Are Nocturnal Hypoxemia and Hypercapnia Associated With Desaturation Immediately After Adenotonsillectomy?

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Abstract

Background—Children who undergo adenotonsillectomy for sleep-disordered breathing frequently have postoperative oxygen desaturations. Nocturnal hypoxia has been shown to predict post-operative respiratory complications, however, other gas exchange abnormalities detected on polysomnography (PSG) have not been evaluated.

Aim—We sought to determine whether hypercarbia seen on preoperative nocturnal PSG can predict postoperative hypoxemia.

Methods—We conducted a retrospective review of 319 children who underwent polysomnography before adenotonsillectomy. Saturation levels were recorded for at least 2 hours postoperatively, and the primary outcome was desaturation (<90%).

Results—The median patient age was 5 years (range, 5 months-17 years). Patients who desaturated postoperatively had higher median peak end-tidal CO_2 (EtCO$_2$) levels (55.5 vs. 52 mmHg; *P*=0.02), lower saturation nadirs (80.5% vs. 88%; *P*=0.048), and were younger (2 vs. 6 years; *P*<0.001) than those without desaturation. Age was significantly correlated with peak

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$EtCO_2$ (r= −0.16), respiratory disturbance index (RDI; r= −0.23), and oxygen saturation nadir (r= 0.25; all *P*<0.01). In unadjusted analysis, age<3 years compared to ≥9 years (odds ratio [OR]=10.09; 95% confidence interval [CI]=2.13–96.26), peak $EtCO_2$>55 mmHg (OR=3.38; 95% CI=1.21–9.47), and RDI ≥10 (OR=2.89; 95% CI=1.05–8.42) were associated with increased odds of desaturation. Multivariable logistic regression on age, race, sex, peak $EtCO_2$, RDI, opioid use, and saturation nadir showed that only age was significantly associated with postoperative desaturation. Patients 0–2 years old were 10.43 (95% CI=1.89–110.9) times more likely to have desaturation than patients 9–17 years old.

Conclusion—Patients less than 3 years of age are most likely to have postoperative hypoxemia after adenotonsillectomy. Gas exchange abnormalities did not correlate with postoperative desaturations, although age and peak $EtCO_2$ did strongly correlate.

MESH compliant words

Sleep Apnea, Obstructive; Tonsillectomy; Postoperative Complications; Desaturation; Polysomnography; Hypercapnia

Introduction

Adenotonsillectomy (AT) is one of the most common pediatric surgeries performed in the United States, with more than 530,000 procedures carried out annually. Historically, the primary indication for AT was recurrent tonsillitis; however, sleep-disordered breathing (SDB) is now the most common indication for AT.[1,2] If left untreated, SDB can result in behavioral problems, including poor school performance, learning impairment, and cardiovascular and metabolic disease.[3–5] AT can potentially prevent these morbidities, but the surgery is often associated with immediate postoperative respiratory compromise, most commonly hypoxemic episodes that result from central nervous system depression or airway obstruction. It has been reported that patients with repetitive hypoxia during sleep have a 20-fold higher incidence of respiratory complications after AT than do patients without hypoxic episodes.[3,6–8] Nevertheless, development of strategies to identify patients at risk for postoperative desaturation have not produced adequate predictive value.

Desaturation and airway obstruction are two of the most common respiratory complications after AT.[9–11] They occur most frequently in younger children and in those with severe obstructive sleep apnea (OSA)[3] and can lead to significant morbidity, including neurologic injury and death.[9] Indeed, catastrophic perioperative respiratory complications are among the leading complaints related to pediatric surgery and AT in closed claim malpractice cases.[2,12] Investigators have found that the incidence of postoperative desaturation can be reduced by 50% or more if opioid dosing is decreased in children with severe OSA, which is diagnosed by preoperative hypoxia during sleep.[3–5]

Although recently published guidelines recommend that all children undergo polysomnography (PSG) before AT,[3] fewer than 10% are sent for PSG before being scheduled for surgery.[13] Extensive data has linked preoperative nocturnal hypoxia and desaturation to increases in opioid sensitivity and postoperative respiratory complications.[14–17] A few published reports support other PSG data as a predictor of

postoperative complications and desaturation in children with OSA, but data to connect these events are limited.[3,6–8,18] Therefore, we sought to determine whether respiratory parameters measured via preoperative PSG can predict postoperative oxygen desaturations. In this retrospective study, we tested the hypotheses that hypoxia (oxygen saturations less than 90%) and hypercapnia (end-tidal carbon dioxide [$EtCO_2$] greater than 55 mmHg) during sleep are associated with postoperative oxygen desaturations in children immediately after AT.

Methods

The study was reviewed and approved by the Johns Hopkins Medicine Institutional Review Board. The patients in this study all received an AT by one of five pediatric otolaryngologists. All children received sevoflurane or desflurane for maintenance of anesthesia, and most children (92%) received intraoperative dosing of an opioid. For each child, we calculated body mass index (BMI)-for-age percentile using the formulas derived from the CDC website (www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/tool_for_schools.html). BMI-for-age percentile is the percentile of BMI normalized for age and gender in children. Obesity is defined as a BMI-for-age percentile of 95% or greater.

Patients were kept in the recovery room for 2 hours postoperatively. It is standard of care in this setting for children to be monitored continuously with pulse oximetry for the duration of their stay in the recovery room. Oxygen desaturations were noted by the bedside nurses, and the level of desaturation was recorded manually. We collected demographic information, PSG measurements, and postoperative pulse oximetry data (Masimo, Irvine, CA) via retrospective chart review and reviewed nursing charts to determine postoperative respiratory complications. We also collected oxygen saturation nadir, which was documented manually by the post-anesthesia care unit (PACU) nursing staff. We did not analyze other interventions that signify respiratory compromise, such as the use of airway adjuncts, ventilation assistance, supplemental oxygen administration, or re-intubation, because these are not routinely recorded by the PACU staff.

We extracted PSG data from patient medical records. Sixteen-channel PSG studies were performed at sleep centers within the greater Baltimore area, including at the Johns Hopkins Sleep Laboratory, with Somnologica or REMLogic (Embla, Broomfield, CO). Recorded signals included electrocardiogram, left and right electrooculograms, electroencephalograms (with leads C3-A2, C4-A1, and O1-A2), tibial and submental electromyelogram, and oxyhemoglobin saturation (Masimo, Irvine, CA). All participants had respiratory effort assessed with both abdominal and thoracic inductive plethysmography (Embla, Broomfield, CO) and had nasal $EtCO_2$ measured (Novametrix, Murrysville, PA). Body position was observed with an infrared video camera, and airflow was monitored with a differential pressure transducer (Pro-Tech, Mukilteo, WA), which was connected to a nasal cannula (Salter Labs, Arvin, CA). If the patient snored during the sleep study, the sleep technologist graded it subjectively on a clinical scale of 1 (least) to 4 (greatest) using guidelines routinely implemented at this institution. Board-certified pediatric sleep medicine physicians interpreted all results. Apnea was defined as the cessation of oronasal airflow for at least two consecutive breaths. Hypopnea was defined as a decrease in oronasal airflow of 50% for at

least two breaths, with an associated desaturation of 3% and/or an arousal. Respiratory event-related arousals were not measured. The respiratory disturbance index (RDI) was calculated as the total number of obstructive apneas and hypopneas per hour of total sleep time. Mild, moderate, and severe OSA were defined as an RDI of 1 to <5 events/h, 5 to <10 events/h, and 10 events/h, respectively. We excluded children with a central apnea index of 6 or more events/h in order to focus on the most prevalent type of sleep apnea and avoid confounding, as many of the children with central events also have neurologic issues that may independently affect the likelihood of postoperative complications. Hypercapnia was defined by peak $EtCO_2$ levels greater than 55 mmHg, and oxygen desaturation was defined as a value less than 90%, as recorded by a pulse oximeter.

Statistical analysis

Patient weight was evaluated with BMI-for-age percentiles. Categorical measures were summarized by using frequencies and percentages, and groups were compared by using Fisher's exact test. Most of the continuous measures were not normally distributed. Therefore, data were summarized as medians and interquartile ranges (IQRs) and compared by Wilcoxon rank-sum tests. Prior to regression analysis, we categorized continuous measures as follows: age as 0–2, 3–8, and 9–17 years (based on a known risk for children under 3 years); BMI-for-age percentile at the median; RDI as <10 vs. 10; saturation nadir as <90% vs. 90% (based on standard clinical cut points); and peak $EtCO_2$ as 55 mmHg vs. >55 mmHg (based on the 75th percentile observed in our study population). Because of the small number of events, we used exact logistic regression analysis to assess the association of demographic and PSG measures with the outcome of postoperative desaturation. We included all variables of interest (race, sex, age, BMI, intraoperative opioid use, and preoperative PSG data [including RDI, peak $EtCO_2$, and saturation nadir]) in the multiple logistic regression model to determine the independent effect of each. Analyses were carried out with SAS version 9.3 (SAS Institute, Inc., Cary, NC). All reported *P* values are two-sided, and significance was set at *P*<0.05.

Results

We conducted a retrospective review of data for 684 consecutive children who underwent AT or tonsillectomy at an urban, academic, tertiary care center during 2006 and 2007. Of those, we excluded 336 (49%) children who did not have preoperative PSG and 29 (4%) children who had coexistent genetic abnormalities. Therefore, the final sample size was 319. The children who were excluded differed significantly from those in the final sample in terms of race (60.6% Caucasian vs. 33.4% Caucasian, respectively; *P*<0.001) and BMI-for-age percentile (median 66.7 vs. 84.6, respectively; *P*<0.001). Patients who did not receive a preoperative PSG were significantly more likely to be normal weight (*P*=0.015) and Caucasian (*P*<0.001). Demographic characteristics and polysomnogram measures of the children in the study are presented in Table 1. The children ranged in age from 5 months to 17 years. Our sample included more overweight patients than normal weight patients and had almost twice as many African-American children as Caucasian children (196 vs. 104, respectively).

Characteristics of study patients with and without desaturations after surgery are presented and compared in Table 1. The groups did not differ in sex, race, BMI-for-age percentile, or opioid use during surgery; however, the children who desaturated were significantly younger than those who did not (median age 2 vs. 6 years; $P<0.001$). For the PSG measures, the groups did not differ significantly in RDI, obstructive apnea, hypopnea, or central apnea indexes. However, the children who desaturated had significantly higher peak $EtCO_2$ levels (55.5 vs. 52.0 mmHg; $P=0.02$) and lower O_2 saturation nadir (80.5% vs. 88.0%; $P=0.048$). Univariate logistic regression analysis revealed that children less than 3 years old were 10.09 (95% CI=2.13–96.26) times more likely to have desaturations after surgery than children 9 years and older ($P<0.001$; Table 2). Likewise, those with $EtCO_2 > 55$ mmHg were 3.38 (95% CI=1.21–9.47; $P=0.02$) times more likely to have postoperative desaturation than were children with $EtCO_2$ 55 mmHg. Additionally, children with RDI 10 were 2.89 (95% CI=1.05–8.42; $P=0.04$) times as likely to desaturate. Saturation nadir levels under 80% on PSG were marginally associated with postoperative desaturations (OR=2.93; 95% CI=0.99–8.24; $P=0.05$).

Results from the multivariable logistic regression of desaturation on age, race, sex, BMI-for-age percentile, $EtCO_2$, RDI, opioid use, and preoperative saturation nadir are presented in Table 3. Because a large proportion of children were missing data on BMI-for-age percentile, we ran a second model that excluded this variable. Only age was significantly associated with postoperative desaturation. Patients who were 0–2 years old were 10.43 (95% CI=1.89–110.9; $P=0.003$) times more likely to have postoperative desaturation than were those 9–17 years old. Age correlated significantly with peak $EtCO_2$ (r = −0.16), RDI (r = −0.23), preoperative oxygen saturation nadir (r = 0.25), and BMI-for-age percentile (r = 0.39; all $P<0.01$). We also performed a subgroup analysis in which we restricted the data to those patients less than 6 years of age and found that only age was significant in the univariate analysis. Multivariable regression of these patients revealed that inclusion of BMI in the model rendered age insignificant ($P=0.09$), suggesting that BMI modified the relationship between age and postoperative respiratory complications and/or the sample size was smaller when BMI was included for young children. We were unable to carry out additional subgroup analysis by age because respiratory complications were rare in children over 6 years, and our sample size was too small.

Discussion

We hypothesized that hypoxia (oxygen saturations less than 90%) and hypercapnia ($EtCO_2$ greater than 55 mmHg) during sleep are associated with postoperative oxygen desaturations in children immediately after AT. Although preoperative peak $EtCO_2$ levels, RDI, and nocturnal oxygen desaturation levels differed by desaturation status, when we controlled for age, neither preoperative oxygen desaturation nor hypercapnia remained significant. However, the fact that postoperative respiratory complications were rare in older children prevented us from further evaluating the relationship between age, hypercapnia, and preoperative oxygen desaturations. Our original hypothesis was based on the notion that during and immediately after anesthesia, central responses to hypercapnia from airway obstruction, ventilatory drive, and arousal responses are blunted. We expected that these effects would especially manifest in younger patients exposed to episodic nocturnal

hypercapnia and hypoxia before surgery. In addition, we recognized that these blunted responses denote greater risk in respiratory compromise when analgesics and anxiolytics are administered in the perioperative setting.

Risk factors that predispose patients to SDB include age, weight, and race. Young age, specifically below 3 years, has been reported previously to be a risk factor for oxygen desaturation[2,19,20] and for postoperative respiratory compromise after AT; however, the mechanism remains speculative.[1,7,18,21–23] Immature respiratory centers and anatomic obstruction, including small airways relative to a large tongue volume, are among some of the proposed causes. In addition, pharyngeal swelling and edema after AT may worsen the anatomic obstruction. Because analgesics and anesthetics cause decreased activity of airway patency musculature, compensation to maintain an open airway is more difficult to manage in the younger age group.

In addition to anatomic obstruction, responses to ventilatory disturbances may be altered in younger children. We found that elevated preoperative hypercapnia was related to postoperative oxygen desaturation on univariate analysis. Children with OSA have an altered response to carbon dioxide, and those with OSA are frequently hypercapnic during sleep.[1,3,5–8,15,18,24,25] Sleeping and waking ventilatory responses and elevated arousal thresholds in response to hypercapnia are abnormal in children with OSA. Habituation to chronic, intermittent hypercapnia results in a blunting of the arousal response. This idea is supported by an observed decrease in hypercapnic arousal threshold after patients are treated for OSA. Arousal threshold changes were not observed in OSA patients exposed to only a hypoxic environment.[3,5–8,11,18,25] Measures of SDB severity, including nocturnal oxygen desaturation[1,2,6–8,11,15,16,18,24] and RDI,[7,11,26,27] have been shown to predict postoperative respiratory complications. Therefore, nocturnal hypercapnia and hypoxia, when combined with an immature and/or dysfunctional respiratory center, may suggest blunted upper airway and ventilatory responses and predispose the patient to respiratory compromise in the postoperative setting. Our study suggests that a relationship may exist and that additional investigation is warranted.

Obesity is associated with varying degrees of upper airway obstruction during sleep and disturbances in hypercapnic ventilatory responses; the latter may be due to neural and/or mechanical loads on upper airway patency. Obesity is a known risk factor for OSA in children.[3,28–36] It increases the prevalence and severity of OSA caused by several factors, including increased mechanical load on the airway, the predominant etiology of OSA in adults.[7,37,38] Young patients who are overweight or obese may not be able to compensate for a large mechanical load on the upper airway and may be more prone to airway collapse than older patients. Interestingly, children who underwent AT without a preoperative PSG were more likely to be normal weight and Caucasian, suggesting that the likelihood of ordering a PSG may be associated with patient race and characteristics like BMI; however, this study was not designed to investigate this question.

Patient race, regardless of weight and respiratory disease, has been linked to the presence of SDB, with African-American children having a higher prevalence than Caucasians.[39–41] This difference is thought to be due to differences in tonsillar growth between African

Americans and Caucasians, but no study as yet supports this speculation. Patients in our study population were predominantly African American, suggesting that our patient population reflects those groups known to be at highest risk for OSA.

This study has some important limitations. As with other retrospective studies, our analysis is limited to the data that are recorded during standard practice. Obtaining data on postoperative desaturation is dependent on patient records and accurate documentation by nursing staff. Postoperative desaturation and airway interventions may be inaccurately reported or under-reported and may not include information on behavioral state (i.e., sleeping vs. awake). Although the standard way to measure CO_2 during PSG is via exhaled gas, this measurement may not accurately reflect CO_2 blood concentrations if supplemental oxygen or positive pressure ventilation is being administered;[42] blood sampling or transcutaneous CO_2 monitoring may be a more accurate way to measure intravascular CO_2.[43–45] Intraoperative analgesic and anesthetic agents can contribute to postoperative respiratory depression; however in this dataset, the time of desaturation was not always recorded, and relation to drug administration and desaturation is crucial to assess causality. Moreover, desaturation is only one sign of respiratory distress; other signs of disturbances in oxygenation and ventilation, including use of airway manipulation, oral airways, or supplemental oxygen delivery, were not consistently reported.

Our analysis of postoperative oxygen desaturation was limited to the 2-hour period immediately after surgery primarily because of the paucity of respiratory evaluations thereafter. However, in our clinical experience, the first 2 hours after surgery represent the period of greatest risk for airway complications. We will use our results to develop hypotheses to test in our ongoing prospective studies.

Conclusion

Although univariate analysis suggested that preoperative peak $EtCO_2$ and RDI were both significant predictors of postoperative desaturations, the effects were attenuated after adjusting for age. The strong correlation between age and peak $EtCO_2$ suggests the presence of a relationship that requires further investigation. Nevertheless, it seems clear that patients less than 3 years of age are more likely than older children to have postoperative hypoxemia after AT.

Acknowledgments

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- **What is already known**: Nocturnal hypoxia predicts post-operative respiratory complications after adenotonsillectomy, however, other gas perturbations including peak hypercarbia have not been evaluated.

- **What this article adds**: In this study, we demonstrate a relationship between nocturnal hypercarbia and OSA disease severity as well as postoperative hypoxia.

- **Implications for translation**: Our results suggests that prospective evaluation is needed to better evaluate the relationship between these factors in order to maximize safety in the perioperative period.

Table 1

Demographic characteristics and polysomnography parameters overall and by desaturation

Characteristic	All patients (n=319)	With desaturation (n=20)[*]	Without desaturation (n=299)[*]	P value[†]
Age, years	5 (3–8)	2 (1–3)	6 (3–8)	<0.001
Male sex, no. (%)	168 (52.7)	11 (55)	157 (53)	0.99
BMI-for-age percentile[‡]	84.6 (53.7–98.5)	85.6 (36.4–99.1)	84.5 (53.7–98.5)	0.92
Race, no. (%)				0.17
Caucasian	104 (32.6)	4 (20)	100 (33)	
African-American	196 (61.4)	16 (80)	180 (60)	
Other or unknown	19 (5.9)	0 (0)	19 (6)	
RDI, events/hour	5.9 (2.3–15.3)	14.1 (3.0–24.5)	5.5 (2.3–13.7)	0.08
Obstructive sleep apnea, no. (%)				0.09
None	40 (12.5)	3 (15)	37 (12)	
Mild	106 (33.2)	3 (15)	103 (34)	
Moderate	59 (18.5)	2 (10)	57 (19)	
Severe	114 (35.7)	12 (60)	102 (34)	
Obstructive apnea index, events/hour	1.9 (0.4–7.6)	6.9 (0.4–10.8)	1.8 (0.4–6.8)	0.11
Hypopnea index, events/hour	2.3 (0.8–6.1)	3.5 (1.1–11.0)	2.3 (0.8–5.6)	0.38
Central apnea index, events/hour	0.0 (0.0–0.2)	0.0 (0.0–0.2)	0.0 (0.0–0.2)	0.99
Peak CO_2, mmHg	52 (49–55)	55.5 (48.5–63.5)	52 (49–55)	0.02
Opioid use, no. (%)	295 (92.5)	17 (85)	278 (93)	0.18
Percent O_2 saturation nadir	88 (81–92)	80.5 (74.5–90.5)	88.0 (82–92)	0.048

Data are presented as medians and interquartile ranges unless otherwise specified.

BMI, body mass index; RDI, respiratory disturbance index; O_2, oxygen

[*] Missing data: for BMI-for-age percentile, 9 with desaturation and 92 without desaturation were missing; for O_2 saturation nadir, 1 without desaturation was missing; for obstructive apnea index and hypopnea index, 7 without desaturation were missing; for central apnea index, 10 without desaturation were missing.

[†] P values are from the Wilcoxon rank-sum test or Fisher's exact test.

[‡] BMI-for-age percentile is the percentile of BMI normalized for age and gender in children. CDC calculator used: http://www.cdc.gov/healthyweight/assessing/bmi/childrens_bmi/tool_for_schools.html.

Table 2

Unadjusted odds ratios for desaturation from exact logistic regression analysis

Characteristic	Odds ratio (95% CI)	*P* value
Sex		
Female	0.90 (0.32–2.48)	0.99
Male	Reference	-
Race		
African-American	2.22 (0.69–9.36)	0.23
Other	*	*
Caucasian	Reference	-
Age, in years		
0–2	10.09 (2.13–96.26)	<0.001
3–8	0.97 (0.15–10.41)	0.99
9–17	Reference	-
BMI-for-age percentile		
<85	0.80 (0.19–3.24)	0.95
85	Reference	-
Obstructive sleep apnea		
RDI < 10 events/hour	Reference	-
RDI 10 events/hour	2.89 (1.05–8.42)	0.04
Peak CO_2		
55 mmHg	Reference	-
>55 mmHg	3.38 (1.21–9.47)	0.02
Opioid use		
No	Reference	-
Yes	0.43 (0.11–2.47)	0.36
O_2 Saturation nadir		
<80%	2.93 (0.99–8.24)	0.05
80%	Reference	-

BMI, body mass index; CI, confidence interval; RDI, respiratory disturbance index, O_2=oxygen

*
Could not be estimated.

Table 3

Odds ratios for desaturation from multivariable logistic regression

Characteristic	Model 1		Model 2	
	Odds ratio (95% CI)	*P* value	Odds ratio (95% CI)	*P* value
Sex				
Female	1.74 (0.36–9.43)	0.65	1.44 (0.45–4.63)	0.66
Male	Reference	-	Reference	-
Race				
African American	1.86 (0.35–13.44)	0.66	2.07 (0.52–10.22)	0.40
Other	*	*	*	*
Caucasian	Reference	-	Reference	-
Age, in years				
0–2	12.56 (1.13–216.4)	0.04	10.43 (1.89–110.9)	0.003
3–8	1.61 (0.20–20.37)	0.95	1.18 (0.19–12.78)	0.99
9–17	Reference	-	Reference	-
BMI-for-age percentile				
<85	0.36 (0.06–1.99)	0.31	-	-
85	Reference	-	-	-
Obstructive sleep apnea				
RDI < 10 events/hour	Reference	-	Reference	-
RDI 10 events/hour	0.71 (0.05–5.78)	0.99	0.96 (0.19–4.30)	0.99
Peak EtCO$_2$				
55 mmHg	Reference	-	Reference	-
>55 mmHg	1.38 (0.18–8.44)	0.98	1.49 (0.38–5.45)	0.70
Opioid use				
No	Reference	-	Reference	-
Yes	0.35 (0.04–4.89)	0.53	0.96 (0.19–6.86)	0.99
O$_2$ Saturation nadir				
<80%	1.24 (0.10–16.94)	0.99	1.16 (0.25–5.54)	0.99
80%	Reference	-	Reference	-

BMI, body mass index; CI, confidence interval; RDI, respiratory disturbance index.

Model 1: Data available on 218 patients, 11 desaturated (BMI data missing on n=100).
Model 2: Data available on 318 patients, 20 desaturated

*
Could not be estimated.