

The role of the nasal valve in patients with obstructive sleep apnea syndrome

Matteo Gelardi¹, Pierluigi Intiglietta², Giuseppe Porro¹, Vitaliano Nicola Quaranta³, Onofrio Resta², Nicola Quaranta¹, Giorgio Ciprandi⁴

¹Otolaryngology, Department of Basic Medical Science, Neuroscience and Sensory Organs, University of Bari Aldo Moro, Bari, Italy; ²Department of Cardiac, Thoracic, and Vascular Science, Institute of Respiratory Disease, School of Medicine, University of Bari, Bari, Italy; ³Pneumology Unity, Di Venere Hospital, Bari, Italy; ⁴Allergy, Casa di Cura Villa Montallegro, Genoa, Italy

Summary. The nasal valve area has the minimal cross-sectional area of the upper airways. A problem at this level may easily induce impaired breathing. Obstructive sleep apnea syndrome (OSAS) is a common disorder. It has been reported that nasal obstruction may be associated with OSAS. The aim of this study was to investigate the role of nasal valve in a group of OSAS patients. Polysomnography was performed. Patients with bilateral valve incontinence had lower SaO₂-nadir than patients with unilateral (or no) one. In conclusion, the present study demonstrates that a bilateral nasal valve incontinence is associated with more severe nocturnal respiratory pattern in patients with OSAS. (www.actabiomedica.it)

Key words: nasal valve, airflow obstruction, obstructive sleep apnea syndrome, polysomnography, Nas-Air®

Introduction

The internal nasal valve is the narrowest portion of the nasal cavity (1). It is formed by the junction of the upper lateral cartilages with the nasal septum. The normal angle between these 2 structures ranges between 10° and 15°. The nasal valve offers the greatest resistance to nasal airflow in the nasal cavity. The narrower the site, the more vulnerable is the nose to pathologic nasal obstruction. Nasal obstruction due to nasal valve abnormalities may result from either dynamic or static problems and is one of the most important and common reasons for nasal obstruction. Despite these facts, nasal valve collapse is a frequently overlooked cause of nasal obstruction (2, 3). In particular, the normal airflow through the nasal valve depends on the Bernoulli principle and Poiseuille law. The Bernoulli principle states that as the flow of air increases through a fixed space, the pressure in that space decreases. If the decrease in pressure overcomes the inherent rigidity of

the flexible nasal sidewall, collapse can occur resulting in obstruction. Clinically, the collapse of the nasal sidewall during inspiration is termed dynamic obstruction. Poiseuille law states that the flow is inversely proportional to the fourth power of the radius, which means that small decreases in the radius of a space have dramatic impacts on the flow of air through the nose. In the clinical setting, an anatomically narrowed portion of the nasal valve is defined as a static obstruction.

On the other hand, obstructive sleep apnea syndrome (OSAS) is a common disorder (4). Notably, 2 meta-analysis pointed out that nasal obstruction represents a common problem in OSAS patients treated with continuous positive airway pressure (CPAP) (5, 6). Very recently, nocturnal nasal obstruction has been found in more than one-third of OSA patients who had, on average, one nasal valve with a smaller minimum cross-section area (7). For these reasons, we aimed to investigate the role of nasal valve in a group of OSAS patients recruited in clinical practice.

Materials and Methods

The present open study included 19 inpatients with OSAS diagnosis.

Inclusion criteria were: adult age and OSA diagnosis according to validated criteria (8). Exclusion criteria were: anatomical clinically relevant problems (e.g. very severe septal deviation and/or turbinate hypertrophy, such as grade IV), disorders and current medications potentially able to interfere with findings.

The patients were visited and undergone also an otorhinolaryngological visit, including anterior rhinoscopy.

Subjective parameters were evaluated by the patients, and include perception of nasal obstruction, sleep quality, and olfaction; they were measured by a visual analogue scale (VAS). VAS score for nasal obstruction ranged from 0 (=completely blocked nose) to 10 (=completely patent nose); VAS score for olfaction ranged from 0 (=no smell) to 10 (=optimal smell); VAS score for quality of sleep ranged from 0 (=worst sleeping) to 10 (optimal sleeping). In addition, VAS was used for assessing the satisfaction for the Nas-Air® (0=bad; 10=best).

Daytime sleepiness was evaluated with the Epworth Sleepiness Scale (ESS): an ESS score of ≥ 10 was considered excessive daytime sleepiness (9). In addition, the STOP-Bang (10) and Restorative Sleep (11) questionnaires, and Mallampati scale (12) were used.

Cardiorespiratory nocturnal monitoring was performed in all patients was done in ambient air and spontaneous breathing using a portable 4-channel/8-track polygraph (WristOx₂, Nonin, the Netherlands). Oxyhemoglobin saturation, heart rate, body posture, oral-nasal air flow, snoring sounds, and thoracic and abdominal movements were recorded in detail. AHI (apnea-hypopnea index), ODI (oxygen desaturation index), TST90 (total sleep time with oxyhemoglobin saturation below 90%), SaO₂-Nadir % and Restoring Sleep were calculated

Clinical characteristics were reported as mean \pm standard deviation (SD) for continuous variables and as percentage for categorial variables. The normal distribution of continuous variables was verified. Continuous parameters were analyzed by Student's T-test, discontinuous parameters were analyzed by X² test. The

percentage of precision of cross-validation was calculated by the "leave-one-out" method. A ROC curve was performed to verify the precision of this method. Significance values assumed for $p < 0.005$. All the analysis have been conducted with SPSS 21 software.

Results

This study included 19 inpatients (mean age 61.1 ± 13.5 years, range 41-87; 4 females). Patients were subdivided in two groups: Group 1 had bilateral incontinence of nasal valve and Group 2 unilateral or absent incontinence.

Clinical data of the two groups are reported in detail in Table 1.

Group 1 had a significantly ($p < 0.05$) lower Nadir of SaO₂ (70.9 ± 11.06) than Group 2 (80.9 ± 9.5) during nocturnal sleep, as reported in Figure 1.

Group 1 had a significantly ($p < 0.05$) less severe Mallampati score than Group 2 (category 5: 36.4% vs 100% respectively) as reported in Table 1.

Group 1 showed a trend for higher AHI in comparison with Group 2 (47.5 ± 34.1 vs 26.7 ± 22.1 respectively).

Discriminating analysis confirmed that AHI and Nadir SaO₂ correctly differentiated 68.4% of patients as reported in Figure 2 with good reliability (AUC=0.78; $p < 0.05$).

Discussion

From a pathophysiological point of view, the nasal valve area represents the narrowest passage in the respiratory tract, causing more than half of the total resistance to nasal respiration in a healthy subject. Therefore, nasal obstruction may have relevant impact on breathing, mainly concerning in patients with OSAS. As it has been reported a relationship between nasal obstruction and respiratory parameters in patients with OSAS, we aimed to confirm these outcomes in a group of inpatients in a real-world setting. In particular, we considered the role of bilateral versus unilateral (or no) incontinence of the nasal valve.

The findings showed that patients with bilateral

Table 1. Clinical characteristics in Group 1 and Group 2

	Group 1 (n=11)	Group 1 (n=8)	P value
Mean age±SD	62.9±12.24	58.62±15.59	0.53
Smokers n (%)	5 (45.5)	4 (50)	0.72
Gender females	2 (18.2)	2 (25)	0.574
BMI Mean±SD	33.7±7.1	30.7±6.3	0.348
Sleepiness n (%)	8 (72.7)	7 (87.5)	0.426
Neck circumference M±SD	41.9±1.6	40.4±2.6	0.169
Weakness n (%)	5 (45.5)	5 (62.5)	0.395
Sleep hours	6.45±0.93	6.75±1.38	0.612
ESS Mean±SD	5.09±3.04	8.5±4.14	0.072
STOP BANG Mean±SD	5.45±1.57	5,25±1.38	0.768
MALLAMPATI			0.018
0	0 (0)	0 (0)	
1	0 (0)	0 (0)	
2	1 (9.1)	0 (0)	
3	6 (54.5)	0 (0)	
4	4 (36.4)	8 (100)	
Turbinate hypertrophy			0.481
1	0 (0)	1 (12.5)	
2	5 (45.5)	3 (37.5)	
	6 (54.5)	4 (50)	
VAS nasal obstruction Mean±SD	6.81±1.77	6.62±2.2	0.841
VAS sleep quality Mean ± SD	4.9±1.92	3.75±2.7	0.314
VAS smell Mean±SD	8.18±2.96	7.37±2.87	0.560
PO ₂ Mean±SD	78.18 ±11.48	80.87±1.21	0.616
PCO ₂ Mean±SD	42.27±5,16	40.62±3.7	0.429
pH Mean±SD	7.42±0.03	7.42±0.02	0.755
HCO ₃ Mean±SD	27.7±2.58	26.1±2.44	0.209
SaO ₂ Mean±SD	94.9±2.84	95.75±1.48	0.416
HR bpm Mean±SD	79.54±12.3	77.25±6.86	0.612
Frequent awakes n (%)	2 (18.2)	2 (25)	0.574
Chocking n (%)	0 (0)	3 (37.5)	0.058
Reported Apnea n (%)	8 (72.8)	7 (87.5)	0.426
Snoring n (%)	8 (81.8)	7 (87.5)	0.624
AHI Mean±SD	47.5±34.1	26.71±22.1	0.127
ODI events/h Mean±SD	44.01±34.7	26.71±22,14	0.394
TST90 Mean±SD	31.83±33.26	21,68±29.4	0.492
Restoring Sleep Mean±SD	47.6±26.2	64.8±24.	0.150
SaO ₂ % (Nocturnal Mean±SD)	89.9±4	92.5±2.65	0.112
SaO ₂ -Nadir % Mean±SD	70.9±11.06	80.9±9.5	0.050

nasal valve incontinence had less severe Mallampati scale scores. On the other hand, patients with unilateral (or no) nasal valve incontinence had higher SaO₂-nadir than patients with bilateral one. In addition, patients of Group 1 showed a trend to have higher AHI than Group 2.

These outcomes suggest that the nasal valve may have a role in OSAS patients as bilateral impairment was associated with a worst nocturnal respiratory pattern. These findings are consistent with previous observations that underlined the relevance of nasal obstruction in patients with OSAS (7). In particular, the

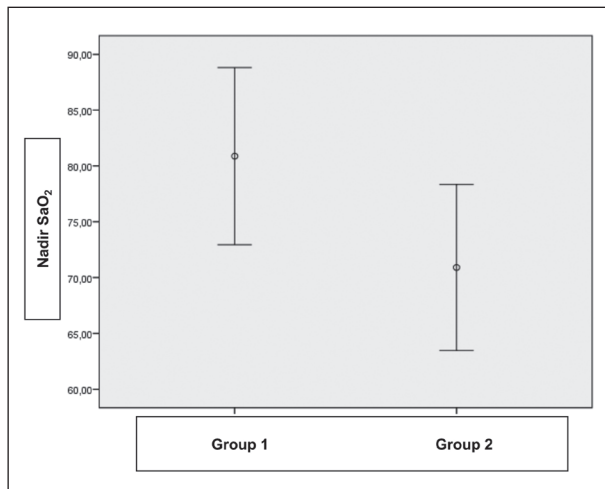


Figure 1. Nadir of SaO₂ in patients of Group 1 and Group 2

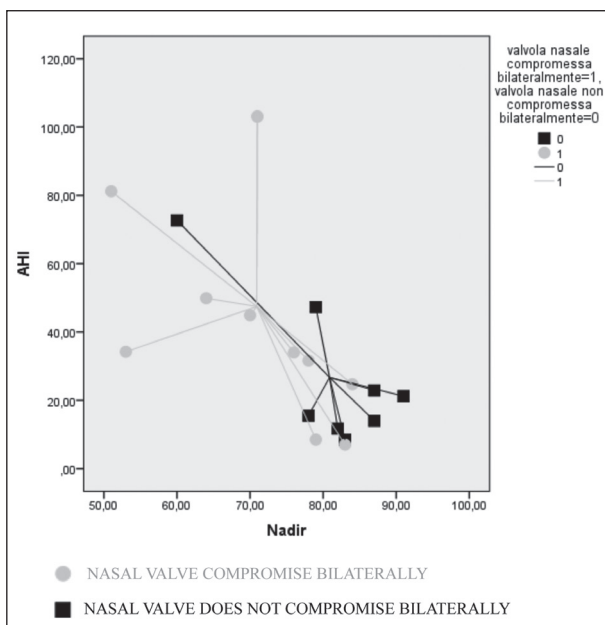


Figure 2. Nadir of SaO₂ and AHI in Group 1 and Group 2

current outcomes suggest that to improve nasal valve function may be clinically relevant in OSAS patients.

However, the present study was conducted in a restricted number of patients and was designed as a cross-sectional study. Thus, further studies should be performed to confirm these preliminary findings.

In conclusion, the present study demonstrates that a bilateral nasal valve incontinence is associated

with more severe nocturnal respiratory pattern in patients with OSAS.

References

1. Hamilton GS. The external nasal valve. *Facial Plast Surg Clin N Am* 2017; 25: 179-94.
2. Leitzen KP, Brietzle SE, Lindsay RW. Correlation between nasal anatomy and objective sleep apnea severity. *Otolaryngol Head Neck Surg* 2014; 150: 325-31.
3. Barrett DM, Casanueva FJ, Cook TA. Management of the nasal valve. *Facial Plast Surg Clin N Am* 2016; 24: 219-34.
4. Heinzer R, Vat S, Marques-Vidal P, et al. Prevalence of sleepdisordered breathing in the general population: the HypnoLaus study. *Lancet Respir Med* 2015; 3: 310-8.
5. Ishii L, Roxbury C, Godoy A, Ishman S, Ishii M. Does nasal surgery improve OSA in patients with nasal obstruction and OSA? A meta-analysis. *Otolaryngol Head Neck Surg* 2015; 153: 326-33.
6. Li HY, Wang PC, Chen YP, Lee LA, Fang TJ, Lin H.C. Critical appraisal and meta-analysis of nasal surgery for obstructive sleep apnea. *Am J Rhinol Allergy* 2011; 25: 45-9.
7. Varendh M, Andersson M, Bjornsdottir E, Hrbos-Strom H, Johannisson A, Arnardottir ES, et al. Nocturnal nasal obstruction is frequent and reduces sleep quality in patients with obstructive sleep apnea. *J Sleep Res* 2018; 27(4): e12631.
8. Chung F, Memsoudis SG, Ramachandran SK, Nagappa M, Opperer M, Cozowicz C, et al. Society of Anesthesia and Sleep Medicine Guidelines on Preoperative Screening and Assessment of Adult Patients With Obstructive Sleep Apnea. *Anesth Analg* 2016; 123: 452-73.
9. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991; 14: 540-5.
10. Shafazand S. Perioperative management of obstructive sleep apnea: ready for prime time? *Cleve Clin J Med* 2009; 76(Suppl 4): S98-103.
11. Drake CL, Hays RD, Morlock R, Wang F, Shikhar R, Frank L, et al. Development and evaluation of a measure to assess restorative sleep. *J Clin Sleep Med* 2014; 10: 733-41.
12. Mallampati SR, Gatt SP, Gugino LD. A clinical sign to predict difficult tracheal intubation: a prospective study. *Can Anaesth Soc J* 1985; 32: 429-434.

Conflict of interest: None to declare

Received: 18 December 2018

Accepted: 8 January 2019

Correspondence:

Giorgio Ciprandi, MD

E-mail: gio.cip@libero.it