Acoustic rhinometry findings in patients with mild sleep apnea

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BACKGROUND: **Nasal obstruction may contribute to the development of obstructive sleep apnea (OSA). Acoustic rhinometry (AR) measures nasal patency and congestion, which are useful parameters in objectively evaluating nasal obstruction. The nasal obstruction produced by allergic rhinitis may contribute to the development of OSA and can be easily assessed with AR.**

OBJECTIVE: **This study was undertaken to assess the degree of nasal obstruction seen in allergic patients with and without OSA.**

STUDY DESIGN AND SETTING: **This study was a retrospective data analysis from a tertiary referral center. The AR data from 10 patients with and 40 patients without mild OSA were compared.**

RESULTS: **The mean congestion factors at the first cross-sectional area (CSA1) on the AR graph were found to be significantly higher in the OSA group than in the non-OSA group ($P = 0.03$). The classification of change in congestion factors demonstrated significant differences at CSA1, CSA2, and CSA3 and in volume (t distributions <0.001, 0.0312, <0.001, and <0.001, respectively). The non-OSA patients noted a significant subjective improvement in nasal congestion after topical nasal decongestion, whereas the OSA patients did not ($P \leq 0.0001$ and 0.064, respectively).**

CONCLUSION: **Although the role of nasal obstruction in OSA is controversial, our study lends evidence to the thought that the nasal obstruction associated with allergic rhinitis is associated with the presence of mild OSA.**

SIGNIFICANCE: **Whether allergic rhinitis is a direct cause of OSA is debatable, but we have shown that greater nasal congestion is related to the presence of OSA in a population of patients with allergic rhinitis.** (Otolaryngol Head Neck Surg 2002;126:475-80.)

Snoring is caused by vibration of the uvula and the soft palate. This leads to an increased respiratory effort and collapse of the upper respiratory airway, which may result in obstructive sleep apnea (OSA). Total or near-total nasal obstruction leads to oral respiration and has been shown to cause increased airway resistance. There is no consensus about the role of nasal obstruction in snoring and mild sleep apnea. There are reports that failed to demonstrate any correlation between snoring and nasal resistance.[1,2] On the other hand, there are opposing reports suggesting that severe nasal deviation may cause sleep disorders and that snoring can be improved after nasoseptal surgery.[3,4]

Acoustic rhinometry (AR) is a relatively new modality of evaluating the function of the nose. Introduced in 1989, the analysis of audible sound waves reflected from the nasal cavity has brought a new perspective to experimental nasal physiology studies because it is an objective, noninvasive technique for assessing the nasal cavity.[5,6] In this method, measurements are taken separately at baseline and after appropriate decongestion or shrinking of the mucosa by α-sympathomimetic agents such as oxymetazoline (Neo-Synephrine).[7] A computer draws a graph plotting the distance from the nostril relative to the cross-sectional area; thus, the 3-dimensional nasal cavity is projected onto a 2-dimensional graph. In this graph,

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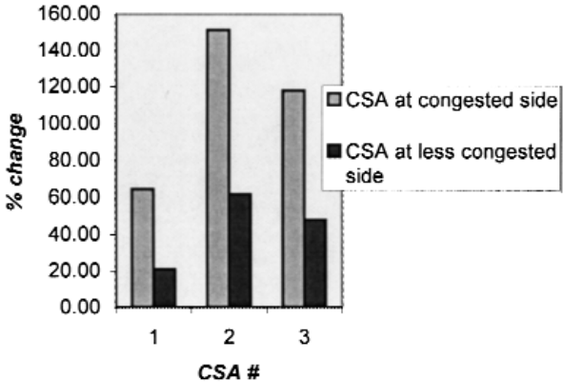

Fig 1. Average percent change in congestion factor in patients with obstructive sleep apnea.

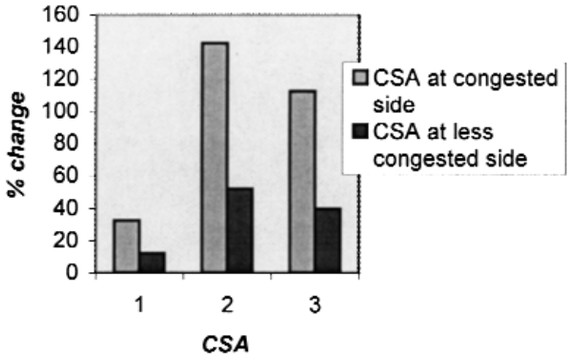

Fig 2. Average percent change in congestion factor in patients without obstructive sleep apnea.

the y-axis represents the distance into the nasal cavity, and the x-axis represents the 2-dimensional area relative to distance. Most subjects demonstrate a sudden decrease, which looks like a valley on the graph, at around 2 cm; this corresponds to the anterior portion of the inferior turbinate and/or the nasal valve. This site is referred to as minimal cross-sectional area 1 (CSA1). At about 4 cm, another valley usually appears, which is termed minimal cross-sectional area 2 (CSA2), and it corresponds to the anterior portion of the middle turbinate. The valley that appears at about 6 cm is termed the minimal cross-sectional area 3 (CSA3), and it roughly corresponds to the middle portion of the middle turbinate and the natural maxillary ostium.[8] This technique can be used as a nasal function test in patients with allergic rhinitis to grade the severity of nasal mucosal congestion by comparing congested and decongested state CSA and volume values (unpublished data). In this study, we compared the AR graphs of allergic patients with OSA to those of allergic patients without OSA.

MATERIALS AND METHODS

We retrospectively examined 50 patients diagnosed as having seasonal and perennial allergic rhinitis. These patients were followed for at least 6 months by one of the authors (J.P.C.) and evaluated on the basis of a detailed history and physical examination. Allergic rhinitis status was confirmed by in vivo or in vitro allergy testing. Patients were divided into 2 groups depending on the presence or absence of both snoring and obstructive mild sleep apnea.

We retrospectively reviewed the patients' clinic charts and noted what medications, if any, they were using at the time they underwent AR testing. All patients were using oral antihistamines and topical nasal corticosteroids for the management of their allergies, and some were undergoing immunotherapy as well. However, none of these patients were using oral or topical decongestants at the time of AR testing. AR was performed during the follow-up examination. The AR graphs were obtained using a 2-microphone AR (Hood Laboratories, Pembroke, MA). Each AR study was performed by an experienced technician in a standard fashion that had been described previously.[5] AR graphs were obtained both at the predecongested state and the postdecongested state. CSA1, CSA2, CSA3, and total volumes from a 0- to 6-cm distance were noted for each state and each side. The congestion factors were calculated for each CSA and volume using the following formula: Congestion factor (%) = [(congested – decongested) CSA or volume/congested CSA or volume value] $\times$ 100.

The mean congestion factors (ie, average of the left and right sides) were calculated for the CSAs and volume values in each group. A Student's *t* test was used to determine the significance of difference between the 2 groups. A *P* value of <0.05 was chosen to determine whether a difference was significant.

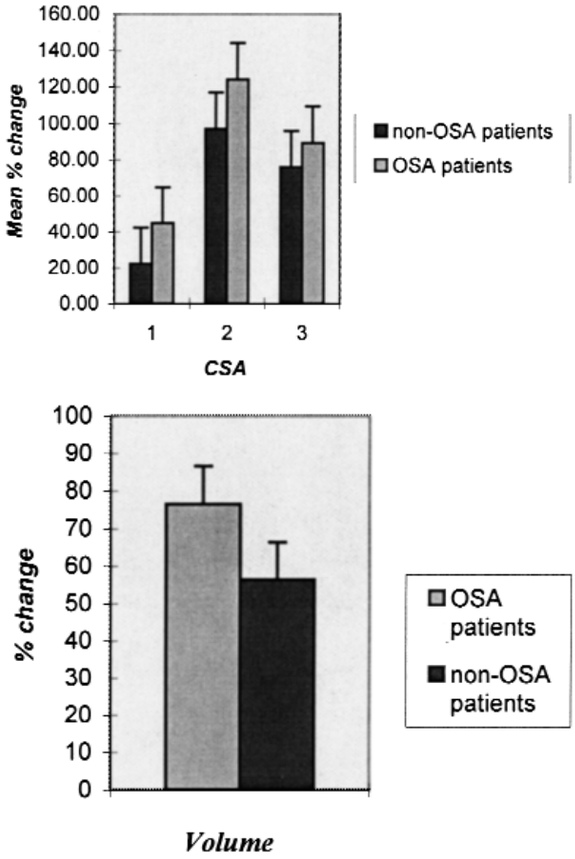

Fig 3. Mean congestion factor for each cross-sectional area and for volume.

RESULTS

Fifty patients were studied, of whom 40 did not snore or have sleep-disordered breathing. The age of the 40 patients ranged from 20 to 63 years (median age 33 years); 17 were women and 23 were men. The presence of mild sleep apnea and snoring was confirmed by polysomnography in 10 patients with an average respiratory distress index (RDI) of 14.5. The mean age was 45 years (age range 29 to 76 years); 2 were women and 8 were men (Table 1).

The CSA1, CSA2, and CSA3 congestion factors for each individual side in patients with and without OSA are depicted (Figs 1 and 2). In comparing the congestion factors for the more congested and less congested sides, a Student's *t* test was used. In all instances, the *P* value was found to be <0.01, in both patients with or without sleep apnea and across all cross-sectional areas.

Table 1. Patient demographics

Type	Male	Female	Total	Age range (y)
With OSA	8	2	10	27-71
Without OSA	23	17	40	17-63

OSA, Obstructive sleep apnea.

In addition, the mean congestion factors (ie, the average of the congestion factors between the more congested and less congested sides) were determined (Fig 3). A Student's *t* test was used to analyze the mean congestion factors. The mean congestion factor at CSA1, CSA2, and CSA3 and volume values were higher in patients with sleep apnea. Only at CSA1, however, did the difference in mean congestion factor between OSA and non-OSA patients achieve significance with a *P* value of 0.03. The CSA2, CSA3, and nasal volume *P* values did not reach significance.

The mean congestion factors were also stratified to determine how dramatic a change took place in the CSAs and volume from the congested to the decongested state. Each group of patients were subgrouped according to their magnitude of change as having normal, mild, moderate, or severe changes in their mean congestion factor at each CSA and for volume. The percentage ranges of normal, mild, moderate, and severe were different from one CSA or volume value to another and based on previously published findings on normal AR values[5] (Table 2 and Fig 4). A Student's *t* distribution was performed to compare the findings in the OSA versus the non-OSA patients across each CSA and volume values. A statistically significant difference in the distribution of mean congestion factors was demonstrated between the OSA and the non-OSA groups. In each case, the distribution of mean congestion factors at each CSA and the volume value was significantly increased in the OSA patients compared with the non-OSA patients.

A visual analog scale (VAS) was completed by 41 of the 50 participating patients (34 of the

Otolaryngology–
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Table 2. Distribution of mean congestion factors as measured by acoustic rhinometry

		Class of mean congestion factor							
		CSA 1	n	CSA 2	n	CSA 3	n	Volume	n
With OSA									
	Normal	0-15	1	0-50	1	0-40	1	0-30	0
	Mild	16-20	0	51-75	1	41-60	2	31-60	4
	Moderate	21-30	2	76-125	3	61-90	2	61-90	6
	Severe	>30	7	>125	5	>90	5	>90	0
Without OSA									
	Normal	0-15	22	0-50	9	0-40	13	0-30	11
	Mild	16-20	3	51-75	11	41-60	10	31-60	19
	Moderate	21-30	9	76-125	16	61-90	9	61-90	12
	Severe	>30	14	>125	12	90	16	>90	6

CSA, Cross-sectional area; *OSA,* obstructive sleep apnea.

40 non-OSA patients, 7 of the 10 OSA patients). The non-OSA patients reported their subjective congestion, as measured by VAS, to be between 0% and 100% before decongestion, with an average score of 40.8%. After application of the decongestant, the congestion assessment ranged between 0 and 70%, with an 18.96% average. Student's *t* test was used to compare the congested and decongested average values ($P < 0.0001$). In the OSA group, subjective congestion ranged from 0% to 85%, with an average of 35.0%. After decongestant, these scores ranged from 0% to 20%, with an average of 10.0% ($P = 0.064$). Student's *t* test was also used to compare the predecongested and postdecongested scores between OSA and non-OSA patients; the *P* value was found to be nonsignificant at 0.057 (Table 3).

DISCUSSION

Our study showed significant differences in the mean congestion factors between OSA and non-OSA patients only at CSA1; this may be a reflection of the fact that AR is most sensitive in the anterior nasal cavity.[6] This difference could also indicate the location of the nasal pathology in OSA. Such a difference at the critical nasal valve region demonstrates that patients with OSA have relatively greater nasal congestion in the anterior nasal cavity, which might contribute to the development of OSA.

At CSA1, CSA2, and CSA3 and for nasal volume, the distributions for the mean congestion factors (ie, normal, mild, moderate, or severe) also showed statistically significant differences between the OSA and the non-OSA patients. At CSA1, this is quite pronounced; 45% (22 of 40) of the non-OSA patients had mean congestion factors in the normal range, compared with 70% (7 of 10) of OSA patients who had severe or marked differences in mean congestion factor (*t* distribution <0.0001). For CSA2, 50% (5 of 10) of the OSA patients, as opposed to 25% (10 of 40) of the non-OSA patients, showed severe changes in the mean congestion factor (*t* distribution 0.0312). CSA3 and nasal volume both showed significant differences with *t* distribution values of < 0.001. The presence of such significant differences between OSA and non-OSA patients speaks to more than a simple coincident relationship between nasal congestion and sleep-disturbed breathing in allergic subjects; objective nasal congestion and sleep-disordered breathing are firmly associated in our study.

The results obtained by VAS showed significant changes in the perception of congestion from the congested to the decongested state in the non-OSA patients, although not in the OSA patients (*t* test P <0.0001 and 0.064, respectively). Both groups, however, did note subjective improvement in their congestion. The degree of predecongested to postdecongested change was

Table 3. Visual analog scale results

Patient group	Average subjective congestion scores (%)				*t* Test *P* value
	Before	SD	After	SD	
With OSA (n = 7)	35.0	29.15	10.29	7.7	0.064
Without OSA (n = 34)	40.85	29.47	18.96	22.46	<0.0001

OSA, Obstructive sleep apnea.
$P \leq 0.05$ considered significant.

compared between the 2 groups, and no significant difference was noted (*t* test $P = 0.0577$). The VAS data suggest that although the changes in congestion may not be significantly different, subjective improvement does exist for both groups of patients. Perhaps the patients with OSA realize that their congestion is lessened with the decongestant spray, although not maximally. The subjective congestion perceptions mirror the findings observed in objective measurements.

The part that nasal obstruction plays in sleep disturbance is a controversial one. Some authors purport it may cause frank OSA syndrome, whereas others minimize the role of nasal obstruction.[9,10]

While awake, nasal airway resistance markedly exceeds that of oral airway resistance. As sleep overcomes a human, however, the relaxation of musculature leads to a reversal in the resistance patterns of the nose and oral cavity. Oral airway resistance increases during sleep in response to flaccidity of the oral and pharyngeal musculature. Increased effort is required to breathe through the inefficient oral cavity, leading to greater negative pressures generated in the pharynx and an increased risk of collapse.[11]

The nasal cavity, having a more rigid frame, has a relatively more constant resistance in both the awake and asleep states. The pharynx still connects the nose to the trachea, but less resistance to airflow makes collapse of the pharynx less likely. The nose appears to be the preferred route of breathing during sleep.

Despite a lesser effect of sleep on the nose compared with the oral cavity, changes in nasal patency do occur. Nasal resistance is known to increase during recumbency, as mucosal congestion takes place.[12] An underlying limitation on nasal airflow, which may be subclinical during the daytime while upright, may become manifest at nighttime shortly after lying down. Any process that produces nasal congestion while awake has an additive effect on nasal airflow during sleep.

Studies have demonstrated a significant correlation between nasal resistance and snoring.[11,13] Patients who experience habitual snoring are more likely to complain of nighttime nasal congestion or discharge and congestion due to allergic rhinitis.

Upper airway resistance syndrome (UARS) is characterized by increased work of breathing during sleep to overcome elevated airway resistance. UARS causes numerous microarousals that fragment sleep, reducing its quality. Patients who have UARS complain of daytime somnolence, which can be objectively assessed with, for example, the multiple sleep latency test. It appears that the nose may play a significant role in the development of UARS.

Sleep fragmentation, a component of UARS, has been shown to reduce a patient's subjective assessments of wakefulness, mood, and attention.[14] The nasal obstruction associated with allergic rhinitis has been demonstrated to fragment sleep and produce a significant increase in microarousals compared with nonallergic patients.[15] The effects of allergic rhinitis can be combated with medical therapy to reduce allergic inflammation. Intranasal topical corticosteroids improve daytime sleepiness, and significantly reduce nasal congestion and increase sleep quality in patients with allergic rhinitis.[16]

Patients with UARS do not have RDIs that are sufficiently high to designate them as having OSA, but some believe that UARS may be a precursor to OSA; a continuum may exist between normal nocturnal breathing, occasional snoring, habitual snor-

ing, UARS, and OSA. For example, patients may progress from one point to another on the continuum as their weight fluctuates. Similarly, nasal obstruction may move a patient up or down the continuum.

A direct linear correlation between nasal resistance and RDI has not been observed, although the 2 indices seem to have an association.[11,13] Complete nasal obstruction from nasal packing has long been suspected of causing apneic episodes. Numerous studies championing for or against packing as a cause of OSA have been published.[17-20] It may be that patients with packing in addition to other risk factors are the main population who may have apneic episodes.[21]

Ragweed-allergic patients have longer and more frequent obstructive apneas during their acute season than they do during their "off" season, which may be attributable to increased nasal resistance.[22] Fixed anatomic obstructions, such as a deviated nasal septum, may contribute to OSA, and surgical repair can improve the RDI.[3,4] Nasal obstruction appears to be a partial etiologic agent in the development of OSA.

CONCLUSION

Our study lends evidence to the thought that the nasal obstruction associated with allergic rhinitis is associated with the presence of mild sleep apnea. Whether allergic rhinitis is a direct cause of OSA is debatable, but we have shown that greater nasal congestion is related to the presence of sleep apnea in a population of patients with allergic rhinitis. The prospective study of a large cohort of patients with allergic rhinitis for the development of OSA over time would be very useful to help delineate the relationship between allergic rhinitis and OSA.

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