

Role of Nose in Sleep-Disordered Breathing

Chan-Soon Park, MD, PhD

Department of Otorhinolaryngology-Head and Neck Surgery, College of Medicine, The Catholic University of Korea, Suwon, Korea

The detailed pathophysiology and mechanisms of sleep-disordered breathing are not completely understood and the role of nose in sleep and obstructive sleep apnea has been still controversial. Considering that nasal obstruction has effects on anatomical factor, neuromuscular factor, and respiratory factor, nose may modulate upper airway collapsibility through anatomical, neuromuscular, and respiratory factors and play an important role of the pathogenesis of obstructive sleep apnea. **Sleep Med Res 2014;5(1):1-4**

Key Words Nose, Sleep, Airway resistance.

INTRODUCTION

Sleep-disordered breathing (SDB) includes a wide range of breathing difficulties during sleep from primary snoring to obstructive sleep apnea (OSA) and its estimated prevalence is from 0.9% to 13% in children.¹

Repeated partial (flow limitation, hypopnea) or complete (apnea) airway obstruction and increased respiratory effort are usually observed in subjects with SDB and these disrupted breathing cause adverse effects on body, growth, academic performance, et al.

The prevalence of habitual snoring (mild form of SDB) has been reported to be 3 to 6 times higher than that of OSA. Although habitual snoring does not always imply the presence of OSA, habitual snoring itself is also related to a variety of adverse effects on children.²

Therefore, it can be said that undisrupted breathing during sleep is very essential for physical and mental health in children and any factors to negatively affect sleep can be negative factors of physical and mental health in children.

Unfortunately, to date, the exact pathophysiology and mechanisms of SDB have not been completely understood and the role of nose in SDB has been much less understood.

The nose accounts for more than 50% of the total resistance of the upper airway and nasal breathing serves important physiological functions, including humidification, heating and filtration.³⁻⁶

In addition to physiologic role of nose, it must be noted that normal respiratory route during sleep is nose and mouth breathing by nasal obstruction can make mandible, hyoid bone downward and backward and finally upper airway narrowed.

Therefore, any conditions to affect normal respiratory route during sleep (nasal breathing) can make breathing during sleep difficult and strenuous, and finally have negative effects on sleep and daily life.

Although our understanding of sleep physiology and the effects of nasal and oral breathing on sleep apneas and total airway resistance has progressed over the last decade, the results of previous studies have been inconsistent. This review highlights the role of nose on physiology of sleep, sleep apnea and SDB.

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Correspondence

Chan-Soon Park, MD, PhD

Department of Otorhinolaryngology-Head and Neck Surgery, St. Vincent's Hospital, College of Medicine,

The Catholic University of Korea, 93 Jungbu-daero, Paldal-gu, Suwon 442-723, Korea

Tel +82-31-249-8304

Fax +82-31-248-5520

E-mail pcs0112@catholic.ac.kr

ANATOMICAL EFFECTS ON NASAL BREATHING DURING SLEEP

The first contributing anatomical factor is postural change, which can contribute to nasal obstruction. Nasal obstruction can be acutely exacerbated by postural reflex mechanisms as well as the changes of hydrostatic pressure on nasal venous circulation.⁷ Kase et al.⁸ performed the nasal response to the supine position. When healthy individuals were placed in the supine position from sitting, there was notable narrowing of nasal cavities, significantly in the more congested side.⁸ Lal et al.⁹ also performed the study used acoustic rhinometry to assess changes in nasal patency after alteration in posture. They demonstrated the change of nasal cavity volumes by acoustic rhinometry after alterations in posture. Another study investigated the relationships between nasal resistance, nasal volumes, and selected sleep parameters using nasal measurements performed in both seated and supine positions.¹⁰ They found a relationship between nasal resistances measured in the supine position and sleep parameters in the non-obese patients.

The second contributing anatomical factor is nasal resistance. Nasal obstruction may predispose to upper airway collapse. According to Poiseuille's law, resistance to airflow varies with the fourth power of the radius. This law suggests that even small obstructed lesions in the nose would have a large effect on overall respiration. Increasing nasal resistance results in much more negative oropharyngeal pressure during inspiration. In previous interventional studies, nasal obstruction was shown to induce apnea and arousal from sleep.¹¹⁻¹³ Several epidemiologic data suggested that nasal obstruction was potential risk factors for obstructive sleep apnea syndrome (OSAS).^{14,15} Young et al.¹⁶ found that nasal obstruction was an independent contributor to OSAS. Blakely and Mahowald¹⁷ found that nasal resistance was higher in OSAS patients than in the control group.

NEUROMUSCULAR EFFECTS ON NASAL BREATHING DURING SLEEP

The upper airway has both passive and active mechanisms for preventing narrowing or occlusion of the passage. Passive mechanisms include the static size, composition, and shape of the airway and soft tissues, and they govern the predisposition to collapse.¹⁸ Active components derive from dynamic respiratory cycle related changes, and they include central drives and tissue deformation, and muscle activity which produced by reflex.

However, it should be addressed that upper airway lacks a fixed rigid structural support, especially oropharynx and hypopharynx. According to the balance of pressure concept,^{19,20} occlusion of upper airway occurs when the P_{crit} becomes greater

than the intraluminal pressure, resulting in a transmural pressure of zero. In normal subjects, P_{crit} is around $-25 \text{ cmH}_2\text{O}$ and P_{close} around $-7.4 \text{ cmH}_2\text{O}$. This difference between P_{crit} and P_{close} may be done by the neuromuscular factor to prevent upper airway collapse during sleep.²¹ The P_{close} can be understood to be a pressure to cause airway collapse without neuromuscular activity whereas the P_{crit} can be said a sum of P_{close} and additional pressure by active neuromuscular reflex activity to prevent upper airway collapse. Patients with OSA have less negative value of P_{crit} and P_{close} , around $-5 \text{ cmH}_2\text{O}$ and $-2 \text{ cmH}_2\text{O}$. Through these facts, I presume that patient with OSA has less effective neuromuscular compensation. Current data suggests that patients without OSA are more sensitive to the negative pressure and effectively control pharyngeal dilator muscle activity during sleep than patients with OSA and are better able to compensate for their deficient anatomy during sleep.²² Even though the basic neuromuscular mechanisms driving this compensation are not well understood, there are some papers to be read for understanding neuromuscular factors. The increased upper airway resistance leads to reflex activation of negative pressure mechanoreceptors located in the larynx, resulting in increased activity in a number of upper airway muscles including the genioglossus and tensor palatine.²³⁻²⁵ The activity of genioglossus is also contributed to the compensation. Genioglossus activity is modulated locally by input from mechanoreceptors in the airway. Negative intraluminal pressure during inspiration results in activation of these mechanoreceptors and reflex activation of the genioglossus muscle for compensatory airway dilatation. Central nervous system controls the activity of genioglossus muscle via the respiratory pattern generator in the medulla and chemoreceptor control will influence the tonic behavior of the genioglossus and thus have some contribution to airway collapsibility.^{26,27}

Up to date, there have been only a few studies on nasal mechanoreceptor related to upper airway compensation, which showed indirect evidences with which I could assume nasal role in sleep-related breathing disorders. In previous animal study, some mechanoreceptors responded to nasal occlusion or persistent negative pressure in the nose. Sekizawa and Tsubone²⁸ reported existence nasal mechanoreceptors in guinea pigs, and some of these mechanoreceptors of nasal mucosa had a relatively low pressure threshold which could be stimulated by normal nasal breathing (mean, -1.41 kPa). In another study, a large number of receptors within the nasal cavity responding to the air jet stimulus were found in the ethmoidal nerve.²⁹ White et al.³⁰ tested the hypothesis that disordered breathing during sleep could result from loss of neuronal input to respiration from receptors located in the nose and proved that nasal anesthesia increased disordered breathing events in human. These results suggest that nasal receptors responsive to air flow may be important in maintaining breathing rhythmicity and preventing airway collapse during sleep. In nasal obstructed sub-

jects, diminished PCO₂ stimulus combined with depressed behavioral activity may activate the nasopulmonary reflex, wherein abnormal nasal trigeminal nerve stimulation decreases downstream ventilation.^{31,32}

When cutaneous supraorbital nerve [cranial nerve (CN) V1] was stimulated, there was no response in the genioglossal muscle. Mucosal lingual nerve (CN V3) stimulation elicited a single, short latency response in the genioglossal muscle.³³ Unfortunately, there was no report about the relationship between mucosal maxillary nerve (CN V2) and hypoglossal muscle. If I could prove relationship between mucosal maxillary nerve and genioglossal muscle, I could explain more easily the neuromuscular role of nose on maintaining upper airway caliber during sleep.

RESPIRATORY EFFECTS ON NASAL BREATHING DURING SLEEP

When I explain respiratory factors, starling resistor model is usually used, which divides upper airway into three segments, non-collapsible nasal and tracheal segments and collapsible pharyngeal segment.³⁴ Airway keeps patent mainly by pharyngeal dilator activity and, partly, lung expansion cause caudal traction of the airway which stretch and give more tension to the airway. If the balance between airway dilating and collapsing forces is disturbed, airway will collapse.²⁶ However classic starling resistor models and traditional flow limitation theory are not exactly applicable to the upper airway. Nasal cavity is not a passive tube as described in starling resistor model.

The Venturi effect should be considered; the velocity of the fluid increases as the cross sectional area decreases, with the static pressure correspondingly decreasing. If the Venturi effect is applied to nasal cavity, narrowed nasal passage can cause increase velocity of air flow, decrease static pressure, which might stimulate nasal mechanoreceptor and modulate collapsibility of the upper airway through a reflex unproved until now.

One more thing to be considered is mouth breathing. Nasal obstruction could induce mouth breathing, which moves the jaw and hyoid bone (including tongue) downward and backward. Therefore, mouth breathing can finally make upper airway narrowed, which increase intraluminal negative pressure and increases airway resistance, causing collapse.³¹

THE EFFECT OF NASAL OBSTRUCTION ON NASAL CONTINUOUS POSITIVE AIRWAY PRESSURE COMPLIANCE

Despite above mentioned relationship between nasal obstruction and SDB, the therapeutic effect of nasal surgery on

SDB remains inconsistent. There have been some reports that nasal surgery improved snoring and obstructive sleep apnea.^{8,35,36} Whereas there have also been some reports that nasal surgery is not supported as solitary intervention of OSA treatment.^{37,38}

Since Sullivan et al.³⁹ first described nasal continuous positive airway pressure (CPAP), CPAP has become the accepted standard therapy for OSA. Even though low risk and efficacy of CPAP, CPAP effectiveness rates are low because of problematic nonadherence and compliance.⁴⁰⁻⁴² The reasons of low compliance was reported many factors including nasal obstruction, discomfort, and claustrophobia.^{40,43} Poirier et al.⁴⁴ showed potential improvement of CPAP compliance rate by correction of nasal obstruction in OSA patients with nasal obstruction (the effect of nasal surgery on nasal continuous positive airway pressure compliance). They reported decreased CPAP pressures following nasal surgery. Although nasal surgery is not solitary curative method of OSA, it could increase CPAP compliance.

CONCLUSION

Nose is a physiologic breathing route during sleep and accounts for more than 50% of the total resistance of the upper airway. Although the results of nasal surgery to improve or cure SDB have been inconsistent, another role of nasal surgery must be considered to assist other treatment modalities, like improving the compliance of nasal CPAP. Therefore, more attention to nose must be paid during physical examination of SDB patients and, anatomical, neuromuscular, respiratory role of nose on airflow must be considered in SDB patients irrespective of nasal obstruction, which may widen our understanding of SDB and give better results to patients subjectively and objectively.

Conflicts of Interest

The author has no financial conflicts of interest.

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