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Nasal resistance for determinant factor of nasal surgery in CPAP failure patients with obstructive sleep apnea syndrome*

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SUMMARY

***Objectives**: Given that criteria for nasal surgery in individuals with obstructive sleep apnea syndrome (OSAS) have not been proposed, we investigated the effectiveness of nasal surgery for CPAP failure in patients with both severe OSAS and nasal obstruction.*

***Patients and Methods**: Conventional nasal surgery was performed in 12 patients who were refractory to treatment by CPAP. The subject group consisted of 12 males (mean age, 54.2 ±9.2 years; range 39–66 years). The effect of nasal surgery was evaluated with data from pre-operative and postoperative polysomunography. The nasal resistance value was first deduced to determine which OSAS patients with CPAP failure should undergo nasal surgery, compared to control values.*

***Results**: Nasal surgery resulted in a significant decrease in nasal resistance, as measured by rhinomanometry, from 0.57 ± 0.31 Pa/cm^3 /sec to 0.16 ± 0.03 Pa/cm^3/sec and rendered all patients tolerant to CPAP. In addition, the lowest nocturnal oxygen saturation significantly increased from 68.3 ± 12.1% to 75.3 ± 7.1% after surgery. Subjectively, Epworth sleepiness scale (ESS) significantly decreased from 11.7 ± 4.1 to 3.3 ± 1.3 after surgery, but the number of apnea and hypopnea episodes per hour did not change significantly. In five patients, for whom it was possible to perform a CPAP titration before nasal surgery, the value decreased significantly from 16.8 ± 1.1 to 12.0 ± 1.9 cmH_2O. The bilateral nasal resistance of the 41OSAS patients with CPAP therapy (control group) was 0.24±0.11 Pa/cm^3/sec. The cut off value for defferentiation between CPAP failure patients and control group was determined as 0.38 Pa/cm^3/sec.*

***Conclusion**: Increased nasal resistance is a determinant of CPAP failure, and the surgical correction of severe nasal obstruction should thus be considered to facilitate treatment of OSAS patients with CPAP.*

Key words: nasal obstruction, CPAP, obstructive sleep apnea syndrome, nasal resistance, nasal surgery

INTRODUCTION

Increased nasal resistance or nasal obstruction has been associated with OSAS [1,2]. Nasal obstruction results in sleep-disordered breathing events including apnea, hypopnea, and snoring [1-8]. An increase in nasal resistance and the consequent shift from nasal breathing to oral breathing presumably contributes to sinkage of the base of the tongue during inspiration in sleeping individuals [4-6,8]. The correction of an obstructed nasal airway is thus considered an important component of OSAS treatment [9].

Incidentally, nasal continuous positive airway pressure (CPAP) is the standard treatment for individuals with moderate to severe OSAS [10] with many studies having demonstrated benefit from long-term use of a CPAP device. However, CPAP often fails in patients with severely narrowed nasal airways. The lack of response in such cases of nasal constriction to medical treatment often necessitates corrective surgery for the obstructive lesion. Friedman et al. [11] have demonstrated that nasal surgery can increase the daily activity, and also allow CPAP pressure to be decreased significantly. Another study also showed that nasal surgery significantly improved sleep quality and decreased daytime sleepiness [12]. However, there has been no study to clarify the relation between nasal resistance and the effectiveness of nasal surgery for OSAS patients.

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Accordingly, we investigated whether nasal resistance was the determinant factor for nasal surgery in CPAP failure patients.

PATIENTS AND METHODS

Patients and Surgery Selection

The study was carried out according to the principles stated in the Declaration of Helsinki. We examined 12 men (mean age, 54.2 ± 9.2 years; range 39 to 66 years) diagnosed with OSAS and daytime sleepiness. These individuals were refractory to treatment by CPAP, with CPAP failure being defined as use for < 4 hours per night on ≥ 70% of nights or lack of symptomatic improvement (or both) [13]. They all manifested nasal obstruction that was resistant to medical treatment and was caused by marked mucosal thickening or by a deviated septum, as revealed by nasopharyngoscopy. The OSAS patients underwent nasal surgery, either submucous resection of the nasal septum (SMR) or inferior tubinectomy (or both). A total of 41 OSAS patients with CPAP therapy diagnosed with OSAS (mean age, 50.2 ± 11.5 years; range, 28 to 75 years) was subjected to rhinomanometry for evaluation of nasal resistance. This study was designed to collect data from standard treatment. It was therefore classified as exempt by the local institutional review board.

Polysomunographic Methods

Standard polysomnography (ALICE3; Respironics, Yongwood, PA, USA) was performed in all OSAS patients. The electroencephalogram (C4-A1 and C4-O2), electrooculogram, electromyogram, and electrocardiogram were recorded continuously, and respiration was monitored with oronasal thermistors and thoracoabdominal strain gauges. Apnea was defined as a cessation of airflow through the mouth and nose for ≥ 10 s, and hypopnea was defined as an obvious reduction in airflow accompanied with either an oxygen desaturation of ≥ 3% or arousal, also for ≥ 10 s. The apnea-hypopnea index (AHI) was determined as the number of apnea and hypopnea episodes per hour. An AHI of ≥ 5 /hr was the basis for a diagnosis of OSAS [14]. The time during which nocturnal oxygen saturation (SpO_2) was < 90% (oxygen desaturation time) and the lowest SpO_2 during sleep were also determined with a pulse oximeter. We assessed percentage contributions of each sleep stage {stage 1, stage 2, stages 3 and 4, and rapid eye movement (REM)} to overall sleep [15].

Table 1. Characteristics in CPAP failure patients.

Case	Nose	Pharynx	Height/cm	Weight/kg	BMI, kg/m^2
T.Y.	DNS, CHR	n.p.	176	91	29,4
T.O.	DNS, CHR	n.p.	180	72	22,2
M.F.	CHR	LPS	165	80	29,4
T.B.	DNS, CHR	n.p.	162	75	28,6
S.A.	DNS, CHR	LPS	165	76	27,9
T.K.	DNS, CHR	n.p.	165	73	26,8
T.T.	DNS, CHR	LPS	157	75	30,4
K.M.	DNS, CHR	n.p.	163	58	22,0
K.S.	DNS, CHR	LPS	170	88	30,4
N.M.	CHR	HLT	165	86	31,7
S.M.	DNS, CS, CHR	LPS	174	58	19,2
Y.I.	DNS, CHR	n.p.	171	76	25,9

Definition of abbreviations: DNS= deflected nasal septum, CHR= chronic hypertrophic rhinitis, CS= chronic sinusitis, LPS= lower position of soft palate, HLT= hypertrophy of the linguinal tonsil, n.p.= not particular.

Surgical Techniques and Postoprative Evaluation

All 12 OSAS patients with nasal surgery had chronic hypertrophic rhinitis and one patient had sinusitis. All nasal surgeries were performed under general anesthesia. Ten patients with both hypertrophy of the inferior turbinate and a deviated nasal septum were subjected to both inferior tubinectomy and SMR with a microdebrider by endoscopy. The remaining two patients underwent only inferior tubinectomy (Table1).

Rhinomanometry

Rhinomanometry was also performed to examine the extent of nasal obstruction. Bilateral nasal resistance at ΔP100Pa was measured by active anterior rhinomanometry with a nasal nozzle and a Rhinorheograph MPR-3100 (Nihon-Kohden, Tokyo, Japan).

Daytime sleepiness

Daytime sleepiness was rated subjectively by each of 12 OSAS patients and 41 control OSAS subjects who can use CPAP with Epworth sleepiness scale (ESS) [16]. In the ESS questionnaire, the subject was asked to rate how likely he would be to doze off or fall asleep in each of eight different situations according to the following scale: 0, no chance; 1, slight chance; 2, moderate chance; 3, high chance. Total ESS scores thus could range from 0 to 24.

Statistical analysis

Data are presented as means ± SD and were analyzed by Wilcoxon's signed rank test. A p-value of < 0.05 was considered statistically significant. The cutoff value for peak negative bilateral nasal resistance at ΔP100Pa for differentiation between patients with CPAP failure and control subjects was determined from a receiver operating characteristic curve. The sensitivity [true positive/(true positive + false negative)], specificity [true negative/(true negative + false positive)], and predictive accuracy [(true positive + true negative)/total group] were determined and expressed as percentages.

RESULTS

Nasal surgery resulted in a significant decrease in nasal resistance from 0.57 ± 0.31 to 0.16 ± 0.03 Pa/cm^3/sec ($p < 0.01$), a significant increase in the lowest SpO_2 from 68.3 ± 12.1 to 75.3 ± 7.1% ($p < 0.05$), and a significant reduction in ESS score from 11.7 ± 4.1 to 3.3 ± 1.3 ($p < 0.05$). Nasal resistance and ESS score of 41 control subjects were 0.24 ± 0.11 Pa/cm^3/sec and 10.6 ± 5.1, respectively

Table 2. Summary of PSG Results before and after nasal surgery for OSAS.

	patient number	before operation	after operation	p value
Height, cm	12	167,8 ± 6,6	167,8 ± 6,6	
Weight, kg	12	75,7 ± 10,3	72,9 ± 6,5	n.s.
BMI, kg/m^2	12	27,0 ± 3,9	26,0 ± 2,8	n.s.
Nasal resistance $Pa/cm^3/sec$	12	0,57 ± 0,31	0,16 ± 0,03	< 0,01
AHI/hr	12	55,9 ± 18,2	47,8 ± 20,4	n.s.
Lowest SpO_2, %	12	68,3 ± 12,1	75,3 ± 7,1	< 0,05
ODT, min	12	111,7 ± 79,1	79,4 ± 67,3	n.s.
% stage 1	12	50,0 ± 18,3	38,2 ± 16,4	n.s.
% stage 2	12	33,7 ± 19,6	37,7 ± 19,6	n.s.
% stage 3+4	12	1,4 ± 2,6	0,3 ± 0,5	n.s.
% REM	12	14,2 ± 3,7	18,6 ± 3,8	n.s.
ESS score	12	11,7 ± 4,1	3,3 ± 1,3	< 0,045
CPAP pressure, cm H_2O	5	16,8 ± 1,1	12,9 ± 1,9	<0,045

Definition of abbreviations: BMI = body mass index, AHI = apnea-hypopnea index, SpO_2 = oxygen saturation levels, ODT = oxygen desaturation time, REM = rapid eye movement.

Although surgery did not significantly affect the oxygen desaturation time or the AHI (Table 2), all patients were able to use CPAP for > 4 hours per night on ≥ 70% of nights after surgery. The cutoff value for nasal resistance in the control group was 0.38 $Pa/cm^3/sec$ and was used to differentiate between patients with CPAP therapy and patients with CPAP failure, yielding sensitivity, specificity, and predictive accuracy values of 91.7% (11 of 12 patients), 97.6% (40 of 41 controls), and 96.2% (51 of 53 subjects), respectively.

We were able to perform CPAP titration in 5 patients both before and after nasal surgery. The value of CPAP decreased significantly from 16.8 ± 1.1 to 12.0 ± 1.9cmH_2O ($p < 0.05$) after surgery in these patients. We were only able to perform CPAP titration in the other 7 patients after nasal surgery. The AHI was 6.2 ± 2.7 /hr after surgery with CPAP, compared with a value of 55.9 ± 18.2 /hr ($p < 0.01$) before surgery (without CPAP).

DISCUSSION

Nasal resistance was significantly decreased after nasal surgery in the CPAP-refractory OSAS patients in the present study. Furthermore, all of the patients were able to use CPAP for > 4 h per night for ≥ 70% of nights after surgery. Our findings suggest that nasal obstruction is a determinant of CPAP failure. Nasal surgery may thus facilitate CPAP therapy in individuals with severe OSAS.

The nasal pathway is the preferred pathway for nocturnal breathing [1-8]. When nasal obstruction becomes severe, however, the oral pathway is utilized instead. An increase in nasal resistance results in a decrease in intraluminal pressure and collapse of the pharyngeal tissues. Nasal obstruction thus leads to various breathing disturbances, including sleep apnea, and a reduced nocturnal lowest SpO_2. As a result of its influence on the so-called nasal pulmonary reflex, nasal obstruction may also result in alveolar hypoventilation. These considerations are consistent with an increase in the nocturnal lowest SpO_2, reduced effective CPAP level, and improvement in the ability to tolerate CPAP after nasal surgery in the patients of the present study. In contrast, AHI and oxygen desaturation time did not differ in the OSAS patients compared before and after nasal surgery. In other studies [11,12,17] in which 89/96 patient were included, changes in major sleep parameters (AI, AHI, lowest SpO_2) were not significant. The difference in postoperative lowest SpO_2 between our data and others requires further study.

Chronic changes in nasal resistance have previously been shown not to be a significant risk factor for the development or severity of OSAS [18], which is consistent with our results. It is therefore possible, at least in some of the patients in our study, that obstructive sleep apnea or hypopnea was caused by multilevel upper airway obstruction rather than by nasal obstruction.

A previous case report demonstrated marked clinical improvement in 3 OSAS patients after repair of a deviated nasal septum [9]. We have now shown that nasal surgery in 12 patients with severe OSAS that was refractory to CPAP therapy resulted in a reduction in nasal resistance and rendered these individuals susceptible to treatment with CPAP. Biemann [19] and Friedman et al. [11] also reported that nasal surgery could enable the CPAP pressure to be reduced. The bilateral nasal resistance of the 12 patients was 0.57 ± 0.31 $Pa/cm^3/sec$ before surgery, with the cutoff value in the control group being 0.38 $Pa/cm^3/sec$. The switch from nasal to oral breathing has been shown to occur when total nasal resistance approaches 0.5 $Pa/cm^3/sec$ [20]. We therefore propose the following guidelines for nasal surgery in patients with severe OSAS. Such surgery is indicated in individuals: (i) with a total nasal resistance of > 0.38

Pa/cm_3/sec; (ii) who are not susceptible to treatment with CPAP; (iii) with nasal obstruction that is not amenable to medical treatment; and (iv) with nasal septum deviation or inferior turbinate hypertrophy.

In conclusion, nasal surgery resulted in a reduced nasal resistance and a consequent improvement in the ability to tolerate CPAP in OSAS patients refractory to this treatment as a result of severe nasal obstruction.

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