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ORIGINAL ARTICLE

Obstructive sleep apnea and history of asthma in snoring children

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Abstract Asthma has been identified as a possible risk factor for Obstructive Sleep Apnea (OSA) in children. It is not known whether parent-reported asthma increases the likelihood of the diagnosis of OSA in snoring children. We hypothesized that snoring children with asthma are more likely to have OSA than snoring children without asthma. This study is a 1-year retrospective review of polysomnogram and questionnaire data collected on 236 patients referred to the University of Maryland Pediatric Sleep laboratory for evaluation of snoring. Of the 236 patients, 58% (137/236) were boys, and 79% (173/219 reporting race) were African-American (AA). The age at referral was 7.2±3.7 years (mean±S.D.). Mean body mass index (BMI) percentile was 73.4±32.3%, with 43.2% (54/125) >95th percentile. A history of asthma was reported by 31.4% (74/236); no subject was symptomatic on the night of the study. We found no increased risk for polysomnographically diagnosed OSA for asthmatics. To the contrary, by logistic regression analysis, a parent/guardian report of asthma decreased the odds of having OSA by 34% (p=0.027), controlling for individual and socioeconomic factors and assessment results. Polysomnographic (PSG) differences between asthmatic and non-asthmatic children were found in only the arousal index (11.0 vs.9.3±6.5/h, p=0.099) and total sleep time (337.1±64.3 vs. 347±65.2 min, p=0.1) In a referral-based group of predominantly AA inner-city snoring children, asymptomatic asthma decreased the likelihood of OSA.

Keywords African-American · Snoring · OSA · Asthma · Polysomnogram

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Introduction

Obstructive sleep apnea (OSA) refers to the occurrence of repetitive episodes of complete or partial upper airway obstruction during sleep, and affects 1–3% of children in the US [1].OSA and primary snoring (PS) can be considered on a continuum, from infrequent partial sleep-related upper airway obstruction without significant hypoxemia or hypercarbia (PS) to apnea with hypoxemia and carbon dioxide retention [2]. Sleep disturbances as a consequence of nocturnal asthma have been reported in children [3], but there is only a small body of literature describing polysomnographic (PSG) features in asthma. Respiratory illnesses such as asthma, which affects 7.5–10% of American children, and atopy are reported to be risk factors for the development of OSA [4–6].

While OSA and asthma are considered separate diagnoses, some have suggested that they may in fact be associated. The pathophysiology of these disorders likely overlap, given that both are affected by inflammation, neural input, and anatomic factors such as obesity [5]. Nocturnal asthma may cause sleep disruption and/or poor quality sleep; unstable asthma treated with oral corticosteroids may increase the risk of OSA [7]. Snoring and noisy breathing are the hallmarks of OSA in children and are also recognized as common complaints of children with asthma [8]. These symptoms are common reasons for referral for PSG evaluation.

The primary aim of this study is to determine whether there is an association between OSA and asthma in pediatric patients referred for PSG for nocturnal snoring. We hypothesized that asthma, as reported on a medical history questionnaire obtained on the night of study would increase the likelihood of OSA. In addition, we expected to find differences in sleep architecture and physiological parameters between asthmatic and non-asthmatic snorers.

Materials and methods

Study sample

Data were obtained from a 1-year (July 2003 to June 2004) retrospective review of clinical charts and overnight PSG results of patients referred to the pediatric sleep laboratory at the University of Maryland for evaluation of obstructive sleep apnea with a chief complaint of snoring. The Institutional Review Board at the University of Maryland approved this study and granted an "exempt protocol" status for the chart review.

Charts and PSG results on 236 patients between the ages of 2 and 15 years were reviewed. Current symptom and history data were obtained from a questionnaire that was completed by the adult caregiver accompanying the child on the night of the study. The questionnaire included patient demographics, the patient's past medical history (including asthma), medication use, sleep, and daytime behaviors. Asthma case definition was determined by an answer of "yes" asthma (item 1), as a listed diagnosis (item 2), or asthma related medication use (item 3, General Medical Information—Appendix) as previously used in Baltimore children [9]. Medical insurance information was available from hospital intake data. We did not directly collect data on household income or other indices of socioeconomic status. As reported by other authors, we used median household incomes from the zip code of the patients' residence using US government census information for the year 2000 as an indirect estimation of socioeconomic status [10, 11]. In addition, we coded zip codes falling within the Baltimore Metropolitan Statistical Area as urban.

Only children aged 2 to 15 years, referred to the sleep laboratory for diagnostic studies for the chief complaint of snoring, were included in this study. Even though our laboratory studies patients up to 21 years of age, we chose the age group of 2–15 for analysis to maintain consistency in the application of pediatric criteria for the diagnosis of OSA. Those referred for oxygen, continuous or bilevel positive airway pressure titration, or evaluation of parasomnias were excluded from the analysis. The pediatric sleep laboratory at the University of Maryland serves largely an inner-city population.

Standard overnight monitoring was accomplished with the Alice 4 diagnostic system (Respironics, Murrysville, PA, USA). The patient slept in a quiet darkened room up to 7 h with a parent or guardian present. The montage included EEG leads O1A2, O2A1, C1A2, C2A1, LOC, ROC, submentalis EMG, EKG, measurements of airflow (oronasal thermistor) and end tidal CO_2 ($ETCO_2$), chest and abdominal impedance using strain gauges, and oxyhemoglobin saturation using a Nellcor pulse oximeter. Respiratory events were described as: 1) obstructive apneas, with cessations of airflow with continued thoracic and/or abdominal respiratory effort lasting two respiratory cycles in duration; or 2) hypopneas, with reductions of airflow >50% associated with ≥4% fall in oxygen saturation or post-event arousal [12]. The severity of OSA was defined by the apnea–hypopnea index (AHI; number of obstructive apneas and hypopneas per hour of sleep). The arousal index included respiratory and non-respiratory arousals per hour of sleep. Baseline and nadir oxyhemoglobin saturation, baseline and peak $ETCO_2$ in addition to sleep stage were recorded. No patient was acutely ill on the night of the study. Polysomnographic scoring was performed in 30-s epochs according to the Rechtshaffen–Kales criteria [13] by the same technician and reviewed by one of two pulmonologists.

When the height and weight were obtained at the time of the overnight PSG, we were able to calculate the body mass index (BMI) based on the Center for Disease Control nutrition and activity website and is expressed in kg/m^2 [14]. BMI percentile was calculated to represent 5th–95th percentile as "normal" and >95th as "overweight" [15]. OSA is defined as an obstructive AHI of five or more events per hour and/or an obstructive apnea index of one or more events per hour [16, 17]. For the purposes of this paper, OSA was defined as AHI>2.

Analysis

Data were analyzed using Stata/SE for Windows (StataCorp. 2005. Stata Statistical Software: Release 9.2. College Station, TX, USA: StataCorp LP). Data were compiled and where appropriate, expressed as mean±SD when data were normally distributed (as identified using the Stata command

"sktest" [18], group differences between the means were assessed using the two sample *t* test. However, when data were non-normally distributed, differences between group medians were compared using the Mann–Whitney *U* test, and are so indicated. In addition, some data were prepared using dichotomous valuations, in which cases chi-square testing was used. Logistic regression was performed to ascertain predictors of OSA controlling for known risk factors such as age, gender, BMI, ethnicity, medical insurance status, and median income for zip code. Differences in OSA prevalences were compared using chi-square. We are unaware of published consensus guidelines for OSA severity in children; therefore, the AHI severity levels used were our best clinical judgment. For some analyses, where indicated, we analyzed data using different definitions of "positive" for OSA: an AHI of >2, or >5 as there is no agreement among clinicians which threshold to use.

Due to the preliminary and clinical nature of the study, in which a type II or beta error is less troublesome and can generate further hypotheses and research questions, we relaxed our assumptions of significance to reject the null hypothesis at the 10% level. In future investigations on this population, the knowledge generated from this preliminary study will permit us to recruit larger samples, therefore permitting us to use more rigorous assumptions of significance.

Results

Clinical characteristics

The average age of the cohort was 7.18±3.74 years (mean± SD), with most of the children <11 years old (n=190 or 80.5%). There were more boys (n=137 or 58%) than girls. Ethnicity data was available on 219 subjects, with the majority being African-American (n=173 or 79%), followed by Caucasians (n=39 or 17.8%). Hispanics and Asians were minimally represented (2.3% and 0.9%, respectively). This reflects the socio-demographic profile of Baltimore City in which 20% of the population is between 5 and 17 years and 90% of these children are African-American [19]. The median household income estimated from zip codes and census data was $36, 661±$15,669. Most of the children in this study were on medical assistance (n=171, 72.5%), showing that they came from households in the lower-income brackets.

Because height was not consistently measured on all subjects on the night of the PSG, BMI was calculated on 124 subjects only. The mean BMI was 22.4±8.27 kg/m^2, and the mean BMI percentile was 74.3±31.7. Most of the children (71/125 or 56.8%) had BMI calculated at <95th percentile. The remaining 54 children were overweight. Weight was available on 207 subjects; the mean was 79.6±55.2 kg.

Asthma was reported by 74 (31.4%) of the caregivers. Table 1 shows the comparison of asthmatic and non-asthmatic subjects with respect to gender, race, AHI, BMI percentile, median household income by zip code, and insurance status. There were no significant differences in any demographic variable between asthmatics and non-asthmatics. Of the 41 subjects with asthma on whom we could calculate BMI, 20 were obese.

Whether OSA was defined as an AHI >2 or >5 there were no significant differences in OSA prevalence for asthmatics compared to non-asthmatics in bivariate analyses. PSG findings with an AHI<2 was diagnosed in 139/236 (58.9%) of all subjects (Table 2). The levels of severity that were analyzed were AHI <2, 2≤5, 5–10, and >10.) OSA, described as AHI≥2 was diagnosed in 97/236 (41.1%) of all snorers referred for a PSG. AHI 2–4.9 was identified in 36 (15.3%), and AHI>5 was seen in 61 subjects. While we hypothesized that asthma would increase the severity of OSA, the distribution of OSA severity was not significantly different between asthmatics and non-asthmatics in the bivariate analyses (data not shown, p=0.622). Please refer to Table 3 for a listing and description of all variables used for analysis.

However, in logistic regressions that included several PSG results as further independent variables and controlled for individual factors and socioeconomic factors (Table 4), we found that reported asthma significantly decreased the odds of OSA (OR [odds ratio] 0.399, 0.160–0.994, p=0.040).

In bivariate analysis, African-American children were significantly more likely to have OSA (chi-square 13.05, p<0.0001), but were no more likely to have asthma (chi-square 0.097, p=0.76). Because this sample is primarily African-American, it was possible that this finding is artifactual (data not shown). We explored the relationship between race and OSA further in the aforementioned

Table 1 Clinical characteristics of asthmatics and non-asthmatics

Variable	Asthma (N=74) N (column %)	Non-asthma (N=162)
Boys, N (%)	43 (58.1%)	94 (58.0%)
African-American, N (%)	53 (71.6%)	120 (74.1%)
BMI >95th percentile	20 (27.0%)	34 (21.0%)
Weight for age >95th percentile	25 (33.8%)	48 (29.6%)
Private Insurance	20 (27.0%)	45 (27.8%)
	$\bar{x}$ ± SD	
Age (years±SD)	7.5±3.6	7.0±3.8
AHI (mean±SD)	6.9±14.9	4.2±6.4
BMI percentile (±SD)	76.9±31.6	71.7±32.6
Estimated median household income ($)	37,889±16,635	36,154±15,416

All NS

Table 2 Distribution of OSA severity and asthma

AHI	Asthma	Non-asthma	Total
	N (column %)		
<2	44 (59.5%)	95 (58.6%)	139 (58.9%)
2–4.9	9 (12.1%)	27 (16.7%)	36 (15.3%)
>5	21 (28.4%)	40 (24.7%)	61 (25.9%)
Total	74	162	236

P=0.622

logistic regression. In this regression, we found that African-American children have 2.85 times higher odds of OSA. (Table 4, p=0.056). Likewise, children on medical assistance were at increased odds of OSA compared to those with private insurance in the bivariate analysis (chi-square 4.68, p=0.031), but that finding was not supported in the logistic regression. The AHI was lower in the African-American group, but the difference was not significant 3.3 vs.5.7 obstructive events/hour, p=0.14). In bivariate analysis, African-Americans were significantly more likely to have an AHI>5 (chi-square 5.40, p=0.02) or to have an AHI>2 (chi-square 20.06, p<0.0001).

Sleep disruption can be a manifestation of nocturnal asthma. We investigated parameters of sleep fragmentation for children with and without asthma by polysomnography.

Table 3 Variable identification and range

Variable name	Value range	Level
Independent/dependent variables		
OSA	0–1	Dichotomous
Asthma	0–1	Dichotomous
PSG results		
Baseline ETCO2 (torr)	23–52	Continuous
Peak ETCO2 (torr)	35–71	Continuous
Baseline SaO2%	88–100	Continuous
Nadir SaO2%	31–97	Continuous
T90 (minutes)	0–205	Continuous
Individual factors		
Boy	0–1	Dichotomous
African- American	0–1	Dichotomous
Age group	1–4	Categorical (1=1–5 yrs, 2=6–10 yrs, 3=11–14 yrs, 4≥15 yrs)
Weight between 5th & 95th percentile	0–1	Dichotomous
Socioeconomic factors		
Private insurance	0–1	Dichotomous
Living in urban area	0–1	Dichotomous
Median income for zip code	6,148–03,879	Continuous

Table 4 Predictors of OSA

OSA	Odds ratio	$P>\|Z\|$	95% CI	
Asthma reported by parent/guardian*	0.3993	0.048	0.1604	0.9939
Baseline $ETCO_2$ (torr)	0.8825	0.266	0.7081	1.0999
Peak $ETCO_2$ (torr)	1.1525	0.112	0.9674	1.3732
Baseline SaO_2%**	1.3170	0.095	0.9530	1.8199
Nadir SaO_2%**	0.9145	0.063	0.8321	1.0050
T90 (minutes)	1.1436	0.230	0.9184	1.4240
boy	1.3534	0.460	0.6068	3.0187
African- American**	2.8500	0.056	0.9717	8.3588
ages 6–10	0.8803	0.786	0.3510	2.2079
ages 11–15	1.2031	0.742	0.4004	3.6148
weight for age <95%ile	0.6877	0.474	0.2469	1.9152
Private insurance	0.3688	0.203	0.0794	1.7134
Living in urban area	1.0000	0.275	0.9999	1.0000
Median income for zip code*	0.3993	0.048	0.1604	0.9939

Logistic regression: Log likelihood=−79.7200404; number of obs = 156 (decreased observations are due to missing data); LR chi^2 (15)= 48.44; Prob >chi^2 =0.0000; pseudo R^2 =0.2330
*Significant at $p\leq0.05$
**Significant at $p\leq0.10$

Total sleep time was significantly decreased in asthmatics at 333±64 min vs. 347±65 min for non-asthmatics (Mann–Whitney U, z=1.656, p=0.10). Asthmatics had an increased arousal index (11.3±7.4) compared to non-asthmatics (9.3± 6.4, p=0.099), with no difference in slow wave sleep as a percentage of total sleep time (SWS/TST%), rapid eye movement as a percentage of total sleep time (REM/TST %), sleep efficiency (time asleep as a percentage of total sleep time), or physiological measurements such as baseline and nadir oxyhemoglobin saturation (SaO_2%), exhaled carbon dioxide ($ETCO_2$), or time spent with oxyhemoglobin saturations <90% (T90).

Discussion

The main finding in this study is that a history of asthma reported by a snoring child's guardian decreases the likelihood of OSA, but is associated with mild sleep disturbance. This finding actually was opposite to what we originally postulated. Surprisingly, there was no major difference between asthmatics and non-asthmatics in parameters that would suggest poor sleep. In the ensuing discussion, we review these findings in light of the currently available literature.

Snoring and noisy breathing are recognized as common symptoms in pediatric patients with asthma [8]. In this regard, several theories have been proposed to explain why

upper airway abnormalities might constitute a risk for asthma or vice versa. OSA and asthma share risk factors such as allergic rhinitis, gastroesophageal reflux, and obesity [20, 21]. In addition, similar cytokine, chemokine, and histologic changes are seen in both OSA and asthma [22]. Abnormalities of the nasopharynx and lower airways may co-exist due to similar airway responses to inflammatory or atopic stimuli [20]. Sleep deprivation, chronic upper airway edema, and inflammation associated with OSA may further exacerbate nocturnal asthma symptoms [22, 23]. The "one airway" hypothesis suggests that upper airway inflammation and intermittent hypoxia from obstruction may influence the expression and severity of disease of the lower respiratory tract [24]. CPAP treatment of OSA improves asthma control in adults, particularly nocturnal attacks [25] and bronchial hyperresponsiveness [26]. Tonsillectomy and/or adenoidectomy (T&A), rather than CPAP, is the first line of treatment in pediatric OSA and is curative in 75–100% of the cases [5]. It is not known if treatment of OSA changes the course of asthma in pediatric populations.

In spite of the above rationale, self-reported asthma was actually inversely correlated with the likelihood of OSA although not with severity. We speculate that this surprising finding is due to a referral bias. It is likely that parents of a child diagnosed with asthma are more vigilant regarding nighttime symptoms and/or are referred more often due to increased interactions with medical professionals. Since asthma symptoms are often worse at night, noisy breathing may have been mistaken for snoring, prompting a referral for a sleep study. In other words, self-reported asthma predicted a lower likelihood of having OSA. We do not have a biologically plausible explanation for this. We speculate that our unexpected finding is a result of parents and caregivers of asthmatic children being more vigilant to nocturnal symptoms. Since both asthma and OSA can have overlapping nocturnal symptoms, there may be some reporting bias on the part of parents, who might choose to "over" report the presence of asthma in children who snore. We did not perform objective assessments of airways obstruction or bronchial hyperreactivity in this retrospective analysis. Finally, we did not evaluate atopy in this cohort, which could predispose to both asthma and OSA. However, to determine if there is a positive relationship between asthma and OSA, a population-based study would need to be performed.

Thirty-three percent of children in this study had guardian-reported asthma, which is three times the national prevalence rate of 10% [9, 28] and statewide asthma prevalence of 10.6% [29]. This rate is also higher than the 20% asthma prevalence rate in some inner-city Baltimore schools for children from the same catchment area [9]. Data were obtained from a clinical questionnaire that was not validated at the time of this study. However, the majority of the clinical questions were similar to that used in the BASS questionnaire, which is a 50-item validated questionnaire for OSA screening in children [27]. Asthma "yes" as a listed diagnosis on school emergency forms, albuterol or inhaled steroid was a method used in a previous study for case definition of asthma in Baltimore children [9].

Body mass index percentiles of participants in this study revealed that more than 40% were obese with a BMI >95th percentile, which may have influenced the high prevalence of asthma in this snoring cohort. The Cleveland Children's Sleep and Health Study found that there was a 1.89-fold increased risk of wheeze, but not asthma in 8- to 11-year-old inner city children with sleep disordered breathing (snoring with or without OSA) [15] and that sleep disordered breathing might explain in part the relationship between obesity and asthma.

Asthma and OSA have common risk factors such as African-American ethnicity, obesity, atopy, and poor socioeconomic status [8, 20, 30, 31]. Consistent with the literature [5, 6, 21, 32] we found that African-American children were 3.13 times more likely to have OSA and 4.9 times more likely to have asthma than other children. There was also an increased risk of OSA in children on medical assistance compared to those with private insurance. Low SES, severe neighborhood disadvantage, and neighborhood distress are proposed risk factors for childhood OSA [33]. This may reflect increased exposure to environmental triggers of airways inflammation as the housing stock of Baltimore's inner city is burdened with cockroach and mouse allergen [34], which contribute to symptoms of allergic rhinitis and asthma. We did not find, however, a difference in OSA severity between African-American and Caucasian children referred for PSG. This may be because African Americans were overrepresented in our cohort. While others have reported that asthma increases the prevalence of snoring [8, 31], it has not been previously reported whether or not a history of asthma increases the risk or severity OSA in children. Chi-square analysis showed that a diagnosis of asthma did not increase the severity of AHI (p=0.549). Indeed, by logistic regression analysis, a self-report of asthma decreased the odds of having OSA (AHI>2) by 40%, controlling for individual and socioeconomic factors.

There are few data on the polysomnographic findings in subjects with asthma. Initial reports over three decades ago by Kales et al.[35] suggest that children with asthma have a decrease in stage 4 sleep, total sleep time, and frequent awakenings when compared to normal controls. Some authors [36] have demonstrated a greater fall in nocturnal saturation in subjects with stable asthma when compared to healthy individuals [36, 37]. In our study, asthmatic snorers had worse sleep quality as judged by indices of sleep

fragmentation than non-asthmatic snorers. This was demonstrated by a slightly higher arousal index, and lower TST. Despite the small increase in arousal index in those with asthma, we are unclear of the clinical relevance of this finding. We do acknowledge the difficulty in scoring of arousals; however, consistency was maintained by having the same technician read all the studies. Increased sleep fragmentation in asthma could also be due to the use of asthma medications such as albuterol, theophylline, and corticosteroids. None of our patients was using these medications the night of the study. While our patients were asymptomatic per parent and technician observation, it is possible that subclinical airways obstruction could have led to greater sleep fragmentation. We did not measure airflow by spirometry during the night to determine if this was the case.

It is unclear if snoring per se alters sleep architecture. In a study of 14 adults, eight "heavy" snorers and six "light" snorers, Hoffstein et al. [38] reported that there was no change in TST, SWS, and REM time between the two groups, but found that snoring intensity did affect sleep efficiency. In another study, the same authors reported that in a group of adult symptomatic snorers and asymptomatic non-snorers, arousals were respiratory in origin in the snorers, but were EEG arousals in non-snorers. The total arousal indexes in both groups, however, were similar [39].

A major limitation of this study was the lack of an objective diagnosis of asthma as well as the fact that there were no data in the questionnaire about the frequency of and severity of the most recent asthma exacerbation. This was a referral-based and not a population-based study, which may explain the high prevalence of reported asthma. A parent report of asthma is the standard for the Behavioral Risk Factor Survey [40], which is a national survey instrument that has been validated. Further study of a cohort of asthmatic children when symptoms are well controlled compared to those with poorly controlled asthma would elucidate further the relationship between pediatric asthma and risk of obstructive sleep apnea.

In conclusion, predominantly African-American inner-city children, referred to the sleep laboratory for an evaluation of snoring, have a high prevalence of self-reported asthma. The odds of OSA were lower in those with prior history of asthma. OSA severity did not change with or without a history of asthma. A higher arousal index in the asymptomatic stable asthmatics suggests a mechanism other than overt nocturnal symptoms of asthma responsible for sleep fragmentation. Objective testing using methacholine or exercise challenges and the use of validated asthma questionnaires to confirm prior vs. current history of asthma will be useful to study the association between asthma and OSA more thoroughly.

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Appendix

DATE: _____

PEDIATRIC SLEEP DISORDERS CENTER
NIGHT SLEEP STUDY QUESTIONNAIRE

CHILD'S NAME _____ ***DATE OF BIRTH*** _____

HOME ADDRESS _____

CITY _____ ***STATE*** _____ ***ZIP CODE*** _____

TELEPHONE _____ ***WORK*** _____

CELL _____

CURRENT PEDIATRICIAN _____

TELEPHONE _____ **FAX** _____

NAME OF PERSON FILLING OUT THIS FORM _____

RELATIONSHIP TO CHILD _____

CLOSEST FRIEND/RELATIVE NOT LIVING WITH YOU _____

ADDRESS _____

CITY _____ **STATE** _____ **ZIP CODE** _____

TELEPHONE _____

RELATIONSHIP TO CHILD _____

- -

HOW MUCH DID YOUR CHILD WEIGH AT BIRTH _____

WAS YOU CHILD BORN AT 9 MONTHS (40 WEEKS)? □No □Yes

IF BEFORE, HOW ANY WEEKS? _____

IF AFTER, HOW MANY WEEKS? _____

FAMILY INFORMATION

1. Do you have other children? If so what are their names, how old are they?

NAME	DATE OF BIRTH
_____	_____
_____	_____
_____	_____
_____	_____

2. Do your other children have problems with sleep? ☐**No** ☐**Yes**

If so, what? _____

_____

3. Does either parent have sleep apnea as diagnosed by a doctor? ☐ **No** ☐ **Yes**

4. Does a grandparent have apnea as diagnosed by a doctor? ☐ **No** ☐ **Yes**

5. Does either parent snore? ☐ **No** ☐ **Yes**

6 Does either parent smoke? ☐ **No** ☐ **Yes**

BRIEF MEDICAL HISTORY

1. Has this child ever had surgery on tonsils, adenoids, or palate? ☐ **No** ☐ **Yes**
(circle all that apply)

If so, what was the date of surgery? _____

If so, where was the surgery performed and who was the doctor?

(Place) _____

(Doctor) _____

GENERAL MEDICAL INFORMATION

1. Does this child have asthma? ☐**No** ☐**Yes**

2. Does this child have any medical problems? If so, please list below:

a. _____ **d.** _____

b. _____ **e.** _____

c. _____ **f.** _____

3. List any medications your child is presently taking (including non-prescription)

a. _____ **d.** _____

b. _____ **e.** _____

c. _____ **f.** _____

4. Is your child on oxygen? ☐ **None** ☐ **Continuous**

☐ **Night only** ☐ **Feeding only**

If so, what are the settings? _____

If so, which equipment company do you use? _____

SLEEP HABITS

1. Does your child have excessive movements during sleep?

☐ **usually still** ☐ **normal movements**

☐ **somewhat restless** ☐ **extremely restless**

2. Does your child have excessive nightmares?

☐ **never** ☐ **rarely** ☐ **most nights**

☐ **more than once/week** ☐ **1 to 4 times/month**

3.Does your child walk during sleep?

☐ **never** ☐ **rarely** ☐ **most nights**

☐ **more than once/week** ☐ **1 to 4 times/month**

4. Does your child talk during sleep?

☐ **never** ☐ **rarely** ☐ **most nights**

☐ **more than once/week** ☐ **1 to 4 times/month**

5. Does your child wake during night, i.e. for a drink, bathroom?

☐ **never** ☐ **rarely** ☐ **most nights**

☐ **more than once/week** ☐ **1 to 4 times/month**

6. If your child is over 5 years old, does he/she wet the bed?

☐ **never** ☐ **rarely** ☐ **most nights**

☐ **more than once/week** ☐ **1 to 4 times/month**

7. Does your child have heavy sweating during sleep?

☐ **never** ☐ **rarely** ☐ **most nights**

☐ **more than once/week** ☐ **1 to 4 times/month**

8. Does your child's lips or skin turn blue during sleep? ☐ No ☐ Yes

9. Do you ever see or hear your child stop breathing during sleep? ☐ No ☐ Yes

10. Do you ever see your child struggling to breathe during sleep? ☐ No ☐ Yes

11. Do you ever shake your child to make him/her breathe?

☐ never ☐ rarely ☐ most nights

☐ more than once/week ☐ 1 to 4 times/month

12. Do you ever watch your child sleeping at night, afraid about his/her breathing?

☐ No ☐ Yes

13. Does the child sleep with you in the same bed? ☐ No ☐ Yes

14. How often does your child snore?

☐ never ☐ rarely ☐ most nights

☐ more than once/week ☐ 1 to 4 times/month

15. How loud is your child's snoring?

☐ mild to quiet ☐ medium to moderately loud

☐ loud ☐ extremely loud

16. Does your child toss and turn in his/her sleep? ☐ No ☐ Yes

17. Does your child have allergies? ☐ No ☐ Yes

(If so, what kind): _____

18. Does your child have ear infections?

☐ never ☐ rarely

☐ 2 to 5 times/year ☐ 6 or more times/year

19. How often does your child have a sore throat?

☐ never ☐ rarely

☐ 2 to 5 times/year ☐ 6 or more times/year

20. Is your child hard to awaken in morning? ☐ No ☐ Yes

21. Does your child have morning headaches? ☐ No ☐ Yes

22. Does your child have bad breath? ☐ No ☐ Yes

23. Is child a daytime mouth breather?

☐ never ☐ sometimes ☐ usually

24. Does your child have a runny nose?

☐ only with a cold ☐ usually runny

☐ **sometimes between colds** ☐ **constantly, every day**

25. Is child sleepy during the day?

☐ **never** ☐ **sometimes** ☐ **usually**

26. Is your child overweight? ☐ **No** ☐ **Yes**

How much weight has your child gained in the past year? _____

27. Describe your child's social interactions:

☐ **withdrawn** ☐ **outgoing** ☐ **typical or average for age**

28. Overall, what grades does your child receive in school? _____

29. Has there been a recent decline in school performance? ☐ **No** ☐ **Yes**

30 How would you describe your child's appetite at present?

☐ **poor** ☐ **excessive** ☐ **typical/average**

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