comprehension and arithmetic skills in children with mouth breathing syndrome: analytical cross-sectional study. Sao Paulo Med J. 2015;133(2):78–83. https://doi.org/10.1590/1516-3180.2013.7630011.*
19. *Masutomi Y, Goto T, Ichikawa T. Mouth breathing reduces oral function in adolescence. Sci Rep. 2024;14:3810. https://doi.org/10.1038/s41598-024-54328-x.*
20. *Kiely JL, Nolan P, McNicholas WT. Intranasal corticosteroid therapy for obstructive sleep apnoea in patients with co-existing rhinitis. Thorax. 2004;59(1):50–55. https://doi.org/10.1136/thorax.59.1.50.*

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