Effect of improved nasal breathing on obstructive sleep apnea

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OBJECTIVES: **The goal was to compare the effect of an improved nasal airway on obstructive sleep apnea (OSA) by use of subjective and objective measures.**

METHODS: **A prospective study of 50 consecutive patients with nasal airway obstruction and OSA was carried out.**

RESULTS: **Subjectively, nasal breathing improved in 49 (98%) patients, whereas snoring decreased or disappeared in 17 (34%); the remaining 33 (66%) patients did not notice any significant change in their snoring. Daytime energy levels increased in 39 (78%) patients and remained unchanged or worsened in 11 (22%). In review of the polysomnographic data, the group overall did not have significant changes in respiratory disturbance index (RDI) or lowest oxygen saturation levels ($LSaO_2$). Continuous positive airway pressure (CPAP) levels required to correct OSA decreased after nasal surgery ($P < 0.01$). Patients with mild OSA showed significant worsening in RDI ($P < 0.05$), whereas $LSaO_2$ levels were improved in the group with moderate OSA ($P < 0.05$). In patients with severe OSA neither the RDI levels nor the $LSaO_2$ changed, but CPAP levels required to alleviate the obstruction after surgery were reduced ($P < 0.01$).**

CONCLUSIONS: **Most patients report improvement in nasal and sleep symptoms after correction of nasal airway obstruction. However, nasal surgery alone does not consistently improve OSA when measured objectively. Depending on the severity of OSA, nasal airway reconstruction may contribute to a decrease in CPAP level and improvement in oxygen saturation. Correction of the obstructed nasal airway should certainly be included in the overall treatment plan for OSA.** (Otolaryngol Head Neck Surg 2000;122:71-4.)

Surgical treatment of obstructive sleep apnea (OSA) is one of the current challenges for the otolaryngologist. Several techniques described in the past for correction of OSA have produced variable success.[1-4] Correction of an obstructed nasal airway is considered an important component in OSA treatment.[5-7] Clinical reports document nasal obstruction–induced OSA,[5,7,8,9] but controversial data exist regarding the role of improved nasal breathing in treating OSA. If nasal obstruction can result in OSA, it would be logical to assume that correction of an obstructed airway can improve OSA.[10,11] Experimental data exist regarding the role of increased nasal resistance and nasal occlusion in OSA.[11,12]

The exact role of the nasal airway in the pathogenesis of OSA is not clear. Reduced nasal cross-sectional area causes increased nasal resistance and predisposes the patient to inspiratory collapse of the oropharynx, hypopharynx, or both.[6,13] Patients with discrete abnormalities of the nasal airway, such as septal deformities, nasal polyps, and choanal atresia, require correction.[8] Nasal mucosal conditions, such as sinusitis and inferior turbinate hypertrophy, may also reduce the nasal cross-sectional area. These sources of nasal obstruction are usually addressed surgically in the management of OSA to decrease nasal resistance and increase nasal airflow.[5,8]

Correction of nasal airway obstruction in combination with several other surgical techniques has been reviewed in the past.[14,15] These studies, however, did not review nasal airway correction as an independent factor to determine its effect on polysomnogram (PSG) results. Only a limited study has reported PSG data after nasal airway correction.[16] Objective analysis of the effect of nasal surgery alone may provide new information about the impact of nasal airway obstruction on OSA. The results of this study may allow a more tailored approach to the treatment of OSA.

METHODS AND MATERIAL

Patient Selection

A prospective study was designed to evaluate 50 adult patients with OSA who required surgical treatment for nasal

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Table 1. Summary of PSG results before and after nasal surgery for OSA

	Mean RDI		Mean $LSaO_2$ (%)	
OSA patients	Preop	Postop	Preop	Postop
Total (n = 50)	31.6	39.5	82.5	84.3
Severe (n = 20)	55.8	56	74.8	75.9
Moderate (n = 14)	19.4	28.9	84.4	88.8
Mild (n = 16)	8.69	18.7	92.3	92.07

Table 2. CPAP titration levels before and after nasal surgery for OSA

	Mean CPAP (cm H_2O)	
OSA patients	Preop	Postop
Total (n = 22)	9.3	6.7
Severe (n = 13)	10.07	7.42
Moderate (n = 4)	9.5	6.5
Mild (n = 5)	7.2	5

airway obstruction between July 1, 1997, and June 30, 1998. Patients were required to have symptomatic subjective documentation and OSA documented on an 18-lead PSG. A second night of PSG with titration of the continuous positive airway pressure (CPAP) was performed in patients who were able to tolerate CPAP treatment. Postoperative PSG and CPAP titration were performed 6 weeks after surgery or later. This study was designed to collect data from standard treatment only and did not alter any treatment plans. It was therefore classified as exempt by the local institutional review board.

The anatomic cause of nasal airway obstruction (septal deviation, inferior turbinate hypertrophy) was documented. Height and weight were recorded, and body mass index (BMI) was calculated. Patients were required to complete a questionnaire regarding nasal obstruction and sleep symptoms before and 6 weeks after surgery. All patients also had CT scans of their paranasal sinuses taken.

Surgery

All patients underwent submucous resection (SMR) of the septum with or without SMR of the bilateral inferior turbinates. Some of the patients also had a history of recurrent sinusitis and underwent endoscopic sinus surgery at the same time. Patients with chronic sinusitis or other chronic diseases were not included in the study. Nasal packing was removed on the first or second postoperative day, and routine postoperative saline nasal irrigation and debridement were performed.

PSG and CPAP Titration

An all-night, attended, comprehensive sleep study was performed with a computerized polygraph to monitor electroencephalogram (C3-A2, C4-A1), left and right electrooculogram, electrocardiogram, chin and anterior tibialis electromyogram, abdominal, and thoracic movement by inductive plethysmograph, nasal buccal airflow, oxygen saturation by pulse oximetry, and throat sonogram. Apnea was defined as cessation of breathing for at least 10 seconds. Hypopnea was a decreased effort to breathe at least 50% less than the baseline and with at least a 4% decrease in oxygen saturation. The respiratory disturbance index (RDI) was calculated as the sum of total events (apneas and hypopneas) per hour. The lowest oxygen saturation levels ($LSaO_2$) were recorded and were also used as criteria to grade the severity of OSA. The degree of OSA was determined by the RDI and the $LSaO_2$ levels: mild (RDI 5-15 and $LSaO_2 > 85\%$), moderate (RDI 15-30 and $LSaO_2$ 65%-84%), and severe (RDI > 30 and $LSaO_2 < 65\%$) OSA.

CPAP titration was performed under the same conditions as described above with the level of nasal CPAP (in centimeters of water) adjusted until RDI was as low as possible without causing disruption of the patients' sleep.

Analysis of Data

The preoperative and postoperative nasal obstruction and sleep symptoms (snoring and daytime energy level) were compared. The mean BMI, before and after surgery, was calculated. The preoperative and postoperative RDI, $LSaO_2$, and CPAP levels were analyzed for the entire group and for the mild, moderate, and severe OSA subgroups with paired *t* tests.

RESULTS

The study group was composed of 41 men and 9 women, with ages ranging from 20 to 71 years. All patients underwent SMR of the septum and of the inferior turbinates bilaterally when indicated. All patients underwent preoperative and postoperative PSG. On the basis of the criteria outlined, 16 patients had mild, 14 had moderate, and 20 had severe OSA. Twenty-two patients, 13 with severe OSA, also underwent a second night of CPAP titration before and after surgery.

Review of the subjective data for the group overall demonstrated significant relief of symptoms after nasal surgery. Before surgery, all 50 patients reported nasal airway obstruction and snoring. Forty-seven (94%) of them reported decreased daytime energy. After surgery, 49 (98%) patients reported improved nasal breathing. One patient, despite a patent airway on physical examination, denied any change in his breathing. With regard to snoring, 33 (66%) patients were not aware of changes in their snoring pattern, 14 (28%) noticed decreased snoring, and 3 (6%) reported that snoring completely resolved after the surgery. Daytime energy levels

increased after surgery in 39 (78%) patients, worsened in 1 (2%), and remained unchanged in 10 (20%).

The average BMI for the entire group did not change significantly after surgery—35 versus 35.7.

Table 1 summarizes the mean preoperative and postoperative PSG results. The preoperative mean RDI level for the entire group was 31.6 versus 39.5 after surgery. The mean RDI change of 7.9 was not significant by the Student *t* test. The severity of OSA did not change after surgery. The $LSaO_2$ level appeared to increase from 82.5% to 84.3% after surgery, but this difference also was not significant. The mean CPAP levels decreased considerably after nasal surgery for OSA (Table 2). This difference was significant for the entire group ($P < 0.01$).

The total patient group was then separated into mild, moderate, and severe OSA subgroups. There were 16 patients in the mild group, 14 patients in the moderate group, and 20 patients in the severe group. The mean RDI in the mild group demonstrated a value of 18.69 after surgery, shifting from a preoperative value of 8.69. RDI significantly increased ($P < 0.05$) after nasal surgery in this group. The mean $LSaO_2$ data did not document significant change shifting from 92.3% to a postoperative value of 92.07% (Table 1).

The preoperative mean RDI values were 19.4 and 55.8 for the moderate and severe groups, respectively. After surgery, the mean RDI levels for the moderate and severe subgroups were 28.9 and 56.0, respectively, showing no significant change (Table 1). It appeared that nasal surgery resulted in increased RDI levels for those with moderate OSA, but this difference was not significant. In patients with moderate OSA, $LSaO_2$ was increased approximately 4%, which was a statistically significant improvement ($P < 0.05$). In those with severe OSA, neither RDI levels nor $LSaO_2$ changed, but CPAP values decreased significantly after surgery ($P < 0.01$) (Table 2). The limited number of patients with CPAP values in the mild and moderate groups did not fulfill the criteria for statistical analysis.

DISCUSSION

Nasal obstruction contributes to snoring, OSA, and alveolar hypoventilation. The resultant disrupted sleep leads to daytime sleepiness and fatigue.[8,17] Moreover, nasal obstruction may produce greater physiologic effects during the sleep than the awake state.[8] Our results indicate that patients' symptoms improved after nasal surgery with regard to nasal breathing, snoring, and daytime energy levels. Correction of an obstructed nasal airway, along with surgical treatment of a deviated nasal septum and inferior turbinate hypertrophy, generally ameliorates symptoms in OSA patients. These findings are consistent with those of other reports.[8,17]

The correction of an obstructed nasal airway is crucial in the treatment of OSA. Nasal obstruction increases nasal resistance and leads to sleep-disordered breathing events, including apnea, hypopnea, and snoring.[8] Nasal septal deformities can predispose patients to apneas and hypopneas.[18,19] In addition, inflammation of the nasal airway mucosa from conditions such as rhinitis and sinusitis might further exacerbate sleep-disordered breathing. Clinical and experimental data show that nasal inflammation causes increased nasal resistance that might lead to OSA.[20,21] If nasal obstruction is present, some authors advocate nasal surgery intervention as part of the routine therapy for OSA.[5-7,19]

Despite the subjective findings, the analysis of the entire group did not reveal objective evidence of improvement in the OSA condition on the basis of PSG studies. This finding is consistent with findings of a previously published report.[16]

Comparison of the preoperative and postoperative RDI levels of the mild, moderate, and severe OSA groups demonstrated increased RDI levels in the mild and moderate OSA groups, but the change was significant only in the mild group after nasal surgery. The results for patients with severe OSA indicated no changes, but individual changes varied drastically from marked elevation of RDI levels in some patients versus improvement in other patients. The unpredictability of postoperative RDI levels requires both discussion and further study. There may be factors allowing for the postoperative PSG to be more accurate and to record a deeper sleep than the preoperative PSG. A deeper sleep may result in more collapse of the upper airway; this is likely to produce higher RDI levels in patients with existing OSA. We propose 2 possible explanations for this. The postoperative PSG is the second or third study for these patients, who may have become accommodated to the test environment and thus slept better. In addition, and perhaps more important, the relief in nasal obstruction may have allowed the patients to sleep more comfortably. Experimental and clinical evidence shows that improved nasal breathing alleviates fragmented sleep and sleep disturbance.[6,22] This is true, however, in patients without true sleep apnea. Our subjective findings support our hypothesis. However, further analysis of sleep architecture is required to explain this phenomenon completely. We could not detect a significant improvement in sleep efficiency to support this hypothesis. Analysis of sleep stages also was not useful to support our postulation. The deeper sleep affected by sleep apnea was more fragmented. There are therefore 2 competing affects occurring in the postoperative patient: improved sleep from a patent nasal airway, but deeper sleep that results in increased apnea and fragmentation. This paradoxical effect of nasal surgery may explain the lack of improvement in RDI levels but certainly

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deserves further study. The same results were published by Series et al.[16]

Olsen and Kern,[8] in their excellent review of nasal influences on OSA, document the importance of the nasal cycle and nasopulmonary reflex in maintaining adequate blood oxygenation. The $LSaO_2$ levels significantly increased only in the moderate group. This may be the affect of the improved nasal airway despite persistent OSA. The data did not demonstrate any $LSaO_2$ change in the mild and severe groups. A lesser degree of collapse in the upper airway of the mild group may have allowed the mild group to have consistent preoperative and postoperative $LSaO_2$. In the severe OSA group, greater collapse of the upper airway with multilevel involvement causing inspiratory occlusions may be the reason for no significant difference of $LSaO_2$. None of these results contradict the necessity of correction of an obstructed nasal airway in the surgical management of OSA.

CPAP is the recommended treatment for OSA. Most patients do not comply with the CPAP treatment for several reasons, such as discomfort of the apparatus, discomfort of the pressured air blowing through the nose, and noise of the machine. CPAP levels were significantly reduced after surgery in the severe OSA group. We think the reduced CPAP levels will increase the compliance rate with the instrument.

The small number of patients with complete CPAP values in the mild (n = 5) and moderate (n = 4) OSA groups restricted us from commenting on these CPAP values changes. In addition, patients with mild or moderate OSA are not likely to use CPAP on a permanent basis. However, several patients in the mild and moderate OSA groups also noted the reduced pressure levels of CPAP allowed for easier use of the apparatus. Series et al[16] mentioned the role of increased compliance with the CPAP device in patients with decreased CPAP requirements after nasal surgery. Correction of an obstructed nasal airway significantly reduced the CPAP to tolerable levels that resulted in increased use of the CPAP device.

Patients with severe apnea also had subjective improvement in their symptoms. This had a 2-fold impact on the outcome. Patients were likely to break their vicious cycle of fatigue and sleepiness when compared with the preoperative state. They also had more energy to implement nonsurgical measures such as weight loss and exercise for treatment of their sleep apnea.

CONCLUSION

Optimal surgical treatment of OSA is still a challenge for the otolaryngologist. Despite subjective improvement, this study shows that the RDI level is not likely to be corrected or improved with nasal surgery alone. The patients undergoing nasal surgery in the treatment of OSA should understand that it is one component of a comprehensive management plan. All patients with OSA should be assessed with PSG both before and after surgery because subjective analysis often does not accurately correlate with objective changes in OSA. One of the most significant benefits of nasal surgery for patients with severe OSA is the resultant decrease in CPAP titration levels that should result in improved compliance of CPAP therapy and reduced morbidity and mortality of OSA.

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