The effect of treatment of nasal and sinus diseases on the success rate of palatal surgery for obstructive sleep apnea

Nassim T. Nassim[a], Hassan M. Elhoshy[a], Ahmed F. A. Fattah[a], Sherif F. Safwat[b] and Ahmed M. Atef[a]

[a]Department of E.N.T., Faculty of Medicine, Cairo University and [b]National Institute of Enhanced Laser Sciences, Cairo University, Cairo Egypt

Correspondence to Nassim T. Nassim, MD, 15 Morad Street, 11121 Giza, Egypt
Tel: +01 223 412 755;
e-mail: nasemtalat@yahoo.com

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Objective
Many patients with obstructive sleep apnea (OSA) have coexisting nasal and sinus problems. In the absence of major anatomical abnormalities, these problems may be overlooked, hence jeopardizing the success rate of palatal surgery. Our objective was to identify the effect of medical management of nasal and sinus diseases on the success rate of palatal surgery in mild to moderate cases of OSA.

Methodology
This prospective study included 28 patients divided in two groups; group A was treated medically with antibiotics and local steroids before palatal surgeries, whereas group B was not treated medically before palatal surgeries. Assessment was made preoperatively and postoperatively using polysomnography.

Results
Group A showed more improvement in the apnea hypopnea index than group B.

Conclusion
Management of nasal and sinus problems improves the results of palatal surgery in cases of mild to moderate OSA.

Keywords:
apnea hypopnea index, Lund–Mackay score, obstructive sleep apnea

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Introduction

Sleep-disordered breathing is a serious disorder that affects a high percentage of the population. In the past few decades, advances have been made in identifying risk factors, understanding the pathophysiologic processes and adverse health sequelae, and in improving diagnosis and treatment. The condition includes a range of abnormal breathing events that include frequent episodes of apnea, hypopnea, and snores or breaths with high airway resistance [1].

Overweight and central ponderosity, aging, male sex, and severe (but relatively rare) craniofacial abnormalities such as acromegaly have been identified as strong risk factors for sleep-disordered breathing. Hypothesized but untested risk factors include problems originating in the nose, including physical obstruction, allergic rhinitis, and chronic sinusitis. Other risk factors include familial predisposition, smoking, alcohol use, menopause, and ethnicity [2].

A biologic basis for nasal obstruction as a cause of sleep-disordered breathing lies in the effect of nasal breathing on resistance and flow velocity, which affects the pressure differential between the atmosphere and the intrathoracic space. Partial or complete obstruction can occur when the intrathoracic negative pressure generated by the inspiratory muscles pulls on the compliant soft tissue in the upper airway, sucking the airway closed. Moreover, the nose accounts for half of the total respiratory system resistance. In this regard, the nose has been described as a variable resistor with a collapsible segment, such that flow limitation in the nasopharynx results in conditions favorable for downstream pharyngeal collapse [3].

It is unfortunate that opening of an obstructed nose using standard nasal procedures is not regularly effective in improving snoring, although it should improve nasal breathing. There are several reports that show subjective improvement; a review of eight papers found the average rate for cessation of snoring to be 41.9%, and that for reduction of snoring to be 85.3%, after correction of nasal obstruction [4].

Nasal congestion is a prominent and troublesome symptom of inflammatory disorders of the upper respiratory tract. It is a major symptom of allergic rhinitis, along with rhinorrhea, sneezing, and pruritus of the eyes, nose, and throat, particularly in patients with perennial allergic rhinitis [5] The symptoms of rhinosinusitis and nasal polyposis are similar; nasal congestion/obstruction is a key feature, accompanied by nasal discharge or postnasal drip, facial pain/pressure, and reduction or loss of smell. In chronic rhinosinusitis, nasal obstruction/congestion is typically the most common major symptom [6]. Typical sleep-related problems observed with these three conditions include sleep-disordered breathing, sleep apnea, and snoring, all of which are associated with nasal congestion/obstruction [7].

Inflammatory disorders of the upper respiratory tract are prevalent in the general population. The sleep impairment associated with these conditions, therefore, is likely to be a common problem. Allergic rhinitis, for example, is thought to affect up to 40% of the population, and its prevalence is increasing [8].

The physiologic tasks of the nose and experimental alteration of nasal resistance support the hypothesis that chronic conditions that increase nasal resistance, including permanent physical obstruction and the congestion and irritation associated with rhinitis and sinusitis, contribute to sleep-disordered breathing. Despite a strong rationale for the hypothesis, few clinical or epidemiologic studies have investigated the association. Such studies are important to determine whether the specific mechanisms identified during basic experimental studies are overcome with habituation and actually translate to measurable health outcomes that persist in daily life [9].

As nasal congestion is a subjective variant, it is more appropriate to use a scoring system [Lund–Mackay score (LMS)] to assess and compare nasal and sinus conditions.

The aim of this study was to assess the effect of management of nasal congestion (measured by LMS) on the success of treatment of snoring [measured using the apnea hypopnea index (AHI)].

Materials and methods

This study is a prospective randomized study that was conducted at the Kasr Elaini Hospital in the period from March 2010 to January 2012; 28 patients with an age range from 22 to 61 years completed the study (18 men and 10 women). After obtaining approval from the scientific committee and informed consent, all patients were subjected to the following:

(1) Detailed history taking and clinical examination, including diagnostic nasal endoscopy
(2) Computed tomography of the nose and paranasal sinuses.
(3) Overnight sleep studies were conducted using standard polysomnography. Results of electroencephalography; electrooculography; submental electromyelography; ECG; and nasal and oral airflow, arterial oxygen saturation (SaO_2), heart rate, and respiratory movement measurements were recorded. Apnea episodes were defined by the absence of ventilation for longer than 10 s, as measured by calibrated inductive plethysmography. Hypopnea was defined as a reduction in ventilation and a reduction in tidal volume to below 50%, without a major change in respiratory frequency. The respiratory disturbance index (RDI) was defined as the mean number of hypopneas and apneas per hour of sleep. Mild OSA severity (OSAS) was defined as an RDI equal to or greater than 10, but less than 20; severe OSAS as an RDI greater than 50 with the lowest SaO_2 being less than 50%; and moderately severe OSAS as falling between the criteria for mild and severe OSAS.

Exclusion criteria were as follows:

(1) Patients with major nasal anatomical abnormalities, for example severe septal deviation and nasal polyps.
(2) Patients with severe OSAS.
(3) Patients with any kind of previous surgery to the nose or palate.
(4) An LMS of less than 5.

Patients were divided randomly into two groups: the first group was given medical treatment in the form of systemic antibiotics for 10 days (amoxicillin/clavulanate) and nasal steroids (fluticasone propionate) for 1 month before palatal surgery; in the second group, no medical treatment was administered before palatal surgery.

The palatal surgeries conducted were as follows:

In the first group: uvulopalatopharyngoplasty (seven), laser-assisted uvulopalatoplasty (three), tonsillectomy (two), and radiofrequency (two).

In the second group: uvulopalatopharyngoplasty (six), laser-assisted uvulopalatoplasty (four), tonsillectomy (one), and radiofrequency (three).

Follow-up examination was carried out after 3 months to assess AHI and LMS for all patients.

Data were statistically described in terms of mean ± SD or frequencies (number of cases) and percentages when appropriate. Numerical variables in the study groups were compared using the Student *t*-test for independent samples. Within group comparisons of numerical variables were made using the paired *t*-test. For comparing categorical data, χ^2-test was performed. The exact test was used when the expected frequency was less than 5. *P*-values less than 0.05 were considered statistically significant. All statistical calculations were performed using the SPSS program (version 15; SPSS Inc., Chicago, Illinois, USA) for Microsoft Windows.

Results

Study included two groups (each contains 14 patients), group A had 10 men (71.4%) and four women (28.6%), whereas group B had eight men (57.1%) and six women (42.9%). The age in group A ranged from 22 to 50 years (mean 41.36), whereas the age in group B ranged from 29 to 61 years (mean 49.21). The *t*-test for data description of the two groups is shown in Table 1.

As regards group A, paired sample statistics are shown in Table 2.

Paired sample correlations are shown in Table 3.

Figure 1 shows pre-AHI and post-AHI in group A; Fig. 2 shows pre-LMS and post-LMS in the same group.

Table 1 *T*-test for data description

	Group	*N*	Mean	SD	SEM
Pre-AHI	Group A	14	17.64	4.845	1.295
	Group B	14	19.43	5.945	1.589
Post-AHI-	Group A	14	3.21	2.940	0.786
	Group B	14	10.00	4.385	1.172
AHI % change	Group A	14	−83.67	12.600	3.368
	Group B	14	−50.06	13.668	3.653
Pre-LMS	Group A	14	13.79	5.102	1.363
	Group B	14	13.93	4.811	1.286
Post-LMS	Group A	14	5.07	2.841	0.759
	Group B	14	12.29	4.615	1.233
LMS % change	Group A	14	−62.13	24.551	6.561
	Group B	14	−12.47	12.243	3.272

AHI, apnea hypopnea index; LMS, Lund–Mackay score.

Table 2 Group A, paired sample statistics

	Mean	*N*	SD	SEM
Pair 1				
Pre-AHI	17.64	14	4.845	1.295
Post-AHI	3.21	14	2.940	0.786
Pair 2				
Pre-LMS	13.79	14	5.102	1.363
Post-LMS	5.07	14	2.841	0.759

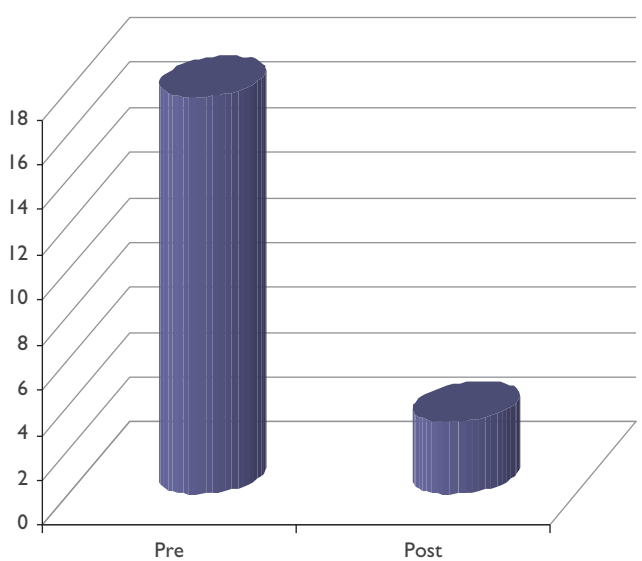
AHI, apnea hypopnea index; LMS, Lund–Mackay score.

Table 3 Paired sample correlations in group A

	N	Correlation	*P*-value
Pair 1			
Pre-AHI and post-AHI	14	0.740	0.002
Pair 2			
Pre-LMS and post-LMS	14	0.627	0.016

AHI, apnea hypopnea index; LMS, Lund–Mackay score.

Figure 1

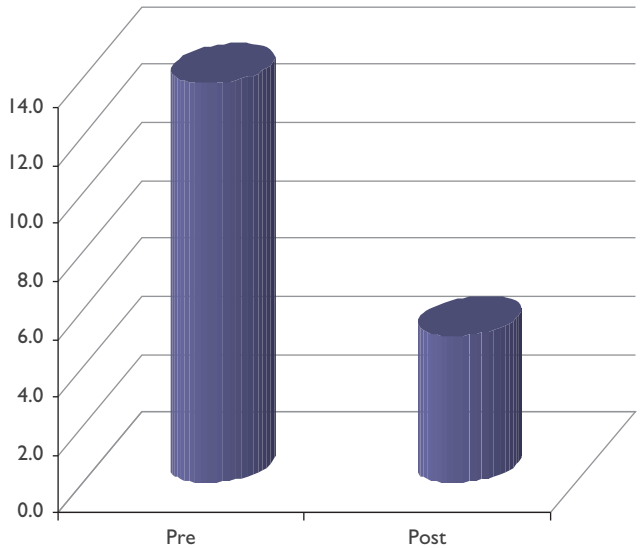


Pre-AHI and post-AHI in group A. AHI, apnea hypopnea index.

As regard group B, paired sample statistics are shown in Table 4.

Paired sample correlations are shown in Table 5.

Figure 2



Pre-LMS and post-LMS in group A. LMS, Lund–Mackay score.

Table 4 Group B, paired sample statistics

	Mean	*N*	SD	SEM
Pair 1				
Pre-AHI	19.43	14	5.945	1.589
Post-AHI	10.00	14	4.385	1.172
Pair 2				
Pre-LMS	13.93	14	4.811	1.286
Post-LMS	12.29	14	4.615	1.233

AHI, apnea hypopnea index; LMS, Lund–Mackay score.

Table 5 Paired sample correlations in group B

	N	Correlation	*P*-value
Pair 1			
Pre-AHI and post-AHI	14	0.817	0.000
Pair 2			
Pre-LMS and post-LMS	14	0.943	0.000

AHI, apnea hypopnea index; LMS, Lund–Mackay score.

Figure 3 shows pre-AHI and post-AHI in group B; Fig. 4 shows pre-LMS and post-LMS in the same group.

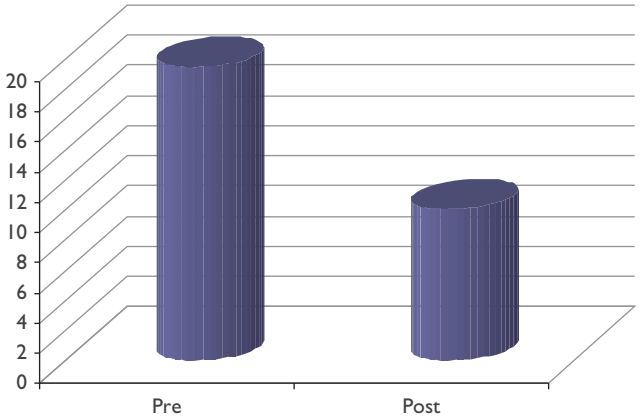
The mean percentage improvement in the AHI score and LMS in group A and group B is illustrated in Figs 5 and 6, respectively:

The mean percentage improvement in AHI score in group A and group B patients is illustrated in Fig. 7.

Discussion

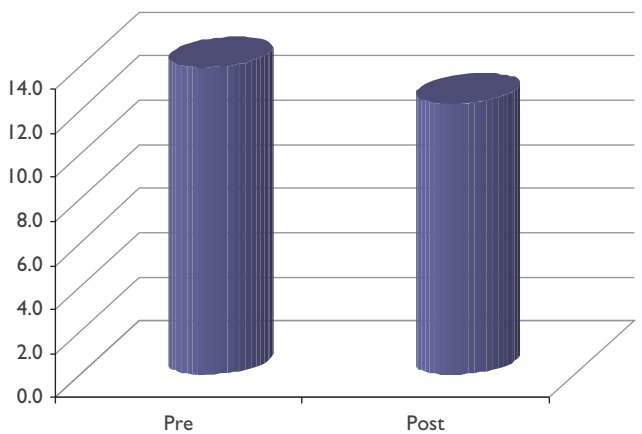
Sleep impairment associated with allergic rhinitis, rhinosinusitis, and nasal polyposis has a significant impact on patients' quality of life. Nasal congestion, one of the most common and bothersome symptoms of these conditions, is thought to be a major cause of sleep impairment and sleep-disordered breathing. Recent research has suggested that poor sleep associated with nasal congestion is

Figure 3



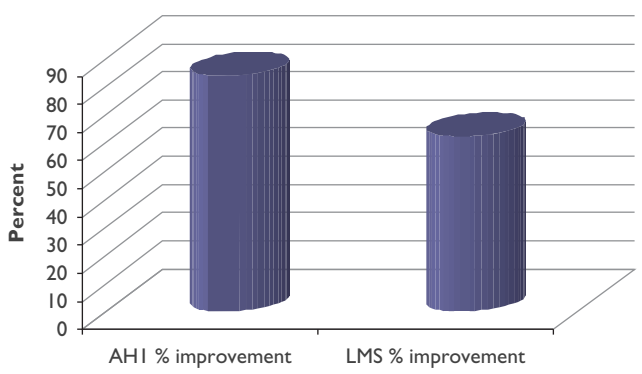
Pre-AHI and post-AHI in group B. AHI, apnea hypopnea index.

Figure 4



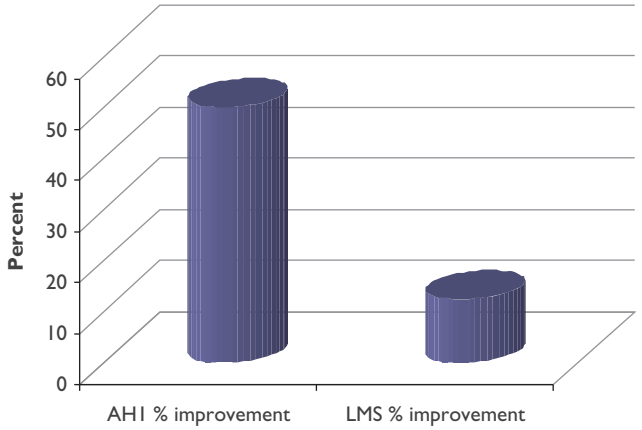
Pre-LMS and post-LMS in group B. LMS, Lund–Mackay score.

Figure 5



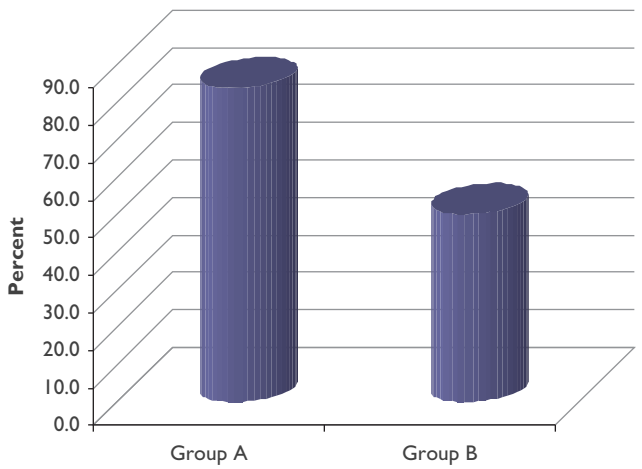
Percentage improvement in AHI and LMS in group A. AHI, apnea hypopnea index; LMS, Lund–Mackay score.

Figure 6



Percentage improvement in AHI and LMS in group B. AHI, apnea hypopnea index; LMS, Lund–Mackay score.

Figure 7



Mean percentage improvement in the apnea hypopnea index score between group A and group B.

an important therapeutic target [10]. Nasal resistances in adults do not correlate with the severity of respiratory disturbances during sleep [11], nor do they represent a risk factor for causing or worsening OSA [12] or a diagnostic predictive factor [13]; however, they are a risk factor for snoring [14]. Although a linear trend between decreased nasal airflow and greater AHI was not observed, habitual snoring was consistently associated with decreased nasal airflow, self-reported stuffiness attributed to allergy, and self-reported night-time nasal congestion or discharge. The lack of a linear relationship between nasal obstruction and severity of sleep-disordered breathing is not consistent with the physiologic hypothesis that increased nasal resistance and decreased flow increase the frequency of airway collapse. In their comprehensive review of research on nasal obstruction and sleep-disordered breathing, Olsen and Kern [14] stated that nasal obstruction is more likely to cause snoring than mild or severe obstructive sleep apnea (with frank apnea and hypopnea), and that the degree of nasal obstruction and severity of sleep-disordered breathing are not directly

correlated. The findings from the Oxford epidemiologic study [15] were consistent with this conclusion.

Self-reported chronic symptoms of rhinitis were significantly related to excessive daytime sleepiness and not feeling rested regardless of the amount of sleep. Sleep-disordered breathing, including habitual snoring, is related to hypersomnolence; therefore, associations between rhinitis and sleepiness may be explained by sleep-disordered breathing. However, it is possible that symptoms of rhinitis, independent of their effect on breathing, may cause cortical arousal and fragmented sleep. This association warrants further investigation for the management of rhinitis, because somnolence due to sleep fragmentation may be compounded by sleepiness caused by daytime use of medication [9]. Treatment of nasal obstruction has three potential goals. It can reduce nasal obstruction, reduce the severity of sleep-disordered breathing (possibly even eliminating it), or facilitate sleep-disordered breathing treatment by allowing the nose to be used more easily as a conduit for positive airway pressure therapy. Because there are multiple causes of nasal obstruction, the combination of history, physical examination, and accurate diagnosis is critical for the selection of a treatment from the long list of medical and surgical treatments [16]. In contrast, the isolated treatment of nasal obstruction does not successfully treat obstructive sleep apnea in most patients. Verse and Pirsig [17] performed a literature review that showed that medical treatment resulted in resolution of obstructive sleep apnea in 9% of patients and surgical treatment in 18%. Nasal corticosteroids can produce small changes in snoring and the AHI, but the degree of improvement varies widely [18]. A few studies specifically concerning surgical treatment showed that there was minimal to no change in the AHI, but some patients showed improvements in sleep quality and symptoms of daytime somnolence [19].

In our study, group A, which showed an improvement in nasal and sinus conditions as reflected by LMS, had better control of AHI than did group B; it is also noticed that when all other factors are standardized, the improvement of AHI is better after control of nasal and sinus conditions using antibiotics and nasal steroids.

Using LMS as an indicator of nasal and sinus conditions is more accurate and measurable than only considering nasal obstruction. Finally, there is evidence showing that nasal treatments can facilitate the treatment of sleep-disordered breathing by decreasing the magnitude of the positive airway pressure necessary to treat sleep-disordered breathing. Schonhofer *et al.* [20] showed a reasonable improvement (range 9.3–6.7 cm of water) on septoplasty, with or without inferior turbinate reduction [21], and a smaller effect (range 8.6–8.0 cm of water) was seen on using an external nasal valve dilator device. Two additional studies have shown that nasal surgery can increase the adherence to CPAP devices [22,23].

Conclusion

Treatment of nasal and sinus problems, even in the absence of major anatomical abnormalities, improves the results of palatal surgeries in controlling AHI in mild to moderate OSA.

Acknowledgements

Conflicts of interest

There are no conflicts of interest.

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