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Utilizing Inspiratory Airflows During Standard Polysomnography to Assess Pharyngeal Function in Children During Sleep

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Summary

Objectives—Obstructive sleep apnea (OSA) is the result of pharyngeal obstruction that occurs predominantly during REM in children. Pathophysiologic mechanisms responsible for upper airway obstruction, however, are poorly understood. Thus, we sought to characterize upper airway obstruction in apneic compared to snoring children during sleep. We hypothesized that apneic compared to snoring children would exhibit an increased prevalence and severity of upper airway obstruction, that would be greater in REM compared to non-REM, and would improve following adenotonsillectomy.

Study Design—Apneic children were assessed with routine polysomnography before and after adenotonsillectomy, and compared to snoring children matched for gender, age, and BMI z-score. In addition to traditional scoring metrics, the following were used to characterize upper airway obstruction: maximal inspiratory airflow (%V_Imax) and percent of time with inspiratory flow-limited breathing (%IFL).

Results—OSA compared to snoring children had similar degrees of upper airway obstruction in non-REM; however, during REM, children with sleep apnea exhibited a higher %IFL ($98 \pm 2\%$ vs. $73 \pm 8\%$, $P < 0.01$) and lower %V_Imax (56 ± 6 vs. $93 \pm 10\%$, $P < 0.01$). In children with OSA, CO₂ levels were elevated during both wake and sleep. Following adenotonsillectomy, upper airway obstruction improved during REM manifest by decreased %IFL (98 ± 2 to $63 \pm 9\%$, $P = 0.04$), increased %V_Imax (56 ± 6 to $95 \pm 5\%$, $P = 0.01$) and decreased CO₂ levels.

Conclusions—Differences in the prevalence and severity upper airway obstruction suggest impaired compensatory responses during REM in children with OSA, which improved following adenotonsillectomy.

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Keywords

obstructive sleep apnea; compensatory responses to sleep apnea; adenotonsillectomy

INTRODUCTION

Obstructive sleep apnea (OSA) is a common disorder occurring in approximately 1–3% of children^{1,2} and is characterized by the development of pharyngeal airflow obstruction during sleep. Resulting alterations in gas exchange and sleep architecture are associated with the development of major clinical sequelae including neurocognitive (hyperactivity³) and poor school performance⁴ metabolic (hyperlipidemia⁵ and hyperglycemia⁶), and cardiovascular morbidity.^{7–9} The prevalence of OSA is increasing dramatically,¹⁰ heightening concerns about its impact on children's present and future health. A significant risk factor for developing OSA during childhood is adenotonsillar hypertrophy,¹¹ and practice guidelines of the American Academy of Pediatrics recommend adenotonsillectomy as first-line therapy for otherwise healthy children with OSA.^{12,13} Normalization of apnea hypopnea indices on polysomnography following adenotonsillectomy, however, is estimated at only 63–79%,¹⁴ exposing many children to the risks of anesthesia and surgery without clear clinical benefit. Clinicians, however, have been hampered by a lack of diagnostic tests to determine pharyngeal function during sleep and to predict treatment responses.

Polysomnography is used to characterize OSA severity by the frequency of discrete episodes of upper airway obstruction assessed by semiquantitative airflows, but it does not predict responses to adenotonsillectomy. In children with OSA, obstructive apnea events occur predominantly during REM,¹⁵ suggesting that pharyngeal obstruction increases during REM. Following upper airway collapse, however, factors independent of pharyngeal function contribute to the frequency of obstructive events including arousal threshold and ventilatory responses to hypoxia and hypercapnia,¹⁶ which vary in REM compared to non-REM.^{17–19} Thus, current polysomnographic metrics to characterize sleep disordered breathing reflect a complex phenotype that is only determined, in part, by the severity of upper airway obstruction. These limitations have precluded full understanding of the pathogenesis of upper airway collapse during sleep standard polysomnography.

Tests to assess pharyngeal function during sleep in children could provide insight into the pathophysiologic etiology of upper airway collapse during sleep, and clinicians with predictors of surgical success. Upper airway obstruction has been quantified by monitoring tidal inspiratory airflows during sleep with detailed studies of pressure–flow relationships to characterize pharyngeal structural and neuromuscular function in adults^{20,21} and children.²² When nasal pressure is manipulated during sleep, decreased maximal inspiratory airflows are exhibited in children with OSA compared to snoring children at similar nasal pressure levels, indicating greater degrees of upper airway collapsibility.^{17,22} Moreover, in children with OSA, upper airway collapsibility is greater in REM compared to non-REM.²³ These methods, however, are laborious, difficult to perform in REM, and are not well tolerated in young children. Thus, we sought to assess pathophysiologic traits responsible for upper airway collapse during standard polysomnography.

Children express a spectrum of obstructive breathing during sleep from simple snoring to complete upper airway obstruction, and these differences become more pronounced in REM than non-REM.¹⁵ We hypothesized that apneic compared to snoring children would exhibit an increased prevalence and severity of upper airway obstruction, that would be greater in REM compared to non-REM, and improve following adenotonsillectomy when assessed by analyzing the level of inspiratory airflow.

MATERIALS AND METHODS

Participants

The first 10 children with OSA by polysomnography (RDI ≥ 2 events/hr) who underwent adenotonsillectomy (AT) and had a second polysomnogram following surgery were consecutively identified. These children were matched group wise by age, gender, and BMI z-score to 10 children diagnosed with primary snoring (RDI ≤ 1 events/hr) who underwent baseline polysomnography and did not require adenotonsillectomy. Informed consent was obtained from one parent/guardian, and assent was obtained from the children. The Johns Hopkins Medicine Institutional Review Board approved the protocol.

Experimental Approach

Assessment of Sleep—Sleep studies were performed for all subjects on EMBLA (Somnologica, Medcare, Inc., Buffalo, NY). Physiologic signals monitored included electroencephalograms (F3-A2, C3-A2, O1-A2), left and right electro-oculograms (EOG), electrocardiogram (ECG modified V2 Lead), thoracic and abdominal inductive plethysmography, submental electromyogram, oxyhemoglobin saturation via pulse oximetry (Ohmeda, Louisville, CO), and airflow via nasal cannula (Salter Adult Nasal Cannulas, Salter Labs, Arvin, CA) attached to a pressure transducer (Gould-Statham P-10, Gould, Inc., Oxnard, CA) and end-tidal CO₂ (Novamatrix Murrysville, PA). Sleep staging, arousals, and respiratory events were performed according to American Academy of Sleep Medicine 2012 criteria²⁴ by a registered polysomnographic technician and a board certified sleep medicine physician. In brief, obstructive apneas were identified when a $\geq 90\%$ drop in inspiratory airflow in the thermistor was detected, and obstructive hypopneas when a $\geq 30\%$ decrease in inspiratory airflow in the nasal pressure transducer was detected that was associated with a $\geq 3\%$ desaturation. Respiratory effort-related arousals (RERAs) were identified as increasing respiratory effort with flattening of the inspiratory airflow contour leading to arousal from sleep that did not meet apnea or hypopnea criteria. The Respiratory Disturbance Index (RDI) was generated by summing the number of obstructive apneas, hypopneas, and RERAs, mixed apneas, and central apneas divided by total sleep time (events/hr).

Analysis

Inspiratory Flow Limitation Metrics—In addition to the respiratory disturbance index (RDI), upper airway obstruction was assessed by the following airflow limitation metrics: percent of time with inspiratory flow limited breathing (%IFL), and maximal inspiratory airflow during flow limited breaths expressed as a percent of peak inspiratory flow during non-flow limited breathing during non-REM sleep (%V_Imax), (See Fig. 1). Breaths were analyzed with a custom computer program that generated five random samples per hour of

sleep that were 1 min in length. Samples were excluded if they occurred during wakefulness or if the integrity of the airflow signal was diminished including evidence of cannula displacement or mouth breathing. Samples were included if they occurred during hypopneas and apneas. Custom-designed software was utilized to detect the start and stop of inspiration, and maximal inspiratory airflow in the first half of inspiration. Breaths with inspiratory flow limitation were identified when rapidly increasing airflow was followed by a plateau in the inspiratory contour (see Fig. 1, right panel) as previously described.^{25–28}

Statistical Analysis—Descriptive statistics were used to describe the characteristics of subject groups as means with standard errors of the mean (SEM) unless otherwise stated. Apnea hypopnea indices, SpO₂, and CO₂, were assessed with signed-rank tests for differences within OSA and primary snoring groups and rank-sum tests were used to assess differences between groups. Demographics, total sleep time, % of non-REM and REM sleep, % inspiratory flow limitation, and % V_Imax were assessed with Students *t*-tests. All statistics were performed with Stata 9 (Stata Corporation, College Station, TX). For all analyses, statistical significance was defined as *P* < 0.05.

RESULTS

Subject Characteristics and Polysomnography Data

Ten children encompassing a range from mild-to-severe obstructive sleep apnea-hypopnea syndrome (RDI ≥ 2 events/hr) were compared to 10 children diagnosed with primary snoring (RDI < 2 events/hr). Apneic children underwent polysomnography before and after surgery, the time between sleep studies was 7.9 ± 3.2 months. Children with OSA and primary snoring were comparable with respect to gender, age, BMI z-score, and gender (See Table 1). There was no difference in total sleep time, sleep efficiency, or percent of sleep time in non-REM and REM in children with OSA before or after AT or compared to snoring children. The arousal index, however, was higher in apneic children prior to adenotonsillectomy compared to snoring children. Adenotonsillectomy was associated with a decrease in the arousal index to a level that was comparable to snoring children (see Table 2).

Gas Exchange During Polysomnography

Children with OSA prior to adenotonsillectomy compared to primary snoring children had a lower overall SpO₂ nadir (84 ± 8 vs. 91 ± 4, *P* = 0.04); however, in the breaths analyzed, there was no difference in average SpO₂ during non-REM (98 ± 1 vs. 97 ± 1, *P* = 0.2) or REM (98 ± 2 vs. 96 ± 1, *P* = 0.06). Children with OSA prior to adenotonsillectomy compared to snoring children had a higher average CO₂ at all points assessed with analyzed breaths, during wake (47 ± 1 vs. 44 ± 1 mmHg, *P* = 0.05), non-REM (49 ± 1 vs. 46 ± 2 mmHg, *P* < 0.01), and REM (54 ± 1 vs. 46 ± 3 mmHg, *P* < 0.01)(see Table 2). Following adenotonsillectomy, gas exchanged improved during sleep, the SpO₂ nadir increased (from 84 ± 8 to 92 ± 1, *P* = 0.01), and CO₂ decreased during both non-REM (from 49 ± 1 to 47 ± 2 mmHg, *P* = 0.04) and REM (from 54 ± 1 to 48 ± 4 mmHg, *P* < 0.01). Moreover, following adenotonsillectomy, CO₂ levels were comparable between snoring and OSA children during wake, non-REM, and REM.

Prevalence and Degree of Upper Airway Obstruction

Children with OSA prior to adenotonsillectomy compared to snoring children had a higher total RDI (14 ± 9 vs. 1 ± 1 events/hr, $P < 0.01$), and higher RDI in both non-REM (RDI 11 ± 19 vs. 1 ± 1 events/hr, $P < 0.01$) and REM (RDI 29 ± 27 vs. 2 ± 3 events/hr, $P < 0.01$) (see Fig. 2, left panel). Adenotonsillectomy led to reduction in total RDI (from 14 ± 9 to 1 ± 1 events/hr, $P < 0.01$), and the RDI during non-REM (from 11 ± 19 to 1 ± 1 events/hr, $P < 0.01$) and REM (RDI from 29 ± 27 to 2 ± 3 events/hr, $P < 0.01$) (see Fig. 2, left panel). After surgery, the total RDI, and the RDI during both REM and non-REM in apneic children was comparable to snoring children.

To characterize upper airway obstruction, in addition to the RDI, the prevalence of upper airway obstruction was assessed by the % inspiratory flow-limited breathing and the degree of upper airway obstruction by the % $V_{I\max}$. The mean number of breaths analyzed in snoring children was 369 (range 139–688) and in apneic children 366 (range 222–545). In children with OSA before surgery, the % inspiratory flow-limited breathing was higher during REM compared to non-REM ($98 \pm 2\%$ vs. $70 \pm 9\%$, $P < 0.04$). In children with OSA compared to snoring children, the prevalence of upper airway obstruction was also higher during REM ($98 \pm 2\%$ vs. $79 \pm 8\%$, $P < 0.01$); no difference, however, was exhibited during non-REM ($70 \pm 9\%$ vs. $62 \pm 12\%$, $P < 0.6$). Adenotonsillectomy in children with OSA led to a decrease in the % inspiratory flow limitation during REM (from 98 ± 2 to $63 \pm 9\%$, $P = 0.04$) (see Fig. 2, right panel) to degrees comparable to snoring children, without a reduction during non-REM. The % $V_{I\max}$ in children with OSA before adenotonsillectomy compared to snoring children was markedly lower during REM (56 ± 6 vs. $93 \pm 10\%$, $P < 0.01$); no difference was observed during non-REM (69 ± 5 vs. $85 \pm 9\%$, $P < 0.1$) (see Fig. 3). Adenotonsillectomy in children with OSA led to an increase in the % $V_{I\max}$ during REM (from 56 ± 6 to $95 \pm 5\%$, $P = 0.01$); however, there was no difference during non-REM (from 69 ± 5 to $82 \pm 6\%$, $P = 0.1$) (see Fig. 3). Following adenotonsillectomy, despite substantial improvements in pharyngeal function and resolution of disease characterized by the traditional RDI metric to characterize sleep disordered breathing, residual upper airway obstruction persisted that was similar to snoring children during non-REM and REM (see Figs. 2 and 3).

DISCUSSION

There were three major findings in our study. First, differences in the severity and prevalence of upper airway obstruction were observed between apneic and snoring children during REM sleep. Both apneic and snoring children maintained tidal inspiratory airflows during non-REM; however, during REM sleep, children with OSA exhibited marked increases in the prevalence and severity of upper airway obstruction. These dynamic changes resulting in decreased pharyngeal patency from non-REM to REM suggest a loss of compensatory neuromuscular responses during REM. Second, following adenotonsillectomy, the prevalence and severity of upper airway obstruction improved in apneic children during REM, consistent with a decrease in pharyngeal mechanical loads. Despite resolution of sleep apnea as assessed by the RDI, inspiratory airflow remained flow limited, indicating significant residual upper airway obstruction that was comparable to that in snoring children.

Third, in children with OSA, CO₂ levels were elevated during sleep and wakefulness. Similar to improvements in upper airway patency, adenotonsillectomy was also associated with decreases in CO₂, which no longer differed from snoring controls. Our findings of decreased pharyngeal patency and elevated CO₂ levels in REM in apneic children that improved following adenotonsillectomy suggests that adenotonsillar hypertrophy loads the upper airway and impairs compensatory pharyngeal neuromuscular and ventilatory neuromuscular responses during REM sleep. Nevertheless, persistent upper airway obstruction remained after adenotonsillar resection that was similar to snoring children, suggesting residual defects in pharyngeal function.

Previously, we have utilized pressure–airflow relationships to determine upper airway collapsibility (Pcrit). During these studies, nasal pressure was manipulated during sleep to induce upper airway obstruction under conditions of either quiescent or increased neuromuscular activity to partition pharyngeal mechanical properties and upper airway compensatory neuromuscular function, respectively.^{29–32} Previous studies of pressure–airflow relationships indicate that in response to pharyngeal obstruction, compensatory upper airway neuromuscular responses are blunted in adults^{20,21} and children^{17,22,33,34} with obstructive sleep apnea. Moreover, increased upper airway collapsibility is seen in children with OSA in REM compared to non-REM sleep²³ likely secondary to further decreases in pharyngeal neuromuscular activity in REM.³⁵ These studies, however, are laborious, difficult to perform in young children and in REM sleep, since they require sleeping with a mask and manipulating nasal pressure.

We found marked differences in airflow dynamics during REM sleep between apneic children before adenotonsillectomy and snoring children. In contrast to snoring children, in whom the severity and prevalence of IFL (% V_Imax and %IFL) did not differ between non-REM and REM sleep, apneic children exhibited a greater severity and prevalence of upper airway obstruction during REM compared to NREM sleep (Figs. 2 and 3). The sleep stage dependent differences in airflow dynamics in REM compared to non-REM suggests a loss of upper airway neuromuscular activity in apneic children which generally wanes in REM sleep.³⁶ In contrast, the ability of snoring children to maintain inspiratory tidal airflow during REM suggests either lower pharyngeal mechanical loads or maintenance of compensatory upper airway neuromuscular responses to upper airway obstruction during REM. The defect resulting in diminished upper airway compensatory neuromuscular activity during REM in apneic children, however, is not certain and might include diminished pharyngeal sensation, differences in the set point of the respiratory central pattern generator or diminished peripheral motor unit control. Taken together, our data suggest that the development of periodic obstructive apneas in REM in apneic children is consistent with decreased compensatory neuromuscular responses to upper airway obstruction.

Adenotonsillectomy in apneic children led to a decrease in the prevalence and severity of upper airway obstruction during REM sleep. Associated with the improvement in upper airway patency, the RDI normalized largely due to a decrease in REM related events. These improvements in pharyngeal function following surgery can be attributed to a reduction in pharyngeal mechanical loads. We found that adenotonsillectomy led to normalization of the RDI. Nevertheless, inspiratory airflow-based metrics were more sensitive than the RDI in

their ability to detect residual upper airway obstruction, which resembled primary snoring even in the absence of apneas, hypopneas, or RERAs. These data are consistent with previous studies of pressure–flow relationships in children with OSA that demonstrated upper airway collapsibility decreased but did not normalize following adenotonsillectomy.¹⁷ The residual impairment in pharyngeal function after surgery indicates that additional as yet unidentified factors contribute to upper airway obstruction during sleep in apneic children.

Following adenotonsillectomy when pharyngeal mechanical loads were reduced and upper airway obstruction decreased, CO₂ homeostasis was improved during both sleep and wakefulness. The decrease in CO₂ levels in apneic children during REM and wakefulness following adenotonsillectomy is consistent with either an increase in alveolar ventilation or a decrease in CO₂ production after removing the airway load. Findings in adults and children support the notion that hypercapnic ventilatory responses are blunted in apneic patients, who also demonstrate concomitant modest increases in CO₂.^{37,38} Conversely, an increase in ventilatory drive could account for striking decreases in CO₂ and in the severity of upper airway obstruction in apneic children during REM sleep after adenotonsillectomy, which restored airflow to levels observed in snoring children. After resecting mechanical loads, tidal airflow would be expected to increase similarly during non-REM and REM sleep. Instead, we found more marked increase in upper airway patency in REM than non-REM sleep (Fig. 3), consistent with the improvement in both anatomic and neuromuscular factors controlling airway patency. Previous studies have demonstrated that the genioglossus is recruited by upper airway obstruction, and further augmented by increases in CO₂ during prolonged periods of airflow obstruction.³⁹ Taken together, our data suggest that after removing adenotonsillar loads, the genioglossus became more effective in restoring upper airway patency. In addition, in apneic children following surgery, CO₂ fell from 54 to 48 mmHg in REM consistent with concomitant increases in ventilation or decreases in CO₂ production. Taken together, our data suggest that compensatory ventilatory and upper airway neuromuscular responses were overwhelmed in apneic children by increased pharyngeal mechanical loads, and these responses improved when loads were reduced after adenotonsillectomy.

Limitations

We acknowledge several limitations in our study. First, airflow data were acquired with a nasal cannula and maximal inspiratory airflow was a relative value referenced to periods of non-flow limited breathing during non REM sleep limiting implications about pharyngeal function. Effort was made to eliminate analysis during periods when the cannula was displaced, or if mouth breathing occurred; however, assessing quantitative changes in airflow with a qualitative signal limits the physiologic implications of this study. Quantification of airflow with a pneumotachograph in future studies would provide greater insight into pathophysiologic factors responsible for upper airway collapse during sleep. Second, our study population was small and because the study was retrospective and drawn from a clinical population, a normal non-snoring control group of children was not included in our study. Nevertheless, the findings in this clinical population reflect real-world responses to adenotonsillectomy, and indicate that residual upper airway obstruction persists during sleep in apneic children. Prospective studies in larger study populations of OSA and non-snoring

children with and without adenotonsillar hypertrophy will provide further insight into effects of adenotonsillectomy on pharyngeal function. Third, pressure flow assessments were not performed in these children for direct comparison of upper airway collapsibility. Comparison of airflow metrics with gold standard measures of upper collapse in future studies will be required to validate these methods. Nevertheless, the marked differences in airflow obstruction exhibited during REM compared to non-REM suggest that sleep stage dependent differences in upper airway obstruction might be utilized to partition pharyngeal mechanical properties and compensatory neuromuscular responses to obstruction during sleep in children.

Implications

Surgical removal of adenoids and tonsillar tissue is one of the most common pediatric surgeries estimated at greater than 500,000 procedures per year, and OSA is the most common indication.⁴⁰ At the present time, there are no known predictors for successful treatment of sleep apnea with adenotonsillectomy. Imaging modalities do not assess the degree of upper airway obstruction across sleep stages, and pressure-flow measurements are not well tolerated in young children during sleep. In contrast, quantification of tidal airflow and the application of inspiratory airflow metrics may ultimately provide reliable measures of pharyngeal dysfunction during sleep. These measures although not currently adopted for clinical evaluation of polysomnography are readily available to clinicians and complement current indices of sleep disordered breathing severity and extend our ability to detect subtle degrees of inspiratory flow limitation during sleep. These airflow metrics may ultimately predict surgical success for children with obstructive sleep apnea. For example, the data demonstrate the greatest improvement in airflow obstruction during REM compared to non-REM, leading us to posit that children with upper airway obstruction predominantly during REM sleep would be more likely to benefit from adenotonsillectomy than children with significant obstruction during non-REM sleep. Moreover, those children with residual obstruction might be more likely to develop obstructive sleep apnea as they reach adulthood, warranting close follow-up of these children into adulthood. To validate this approach, inspiratory airflow should be quantified with a pneumotachograph during routine polysomnography to assess pharyngeal patency. To develop a model to predict surgical responses, further work is required that should include a larger sample size, and examination of the natural history of residual upper airway obstruction following adenotonsillectomy in order to determine the extent to which these measurements predict long-term outcomes.

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Fig 1.

Assessment of inspiratory flow limitation. Three breaths of non-flow limited (Left panel) and flow limited breathing (right panel) during non-REM sleep are depicted. Inspiratory flow-limited breathing is indicated by the flat inspiratory flow contour and increased inspiratory time relative to total respiratory time. Maximal inspiratory airflow was defined during the non-flow limited period during non-REM sleep. Exp, expiration; Insp, inspiration; T_I , inspriatory time; T_{TOT} , total respiratory time; $V_{I\max}$, maximal inspiratory airflow in the first half of inspiration.

Fig. 2.

RDI and prevalence of upper airway obstruction. RDI and prevalence of upper airway obstruction are displayed for children with OSA before and after adenotonsillectomy and snoring children. Respiratory disturbance indices during non-REM and REM (left Panel) and percent of inspiratory flow limitation (right panel). Data presented are mean $\pm$ standard error of the mean. * indicated REM compared to non-REM, $P = 0.05$; † indicates OSA before adenotonsillectomy compared to snoring children, $P = 0.05$, + indicates OSA before compared to after adenotonsillectomy, $P = 0.05$. RDI, respiratory disturbance indices; %IFL, percent of inspiratory flow limitation; AT, adenotonsillectomy; OSA, obstructive sleep apnea.

Fig. 3.

Degree of upper airway obstruction. Maximal inspiratory airflow is displayed for children with OSA before and after adenotonsillectomy and snoring children. Data presented are mean $\pm$ standard error of the mean. † indicates OSA before adenotonsillectomy compared to snoring children; * indicates OSA before compared to after adenotonsillectomy, $P = 0.05$. %V_Imax, maximal inspiratory airflow; AT, adenotonsillectomy, OSA, obstructive sleep apnea.

TABLE 1

Demographics

	OSA	Snoring	P-value
Gender (M:F)	5:5	6:4	0.7
Age (years)	5 ± 3	5 ± 2	0.6
Height (cm)	115 ± 20	114 ± 19	0.9
Weight (kg)	29 ± 14	24 ± 9	0.2
BMI (kg/m ²)	21.3 ± 6.9	19.0 ± 3.9	0.2
BMI z-score	1.4 ± 1.7	0.8 ± 1.3	0.4

All values are presented as mean ± standard deviation. BMI, body mass index.

TABLE 2

Polysomnography Data

Polysomnography data	OSA (Pre-AT)	OSA (Post-AT)	Snoring
TST (min)	386 ± 31	372 ± 37	369 ± 37
SE%	94 ± 4	91 ± 8	89 ± 7
Arousal indices (number/hr)	13 ± 5 ^{I, *}	9 ± 2	8 ± 3
NREM % of TST	75 ± 17	78 ± 6	80 ± 6
REM % of TST	28 ± 16	21 ± 5	19 ± 6
RDI total (events/hr)	12.6 ± 9.1 ^{I, *}	1.0 ± 0.8	0.6 ± 0.4
OAHI non-REM (events/hr)	9.5 ± 9.1 ^{I, *}	0.3 ± 0.4	0.1 ± 0.1
RERAs non-REM (events/hr)	1.1 ± 1.4	0.4 ± 0.4	0.2 ± 0.3
OAHI REM (events/hr)	36.0 ± 33.1 ^{I, *}	1.4 ± 1.7	0.8 ± 1.7
RERAs REM (events/hr)	1.4 ± 1.3	0.8 ± 1.4	1.1 ± 1.3
Gas exchange			
Low SpO ₂ %	84 ± 8 ^{I, *}	92 ± 1	91 ± 4
Average SpO ₂ % NREM	97 ± 1	98 ± 1	98 ± 1
Average SpO ₂ % REM	96 ± 2	97 ± 1	98 ± 1
Average CO ₂ wake (mmHg)	47 ± 11	45 ± 1	44 ± 1
Average CO ₂ NREM (mmHg)	49 ± 1 ^I	47 ± 2	46 ± 2
Average CO ₂ REM (mmHg)	54 ± 1 ^{I, *}	48 ± 4	46 ± 3

All values are presented as mean ± standard deviation. AT, adenotonsillectomy; TST, total sleep time; SE, sleep efficiency; RDI, respiratory disturbance index; OAHI, obstructive apnea hypopnea index, RERAs, respiratory effort related arousals, SpO₂, oxygen saturation; CO₂, carbon dioxide.

^I OSA pre-AT versus snoring.

^{*} *P* 0.05 OSA pre-AT versus OSA post-AT.