sciendo

DOI: 10.2478/rjr-2020-0008

Romanian Journal of Rhinology, Volume 10, No. 38, April - June 2020

EDITORIAL

The role of the nose in snoring and obstructive sleep apnea syndrome

Adriana Neagos, MD, PhD, Lecturer

ENT Department, Targu Mures University of Medicine and Pharmacy, Targu Mures, Romania

Sleep-related breathing disorders (SRBD) are part of the multitude of sleep disorders, which include narcolepsy, insomnia, parasomnia, restless legs syndrome, professional sleep disorders, jet lag-related sleep disorders, or sleep disorders caused by mental illness.

In the category of breathing-related sleep disorders are included primary snoring, obstructive sleep apnea syndrome (OSAS), as well as the upper airway resistance syndrome, which occupies an intermediate position. Add to these the central apnea (e.g. Cheyne-Stokes respiration), as well as the mixed apnea.

Obstructive sleep apnea, as well as primary snoring and upper airway resistance syndrome, represents a frequent pathology and a particular medical field in otorhinolaryngology.

Sleep apnea is defined as a cessation of airflow to the lungs for more than 10 seconds. The severity of sleep apnea is defined primarily by the apnea-hypopnea index (AHI) that represents a value given by the number of apneas and hypopneas per unit time of 1 hour. Hypopnea is characterised only by the reduction of airflow, without its complete cessation.

In OSAS and in chronic snoring, the anatomical obstruction on several levels of the upper airway is frequently present. The nose is considered to be the first structure of the upper airway. In this context, nasal obstruction contributes to the onset of SRBD[1].

Narrowing or obstruction of the upper airway during sleep has been attributed to different factors. In addition to the modified upper airway anatomy, there is the insufficient activity of the upper airway dilator muscles and the increase in the collapse capacity of the upper airway. Upper airway dysfunction has a progressive evolution, starting with snoring and airflow limitation, which causes mechanical trauma with progressive upper airway injury. Recent studies show that the upper airway collapse occurs at the end of the expiratory phase, preceding apnea.

The size of the craniofacial structures was correlated with the prevalence and severity of OSAS. In this context, the anatomical variation that decreases the volume of the skull, mandibular retroposition, micrognathia, retrognathia, were found to be the determining factors in the occurrence of OSAS[2].

Associated with the above mentioned, narrowing of the upper airway can be produced by the lateral pharyngeal walls, as a result of an edema or fatty tissue deposition, tonsillar hypertrophy, changes in the soft palate, uvula and tongue[3,4].

The upper airway behaves like a Starling resistor, the collapse occurring independently of the suction forces (aspiration) of the diaphragm, but in close connection with the existing balance between the pressure in the airway and the pressure exerted by tissues on the same side. The upper airway collapsibility remains significantly related to the excessive pressure applied to it, just like the pressure at the end of the expiration. As a result, upper airway collapse, which occurs at the end of the expiration, is due to the lack of activity of the upper airway dilator muscles.

The pathophysiological mechanisms may explain in many cases the involvement of the nasal pathology in the sleep-related breathing disorders. This mechanism also includes, besides the Starling resistor model, the upper airway instability due to the lack of bone and cartilaginous structures, as

Corresponding author: Adriana Neagos, MD, PhD, Lecturer, ENT Department, Targu Mures University of Medicine and Pharmacy, 8 Lapusna Street, 50342, Targu Mures, Romania
e-mail: neagos.adriana@gmail.com

well as the nasal ventilation reflex, the nitric oxide (NO) involvement.

The nose provides more than 50% of the total resistance in the upper airway; nasal breathing allows the accomplishment of the nasal function: humidification, heating and filtration of the inspired air. The pathophysiological mechanisms that explain the relationship between nasal flow and breathing during sleep include the Starling model. The Starling resistor model shows the upper airway as a tube with a primary site with a partial collapse capacity at the level of the nasal region and a segment with the possibility of collapse from the outside at the level of the oropharynx. This model shows that the obstruction in the nose followed by a reduction in the nasal airflow will generate a suction force, the negative intraluminal force, located in the oropharynx. This effect is exacerbated in the supine position, when nasal resistance tends to increase. Increased nasal resistance reactively causes mouth breathing; this switch between the two types of breathing is physiological, being disadvantageous in the situation of unstable oral breathing. During sleep, the upper airway resistance is lower in patients breathing through the nose than in those with mouth breathing, unlike the waking state, in which airway resistance is the same during nasal and oral breathing. During sleep, there is a decrease in the fraction of mouth breathing from 7.6 to 4.3%. When the airway is completely obstructed, there is an automatic change of nasal breathing with oral breathing, achieved at a high cost to the body. Oral breathing is associated with a 2.5-fold increase in upper airway resistance, by narrowing of the pharyngeal lumen, decrease in pharyngeal diameter in the tongue base region, due to posterior retraction of the tongue, and the increase in oscillations of the soft palate and pharyngeal tissue. All these are involved in the onset of SRBD.

An important factor is also the nasal ventilation reflex. Experimental application of anaesthetic to the nasal mucosa, in healthy patients, causes a significant increase in episodes of obstructive and central apnea, at the same similar value reported by complete nasal obstruction. Similar results confirm that the activation of nasal receptors during nasal breathing has a positive effect on spontaneous ventilation. Nasal airway bypass reduces the activity of nasal receptors, by deactivating the nasal ventilation reflex, and reduces spontaneous breathing, which may be associated with the exacerbation of OSAS and with an increased duration of apnea episodes[5,6].

Understanding nasal and sleep physiology, and in particular the knowledge of the importance of nasal and oral breathing, has led, in the last decade, to a real progress in understanding the connection between total upper airway resistance and sleep apnea.

Hippocrates, in “de Morbis Popularibis”, noted the association of nasal polyposis with restless sleep. Most people present sleep disorders during episodes of virally induced nasal congestion. Many specialists take into consideration the connection between nasal breathing and sleep quality, as well as the sleep disorders arising as a result of the architectural modification of the nostrils. Since the literature was not fully conclusive, multiple studies have been carried out in this regard over time, resulting in the publication of new articles.

From a clinical point of view, studies carried out on groups of patients try to show that nasal obstruction is associated with chronic snoring and mild sleep apnea syndrome. In this context, there was found no linear correlation between the degree of nasal obstruction and the severity of sleep respiratory syndrome, so that it is thought that nasal obstruction is not the main cause of moderate and severe OSAS in most patients[7].

Randomized studies have shown that in patients with associated nasal pathology, such as allergic or nonallergic rhinitis, intranasal corticosteroid administration may influence sleep quality, and it can be extremely useful for those patients with mild OSAS. Also, nasal surgery can influence the quality of life and snoring in a group of patients with mild sleep-breathing disorder associated with nasal septum deviation, but it is not an effective treatment for patients with OSAS. On the other hand, patients who cannot tolerate the specific therapy with the CPAP mask, if the evaluation of the upper airway reveals an obstruction of the nasal airway, nasal surgery can influence CPAP compliance and adherence.

The reduction in nasal breathing and nasal airflow have a significant effect on sleep quality. In many studies, there is an increase in the value of the AHI in normal patients without associated pathology. In other studies, complete or partial artificial induction of nasal obstruction produces a worsening in the quality of sleep, with the increase in the number of apneas and intensification of micro-arousals. Studies on patients with nasal tampons applied for the epistaxis-type vascular pathology show a significant intensification of apneas as well as a significant alteration of sleep.

Nasal obstruction can occur through structural abnormalities (nasal septum deviation, inferior turbinate hypertrophy), inflammatory conditions (acute rhinitis, acute and chronic rhinosinusitis, nasal polyposis) or, rarely, neuromuscular disorders. Many observational studies show that nasal

congestion is associated with daytime sleepiness and snoring. In patients with nocturnal nasal congestion, there is an increase in the incidence of snoring and the maintenance of daytime fatigue. The polysomnography and the rhinomanometry show a correlation between nasal resistance in the lying position and habitual snoring. Nasal obstruction during wakefulness, evaluated by posterior rhinomanometry, is an independent risk factor for OSAS, making 2.3% of the total variations of the AHI index.

Multiple clinical studies show that allergic rhinitis affects 9-42% of the population. The way in which this pathology produces restless sleep and daily fatigue is not fully clarified. The occurrence of nasal obstruction due to inflammatory mediators such as IFN-gamma, TNF-alpha, IL-lb, IL-4 and IL-10, and posture changes during sleep, may have a direct effect on sleep regulation. In patients with allergic rhinitis, a direct association between nasal resistance and the severity of the sleep-related breathing disorder has been demonstrated. Reversible nasal obstruction influences sleep quality much more frequently, the correlation between this and the SRBD being much more pronounced than in the case of structural anomalies such as nasal septum deviation.

CONCLUSIONS

Nasal obstruction, especially the reversible one, is associated with chronic snoring and mild OSAS. A direct association between the degree of nasal obstruction and the severity of sleep-related breathing disorders is not fully demonstrated and certified, since nasal obstruction is not a factor contributing to the appearance of moderate and severe apnea.

In this context, the multitude of therapeutic possibilities of SRBD must be adapted to each case.

Nasal surgery alone, as a treatment of OSAS, has no efficacy, being useful only for symptom reduction, or as an integral part of multilevel surgery. In the case of therapeutic failure through CPAP, the evaluation of the upper airway can have a positive influence on CPAP compliance. In general, patients who respond positively to medication, nasal dilators, may be candidates for surgical treatment. In patients with OSAS and associated nasal obstruction, nasal surgery can reduce the required CPAP pressure.

REFERENCES

1. Cai Y, Goldberg AN, Chang JL. The nose and nasal breathing in sleep apnea. Otolaryngol Clin North Am. 2020;pii:S0030-6665(20)30004-9. DOI: 10.1016/j.otc.2020.02.002. [Epub ahead of print].
2. Awad MI, Kacker A. Nasal obstruction considerations in sleep apnea. Otolaryngol Clin North Am. 2018;51(5):1003-9. DOI: 10.1016/j.otc.2018.05.012.
3. Georgalas C. The role of the nose in snoring and obstructive sleep apnoea: an update. Eur Arch Otorhinolaryngol. 2011;268(9):1365-73. DOI: 10.1007/s00405-010-1469-7.
4. Meen EK, Chandra RK. The role of the nose in sleep-disordered breathing. Am J Rhinol Allergy. 2013;27(3):213-20. DOI: 10.2500/ajra.2013.27.3876.
5. Friedman M, Maley A, Kelley K, Leesman C, Patel A, Pulver T, et al. Impact of nasal obstruction on obstructive sleep apnea. Otolaryngol Head Neck Surg. 2011;144(6):1000-4. DOI: 10.1177/0194599811400977.
6. Kotecha B. The nose, snoring and obstructive sleep apnoea. Rhinology. 2011;49(3):259-63. DOI: 10.4193/Rhino10.165.
7. Kohler M, Bloch KE, Stradling JR. The role of the nose in the pathogenesis of obstructive sleep apnea. Curr Opin Otolaryngol Head Neck Surg. 2009;17(1):33-7. DOI: 10.1097/MOO.0b013e32831b9e17.