# 13 Breath of Life: Nasal Airway Obstruction in Sleep Apnea Patients

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## INTRODUCTION

Nasal airway obstruction (NAO) frequently occurs in patients with obstructive sleep apnea (OSA) and can influence the symptoms and severity of OSA to varying degrees. An increase in nasal airway resistance (NAR) can lead to mouth breathing.

The earliest literature linking mouth breathing to sleep-related health issues comes from the work of American traveler George Catlin, who discussed this in his 1862 book "The Breath of Life" (later retitled "Shut Your Mouth and Save Your Life").[1] Catlin posited that Native Americans were healthier than Europeans because they slept on their backs and breathed through their noses instead of their mouths. However, his contemporaries did not widely accept his observations, and his findings did not gain significant recognition in subsequent years.

It wasn't until 120 years later that researchers began to reconsider the importance of nasal breathing. In 1981, Taasan V et al.[2] and Olsen KD et al.,[3] in two separate studies, found that artificial nasal obstruction using nasal packs worsened polysomnographic (PSG) variables in sleep disordered breathing (SDB) patients and could even induce apneas and hypopneas to varying degrees in healthy individuals. Following these observations, in 1997 the first population-based study came from the University of Wisconsin, where they found that nasal obstruction was an independent contributor to OSA.[4]

Further studies have shown that nasal obstruction increases pharyngeal airway collapse and PSG variables significantly. These studies have found a positive correlation between NAO, NAR and Apnea-Hypopnea Index (AHI) and oxygen desaturation index (ODI) even in nonobese subjects.[5–8]

## PATHOPHYSIOLOGY

Considering Poiseuille's law, airway resistance is proportional to the length of the airway and inversely proportional to the fourth power of the radius.[9] Since the nose

is the primary entry point for inhaled air, any nasal pathologies, causing even a very small change in the size of the nasal airway can lead to a significant increase in NAR. Given that the nose accounts for more than two-thirds of total upper airway resistance and is the primary route for breathing during sleep, it likely plays a significant role in the pathophysiology and mechanisms of sleep disordered breathing.[10,11]

Various pathophysiological mechanisms have been proposed to explain the impact of nasal obstruction on sleep-disordered breathing. These include:

## STARLING RESISTOR MODEL

Upper airway is like a starling resistor, a collapsible tube bound on either end by fixed segments (nasal passage and trachea) - See Figure 1. Obstruction at the inlet (i.e. the nasal airway) produces collapsing forces that are manifested downstream in the collapsible segment, the pharynx.[12]

During inspiration, the negative pressure generated in the intrapleural space is transmitted along the entire upper airway, including the pharynx, retroglossal and retropalatal space. When the nasal airway is obstructed, lungs must generate more negative pressure for sucking in the same air to maintain stable airflow. This creates more negative pressure in the pharynx, causing more collapse of the pliable pharyngeal airway.[12,13]

## UNSTABLE MOUTH BREATHING

As nasal resistance increases, the maladaptive mouth breathing fraction also rises. In healthy individuals with normal nasal resistance, only about 4% of inhaled

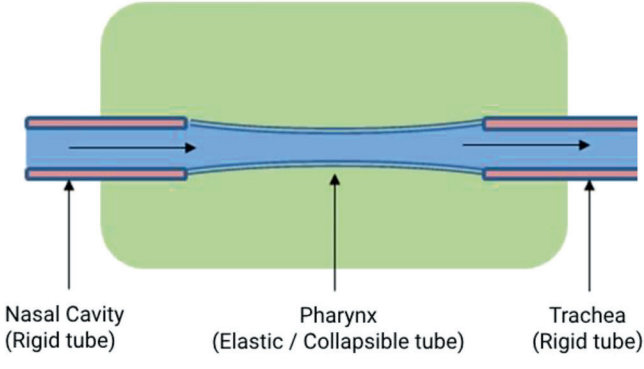


**Figure 1**. Starling resistor model. When the inward pressure increases, the intrapharyngeal pressure decreases and the pharyngeal lumen collapses.

ventilation during sleep is through the mouth. Mouth breathing is defined as occurring when more than 30% of airflow passes through the oral cavity.[14]

Nasal breathing serves essential physiological functions such as humidification and filtration and is the preferred route for breathing during sleep. Bypassing normal nasal physiology through unfavorable and unstable oral breathing can negatively impact sleep quality.[15]

When the mouth opens, the mandible moves backward and downward, pushing the tongue into the pharynx. On an average, a 1.5 cm opening of the mouth, measured as the distance between the upper and lower incisors, results in a posterior movement of the mandible by 1 cm, leading to a 2.5-fold increase in the upper airway resistance.[14] In addition, the length and tension of the pharyngeal muscles also decreases during mouth opening, leading to increased collapse of the pharyngeal walls.[15]

As the cross-sectional area of the pharyngeal lumen decreases, airflow velocity increases and intraluminal pressure decreases to maintain equivalent flow due to venturi effect. Decreased intraluminal pressure potentiates further risk of pharyngeal collapse during inspiration.[16]

## NASAL SENSORY INPUT AND NEURAL REFLEX PATHWAYS

The genioglossus muscle (GG), an extrinsic muscle of the tongue, is the primary muscle responsible for protruding the tongue. It acts as a dilator of the upper airway by controlling the tongue's position and moving the hyoid bone forward and upwards.[17,18] Studies have shown that pressure sensors in the upper airway reflexively modulate genioglossus muscle activity.[19] The thermoreceptors in the nasal mucosa also have afferent relations to the genioglossus muscle. Application of topical anesthetic agents to the nasal cavity resulted in the abolishment of these trigemino-hypoglossal reflex pathways and caused apneas and hypopneas in subjects.[20,21]

It is also studied that GG muscle activity is inhibited by both passive and active jaw openings through a reflex activated by the afferents from the temporomandibular joint.[22] This further worsens the unstable mouth breathing.

McNicholas et al. has directly measured ventilation during conditions of altered nasal airflow in humans and found that a neural feedback mechanism from the airflow sensors in the nose affects the minute ventilation. It was noted that minute ventilation and pharyngeal muscle tone are higher in nasal breathing compared with oral breathing.[23]

## ROLE OF NITRIC OXIDE

Nitric oxide (NO) is abundantly produced in the nasal and sinus epithelium, with its production reliant on nasal airflow.[24] This NO is transported to the lungs with inspired air, aiding in the reduction of ventilation-perfusion mismatching.[25] When NO levels are reduced, it can lead to a failure in maintaining oropharyngeal muscle tone, cause ventilation-perfusion mismatch, and negatively impact breathing rate and sleep regulation.[25,26] Although this pathway is likely significant in the development of OSA, the exact mechanisms are not yet fully understood.

## POSITIVE POSTURE REACTION AND NOCTURNAL NASAL CONGESTION

The condition of experiencing nasal obstruction while lying down is a normal physiology. This was first described by Rundcrantz in 1969.[5,27] The condition worsens nasal obstruction in patients with OSA. The underlying physiology of nasal congestion on decubitus remains unclear. A review of the literature has identified three disorders that may contribute to the occurrence of nasal congestion. These disorders, individually or in concert with one another, could influence the development of nasal congestion:

1. Venous stasis: In the erect position, the pressure within the jugular vein is nearly zero, but it increases from 4.5 mmHg to 11 mmHg when lying down.[28]
2. Baroreceptor mediated Reflex: This hypothesis suggests that a neural reflex mediated by pressure receptors induces ipsilateral nasal congestion and contralateral decongestion when lying down.[29]
3. Sympathetic Withdrawal and Parasympathetic Preponderance: This hypothesis suggests that the predominance of parasympathetic autonomic nervous system activities during a positional change from sitting to a supine position results in vascular dilation of the nasal mucosa.[30]

Various large population-based studies have shown that nocturnal nasal congestion is a strong independent risk factor for snoring and SDB.[31,32] Recently Värendh et al. in their study among 810 untreated OSA patients observed that nasal congestion happened in two-thirds of OSA patients during sleep. These nasal obstructions were associated with increased daytime sleepiness and reduced mental health quality-of-life score.[33]

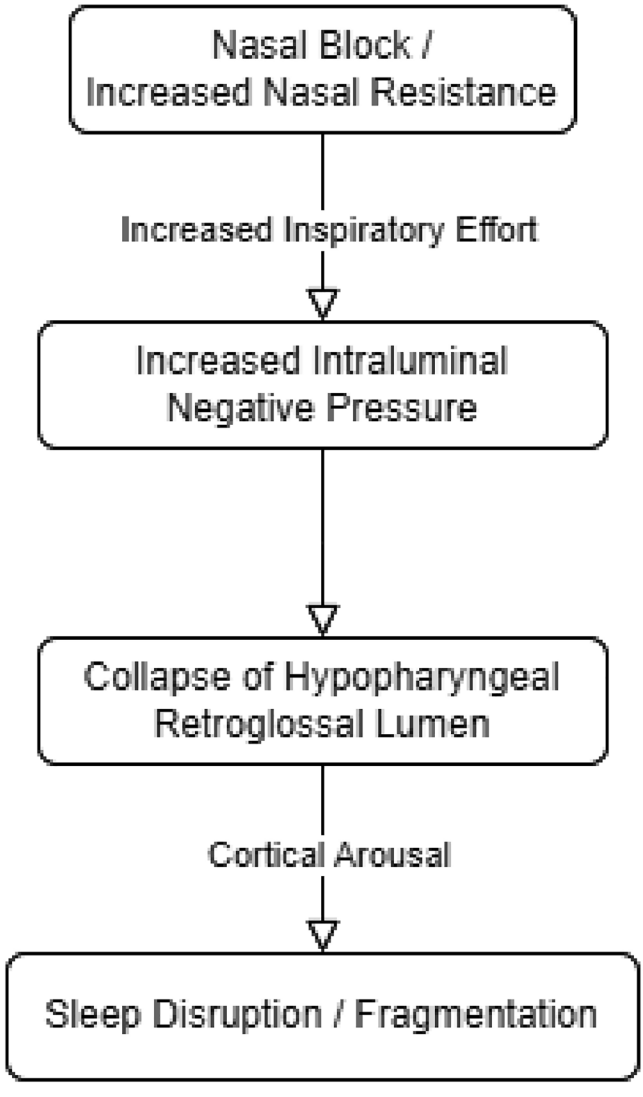


**Figure 2**. Flow diagram depicting how increase in nasal resistance causes micro sleep fragmentation.

## EFFECTS OF NASAL TREATMENT

As discussed earlier, nasal obstructions caused by either anatomical abnormalities or inflammatory diseases (Table 1) are linked to a higher risk of snoring, apnea, and hypopnea episodes, reduced sleep quality, and increased daytime sleepiness. But the effectiveness of nasal treatments for OSA remains controversial. While numerous studies have shown that nasal treatments can improve subjective

**Table 1**. Anatomical and inflammatory conditions which can cause change in nasal cross sectional area.

| Anatomical Causes | Inflammatory Conditions |
|---|---|
| Deviated Nasal septum | Allergic rhinitis |
| Nasal deformities/injuries | Nonallergic (vasomotor) rhinitis |
| Inferior turbinate hypertrophy | Viral/Bacterial/Fungal rhinosinusitis |
| Middle turbinate concha bullosa | Rhinitis of pregnancy |
| Nasal polyps | Rhinitis medicamentosa |
| Inverted papilloma | Atrophic rhinitis |
| Neoplasm | Wegener granulomatosis |
| Septal perforation | Sarcoidosis |
| Nasal synechia | Medications (e.g., β-blockers, estrogens) |
| Nasal valve collapse | Tobacco use |
| Choanal atresia | |
| Pyriform aperture stenosis | |
| Adenoid hypertrophy | |
| Nasopharyngeal cysts/tumors | |
| Meningocele/encephalocele | |
| Foreign body | |

symptoms related to OSA and enhance quality of life (QOL), their impact on objective parameters like the AHI has been inconsistent.

The prevalence of nasal obstruction is not well documented; however, it is estimated that more than 30% of patients with OSA have nasal diseases.[34] In a recent study, it was noted that when compared to healthy controls, OSA patients have increased prevalence of sinonasal disease as measured on a visual analog scale and using the SNOT-20 questionnaire.[35] Krakow et al, conducted a study among large sample of sleep center patients and observed that the prevalence of non allergic rhinitis in general adult population is estimated to be 7 to 19 %, but it was observed to be in 45% of adult OSA patients.[35] Two third of Non Allergic Rhinitis patients experienced nasal obstruction symptoms in night. It was also studied that sleep quality was poor in OSA patients with chronic rhinosinusitis, and Non Allergic Rhinitis.[35–37]

## NASAL SURGERY IN OSA

Regarding the role of nasal surgeries in obstructive sleep apnea patients, Studies have given controversial results. This is because data sets are polluted by heterogeneous patient populations with varying severities and etiologies of OSA and nasal pathologies, inconsistent surgical methods, criteria for patient selection, and small sample sizes.

In addition, some studies have shown the poor correlation between subjective symptoms of nasal obstruction and objective nasal resistance or volume which also affects the results. Though nasal surgeries alone fail to reduce AHI, all studies till date have shown that it reduces excessive daytime sleepiness (EDS). This is an important finding, as AHI often does not correlate with subjective symptoms of OSA, and studies have demonstrated that patients with OSA and EDS are at significantly higher risk for cardiovascular and metabolic disease.[38–40]

Surgical interventions to address nasal obstruction include, but are not limited to, nasal valve correction, septoplasty, turbinate reduction, functional rhinoplasty, sinus surgery and adenoidectomy.

## Assists CPAP Usage

CPAP therapy is considered the first-line therapy for moderate-severe OSA. CPAP is highly efficacious but the adherence rate is not high. Nasal obstruction is a common complaint among CPAP defaulters, with an estimated prevalence of 25% to 45%.[34]

Nasal surgery reduces nasal resistance and lowers the required optimal positive airway pressure. It also alleviates daytime sleepiness, enhances overall quality of life and subjective sleep quality, improves the lowest oxygen saturation, and reduces the apnea-hypopnea duration.[41–43] This, in turn, increases the acceptance of CPAP.

Sugiura et al. demonstrated that for each 0.1 Pa/cm³/s increase in nasal resistance, the odds ratio of non-acceptance of CPAP increases 1.48-fold.[41] Nakata et al., demonstrated that performing septoplasty and inferior turbinate reduction in CPAP non-adherent patients resulted in a reduction in nasal resistance from 0.57 to 0.16 Pa/cm³/s. Postoperatively, all patients became CPAP adherent.[43]

However, despite these improvements in various PSG parameters, nasal surgery alone does not significantly reduce the AHI.[41–43] Kempfle et al has shown that nasal surgery is a cost-effective strategy to improve CPAP compliance in OSA patients with nasal obstruction.[44]

## Improves Oral Appliance Usage

Nasal airway resistance is one of the most significant predictors of oral appliance response. Higher levels of NAR can negatively impact treatment outcomes with mandibular advancement devices (MAD).[45,46] Since nasal surgery reduces NAR, it may enhance the effectiveness of oral appliance therapy in individuals with nasal obstruction.

## Improves Outcomes in Multilevel Surgeries

A large multicentric study conducted across nine tertiary clinical centers in eight countries evaluated the role of nasal surgery as part of multi-level surgeries for OSA. The study divided patients into two groups: those who underwent nasal surgery (nose group) and those who did not (no nose group). The results showed that patients in the nose group experienced a greater percentage change in AHI and daytime sleepiness. The success rate of surgery was 68.2% for the nose group, compared to 55.0% for the no nose group.[47]

## Types of Nasal Surgeries

The detailed discussion of nasal surgical procedures and their steps is beyond the scope of this chapter. However, the most common nasal surgeries performed on sleep apnea patients include:

- Septal Surgery - septoplasty, septorhinoplasty etc.
- Turbinate reduction, which can involve volumetric reduction using radiofrequency devices or microdebrider-assisted reduction.
- Nasal valve surgeries including piriform plasty.
- Endoscopic sinus surgeries, particularly in cases of associated rhinosinusitis.
- Expansion surgeries - Distraction osteogenesis maxillary expansion, posterior palatal expansion via subnasal endoscopy (2PENN), Endoscopically-assisted surgical expansion (EASE) etc.
- Adenoidectomy.

## Who Benefits the Most with Nasal Surgery?

The effectiveness of nasal surgery in treating sleep apnea is influenced by several patient-specific factors. Addressing these factors pre-operatively can improve surgical success rates.

Morinaga et al. observed that nasal surgery is more effective in patients with a high-positioned soft palate and/or a wide retroglossal space. Conversely, a low-positioned soft palate, elevated modified Mallampati score (Friedman tongue position, FTP), and a narrow retroglossal space are associated with less significant reductions in AHI after nasal surgery.[48] Similarly, Li et al. found that AHI reduction occurred after nasal surgery alone in OSA patients with a lower body mass index and lower tongue positions (FTP I or II).[49] Kim et al. noted significant reductions in AHI in patients who underwent isolated nasal surgery for sleep apnea and had moderate to severe nasal obstruction based on preoperative questionnaire scores.[50]

# NON SURGICAL/MEDICAL MANAGEMENT OF NOSE IN OSA

## Intranasal Corticosteroids

Research has demonstrated that a 4-week treatment with intranasal fluticasone significantly reduces the median AHI and improves oxygen saturation nadir in patients with inflammatory nasal airway compared to a placebo.[51,52] The overall literature indicates that intranasal steroids (INS) have a modest positive therapeutic effect on OSA in patients with concurrent rhinitis.

## Nasal Decongestants

Compared to a placebo, sympathomimetic nasal decongestants (such as oxymetazoline and xylometazoline) significantly increased REM sleep, reduced the arousal index and AHI, and improved oxygen saturation and the lowest oxygen saturation during sleep (LSAT).[53,54] These findings indicate that pharmacological enhancement of nasal patency in OSA patients can have positive effects on the severity of OSA and on sleep architecture.

## Nasal Dilators

In 2016, Camacho et al. conducted a meta-analysis of 14 studies on nasal dilators, both external and internal, involving 147 subjects with OSA. This remains the only meta-analysis on the role of nasal dilators. The study found no significant changes in AHI, LSAT, or daytime sleepiness with the use of nasal splints. However, they did observe that nasal dilators help reduce CPAP pressure, which can improve adherence.[55]

# CONCLUSION

Patients with obstructive sleep apnea frequently experience nasal obstruction. The relationship between nasal obstruction and OSA is complex, involving several pathophysiologic connections. These include the potential risk of increased pharyngeal collapse due to high nasal resistance, a higher likelihood of mouth breathing when nasal obstruction is present, and the blunting of the nasal ventilatory reflex caused by decreased nasal airflow. This complexity necessitates an individualized approach tailored to each patient when considering the nasal airway.

Decisions to treat nasal obstruction in OSA patients should be based on clinical examination and shared decision-making regarding therapy goals. The literature supports using steroid nasal sprays as a first-line treatment for patients with OSA

and nasal obstruction, as well as for those who are noncompliant with CPAP due to nasal obstruction. When medical management is insufficient, nasal surgery can be considered to improve subjective symptoms and sleep quality, including CPAP adherence. While some patients may benefit from nasal surgery alone, it should generally be viewed as an adjunct to other medical and surgical treatments. Further research is needed to better understand the predictors of surgical success and OSA improvement.

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