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Anterior Nasal Resistance in Obese Children with Obstructive Sleep Apnea Syndrome (OSAS)

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Abstract

Objective/Hypothesis—To evaluate nasal resistance in obese children with and without OSAS, study the correlation between nasal resistance and severity of OSAS using the apnea-hypopnea index (AHI), and examine the association of gender and body-mass index (BMI) with this measurement.

Study Design—Retrospective analysis

Methods—Active anterior rhinomanometry was used to determine anterior nasal resistance (aNR) during wakefulness in the supine position during tidal breathing. Thirty obese children with OSAS (age 13.8±2.6 yrs, BMI-Z score 2.6±0.4) and 32 matched obese controls (age 13.6±2.3 yrs, BMI-Z score 2.4±0.4), were studied. Unpaired t-tests and Spearman correlation were performed.

Results—The OSAS group had significantly higher aNR than the non-OSAS group during inspiration (p=0.012) and expiration (p=0.003). A significant correlation between inspiratory aNR and AHI was found for the OSAS group (r=0.39, p=0.04). The aNR did not correlate with BMI-Z score or with either gender.

Conclusions—We noted a higher aNR in obese children with OSAS as compared to obese controls and the aNR on inspiration correlated significantly with AHI. These findings suggest that a causal or augmentative effect of high inspiratory aNR may exist for obese children who exhibit OSAS.

Keywords

AAR: Active anterior rhinomanometry; Obesity; OSAS: Obstructive sleep apnea syndrome

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Conflict of Interest: None

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) is a respiratory disorder characterized by repeated episodes of flow limitation or complete cessation of flow due to partial narrowing or complete occlusion of the pharyngeal airway during sleep ¹. These respiratory events are followed by alterations in gas exchange arousals leading to disruption of normal sleep pattern.

OSAS affects 2–4% of children in the general population ². However, obese children have a much higher prevalence of the disorder that may approach 50% ^{3–5}. Thus, obesity is an important risk factor for the development of OSAS in children. Several studies suggest that particular anatomical factors around the pharyngeal airway including lymphoid and parapharyngeal fat pad tissues in obese children induce sleep apnea by narrowing the upper airway ^{6–8}. For a given inspiratory flow rate in the airway, increased airway resistance anterior to a given point in the airway will increase the magnitude of negative pressure loading at that point, favoring its narrowing and/or collapse. Additionally, this will be facilitated if there is no increased reflex activation of airway to maintain the airway patency. Distal to the choanae, the pharynx is particularly liable to collapse, especially in the region of the soft palate, tonsils and adenoids. Likewise, the oropharynx is susceptible to collapse due to the tongue, tonsils and the distensible nature of the surrounding structures comprising the airway.

Although adenotonsillectomy is considered the first-line treatment in OSAS in obese children with adenotonsillar hypertrophy, up to 50% may still have unresolved OSAS after their surgery ^{9–11}. This suggests that other factors not ameliorated by adenotonsillectomy contribute to OSAS in obese children. Factors to be considered include: low upper airway muscle tone, increased parapharyngeal fat and upper airway tissue fat content, altered chest-wall mechanics, all that could increase upper airway collapsibility during sleep; as well as anatomical abnormalities of nasal passages such as: nasal septal deviation, nasal turbinate hypertrophy, and allergic rhinitis, that could increase upper airway nasal resistance and perpetuate OSAS in these subjects.

The relationship between nasal resistance and OSAS is not well defined ¹². Several studies using a standardized method known as active anterior rhinomanometry (AAR) have shown that adults with OSAS have a higher anterior nasal resistance (aNR) compared to controls ^{13,14}. However, data in children and particularly in obese children is lacking. Thus, the main aim of the study was to use a standardized method of AAR to evaluate the relationship between aNR and the occurrence of OSAS in obese children as compared to controls. We hypothesize that obese children with OSAS have an increase in aNR that pre-loads the nasopharynx and oropharynx and that this resistance will correlate with severity of the disorder. Such an abnormality may also help explain the low response to AT in these subjects. A secondary aim was to examine the role of gender and body mass index (BMI) on aNR in these groups.

METHODS

Subjects and Procedures

All children were recruited at the Children's Hospital at Montefiore (CHAM), Bronx, NY, USA. The study was approved by the Committee of Clinical Investigations at Albert Einstein College of Medicine, Bronx, NY. Sixty-four obese children with intact tonsils and adenoids in the age range of 8 to 17, (BMI > 95th percentile for age and gender) were initially enrolled into the study. After an overnight polysomnography (PSG) study, each subject was classified as an OSAS or a Control. Subjects were excluded if they had abnormal development or a known metabolic or endocrine disorder. Subjects were also excluded if they had complete unilateral or bilateral nasal obstruction, which precludes measuring nasal resistance with anterior rhinomanometry. Thus, 2 of the OSAS subjects were excluded from final analysis due to unilateral nasal obstruction.

Overnight Polysomnography

To evaluate for the existence and severity of OSAS an overnight polysomnography (Xitek, Oakville, ON, Canada) was conducted at the Sleep Disorders Center at the CHAM. OSAS was defined if the apnea index (AI) was ≥ 1 /hour and/or the AHI was ≥ 5 /hour. Sleep scoring was performed according to standard criteria as defined by the American Academy of Sleep Medicine ¹⁵.

Active Anterior Rhinomanometry

To measure aNR we used the AAR technique ¹⁶. Accordingly, transnasal pressure and airflow were measured separately in each nostril during quiet tidal breathing in the supine position during wakefulness. Airflow and pressure were collected by a research grade clinical Rhinomanometer (GM Instruments NR6, Kilwinning, KA13 6U, UK) with a face mask. Patients were allowed to blow their nose prior to test if needed. We used disposable foam plugs fitted with a pressure sensing tube and inserted into one nostril to carry out the pressure measurement while airflow through the other nostril was measured by a pneumotachometer. Measurement of the opposite nostril was performed in the same fashion. Thus, pressure measured in the occluded nostril is equivalent to the driving pressure at the choanal junction because the air column in the occluded nostril is stagnated. Subjects maintained a closed mouth during the measurement. Measurements included four breathing phases: ascending and descending phases during inspiration and expiration.

Unilateral nasal resistance (uNR)—Nasal resistance of each nostril was defined at the 150 Pa standard transnasal pressure value (Figure 1). Twelve respiratory cycles were recorded and averaged for each subject during inspiration and expiration. Measurements were discarded if flow and pressure waveforms were atypical of normal breathing.

Anterior nasal resistance—aNR was calculated using the two parallel resistances addition: $(uNR_R \times uNR_L) / (uNR_R + uNR_L)$, where uNR_R and uNR_L are Right and Left unilateral measurements, respectively.

Statistical Methods

Demographics, polysomnographic, and rhinomanometry data are presented as mean $\pm$ standard deviation (SD) values for OSAS and control groups. Endpoints between groups were compared using two-tailed unpaired t-tests and Spearman correlation analysis was performed to evaluate the association between aNR and: AHI, gender, and BMI-Z score for all subjects. A $p < 0.05$ was considered significant. A commercial statistical software package (SigmaPlot 12, San Jose, CA, USA) was used for all statistical computations.

RESULTS

Demographic characteristics are summarized in Table 1. Obese control subjects ($n = 32$) with the mean age of 13.6 ± 2.3 years (range 8–17 years) and obese OSAS subjects ($n = 30$) with mean age of 13.8 ± 2.6 years (range 8–17 years) were studied. 12 females (37%) in control and 9 females (30%) in OSAS were included. BMI Z-scores of control and OSAS patients were 2.4 ± 0.4 and 2.6 ± 0.4 , respectively. The control group included: 17 Hispanic, 11 African American and 4 other ethnicities and the OSAS group included: 16 Hispanic, 11 African American and 3 other ethnicities. Eleven of the 32 (34%) of controls and 6/30 (20%) of the OSAS subjects had history of allergic rhinitis ($p = \text{NS}$). There were no significant differences in demographic characteristics between groups.

Polysomnographic data are presented in Table 2. Significant differences between controls and OSAS were noted, particularly in regard to: Apnea Index (AI, events/hr) 0.1 ± 0.3 vs. 3.8 ± 5.9 ($p = 0.002$), AHI 1.5 ± 1.6 vs. 24.2 ± 25.0 ($p < 0.001$), SpO_2 Nadir (%) 92.9 ± 3.3 vs. 82.1 ± 6.9 ($p < 0.001$). Arousal Index (events/hr) was also significantly higher in OSAS (24.3 ± 20.2) than control (6.8 ± 3.6) ($p < 0.001$). For all subjects the correlation coefficient between AI and AHI was 0.73 ($p < 0.001$).

Results of aNR are presented in Table 3. Inspiratory and expiratory aNR were significantly higher in OSAS subjects than in controls. The mean inspiratory aNR for controls was 0.58 ± 0.36 vs. 1.47 ± 1.92 kPa/l/s, in OSAS ($p = 0.012$), and the mean expiratory aNR for controls was 0.62 ± 0.35 kPa/l/s vs. 1.48 ± 1.53 kPa/l/s in OSAS ($p = 0.003$). The correlation between aNR and AHI as a measure of severity of OSAS was significant for inspiratory resistance ($r = 0.39$, $p = 0.04$, Figure 2), but not for expiratory resistance.

Finally, since aNR could be affected by both body fat composition and gender we analyzed our data accordingly. The subgroups were composed of 12 female controls (age 13.7 ± 1.8 years), 20 male controls (age 13.5 ± 2.6 years), 9 female OSAS (age 13.1 ± 3.1 years), and 21 male OSAS (age 14.1 ± 2.4 years). No significant differences were noted in demographic characteristics of the four subgroups. In this latter analysis, we found no correlation between aNR and each gender or between aNR and BMI-Z score.

DISCUSSION

The possible contribution of obstruction of the nasal passages to the occurrence of OSAS has been of interest for more than a decade. However, the relationship between nasal resistance and OSAS is still ill-defined and no consensus is found in reviewing previous

studies. Several studies reported that nasal resistance measured by a posterior rhinomanometry¹⁷ in the sitting position correlated positively with increasing AHI in non-obese middle age subjects^{14,18}. More recently, Tagaya et al.¹³ expanded on the significance of aNR in the obese adult population with OSAS, using a more recent and standard technique of AAR¹⁶, as in our study. These authors have shown that increased aNR caused by obstructed nasal passages could play an important role in the causation of OSAS. In a pediatric study of children between 4 and 7 year of age Rizzi et al.¹⁹ determined that aNR correlated positively with the severity of OSAS. They performed their measurements in an erect position in contrast to the supine position used in this study.

Conversely, other investigators have presented results showing that aNR was not a significant risk factor in the severity of OSAS in adults²⁰. Their rhinomanometry was performed in the seated posture during wakefulness. De Vito et al.²¹ also analyzed AAR data obtained from both the seated and supine position. However, they could not find any statistical significance between aNR and the severity of OSAS in the middle age population. In a recent study of awake adults with OSAS, Maria Masdeu and colleagues²² utilized AAR and acoustic rhinometry which provides a cross sectional area profile of the nasal passages. They compared these in both supine and seated posture but they did not find a correlation between either of these two measurements and the severity of OSAS.

Disagreement in these previous studies could result from the differences in measuring techniques, postural differences, or the surveyed populations. Rhinomanometry measurements themselves have also been historically questioned due to apparent inconsistencies. Thus, the International Rhinology Society established the universal standard called four-phase Rhinomanometry to determine aNR¹⁶. Four-phase AAR has advantages as the need for patient cooperation is minimized and results are quickly produced. It should be noted that anterior rhinomanometry measures resistance from the nasal opening back to the choanal junction. Posterior rhinomanometry measures that resistance combined with resistance due to the airway segment from the choanal junction to the pressure sensor location, usually placed in the region of the oropharynx. Thus, posterior rhinomanometry will by definition also include any resistance due to the nasopharynx. In subjects with OSAS and/or obesity, the resistance of the nasopharynx is an important factor but can confound the question of how anterior nasal resistive loading alone relates to OSAS. This study utilized the standardized anterior method. Additionally, all data were acquired in the supine position to mimic the sleeping position as much as possible because postural change may induce alterations in nasal resistance.

On describing the role of obesity in OSAS, obesity is known to be a primary risk factor²³ and has also been linked to increased aNR²⁴. Our data show that the obese OSAS group had a higher aNR than obese controls, but were not significantly more obese. On that basis, it appears that higher aNR is not likely to be due to anatomical effects of fat deposition that would reduce airway caliber of the nasal vestibule, valve, or turbinates. Rather, this suggests that having a high aNR for any OSAS-independent reason may be a risk factor for precipitating OSAS in obese subjects already susceptible, or making OSAS worse if they already have it. However, we cannot exclude the possibility that the higher aNR found in the OSAS group could be caused or exacerbated by effects of having pre-existing OSAS. For

example, it is possible that chronic snoring, chronic colds, sinus infections and metabolic changes may serve to cause inflammation of the turbinates and contribute to OSAS in a positive feedback effect.

In this study we show a new finding in which aNR is strongly associated with the severity of OSAS in obese children. In contrast, Virkkula et al.¹⁴ reported weak correlation between aNR and sleep parameters in middle-age obese subjects. However, they attributed the relationship to failed AAR measurement by severe obstruction in either or both nostrils. Since it is generally considered that 50 – 75% of total respiratory airway resistance is due to the normal nasal passage²⁵, anterior nasal resistance should be considered as an important risk factor in sleep medicine. As an example, patients whose nasal passage is obstructed seriously frequently alter to oral breathing to compensate for high resistance during sleep, which may predispose them to apnea as oral breathing may reduce pharyngeal airway size by changing the tongue and jaw's location²⁶.

The higher aNR observed in obese children with OSAS could result from various anatomical abnormalities, the deformation of nostrils by septal deviation, flow restricted by the inflamed nasal mucosa, hypertrophied nasal turbinates, nasal polyps and paranasal sinus disease^{27,28}. We can postulate that flow limitation through these mechanisms could create large negative pharyngeal pressures and induce OSAS. Consequently, a well defined assessment of the functional capability of the nose in obese children with OSAS can give additional information regarding the pathophysiology of their OSAS. Identification of structural problems in the nasal pathway that would increase aNR in patients with OSAS may provide an indicator of their OSAS severity. Treatment of this obstruction may also serve to improve compliance with nasal continuous positive airway pressure therapy²⁹.

Finally, we examined into the effects of gender and BMI-Z score on aNR and could not find a significant correlation for either. However, larger studies are needed to confirm this finding due to the relative low sample size and the fact that all subjects were obese and were within a narrow BMI-Z range.

CONCLUSION

From the current study using four-phase AAR, we conclude that obese children with OSAS have higher aNR than children without the sleep disorder. Male and female do not have significantly different aNR. This clinical investigation suggests that the increase of aNR from the obstruction of the nasal passage could play an important role to develop or exacerbate OSAS in obese children. Thus, the importance of aNR in obese children with OSAS should be considered in the future studies of OSAS.

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FIGURE 1.

Diagram with flow vs. pressure changes from a control (Top, AHI: 0, aNR: 0.256 kPa/l/s on inspiratory, aNR: 0.258 kPa/l/s on expiratory) and an OSAS subject (Bottom, AHI: 5.7, aNR: 1.149 kPa/l/s during inspiratory, aNR: 1.297 kPa/l/s during expiratory). Green line: pressure reference value at 150 Pa, blue curve: a breathing cycle of the right nostril, red curve: a breathe cycle of the left nostril.

FIGURE 2.

Correlation between anterior nasal resistance and AHI during inspiration in OSAS group.

TABLE 1

Demographics

	Control (n=32)	OSAS (n=30)	p
Age (year)	13.6 ± 2.3	13.8 ± 2.6	NS
Height (cm)	160.7 ± 12.0	161.6 ± 14.1	NS
Weight (Kg)	92.3 ± 28.7	104.3 ± 37.8	NS
BMI (Kg/cm ²)	35.1 ± 8.4	38.9 ± 10.5	NS
BMI Z-score	2.4 ± 0.4	2.6 ± 0.4	NS
Gender (F/M, %F)	12/20 (37%)	9/21 (30%)	NS
Ethnicity (Hisp/AA/other)	17/11/4	16/11/3	NS

Mean ± SD, Hisp: Hispanic, AA: African American

TABLE 2

Polysomnographic Data

	Control (n=32)	OSAS (n=30)	p
TST (hrs)	6.2 ± 0.9	6.1 ± 1.3	NS
Sleep Efficiency (%)	83.0 ± 9.8	80.6 ± 14.3	NS
AI (events/hr)	0.1 ± 0.3	3.8 ± 5.9	0.002
AHI	1.5 ± 1.6	24.2 ± 25.0	< 0.001
Baseline SpO₂ (%)	98.8 ± 1.7	98.9 ± 1.4	NS
SpO₂ Nadir (%)	92.9 ± 3.3	82.1 ± 6.9	< 0.001
Baseline ETCO₂ (mmHg)	42.8 ± 11.5	39.6 ± 6.5	NS
Peak ETCO₂ (mmHg)	50.4 ± 8.8	51.6 ± 4.4	NS
Arousal Index (events/hr)	6.8 ± 3.6	24.3 ± 20.2	< 0.001

TST: total sleep time, AI: apnea index, AHI: apnea hyponea index

TABLE 3

Anterior Nasal Resistance

Phase	Control (n=32)	OSAS (n=30)	p
Inspiration aNR (kPa/l/s)	0.58 ± 0.36	1.47 ± 1.92	0.012
Expiration aNR (kPa/l/s)	0.62 ± 0.35	1.48 ± 1.53	0.003

aNR: anterior nasal resistance