The importance of nasal resistance in obstructive sleep apnea syndrome

P. KERR, MD, T. MILLAR, BSc, P. BUCKLE, MD and M. KRYGER, MD, FRCPC Winnipeg, Canada

Abstract. The importance of nasal airflow resistance in the pathogenesis of obstructive sleep apnea syndrome (OSAS) remains contentious. We performed formal nocturnal polysomnography (PSG) on OSAS patients under conditions of baseline and reduced nasal resistance to answer two main questions. First, to what degree does baseline nasal airflow resistance influence upper airway collapse in OSAS patients? Second, in what proportion of the OSAS population is baseline nasal resistance contributing to the pathogenesis of upper airway collapse? Our study group consisted of 10 patients with a wide range of OSAS severity. Six of these patients had symptoms and clinical evidence of chronic nasal obstruction which, in some, was associated with markedly elevated nasal resistance. A placebo (normal saline) was instilled in the nose of each patient on the night of baseline data collection. On the treatment night of the study, nasal resistance was reduced by application of topical vasoconstrictor and insertion of vestibular stents designed to dilate the area of the nasal valve. Posterior rhinomanometry was used to measure resistance to nasal airflow immediately before and after each PSG study. Although treatment was associated with a subjective improvement in sleep quality and mean drop in nasal resistance of 73% ($P < 0.001$), there was no significant improvement in sleep architecture, nocturnal oxygenation, or the amount of apnea experienced by patients. The most significant improvement was a reduced number of arousals/hour from 52.4 ± 12.4 on placebo to 43.7 ± 10.2 on treatment ($P < 0.04$). We conclude that baseline nasal airflow resistance is of minor importance in the pathogenesis of upper airway collapse in OSAS patients, including most of those with symptoms and signs of chronic nasal obstruction. Reduced nasal resistance is associated with fewer arousals which may account for the subjective improvement in sleep quality noted on treatment.

Obstructive sleep apnea syndrome (OSAS) is caused by inspiratory collapse of the upper airway during sleep. Collapse generally occurs somewhere in the pharynx, but the precise region and mechanism of collapse varies from patient to patient. The three factors thought to be involved in the pathogenesis of airway collapse in OSAS are narrowing of the airway at the site of obstruction, hypotonicity of the pharyngeal musculature, and increased negative intrapharyngeal pressure[1].

The nasal passages can theoretically influence upper airway collapse via several different mechanisms. The most likely involves increasing negative intrapharyngeal pressure by contributing to inspiratory airway resistance upstream to the region of collapse. Control of pharyngeal muscles may be influenced by nasal airflow receptors which both regulate respiration and alter airway tone through undefined pathways. The presence and role of these receptors are matters of conjecture[2,3]. Finally, total or near total nasal obstruction forces a patient to rely on the transoral route of breathing which has been shown to be of greater resistance and may be prone to collapse during sleep[4].

Many studies indicate an association between nasal obstruction and apnea. However, the precise nature of this relationship is far from clear and the importance of resistance to nasal airflow in the pathogenesis of airway collapse in OSAS patients remains contentious. We performed formal nocturnal polysomnography (PSG) on a group of OSAS patients under conditions of baseline and reduced nasal resistance in an attempt to answer two main questions. First, to what degree does baseline nasal airflow resistance influence upper airway collapse in OSAS patients? Second, in what proportion of the OSAS population is baseline nasal resistance contributing to the pathogenesis of upper airway collapse? The answers to these questions enable one to better determine how aggressively nasal abnormalities should be treated in these patients and what sort of results can be expected.

Address reprint requests to Dr. M. Kryger, St. Boniface General Hospital, Faculty of Medicine, Respiratory Diseases, Sleep Laboratory, 351 Tache Avenue, Winnipeg, Manitoba, Canada R2H 2A6.

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METHODS

Ten OSAS patients both with and without symptoms of chronic nasal obstruction were included in the study. Each subject provided a full medical history with emphasis on sleep and nasal respiration. The upper airway was carefully assessed with emphasis on the nasal passages. Each patient underwent three consecutive nights of polysomnographic study. Night 1 was considered an orientation night for acclimatization to the lab environment. No data from Night 1 were included in the results. Placebo and treatment were applied in random order on Nights 2 and 3 (experimental nights). The placebo night involved instillation of normal saline (NS) into both nasal passages followed by baseline PSG data acquisition. On the treatment night, nasal resistance was

reduced by application of a topical nasal vasoconstrictor (.05% oxymetazalone) and insertion of vestibular stents designed to dilate the area of the nasal valve. On both the experimental nights, posterior rhinomanometry was used to measure resistance to nasal airflow immediately before and after each PSG study.

The patients filled out questionnaires on quality of sleep, ease of nasal respiration, and excessive daytime sleepiness after both the treatment and placebo nights to determine the degree of subjective improvement associated with decreased nasal airflow resistance. The patients also performed digit symbol substitution and symbol copying tests immediately after awakening from both experimental nights. These two brief (three-minute) tests are measures of concentration and were used to ascertain whether there was any obvious cognitive improvement after treatment.

A paired t-test was used to test statistical differences between placebo and treatment measures.

Details of PSG instrumentation

Each night of the study involved eight hours of overnight monitoring using standard polysomnographic techniques. During the sleep studies, surface electrodes were applied to obtain an electroencephalogram, electrooculogram, electromyogram, electrocardiogram, and recording of the heart rate. Arterial oxygen saturation (SaO_2) was recorded with a pulse oximeter (Biox 3700; Biox Inc., Boulder, Colo.) set on its fastest response. Respiration was measured using inductance plethysmography (Respitrace; Ambulatory Monitoring, Ardsley, N.Y.) and airflow was inferred from expired CO_2 (Datex 223 CO_2 analyzer; Datex Instrumentarium, Finland). Patient position was monitored either by continuous videotaping or by Vitalog sleeping position sensor. A polygraph (Model 78D; Grass Instruments, Quincy, Mass.) was used to record all the above variables on paper. End tidal CO_2, SaO_2, heart rate and plethysmography data were also stored on floppy disk via an IBM-compatible computer.

The complete polygraphic record was scored manually for sleep stage and arousals according to established criteria[5]. The manually acquired sleep stage and arousal data were then added to the records of cardiorespiratory variables on the mass storage medium. All subsequent record analyses were computerized. Apneas and hypopneas were identified and quantified by computer-based analysis of oxygen saturation[6]. The obstructive nature of the apneas was verified by visual inspection of each chart.

The main cardiorespiratory parameters analyzed were the apnea hypopnea index (AHI = number of apneas and hypopneas per hour of sleep), proportion of time spent apneic, proportion of time with SaO_2 less than 90%, mean overnight SaO_2, mean SaO_2 at nadir of desaturation episodes, and mean overnight heart rate. Arousal index (number of arousals per hour), sleep efficiency (proportion of time spent asleep during monitoring), and time spent in each stage of sleep were the parameters of sleep architecture included in the analysis.

Details of rhinomanometry

A posterior rhinomanometer was used to measure nasal resistance. Airflow was monitored using a pneumotachometer capable of maintaining laminar flow at rates up to 12 L/second attached to a Respironics nasal continuous positive airway pressure (NCPAP) mask. Nasal inlet pressure was monitored with a probe attached to one of the ports on the NCPAP mask. Nasal outlet pressure was measured using a probe that was held in the subject's mouth. Both flow and pressure probes were linked to Celesco CD 10D pressure transducers which in turn were patched directly into the analog to digital conversion (ADC) board of our IBM microcomputer. Both transnasal pressure and flow were simultaneously recorded at a rate of 40 samples/second. The flow and pressure data were ultimately displayed on an XY co-ordinate system similar to those described by other authors[7]. Nasal resistance was defined as pressure/flow (as recommended by the Committee Report on Standardization of Rhinomanometry) and calculated at a flow rate of .4 L/second[8].

Rhinomanometry was performed within one hour of the start and end of polysomnographic studies done on experimental nights. Measurements were made in a controlled environment at least 15 minutes after application of placebo or treatment and 15 minutes after the patient assumed the supine position. Subjects were told to refrain from exertion prior to each study. The patients were not allowed to use any medications of their own that could potentially alter nasal resistance while they were taking part in this study.

The nasal resistance values documented in this report represent an average of the pre- and post- PSG rhinomanometry results. They are thought to be a good estimate of mean overnight resistance. We found little variation in the pre- and post-PSG measurements on both the placebo and treatment nights. Nasal resistance only rose an average of 0.4 cm H_2O/L/s over the treatment night as the vasoconstrictor effect wore off. Continuous monitoring of nocturnal nasal airway resistance using a transnasal pressure probe was considered. However, it was felt that this sort of instrumentation would alter resistance significantly. Therefore, these techniques were not employed.

RESULTS

All 10 OSAS patients included in this study were males. The age range of the patients was 29 to 68 years with a mean age of 51 years. The body mass (BMI) in these patients ranged from 25.9 to 38.9 with a mean of 32.0 kg/m^2. All patients presented to the sleep laboratory with snoring, apnea and/or excessive daytime sleepiness. Six of the 10 patients had signs and symptoms of chronic nasal obstruction on the basis if a deviated nasal septum, narrow nasal valve region, mucosal swelling, or a

combination of these factors. All of these six patients were considered candidates for medical or surgical treatment of their nasal pathology. Two were undergoing a trial of intranasal medication as treatment for their symptoms of nasal obstruction and two were being considered for nasal surgery at the time they were referred to the sleep lab.

Table 1 is data from the questionnaires completed by the patients after each experimental night. The majority of patients found nasal respiration to be easier and sleep quality to be better while on treatment.

Figures 1 and 2 show the nasal resistance and apnea hypopnea index during placebo and treatment nights for each patient. In all subjects there was a drop in nasal resistance while on treatment. The drop in nasal resistance relative to baseline for individual patients ranged from 22 to 94%. The average drop in nasal resistance for the entire group was 73% ($P < .001$). Despite this marked reduction in resistance to nasal airflow, patients did not experience a significant improvement in the number of obstructive apneas and hypopneas/hour of sleep. Only one patient demonstrated a drop in AHI that could be considered clinically significant. His AHI on placebo was 33 and on treatment was 13. However, when the AHI for this patient was normalized for body position, the improvement in his condition was much less evident. While lying on his back, he had an AHI of 39 on placebo and 34 on treatment. While lying in any other position, he had an AHI of 2 on placebo and 4 on treatment. Another patient experienced an unexplained rise in AHI from 35 on placebo to 54 on treatment. This difference was still evident even after normalization for position.

Tables 2 and 3 summarize the effects of lowered nasal airway resistance on other cardiorespiratory parameters and sleep architecture. Average heart rate was the only cardiorespiratory parameter that demonstrated a statistically significant change dropping from 66.0 ± 3.1 beats/minute to 62.8 ± 2.8 beats/minute on treatment ($P < 0.01$). Sleep architecture in general improved minimally and only the decrease in arousal index from 52.4 ± 12.4 on placebo to 43.7 ± 10.2 on treatment attained statistical significance ($P < 0.04$).

An identical analysis of sleep ar-

Table 1. *Subjective Effects of Lowered Nasal Resistance on Sleep and Nasal Respiration in 10 Patients*

	Number of patients with subjective improvement while on	
	Placebo	Treatment
Nasal respiration	1	8
Sleep quality	2	6
Excessive daytime sleepiness	1	2

Table 2. *The Effect of Lowered Nasal Resistance on Other Cardiorespiratory Parameters During Sleep*

Parameter	Mean for all patients (± SE)	
	Placebo	Treatment
Time apneic (%)	45.8 ± 10.6	42.9 ± 11.1
Time with SaO2 < 90% (%)	21.1 ± 5.6	21.9 ± 6.3
*SaO2 (%)	92.0 ± 1.0	92.1 ± 0.9
*SaO2 at nadir (%)	84.6 ± 1.9	84.6 ± 1.6
*Heart rate (BPM)	66.0 ± 3.1	62.8 ± 2.8†

† $P < .01$.
* Values are overnight means.

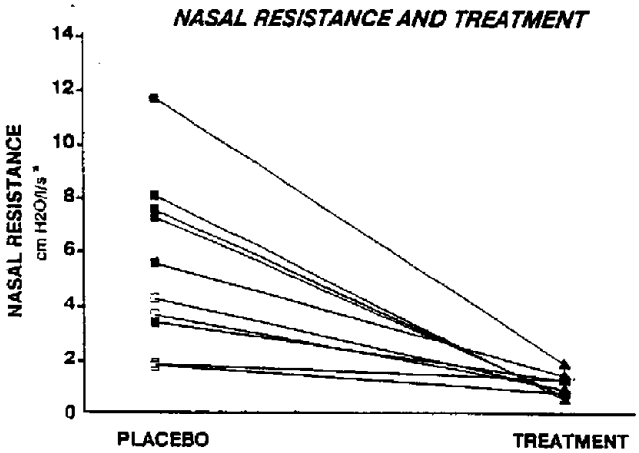

Fig. 1. Treatment effectively reduced nasal resistance in all patients. Solid markers represent subjects with chronic nasal obstruction. Open markers represent subjects without complaints of nasal obstruction (*measured at a flow rate of 0.4 L/s).

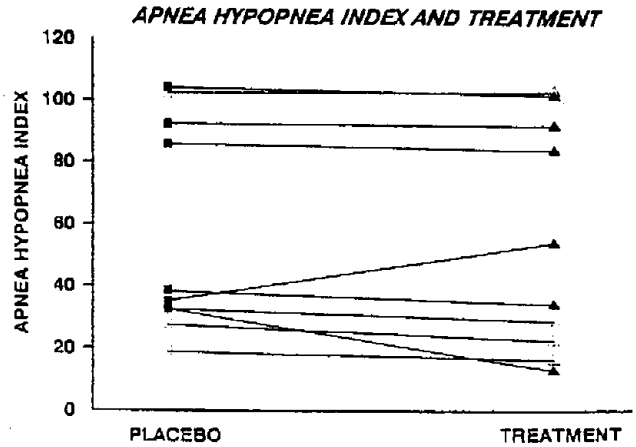

Fig. 2. Treatment did not significantly affect apnea hypopnea index. Solid markers represent subjects with chronic nasal obstruction. Open markers represent subjects without complaints of nasal obstruction.

Table 3. *The Effect of Lowered Nasal Resistance on Sleep Architecture*

Parameter	Mean for all patients (± SE)	
	Placebo	Treatment
Sleep efficacy (%)	89.0 ± 1.8	92.4 ± 1.4
Time in REM sleep (%)	20.1 ± 2.3	22.3 ± 1.4
Time in slow wave sleep (%)	5.3 ± 2.6	7.0 ± 2.4
Arousals per hour	52.4 ± 12.4	43.7 ± 10.2†

* P < 0.04.

Table 4. *The Effect of Treatment on Nasal Resistance and Apnea Hypopnea Index in Different Patient Groups*

Group	No. of Patients	Nasal Resistance* (Mean ± SE)		Apnea Hypopnea Index (Mean ± SE)	
		Placebo	Treatment	Placebo	Treatment
Patients with chronic nasal obstruction	6	7.2±1.1	1.0±0.2**	64.9±13.4	63.2±14.4
Patients who noted better sleep quality while on treatment	6	4.8±0.9	0.9±0.1***	52.9±14.7	53.5±14.7

* in cm H20/L/SEC at a flow rate of 0.4 L/sec.
** P < 0.0003.
*** P < 0.004.

chitecture, apnea hypopnea index, and other cardiorespiratory parameters was performed on two sub-groups of our subjects. One sub-group was composed of the six patients who had symptoms of chronic nasal obstruction and the other included the six patients who reported that their sleep quality had subjectively improved on treatment. Treatment successfully lowered nasal airflow resistance in both groups but did not significantly alter AHI (*Table 4*). The amount of time spent in rapid eye movement (REM) sleep increased slightly from 15.8% ± 2.6% on placebo to 18.6% ± 2.6% (P < 0.01) on treatment in the sub-group of patients complaining of chronic nasal obstruction. Heart rate also dropped significantly in this group from 66.8 ± 5 beats/minute on placebo to 62.1 ± 4.4 beats/minute (P < 0.006) on treatment. No other parameter improved significantly in either sub-group while on treatment despite the significant drop in nasal resistance.

Comparison of digit symbol substitution and symbol copying scores failed to reveal a significant difference between tests performed after placebo nights and those performed after treatment nights.

DISCUSSION

We found that substantially reducing nasal resistance had no effect on apnea hypopnea index even in patients with very high baseline resistances. Although many previous studies indicate an association between nasal obstruction and sleep apnea, the precise nature of this relationship is far from clear and the importance of nasal airflow resistance in the pathogenesis of OSAS remains contentious.

The nasal passages can theoretically influence upper airway collapse in the OSAS population by three different mechanisms. First, total or near total nasal obstruction forces a patient to rely on the transoral route of breathing which may be less stable and prone to collapse during sleep. This degree of nasal obstruction is rare and therefore is unlikely to be of pathogenic importance in the majority of OSAS patients. In addition, studies attempting to verify this theory have yielded conflicting results. Several authors have reported increased amounts of sleep apnea experienced by non-OSAS adult subjects during periods of acute artificially-induced total nasal obstruction[4,9-12]. However, there is disagreement regarding the predominant nature of apnea induced (*i.e.*, central or obstructive) and the clinical significance of the effects are unclear. The increases in central apnea observed in some studies suggest that the nose may play a bigger role in control of breathing than in determining airway patency.

A second theory relating the nasal airway to OSAS is based on the possible presence of receptors sensitive to nasal airflow that maintain pharyngeal muscle tone through undefined reflex pathways. Two studies have failed to clearly verify this hypothesis. Both White, *et al* and McNicholas, *et al* studied healthy, non-OSAS adults polysomnographically after blocking nasal airflow sensation with topical anesthetic. Both of these studies demonstrated small increases in the number of nocturnal disordered breathing events during periods of nasal anesthesia. However, these abnormal breathing episodes included both central and obstructive events, and the increase in obstructive apnea was not statistically significant in either study[2,3].

The third mechanism by which the nose could influence airway collapse in OSAS relates to its contribution to airway resistance upstream to the region of collapse. Elevated upstream airway resistance (*i.e.*, nasal obstruction) could result in lower intrapharyngeal pressure and a predisposition to collapse. Studies in non-OSAS subjects with partial nasal occlusion have again failed to clearly support this theory. Lavie, *et al* monitored adults with artificially-induced unilateral nasal obstruction during sleep and found only mildly disordered breathing that was of central origin[10]. In a study by McNicholas, *et al* patients who suffered from allergic rhinitis were

studied polysomnographically both during ragweed season when they were symptomatic and out of ragweed season when their symptoms were improved. Despite a two-fold increase in average nasal resistance during ragweed season, the mean number of obstructive apneas only increased from 0.7/hour to 1.7/hour[13]. The results of this study suggest that commonly encountered degrees of nasal obstruction only marginally increase the frequency of obstructive apnea in normal adults. One can argue that partial nasal occlusion may still be of great importance in the OSAS population because of increased pharyngeal compliance or collapsibility, but this is yet to be proven.

Several studies have attempted to demonstrate that surgical treatment of OSAS patients with nasal abnormalities will reduce the severity of their apnea. These are the only reports that try to directly demonstrate the importance of nasal resistance in the OSAS population. Unfortunately, these studies have all included only small numbers of highly selected patients (*i.e.*, those with severe nasal abnormalities) and have yielded inconsistent results that are difficult to interpret. All of these series neglect to quantitate nasal resistance or even objectively demonstrate lowered resistance post-surgery. Ruben, *et al* published the largest series of OSAS patients assessed polysomnographically both pre- and post-nasal surgery. Although the majority of the nine patients in this study seemed to improve after nasal surgery, the AHI of some patients worsened or remained unchanged and the results did not attain statistical significance[14]. Dayal, *et al* reported six patients who underwent nasal surgery as treatment for OSAS. PSG was again performed pre- and post-operatively. Although all the patients subjectively improved postoperatively, the AHI improved in only three patients, worsened in two, and remained essentially unchanged in one[15]. In a discussion of the surgical management of OSAS, Simmons, *et al* reported the results of treating three OSAS patients complaining of nasal obstruction with nasal surgery alone. Two of the patients were not improved. The third, although symptomatically better, had no improvement in his OSAS when assessed objectively[16]. Finally, Olsen, *et al* reported a single case of a 55-year-old male with chronic nasal obstruction and severe OSAS (AHI 72) who had his apnea abolished with only nasal surgery[17]. The interesting point in this case is that the patient's nasal resistance was low even pre-operatively (1.9 cm H_2O/L/second). Although the patient's post-operative nasal resistance was slightly lower (1.4 cm H_2O/L/second), it is unlikely that the vast improvement in AHI was due to such a small absolute drop in nasal resistance. At best, these studies have only revealed a possible role for nasal resistance in some highly selected OSAS patients.

We felt that the relationship of nasal airflow resistance and OSAS was in need of more careful assessment and designed our study to answer two main questions. First, to what degree does baseline nasal airflow resistance influence upper airway collapse in OSAS patients? Second, in what proportion of the OSAS population is baseline nasal resistance contributing to the pathogenesis of upper airway collapse? To address these questions we gathered a group of 10 patients with a wide range in OSAS severity and baseline nasal resistance. Six of these patients were symptomatic for chronic nasal obstruction and were candidates for surgical or medical treatment of their nasal disorders. All patients were assessed polysomnographically under conditions of carefully controlled baseline and reduced nasal resistance.

To lower nasal resistance as much as possible on the treatment night, we elected to use vestibular stents designed to dilate the region of the nasal valve and a topical vasoconstrictor. Previous reports have demonstrated the efficacy of devices that dilate the nasal valve region in reducing a resistance to nasal airflow[18,19]. The combination of stents and vasoconstrictor proved to be an effective technique for reduction of nasal resistance in each of our patients and its reversibility enabled us to randomize placebo and treatment nights. The reduction in nasal resistance we achieved was almost certainly as great or greater than that expected with the use of usual therapeutic means such as surgery.

The majority of patients sensed improvement of their nasal respiration, felt their nocturnal breathing was more comfortable, and thought their sleep was more restful while on treatment. Despite these subjective improvements and an average drop in nasal resistance of 73%, the only sleep parameter in our study group that improved significantly was the arousal index. The most important mechanism of arousal in OSAS may be increased respiratory effort and amplitude of intrapleural pressure swings[20]. If this is true, the observed drop in arousal index may indicate that lowered nasal resistance had at least partially relieved airway obstruction and reduced the work of breathing in our patients. Although other parameters such as AHI, sleep efficiency, the amount of time in REM sleep and the amount of time in slow wave sleep showed signs of subtle improvement, these changes were of neither statistical nor clinical significance. Oxygenation was not improved during treatment nights. The drop in heart rate on treatment may be a reflection of fewer arousals, may be a subtle indication that subjects had decreased work of breathing, or may represent a systemic reaction to the use of topical vascoconstrictor (alpha agonist).

As discussed in the results, a significant drop in AHI was seen in one patient while on treatment. However, the improvement in his condition was more likely secondary to a change in sleep position than the drop in nasal resistance. Another patient had a significant rise in AHI on treatment for which no cause could be identified. These two cases illustrate the potential

variability of PSG scores and stress the need for carefully controlled studies.

We analyzed the data of two sub-groups that were expected to attain more benefit from treatment than the study group as a whole. One sub-group consisted of patients with chronic nasal obstruction and the other consisted of those who noticed greatest subjective improvement in sleep quality during the night of lowered nasal resistance. Apart from an increased REM sleep time on treatment seen in the patients with chronic nasal obstruction, analysis of both sub-groups failed to reveal an improvement on treatment that was any more impressive than that seen in the whole group.

We believe these results indicate that baseline nasal airflow resistance generally plays a minor role in generating upper airway collapse in the majority of OSAS patients, including most of those with symptoms and signs of chronic nasal obstruction. The only OSAS patients for which this may not be true are those with very high grade nasal obstruction (*i.e.*, total or near total occlusion throughout the night). We believe these patients account for a very small portion of the OSAS population. In the six months required to complete this study, we screened all the OSAS patients seen in our laboratory who complained of chronic nasal obstruction and did not find any with nasal resistance greater than 10 cm H_2O/L/second other than the one included in our study. Other authors have also demonstrated that patients with nasal resistance greater than 10 cm H_2O/L/second at a flow rate of 0.4/L/second are extremely rare even in the population with chronic nasal obstruction[21,22].

Our findings agree with recent work by Blakley, *et al* in which they demonstrated an absence of correlation between apnea hypopnea index and nasal resistance and concluded that the nose is not generally a major factor in the development of snoring and apnea[23]. Studies that have demonstrated an increased frequency of apnea and sleep disruption in non-OSAS patients with nasal obstruction leave little doubt that the nose plays some role in control of breathing and affecting pharyngeal airway patency. However, this does not necessarily means that nasal obstruction is an important factor in the pathogenesis of airway collapse in the OSAS population. Indeed, our study suggests it generally is not.

Previous reports have demonstrated a poor correlation between symptomatic improvement of OSAS patients on treatment and reduced AHI[24]. This problem was evident in our subjects when lowered nasal resistance was used as treatment. As AHI has been clearly correlated with increased morbidity and mortality rates, it is important to objectively assess patients post-treatment to ensure that apnea frequency has been adequately reduced[25,26]. This is especially important if the treatment used appears to be of questionable efficacy, as is the case with procedures designed to reduce nasal resistance.

Several studies such as those by Zwillich, *et al* and Lavie, *et al* have noted that an increased arousal index is one of the more prominent and consistent effects of nasal obstruction on sleep in the non-OSAS population[12,27]. Our study suggests this is also true for OSAS patients. A reduction in arousal index may account for the subjective improvement in sleep quality seen in most patients during periods of lowered nasal resistance.

The predominance of mouth breathing during sleep in the OSAS population may explain why baseline nasal resistance is of little importance in this syndrome. To investigate this possibility, we performed a further sleep study on one of our patients under conditions of reduced nasal resistance and forced him to breathe through only his nose by closing his mouth with a chin strap. Although the apnea was not abolished, there were striking differences noted when the data from this night was compared to that acquired on the regular treatment night of our study. By forcing the patient to breathe through his nose, both the number and duration of apneas was reduced, resulting in a 23% drop in the total amount of time spent apneic. Oxygenation was clearly improved. The proportion of time spent with an SaO_2 of less than 90% dropped from 41 to 22% and the average nadir SaO_2 with each apneic episode dropped from 22 to 15%. These results both implicate transoral respiration as an important factor in the pathogenesis of OSAS and support our suggestion that the nasal airway is of little importance because it is generally bypassed. The fact that apnea was not completely controlled despite being forced to breathe through very low resistance (1.3 cm H_2O/L/s) nasal passages implies that airway collapse often occurs in these individuals even without the influence of lowered intrapharyngeal pressure. We hope to confirm these findings in the future by studying a series of OSAS patients during periods of oral occlusion.

CONCLUSIONS

Resistance to nasal airflow likely plays some role in control of breathing and influencing pharyngeal airway patency. However, our results indicate that baseline nasal airflow resistance is of minor importance in the pathogenesis of airway collapse in OSAS patients, including most of those with signs and symptoms of chronic nasal obstruction. Despite a marked reduction in nasal resistance and a subjective improvement in sleep quality while on treatment, our patients showed no significant improvement in most sleep parameters including apnea hypopnea index. Arousal index and mean heart rate were reduced which suggests that lowered nasal resistance partially relieved airway obstruction and reduced the work of breathing.

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Sommaire. L'importance de la résistance du flot aérien nasal dans la pathogenèse du syndrome de l'apnée obstructive du sommeil (OSAS) rest litigieuse. Nous avons effectué des polysomnogrammes nocturnes (PSG) sur des patients souffrant d'OSAS avec une résistance nasale de base puis avec une résistance réduite, dans le but de pouvoir répondre à deux importantes questions. Premièrement, à quel degré, une résistance de base du flot aérien nasal peut influencer le collapsus des voies respiratoires supérieurs des patients OSAS? Deuxièmement, dans quelle proportion d'une population à OSAS, la résistance nasale de base interviendrait dans la pathogenèse du collapsus des voies respiratoires supérieures? Notre étude se fait sur un groupe de 10 patients souffrant d'OSAS à différents degrés de sévérité. Six de ces patients avaient des symptômes et une évidence clinique d'obstruction nasale chronique, associés pour quelques uns d'une élévation marquée de la résistance nasale. La nuit de l'enregistrement des données de base, un placebo (sérum physiologique salin) a été instillé dans les narines de chaque patient. La nuit d'enregistrement, où le traitement a été appliqué, la résistance nasale a été réduite par l'application topique d'un vasoconstricteur et par l'insertion d'un moule vestibulaire pour dilater la zone de la valve nasale. Une rhinomanométrie postérieure donnait les mesures de la résistance du flot aérien nasal immédiatement avant puis après chaque étude du PSG. Bien que le traitement fut suivi d'une amélioration subjective dans la qualité même du sommeil et par une chute de la résistance nasale de 73% ($P < 0.001$), on ne retrouvait pas d'amélioration significative dans l'architecture du sommeil, l'oxygénation nocturne ou le nombre d'apnée effectué par les patients. L'amelioration la plus évidente était la diminution du nombre de réveil/heure de 52.4 ± 12.4 avec le placebo contre 43.7 ± 10.2 avec le traitement ($P < 0.04$). En conclusion, la résistance de base du flot aérien nasal reste d'une importance mineure dans la pathogenèse du collapsus des voies respiratoires supérieures chez les patients à OSAS, même chez les patients symptomatiques d'une obstruction nasale. Toute diminution de la résistance nasale par des traitements, n'améliore que subjectivement la qualité du sommeil, en réduisant les réveils.

REFERENCES

1. Block AJ, Faulkner JA, Hughes RL, *et al:* Factors influencing airway closure. *Chest 1984:* 86, 114–122.
2. White DP, Cadieux RJ, Lombard RM, *et al:* The effects of nasal anesthesia on breathing during sleep. *Am Rev Respir Dis 1985:* 132, 972–975.
3. McNicholas WT, Coffey M, McDonnell T, *et al:* Upper airway obstruction during sleep in normal subjects after selective topical oropharyngeal anesthesia. *Am Rev Respir Dis 1987:* 135, 1316–1319.
4. Olsen KD, Kern EB, Westbrook PR: Sleep and breathing disturbance secondary to nasal obstruction. *Otolaryngol Head Neck Surg 1981:* 89, 804–810.
5. Rechtschaffen A, Kales A: *A manual of standardized terminology, techniques and scoring system for sleep states of human subjects.* NIH publication number 204. Washington, D.C.: USGPO, 1968.
6. George CF, Millar TW, Kryger MH: Identification and quantification of apneas by computer-based analysis of oxygen saturation. *Am Rev Respir Dis 1988:* 137, 1238–1240.
7. Broms P, Jonson B, Lamm CJ: Rhinomanometry: A system for numerical description of nasal airway resistance. *Acta Otolaryngol 1982:* 94, 157–168.
8. Clement PAR: Committee report on standardization of rhinomanometry. *Rhinology 1984:* 22, 151–155.
9. Taasan V, Wynne JW, Cassisi N, *et al:* The effect of nasal packing on sleep disordered breathing and nocturnal oxygen desaturation. *Laryngoscope 1981:* 91, 1163–1172.
10. Lavie P, Fischel N, Zomer J, *et al:* The effects of partial and compiete mechanical occlusion of the nasal passages on sleep structure and breathing in sleep. *Acta Otolarygol 1983:* 95, 161–166.
11. Suratt PM, Turner BL, Wilhoit SC: Effect of intranasal obstruction on breathing during sleep. *Chest 1986:* 90, 324–329.
12. Zwillich CW, Pickett C, Hanson FN, *et al:* Disturbed sleep and prolonged apnea during nasal obstruction in normal men. *Am Rev Respir Dis 1981:* 124, 158–160.
13. McNicholas WT, Tavlo S, Cole P, *et al:* Obstructive apneas during sleep in patients with seasonal allergic rhinitis. *Am Rev Respir Dis 1982:* 126, 625–628.
14. Rubin A-HE, Eliaschar I, Joachim Z, *et al:* Effects of nasal surgery and tonsillectomy on sleep apnea. *Bull Europ Pathophysiol Resp 1983:* 19, 612–615.
15. Dayal VS, Phillipson EA: Nasal surgery in the management of sleep apnea. *Ann Otol Rhinol Laryngol 1985:* 94, 550–554.
16. Simmons FB, Guilleminault C, Dement WC, *et al:* Surgical management of airway obstruction during sleep. *Laryngoscope 1977:* 87, 326–338.
17. Olsen KD, Suh KW, Staats BA: Surgically correctable causes of sleep apnea syndrome. *Otolaryngol Head Neck Surg 1981:* 89, 726–731.
18. Haight JSJ, Cole P: The site and function of the nasal valve. *Laryngoscope 1983:* 93, 49–55.
19. Petruson B: Improvement of nasal airflow by the nasal dilator Nozovent. *Rhinology 1988:* 26, 289–292.
20. Gleeson K, Zwillich C, White DP: The influence of increasing ventilatory effort on arousal from sleep. *Am Rev Respir Dis 1990:* 142, 295–300.
21. Schwartz HJ, Arnold JL, Decker MJ, *et al:* Posterior rhinomanometry as a rapid screening test for nasal dysfunction. *Am J Rhinology 1990:* 4(2), 1–5.
22. McCafferty TV, Kern EB: Rhinomanometry. In: English GM, ed: *Otolarygology Vol. 2.* Philadelphia: Harper and Row, 1986, pp 12–14.
23. Blakley BW, Mahowald MW: Nasal resistance and sleep apnea. *Laryngoscope 1987:* 97, 752–754.
24. Wetmore SJ, Scrima L, Snyderman NL, *et al:* Postoperative evaluation of sleep apnea after uvulopalatopharyngoplasty. *Laryngoscope 1986:* 96, 738–741.
25. He J, Kryger MH, Zorick FJ, *et al:* Mortality and apnea index in obstructive sleep apnea. *Chest 1988:* 94, 9–14.
26. Hung J, Whitford EG, Parsons RW, *et al:* Association of sleep apnea with myocardial infarction in men. *Lancet 1990:* 336, 261–264.
27. Lavie P, Gertner R, Zomer J, *et al:* Breathing disorders in sleep associated with "microarousals" in patients with allergic rhinitis. *Acta Otolarygol 1981:* 92, 529–533.