Sleep Disordered Breathing in Children in a General Population Sample: Prevalence and Risk Factors

Edward O. Bixler, PhD[1]; Alexandros N. Vgontzas, MD[1]; Hung-Mo Lin, PhD[2]; Duanping Liao, MD, PhD[2]; Susan Calhoun, PhD[1]; Antonio Vela-Bueno, MD[1]; Fred Fedok, MD[3]; Vukmir Vlasic, MD[4]; Gavin Graff, MD[4]

[1]Sleep Research & Treatment Center, Departments of Psychiatry, [2]Public Health Sciences, [3]ENT, and [4]Pediatrics, Penn State University College of Medicine, Hershey, PA

Study Objectives: Assess the prevalence based on clinically meaningful criteria (i.e., blood pressure) and identify risk factors of sleep disordered breathing (SDB) in a representative sample of elementary school children.

Design: A random sample of the local elementary school children (K-5) were assessed using a two-phased strategy. In phase I a brief questionnaire was completed by a parent of each child in local elementary schools (N = 5,740), with a response rate of 78.5%. In phase II, randomly selected children and their parent spent a night in our sleep laboratory (N = 700) with a response rate of 70.0%.

Setting: University sleep laboratory

Participants: Children enrolled in local elementary schools.

Intervention: None

Measurement & Results: Each child was assessed with a full polysomnogram and completed a history/physical examination including an electrocardiogram, otolaryngology examination, and pulmonary evaluation. The prevalence of moderate SDB (apnea-hypopnea index ≥ 5) was 1.2%. The independent risk factors included nasal abnormalities and minority associated only with mild (1 < AHI < 5) SDB and snoring and waist circumference associated with all levels of SDB. Tonsil size, based on visual inspection, was not an independent risk factor.

Conclusion: The prevalence of AHI ≥ 5 was 1.2% in a representative sample of elementary school children. Risk factors for SDB included waist circumference, nasal abnormalities (e.g., chronic sinusitis/rhinitis), and minority. The strong linear relationship between waist circumference and BMI across all degrees of severity of SDB suggests that, as in adults, metabolic factors may be among the most important risk factors for SDB in children.

Keywords: SBD, children, prevalence, risk factors

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THE PREVALENCE OF SLEEP DISORDERED BREATHING (SDB) IN CHILDREN, BASED ON OBJECTIVE FINDINGS, HAS BEEN ESTIMATED IN SEVERAL STUDIES.[1-10] These prevalence estimates have varied widely from 0.7% to 13.0%. This wide range of prevalence is at least partially due to the fact that SDB in children was defined based on an assortment of methods of assessing for the presence of SDB. These methods included: only hemoglobin oxygen saturation (SpO_2)[2,8]; SpO_2 + airflow[3,4]; and SpO_2 + airflow + effort.[5,7,9] Only 3 of these studies used a full polysomnogram (PSG),[1,6,10] however, the number of subjects evaluated in these PSG studies was very small (N = 12–50). The majority of these studies had relatively narrow age ranges,[2-4,6-10] while others had relatively wide age ranges.[1,5] Some of these studies were primarily focused on ages < 6 years [2-4,8] while others included subjects as old as 18 years.[1,5]

Another limitation of the available studies is that they have not systematically assessed a wide range of risk factors that may contribute to SDB in children. This may reflect the paucity studies large enough to evaluate potential variables. Thus, the objectives of this study were (1) to establish the prevalence of SDB based on cutoff points of respiratory events that we have previously observed to be associated with a clinically significant risk (i.e., elevated systolic blood pressure), and (2) to identify independent risk factors for SDB. This study is based on the largest population-based sample of elementary school-aged children available to date.

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Address correspondence to: Edward O. Bixler, PhD, Penn State University College of Medicine, Sleep Research & Treatment Center, Department of Psychiatry MC:H073, 500 University Dr, Hershey PA 17033; Tel: (717) 531-5556; Fax: (717) 531-6491; E-mail: ebixler@hmc.psu.edu

METHODS

This study was designed as a 2-phase study: the first phase involved collection of general information from parents about their children's sleep and behavioral patterns.[11] The second phase involved collection of more detailed data in our General Clinical Research Center (GCRC) on a randomly selected subset from the first phase. The study was reviewed and approved by our institutional review board as well as the GCRC review board.

This study was completed over a 5 year period. For the first phase each year elementary schools (K-5) with approximately 1,500 students were selected. In order to establish a representative sample, we sent a questionnaire home with every child in the selected schools with consent forms to be completed by the parent. The questionnaire used was based on the validated survey published by Stradling.[2] We added questions regarding height, weight, age, gender, race, and ethnicity. We defined minority as any non-Caucasian response for race or ethnicity. Over the course of 5 years, we assessed all 18 public elementary schools within 3 school districts within Dauphin County. We sent home 7,312 questionnaires; 5,740 were returned, for a response rate of 78.5%.

Table 1—Prevalence, 95% Confidence Interval, and [Sample Size] of SDB Status in Children

Total Sample	None	Primary Snore	1 ≤ AHI < 5	AHI ≥ 5
(N = 700)	58.3% (54.7, 62.0) [409]	15.5% (12.9, 18.3) [108]	25.0% (21.9, 28.3) [175]	1.2% (0.6, 2.2) [8]
Age (years)				
5-8 (N = 306)	59.5% (53.9, 64.8) [182]	16.0% (12.3, 20.5) [49]	24.3% (19.7, 29.3) [74]	0.2% (0.1, 1.8) [1]
9-12 (N = 394)	57.5% (52.7, 62.4) [227]	15.0% (11.7, 18.8) [59]	25.5% (21.6, 30.2) [101]	2.0% (0.9, 3.6) [7]
BMI Percentile				
< 85 (N = 497)	62.6% (58.2, 66.7) [311]	14.5% (11.7, 17.9) [72]	22.3% (18.9, 26.2) [111]	0.6% (0.2, 1.8) [3]
> = 85 < 95 (N = 102)	53.8% (44.3, 63.3) [55]	12.8% (7.6, 20.6) [13]	31.4% (23.2, 40.9) [32]	2.0% (0.6, 6.8) [2]
> = 95 (N = 101)	42.0% (33.3, 52.3) [43]	22.8% (15.7, 31.9) [23]	32.0% (23.4, 41.3) [32]	3.2% (1.1, 8.4) [3]

The procedure for Phase II of this study was initiated each year by randomly selecting 200 children based on stratification for grade, gender, and risk for SDB from the current year's returned questionnaires. We studied 704 children in this phase. Four children did not complete the PSG recording; thus 700 children were included in this study, for a response rate of 70.0%. Each child with a parent spent one night in the sleep laboratory. A second consent for the more detailed evaluation was signed by the parent and assented to by the child. During this time the child had a detailed evaluation including a physical examination and a 9-hour PSG. The standardized physical examination included a visual evaluation of the nose and throat by an ENT specialist and an evaluation of the respiratory function by a pediatric pulmonologist. The physical examination was completed in the evening prior to the PSG and included measurements of height, weight, hip circumference, waist circumference, and neck circumference, and blood pressure. Blood pressure was measured using an automated system (vital signs monitor #006-001-01, Welch Allyn, Skaneateles Falls, NY). Height was measured in cm using a stadiometer (model # 242, SECA Corp. Hanover, MD) and weight was assessed in kg (model 758c, Cardinal Scale Manufacturing, Webb City, MO). The waist was measured in cm at the top of the iliac crest and the neck at the cricothyroid membrane.

All subjects were evaluated in our GCRC for one night in sound-attenuated and temperature-controlled rooms. During this time the child's sleep was continuously monitored for 9 hours (Gamma Research Data Acquisition and Analysis System; Grass-Telefactor, West Warwich, RI). A 4-channel electroencephalogram, a 2-channel electroculogram, and a single-channel electromyogram were recorded. The sleep records were subsequently scored independently according to standardized criteria.[12] Respiration was monitored throughout the night with a nasal pressure cannula (MP 45-871 ± 2 cm H_2O, Validyne Engineering, Northridge, CA), thermocouple at the nose and mouth (model TCT R, Grass-Telefactor), and thoracic and abdominal strain gauges (model 1312 Sleepmate Technologies Midlothian, VA). Snoring sounds were monitored by a microphone attached to the throat (model 1250 Sleepmate Technologies, Midlothian, VA). A single-channel EKG was also recorded. All-night SpO_2 was obtained from the finger (model 8800, Nonin Medical, Plymouth, MN).

We defined sleep apnea using criteria that are currently used clinically.[13,14] All records were double scored for SDB. An apnea was defined as a cessation of airflow ≥ 5 seconds and an out of phase strain gauge movement. A hypopnea was defined as a reduction of airflow of approximately 50% with an associated decrease in oxygen saturation (SpO_2) ≥ 3% or an associated arousal. Snoring was monitored objectively (PSG) as well as by parent report. We have previously reported that these 2 sources of data do not overlap well ($\kappa = 0.109$) and that objective snoring was more strongly associated with blood pressure and AHI than subjective snoring.[15] Thus, we used the objective snoring for all analysis in this study.

For analysis purposes we categorized SDB into primary snore (AHI < 1 and snore), mild SDB (1 ≤ AHI < 5), and moderate SDB (AHI ≥ 5). We did not assess the clinical threshold of AI > 1 because we had previously observed that this threshold was not associated with elevated blood pressure while AHI ≥ 5 was.[15] The BMI was adjusted for age and gender based on CDC criteria.[16] We established compensatory weights to obtain estimates of the original target population as well as to adjust for the age and gender distribution of the nation as a whole. Univariate comparisons were made using ANOVA or χ^2. Logistic regression was used for the multivariate analysis. All analyses were performed using SAS version 9.1.

RESULTS

The final sample of 700 children consisted of 52.2% girls. The age range was 5–12 years, with an average age of 111.0 ± 0.8 months. Approximately one quarter (23.8%) of our sample was minority (Black not Hispanic = 13.8%, Hispanic = 6.3%). Our sample tended to be taller than expected with an average height percentile of 58.6 ± 1.1 as well as heavier with an average weight percentile of 61.1 ± 1.1. The average AHI was 0.8 ± 0.06 with a maximum value of 24.6 and an interquartile range of 0.12, 0.38, and 1.07. The prevalence of AHI associated with primary snore was 15.5%, with mild SDB was 25.0%, and with moderate SDB was 1.2% (Table 1).

The distribution of demographic factors and potential risk factors for SDB is described in Table 2. These data suggest that minority, BMI, waist circumference, tonsil size, nasal drainage, turbinate hypertrophy, and long soft palate were significantly associated with AHI. Factors that were clearly not associated with SDB included middle ear effusion, abnormal uvula, macroglossia, retrognathia, chronic cough, and wheeze (all P > 0.30).

In order to establish the relative independent contribution of these risk factors we further analyzed these data from a multivariate perspective using a stepwise logistic regression (retention criteria P < 0.05). We included in the initial models all variables with a univariate association of P < 0.30 (as indicated in Table 1). We assessed the 3 levels of SDB each compared

Table 2—Distribution of Demographic Factors and Potential Risk Factors for Sleep Disordered Breathing in the Population*

	(Non-SDB)	(Primary Snore)	1 ≤ AHI < 5 (Mild SDB)	AHI ≥ 5 (Moderate SDB)	P
Female	51.0%	52.0%	53.2%	88.5%	0.191
Age (months)	111.2 ± 1.0	108.1 ± 2.1	111.9 ± 1.6	121.1 ± 6.0	0.221
Minority	18.4%	28.8%	34.4%	0.0%	< 0.0001
BMI Percentile	58.5 ± 1.4	63.9 ± 2.9	65.2 ± 2.2	67.5 ± 14.0	0.041
Waist (cm)	63.5 ± 0.4	66.3 ± 1.2	66.4 ± 0.9	73.4 ± 5.0	< 0.0001
Neck (cm)	28.6 ± 0.3	28.7 ± 0.4	29.5 ± 0.4	29.5 ± 0.5	0.325
Tonsil size:					0.032
Abnormal	34.8%	48.5%	46.9%	28.8%	
Normal	58.1%	41.5%	45.3%	63.4%	
Removed	7.1%	10.0%	7.8%	7.8%	
Mouth breath	4.8%	8.3%	9.5%	7.8%	0.185
Stertor	1.3%	4.8%	0.3%	0.0%	0.034
Nasal drainage	20.7%	33.7%	32.7%	7.8%	0.003
Turbinate hypertrophy	13.9%	22.3%	28.4%	0.0%	< 0.0001
Middle ear effusion	3.3%	4.6%	1.3%	0.0%	0.409
Cervical adenopathy	17.9%	28.1%	20.6%	19.3%	0.175
Long palate	5.3%	7.7%	13.0%	28.8%	0.002
Abnormal palate	1.6%	2.7%	4.9%	0.0%	0.170
Abnormal uvula	1.7%	5.0%	7.2%	0.0%	0.655
Macroglossia	1.5%	1.8%	3.0%	7.8%	0.439
Retrognathia	0.2%	0.0%	0.3%	0.0%	0.948
Chronic sinusitis	9.7%	10.6%	17.2%	7.8%	0.075
Chronic cough	6.1%	2.3%	6.3%	11.5%	0.367
Wheeze	10.4%	12.8%	11.5%	13.6%	0.897
Enuresis	10.8%	16.7%	12.5%	0.0%	0.258

* Analysis for binary variables = Chi Square and for continuous = ANOVA

to no-SDB (Table 3). These systematic models enabled us to identify changing patterns of association at various thresholds of severity of SDB. In general, waist circumference was consistently a significant predictor of SDB at all levels of severity. At the lowest threshold (i.e., primary snoring), an association with waist, age (negative), cervical adenopathy, and minority was observed. With mild SDB, waist, nasal abnormalities (e.g., chronic sinusitis/rhinitis, turbinate hypertrophy, nasal drain), and minority were significant risk factors. Finally, with moderate SDB, waist and long soft palate were identified as risk factors. At no level was tonsil size, as assessed by visual inspection, retained as a significant risk factor for SDB.

DISCUSSION

We observed a prevalence of 1.2% for moderate SDB, 25.0% for mild SDB, and 15.5% for primary snoring. These prevalence estimates were based on a representative sample of children randomly selected from the local population of all children enrolled in elementary schools (K-5) within the 3 school districts closest to the medical center. Previous estimates using this same threshold (AHI ≥ 5) have varied greatly ranging from 0.9% to 13.0%.[5,8,9] A major reason for this wide range of estimates may be due to small sample sizes of previous studies, varied criteria for SDB, and differences in the age range. We found only one study that used AHI ≥ 5 and a roughly similar age range (8-11 years), and it reported a prevalence of 0.9%.[9] This study was based on a cross-sectional sample which oversampled preterm and African American children. The final analysis only evaluated the independent contribution of race, preterm status, and BMI.

We have previously reported that SDB in children is an independent risk factor for elevated systolic blood pressure in this sample.[16] This association was observed for a threshold of AHI ≥ 5 (P < 0.001) and to a lesser extent with AHI = 3 (P = 0.10). However, elevated blood pressure was not observed associated with AI ≥ 1, REM AHI, or primary snoring. Thus, we suggested that the threshold of AHI ≥ 5 warranted treatment for SDB in children.

A unique contribution of our study is that we assessed a wide range of potential risk factors. The assessment of these potential risk factors at various thresholds of AHI within our sample suggested possible mechanisms that should be considered. First, at milder AHI thresholds, nasal anatomic factors were more predominant (e.g., chronic sinusitis/rhinitis, turbinate hypertrophy, nasal drain). As such, these factors were significant predictors of milder SDB in this population-based sample. Several studies have also reported that obesity is a risk factor for SDB in children.[9,17,18] Waist circumference as a possible surrogate for metabolic factors has not been assessed in children. In our study, waist circumference, but not neck circumference, was a significant and strong predictor of SDB at all levels of severity. This finding suggests that in children, metabolic factors may play a contributing role in the mechanism of SDB, as seen in adults. It should be emphasized that when we replaced waist circumference with BMI in the list of variables to be included in the initial model, BMI was retained as a significant risk factor in all AHI cutoff thresholds to define SDB (data not shown).

Table 3—Final Model of Logistic Regression

	β	OR (95% CI)	P
Primary Snore vs No-SDB			
Waist (cm)	0.048	1.05 (1.02, 1.08)	< 0.001
Age (mo)	−0.017	0.98 (.97, .99)	0.010
Cervical adenopathy	0.687	1.2 (1.1, 3.4)	0.014
Minority	0.574	1.8 (1.0, 3.1)	0.038
1 ≤ AHI < 5 vs No-SDB			
Minority	0.805	2.2 (1.4, 3.4)	< 0.001
Turbinate hypertrophy	0.716	2.0 (1.3, 3.3)	0.003
Waist (cm)	0.031	1.03 (1.01, 1.05)	0.003
Long palate	0.975	2.7 (1.3, 5.3)	0.006
Chronic sinusitis	0.715	2.0 (1.2, 3.6)	0.013
Nasal drain	0.546	1.7 (1.1, 2.7)	0.017
AHI ≥ 5 vs No-SDB			
Waist (cm)	0.030	1.09 (1.03, 1.16)	0.005
Long palate	0.875	6.9 (1.2, 38.3)	0.025

Note: Waist circumference replaced BMI in all models

This finding of a similar property of BMI and waist circumference in their association with SDB further supports the metabolic mechanism for SDB. Moreover, it can be argued that the previous finding of BMI being independently associated with the failure of surgical treatment of SDB in children[19] further supports the hypothesis of a metabolic mechanism for SDB in children.

It is commonly assumed that adenotonsillectomy is the first line treatment for SDB in children. In reviews of available studies there is insufficient data to support the routine use of adenotonsillectomy in children with SDB.[20-22] In a sample of children referred to the Rainbow Babies and Children's Hospital, adenoid size based on lateral roentgenography was not found to be associated with SDB.[23] Research by Gozal and coworkers has also raised concerns regarding the efficacy of adenotonsillectomy in the treatment of SDB in children; they have suggested that at least 20% of children treated surgically will fail this treatment based on a threshold of AHI > 5[24]; if the threshold for failure is lowered to AHI > 1, the failure rate may be as high as 75%.[17,19] In addition, in focused samples using the threshold of RDI > 5, the failure rate in a sample of younger children (1-3 years) was 65%,[25] and in a sample of severe SDB (RDI > 30), the failure rate was 64%.[26] The basis of these controversies is whether tonsil size is an independent significant risk factor for SDB. In our data, after systematic modeling, tonsil size was not a significant risk factor for SDB, regardless of the threshold used to define SDB. This finding, however should not be interpreted as indicating that tonsils or adenoids are not at times the primary mechanism for SDB in children. Instead, it should be considered as an important call for consideration of alternative treatments. This becomes especially important when one considers that there is morbidity and rarely mortality associated with these surgical treatments. There is even some suggestion that morbidity rates are increased in those children with SDB.[24]

Although previous studies have reported positive findings between children with moderate to severe SDB and neuropsychological functioning,[27-31] we did not confirm this finding in this sample.[32] The strongest support for the position that SDB causes neurocognitive and behavioral deficits is found in a study[33] demonstrating improvement in cognitive test scores following adenotonsillectomy in children with mild to moderate SDB, and in 3 additional studies[2,31,34] demonstrating improvement in behavior and attention after adenotonsillectomy. It is difficult, however, to conduct blinded, placebo-controlled studies in these pre-post studies; therefore it is possible that the reported improvement in attention and cognition is the result of placebo effect and/or regression to the mean, rather than treatment effects. The most parsimonious explanation for the differences between the neurocognitive findings in our study and the findings of others is the lack of children with more severe SDB in our sample that limited our power. It is certainly reasonable to assume that the more severe the SDB, the more impact on neuropsychological functioning.

Previous literature supporting mechanisms other than the metabolic pathway comes from several sources. Partial or complete nasal obstruction has been shown to increase SDB, especially snoring, in normal adults[35] and in children.[36] Sinusitis/rhinitis is associated with at least partial obstruction of the nasal passage. It has been shown to be a risk factor for SDB in adults[37-40] and in children.[10,41] Snoring occurs when the soft palate or pharyngeal tissues vibrate.[42] Thus, soft palate length has been shown to be a risk factor for SDB in adults[43] as well as in children.[44]

In adults with SDB, it has been shown that systemic and local inflammatory mechanisms are an important contributing factor.[45] Treatment of nasal congestion in children with SDB with anti-inflammatory agents has been demonstrated to be effective with several compounds. Intranasal corticosteroids, fluticasone propionate and budesonide have both been shown to effectively reduce SDB in children.[46,47] In addition, in a study that did not assess SDB in children, the anti-inflammatory aqueous nasal beclomethasone has been shown to reduce adenoid hypertrophy as well as nasal airway obstruction in children.[48] More recently montelukast (a leukotriene receptor antagonist) has been shown to reduce SDB in children as well as to decrease adenoid size.[49] In a subsequent study, it was reported that combining budesonide and montelukast improved the residual SDB in children post adenotonsillectomy.[50] These data combined with the findings of this study suggest that a thorough evaluation and treatment of nasal abnormalities may be beneficial for this category of SDB in children.

Obesity in children as in adults is associated with an increased risk for SDB. There are reports of increased adipose tissue in the uvula as well as in the soft palate.[51] In our sample of children, we observed an independent association of waist circumference but not neck circumference with SDB, suggesting a systemic rather than a local influence of obesity. Adipose tissue is capable of producing the proinflammatory molecule TNF,[52] and systemic administration of TNF in animal models has been shown to be associated with a reduced muscle and diaphragm contractility.[53-56] In humans, systemic administration of a TNF antagonist (etanercept) was associated with a reduction of apnea and sleepiness.[57] Further support for the systemic inflammation mechanism associated with SDB in children comes from the finding that C-reactive protein[58,59] and plasma adherence molecules[60] have been reported to be elevated in these children.

In adults, it has been demonstrated that metabolic factors are associated with SDB, which may be the mechanism for the cardiovascular risk observed in these patients. In a similar manner in children with SDB it has been reported that fasting insulin levels independent of BMI are associated with the severity of SDB.[61,62]

Minority, specifically African-American, has been reported to be associated with SDB in adults[18] as well as in children.[5,9] In our sample we observed that minority status was an independent risk factor for primary snoring and mild SDB. It is unclear whether this added risk is due to a specific physiologic characteristic or to sociocultural differences.

This study does have limitations which need to be considered. First, because this study is a representative sample of the general public, the severity of SDB observed was not as high as might be seen in a clinical sample (max AHI = 24.6). We did not complete an estimate of the night-to-night variability, which could have influenced our prevalence estimate. Further, the tonsil size evaluation was completed only by visual inspection rather than by a more accurate but more expensive imaging method in this large sample. However, visual inspection is the usual method used in a clinical setting. Due to the relatively low prevalence in the most severe AHI group (AHI $\geq$ 5), we may have a reduced power. However, risk factors of BMI and waist circumference were consistently present across several AHI thresholds, suggesting a quite robust association. Because this was an epidemiologic study, rare factors such as retrognathia did not enter into our models. This should not be interpreted as indicating that these rare factors are not at times the primary mechanism for SDB in a specific child. The contribution of other risk factors (e.g., minority) in more serious SDB needs to be evaluated in larger studies.

In conclusion, we observed a prevalence of 1.2% for moderate SDB based on a threshold of AHI $\geq$ 5 in elementary school children (K-5). Waist circumference and BMI were consistent independent risk factors across thresholds of severity of SDB. This suggests that in children, as in adults, metabolic and inflammatory factors may be an important mechanism in the development of SDB. Tonsil size was not an independent risk factor at any AHI threshold used to define SDB, suggesting that adenotonsillectomy may not always be the best first-line treatment. Finally, nasal abnormalities (e.g., chronic sinusitis/rhinitis) were significant risk factors in children with milder SDB, suggesting that the evaluation and treatment of these abnormalities might be beneficial for this category of patients.

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