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Development of a Simplified Pediatric Obstructive Sleep Apnea (OSA) Screening Tool

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A thesis submitted in partial fulfillment of the requirements for the degree of Master of Science in Dentistry at Virginia Commonwealth University.

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Abstract

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Background: Obstructive sleep apnea has become recognized as one of the most common, under-diagnosed chronic diseases. Recently studies have shown increased numbers among the pediatric and adolescent population. OSA in children is associated with behavioral problems, poor school achievements, and in severe cases, pulmonary hypertension. OSA is often the Achilles heel of pediatric sedation and analgesic programs; during sedation, children with OSA have an increased vulnerability of their airway undergoing pharyngeal collapse and of having upper airway obstruction. Consequently, pediatric dentists who practice sedation dentistry should exercise extra precautions when treating patients with risk of sleep apnea. Currently there is no screening tool used in pediatric dentistry for diagnosing OSA during the pre-operative appointment or consultation for patients undergoing minimal and moderate oral conscious sedation. The purpose of this study was to develop and test a concise and easy-to-use questionnaire as a screening tool to aid in the diagnosis of OSA in pediatric patients. **Materials and Methods**: A retrospective chart review of 180 patients under the age of 18, who completed a polysomnogram at the VCU Center for Sleep Medicine between February 2011 and February 2013. A validated adult questionnaire, STOPBANG, was modified using more typical pediatric

risk factors for OSA: presence of snoring (S), tonsillar hypertrophy (T1), tiredness; pESS>10 (T2), observed obstruction (O), neuroPsych-behavioral symptoms such as ADHD or daytime irritability (P), BMI percentile for age (B), age at diagnostic screening (A), presence of neuromuscular disorder (N), and presence of genetic/congenital disorder (G). A positive scoring from these variables was measured against the standard OSA measure, Apnea-Hypopnea Index. A multiple logistic regression analysis tested for relationships.

Results: There was a statistically significant relationship *P*= .0007 for the S(T1)OPBANG scale, with a minimum of 4 variables needed to have a sensitivity of 57% and a specificity of 78%. There was also a statistically significant relationship *P*= .0040 for the S(T2)OPBANG, the cutoff>5 yielding sensitivity=36%, and specificity=90%. Only obstruction, BMI, and age showed a strong significant relationship to OSA. The presence of an obstruction was positively related to apnea (P = 0.0010). Most of the other components had an odds-ratio larger than one (indicating a nominally positive relationship). **Conclusions:** While both STOPBANG screening tools showed a statistically significant relationship, only obstruction, BMI, and age showed a predictive relationship to OSA. Consequently, consideration of other risk factors may be beneficial for future studies.

Introduction

Sleep-disordered breathing encompasses a wide range of upper airway disorders from primary snoring (PS) to obstructive sleep apnea (OSA). OSA results from impedance to air flow in the upper airway during sleep; these periodic obstructions of the upper airway interfere with normal respiratory gas exchange and subsequently interrupt sleep.[1,2] OSA is measured by the Apnea-Hypopnea index, which represents the total apneas and hypopneas per hour of sleep.[3] Apnea denotes the cessation of airflow and is classified based on the pattern of respiratory effort. Hypopnea is a reduction in airflow.[4]

OSA has become recognized as one of the most common, under-diagnosed chronic diseases.[5-7] People of all ages are affected with OSA, recently studies have shown increased numbers among the pediatric and adolescent population.[8] The prevalence of obstructive sleep apnea (OSA) in children is estimated to be 1-3%,[9] while primary snoring occurs in 3-12% of the pediatric population.[10] A child with an AHI of greater than 5 events an hour emerges as having clinically significant OSA.[11] Even brief obstructive apneas may be associated with significant hypoxemia due to children having a faster respiratory rate and lower functional residual capacity than adults.[12] Mild cases of pediatric OSA are recognized and at times treated; however, measurable effects on development, cardiopulmonary or metabolic systems have been difficult to validate. OSA is associated with behavioral problems, poor school achievements, and in severe cases, pulmonary hypertension.[2] Many studies have been conducted to identify adverse effects of

sleep disorders, yet few studies have examined how health care providers may identify and treat sleep disorders.[13]

Dentists who see their patients more frequently than their primary care doctors have a greater opportunity to observe signs and symptoms of OSA.[8] However many potential sleep disorders in children are unrecognized and under-reported, and overall the condition is under-diagnosed.[14] Dentists who practice sedation dentistry should exercise extra precautions when treating patients with risk of sleep apnea. Minimal and moderate oral conscious sedation and general anesthesia are commonly used in pediatric dentistry; thus pediatric dentists have an acute responsibility to be able to identify patients who may have OSA.[7] During sedation, children with OSA have an increased vulnerability of their airway undergoing pharyngeal collapse and of having upper airway obstruction.[9] The risk of postoperative respiratory complications amongst the pediatric population ranges from 0 to 1.3%; however for children with OSA the rates have been reported to be 16-27%.[15,16] The incidence of OSA in children is most elevated between ages 2 to 6 years old. In this age range pharmacologic measures are most often used to complete diagnostic and therapeutic procedures.[9]

While polysomnography (PSG) remains the gold standard to diagnose OSA, there are many challenges due to the limited number of sleep laboratories and the high cost of using PSG on each child who snores and may be at risk.[10] Polysomnography provides information pertaining to sleep patterns but fails to identify behavioral sleep disturbances.[17] Available non-PSG screening tests have poor sensitivity for milder OSA, and overall poor specificity.[10] In extreme cases of OSA the clinical severity is easily determined with or without PSG, however it is the mild to moderate cases that are most difficult to diagnose. Moreover, there remains a challenge to differentiate PS from OSA in a "cost-effective, reliable, and accurate manner before

recommending invasive or intrusive therapies, such as surgery or continuous positive airway pressure".[10]

Sleep questionnaires that are completed by the parent and child are a crucial component of behavioral and physiological sleep assessment. Pediatric questionnaires are mostly retrospective in that the parents report on past sleep patterns and behaviors that are typical of their child. Questionnaires have been used alone or with other sleep assessment tools. Within the last 20 years, the number of pediatric sleep questionnaires has greatly increased.[17] Moreover, a review of sleep questionnaires from 2011 stated that there were "too many tools being used that have not undergone careful and methodical psychometric evaluation, and as such may be fraught with biased or invalid findings".[18]

In 2008, Chung et al developed and validated a STOP questionnaire as a screening tool for OSA in patients 18 years and older. This questionnaire asks four yes or no questions: do you snore loudly?, do you feel tired during the daytime?, has anyone observed you stop breathing during your sleep?, and do you have high blood pressure? These questions along with body mass index, age, neck size, and gender (BANG) were found to have a sensitivity of 83.6, 92.9, and 100% (for mild, moderate, and severe OSA, respectively).[19] In 2006, the American Society of Anesthesiologists (ASA) Task Force on Perioperative Management of Patients with Obstructive Sleep Apnea recommended a checklist as a routine screening tool to utilize in surgical patients who may have OSA. The ASA checklist has 12 items for adults and 14 items for children, but was only validated by Chung for its use on adults.[20]

In 2011 Spruyt and Gozal published a review on Pediatric sleep questionnaires that examined 57 sleep measures that were used to screen children for sleep disorders including OSA.[18] Only 2 questionnaires fulfilled all desirable criteria: The Sleep Disturbance Scale for

Children (SDSC) at a cut-off score of 39 provided a sensitivity of 0.89 and a specificity of 0.74 [21]; and The Sleep Disorders Inventory for Students-Children (SDIS-C) showed a sensitivity of 0.91 and a specificity of 0.62 for the category of Obstructive Sleep Apnea Syndrome.[22] This review documented that few standardized screening tools exist thus far to determine risk for OSA in children.[18] Pediatric Dentists and anesthesiologists alike would benefit from a standard screening tool, similar to the STOPBANG, to determine if OSA may exist in potential sedation and anesthesia pediatric patients.

Complications arise with diagnosing pediatric OSA because its presentation can differ from that of an adult. The exact pathophysiology of OSA in children has not been determined but it appears to result from both upper airway narrowing and upper airway hypotonia. Thus children with craniofacial abnormalities and neuromuscular disorders are at increased risk. The patency of the upper airway depends on pharyngeal dilating muscles. Children with neuromuscular diseases frequently present upper muscle airway weakness that can cause the airway to collapse. Upper airway narrowing is often a result of adenotonsillar hypertrophy. Despite past studies in which children with OSA typically have larger tonsils and adenoids than children who do not, the size of tonsils and adenoids does not predict the disease in individual persons. While excessive daytime sleepiness is a major complaint in adults with OSA, it is less common in children. This is most likely due to children maintaining their sleep architecture as they have a higher waking threshold than adults. Snoring has also been noted in both children and adults who have OSA. In prepubertal children, OSA occurs equally in both genders. However in adults, OSA occurs twice as often in males as in females. A large predisposing factor to OSA in adults is obesity. Obesity also appears to be a risk factor in children.[12] Capdevila proposed the existence of two distinct types of OSA in children. Type I is associated with adenotonsillar hypertrophy in the absence of

obesity. Type II presents with "milder upper airway lymphadenoid hyperplasia" and obesity.[23] It is fundamental to be able to identify both phenotypes that my present with signs of OSA.

OSA is often the Achilles heel of pediatric sedation and analgesic programs,[9] thus it is imperative that pediatricians and pediatric dentists be able to identify a child who may be at risk for OSA so that appropriate referrals for a definitive diagnosis can be made. Currently there is no screening tool used in pediatric dentistry for diagnosing OSA during the pre-operative appointment or consultation for patients undergoing minimal and moderate oral conscious sedation.

The purpose of the study was to develop a concise and easy-to-use questionnaire as a screening tool to aid in the diagnosis of OSA in pediatric patients.

Materials and Methods

This project was approved under exempt status from the Virginia Commonwealth University Institutional Review Board (VCU IRB #: HM15027).

This was a retrospective chart review of data previously collected as part of a routine screening exam for patients referred for a sleep study. The original data was collected at the VCU Center for Sleep Medicine for patients who presented from February 1st 2011 to February 1st, 2013, with no previous sleep disorder diagnosis. Study data were collected and managed using REDCap electronic data capture tools hosted at Virginia Commonwealth University. REDCap (Research Electronic Data) is a secure, web-based application designed to support data capture for research studies.[24] Prior to data collection, an excel file was created at the VCU Center for Sleep Medicine and included all patients under the age of 18 who had completed a polysomnography and thus had a definitive sleep disorder diagnosis. The excel file listed the following information for each patient: patient name, medical record number, age at the time of the study, and date of the polysomnogram.

In order to be included in the study patients must have met the following criteria:

- Age < 18
- Completed polysomnography with sleep disorder diagnosis
- Sleep Questionnaire completed and scanned into chart

Approximately 180 patients completed a polysomnography within the above time frame and were listed in the excel file. Using this listing of patients, data was gathered from 2 sources: Cerner, the Medical College of Virginia (MCV) electronic medical record, and the Sleep Center database. Paper charts for all patients prior to 2011 were currently inaccessible, so only 180 charts were utilized in this study. A data collection sheet was fabricated to document each variable needed in this study (Appendix 1). No patient identifiers were collected or used during this study. The following variables were collected for this study: age of patient at time of PSG, gender, race, height, weight, body mass index, presence of snoring, presence of tonsillar hypertrophy, obstruction while sleeping, presence of neurobehavioral symptoms, daytime tiredness or irritability, presence of neuromuscular disorders, presence of genetic disorders, the patient's Epworth scale score, and Apnea-Hypopnea index (AHI). The methods for determining each are given below.

Date of PSG

The date of the polysomnogram was recorded from the excel file after being verified on Cerner.

Age

Age at time of sleep disorder diagnosis was calculated using the electronic Cornell University age calculator (http://www-users.med.cornell.edu/~spon/picu/calc/agecalc.htm) in which the date of birth and date of polysomnography are entered to compute a patient's age in years and months. Partial months were rounded down to whole numbers. As a risk factor, ages younger than 3 or older than 12 years were coded as "yes".

Gender and race

Race (when specified) was recorded as Caucasian, African American, Asian, Hispanic, or written in under "Other". Gender was also recorded. Information on both of these variables was accessed under Patient Demographics in Cerner.

Height, weight, body mass index

If not provided in the chart, body mass index was calculated using the Centers for Disease Control and Prevention (CDC) BMI calculator for children and teens (http://apps.nccd.cdc.gov/dnpabmi/). The calculator computes BMI and the "BMI-for-age" percentile using the patient's gender, age at time of measurement, height and weight. BMI-for-age percentiles greater than 95% are coded as risk factor "Yes".

Presence of tonsillar hypertrophy

This characteristic was coded as "Yes" when tonsillar hypertrophy was noted in the neurologist's preoperative note. Many patients who were being screened for OSA had recently seen an otolaryngologist for tonsil evaluation. Preoperative notes were reviewed and tonsils recorded as a 3+ or 4+ were marked "yes" for tonsillar hypertrophy. If a patient had a tonsillectomy, or if it was noted that the patient had tonsils 2+ or smaller, then tonsillar hypertrophy was marked as a "No". "Unknown" was recorded when tonsils were not mentioned on any neurologist or otolaryngologist record during the time frame of the OSA screening.

Presence of neurobehavioral symptoms

Daytime neurobehavioral symptoms encompass attention deficit disorder with hyperactivity (ADHD), attention deficit disorder without mention of hyperactivity (ADD), and oppositional defiant disorder (ODD). These diagnoses were noted in the diagnosis section or at the top of the polysomnogram under patient's history. For example, the top of a typical polysomnogram report

may read: "History: This 8 year-old female with a history of ADHD, Kabuki syndrome is being evaluated for obstructive sleep apnea contributing to daytime fatigue and hypersomnolence and loud snoring." To verify these diagnoses the patient's "Diagnoses and Problems" section was reviewed in Cerner. If no disorder was mentioned in these two sections or in the other sleep medicine documents, than the patient was recorded as not having any neurobehavioral symptoms.

Presence of neuromuscular disorders

This characteristic was coded as "Yes" when a neuromuscular disorder was recorded under the patient's "Diagnosis" or if it was documented at the top of the polysomnogram report under patient's history. "No" neuromuscular disorder was recorded when it was not mentioned in any of the above.

Presence of genetic disorders

This characteristic was coded as "Yes" when a genetic or congenital disorder was recorded under the patient's "Diagnosis" or if it was found documented at the top of the polysomnogram report under patient's history. "No" genetic or congenital disorder was recorded when it was not mentioned in any of the above.

Sleep Study Questionnaire

Prior to undergoing a sleep study, each patient along with their parent would complete a sleep questionnaire called "The VCU Center for Sleep Medicine: New Patient Questionnaire" (Appendix 2). This five page document consisting of the Sleep-50 Questionnaire, gave each patient the opportunity to complete the Epworth Scale, give a detailed medical history, and respond to specific statements about a patient's sleep hygiene[25]. Patients were asked to rate to what extent a statement was applicable to them by circling 1, 2, 3, or 4. The number one

corresponded to “Not at all”, 2 indicated “somewhat”, 3 “rather much” and 4 “very much”. The prefabricated data sheet asked data collectors to determine whether the patient believed they snored, had sleep obstruction, or was excessively tired or irritable during the day. Data collectors recorded “Yes”, “No” or “unknown” to these variables. If the patient had circled 3 or 4 corresponding to a statement than that was considered a positive (yes) for whether the patient had the symptom specific to that statement. A 1 or 2 indicated a negative response for the symptom. The following variables below utilized the Sleep Study Questionnaire.

Presence of snoring

This characteristic was recorded solely based on the patient reporting symptoms and not on whether the patient snored during the polysomnography. Snoring was coded as “Yes” when snoring was noted on patient’s referral form, noted in the neurologist’s preoperative notes, or when the parent and patient recorded a “3” or “4” when responding to the question “I am told that I snore”. In the sleep questionnaire “1” corresponds to Not at all, a “2” simulates “somewhat”, a “3” denotes rather much while “4” means very much that the patient has certain symptoms. “No” was recorded when a patient selected 1 or 2 when responding to the question “I am told that I snore”, or when the neurologist had noted that the patient did not snore. “Unknown” was recorded when snoring was not mentioned in the doctor’s preoperative note, the referral form, or when the sleep questionnaire was not completed.

Obstruction while sleeping

This characteristic was recorded based on the patient’s symptoms. Sleep obstruction was coded as “Yes” when sleep obstruction was noted on the patient’s referral form, noted in the neurologist’s preoperative notes, or when the parent and patient recorded a “3” or “4” when responding to the questions: “I am told that I hold my breath when sleeping” and “I am told that I

wake up gasping for air". "No" was recorded when a patient selected 1 or 2 when responding to the same questions, or when the neurologist had noted no past history of obstruction. Unknown was recorded when sleep obstruction was not mentioned in the doctor's preoperative note, the referral form, or when the sleep questionnaire was not completed.

Daytime tiredness or irritability

This characteristic was coded as "Yes" when the patient recorded a "3" or "4" when responding to the majority of the following questions: "I feel tired at getting up", "I feel sleepy during the day and struggle to remain alert", "I would like to have more energy during the day", "I am told that I am easily irritated", or "I have difficulty in concentrating at work or school". No was recorded when the patient had marked a "1" or "2" to a majority of these questions. Unknown was recorded if the sleep questionnaire had not been completed.

Apnea-Hypopnea index (AHI)

As previously mentioned, OSA is primarily diagnosed by a patient's apnea-hypopnea index (AHI). The AHI represents the average number of apneas and hypopneas per hour of sleep[10] and was obtained from each polysomnogram. In children, more than one obstructive apnea event of any length per hour of sleep is considered abnormal.[26,27] Based on these recommendations, in this study mild AHI was defined as >1.5, moderate >5 and severe >15. For the purposes of analysis, AHI was categorized two ways. One categorization characterized apnea as: none (AHI $\leq$ 1.5), mild (AHI > 1.5), moderate (AHI > 5), or severe (AHI > 15). The primary categorization was a binary outcome: apnea negative (AHI $\leq$ 5) or apnea positive (AHI > 5).

S(T1)OPBANG

There were two scales evaluated, differing only in the "T" variable. S(T1)OPBANG was the sum of the presence of snoring (S), tonsillar hypertrophy (T1), observed obstruction (O), neuro-

psych-behavioral symptoms such as ADHD or daytime irritability (P), BMI percentile for age (B), age at diagnostic screening (A), presence of neuromuscular disorder (N), and presence of genetic/congenital disorder (G). Yes values were scored as 1 and all other values (No and unknown) were scored as zero in the calculation of the sum.

S(T2)OPBANG

The S(T2)OPBANG score was calculated as above but tiredness evaluated through the Epworth scale; pESS>10. (T2) replaced tonsillar hypertrophy.

Purpose

The primary aims of the study were to test two scales for the identification of sleep apnea in children. The secondary aims were to test each of the components of the sleep apnea scale.

Data analysis

All the information from the data sheets was entered into a REDCap database for analysis. A second party for input errors checked all information entered. All analyses were performed using SAS software by the project biostatistician, Dr. Best. All of the patients were described according to the STARD standard for reporting diagnostic accuracy.[28] This includes complete reporting of patients excluded from study. The statistical methods included screening of each diagnostics characteristic (using chi-square analysis) and a multiple logistic regression analysis of the OSA diagnosis to determine which diagnostics characteristics are associated with the diagnosis. Final reporting included odds-ratios and 95% confidence intervals on all estimates. Using the projected 250 charts that were initially thought to be available, and estimating the prevalence OSA of at least 25% and odds-ratios of at least 2, the study had approximately 80% power (at alpha=0.5).

Results

There are three major sections included in the Results: The first section is a description of the results of the process from the identification of potential subjects to the building of the final analysis dataset, and a description of the subjects analyzed. The second section includes the testing of the primary aims. The third section denotes the exploration of each of the components and the proposal of a revised scale.

Data gathering and Description of subjects

The sleep study questionnaire was completed on paper and scanned into the patient's chart in Cerner. Occasionally sleep study questionnaires were not scanned into Cerner or parents and patients did not complete the sleep study questionnaire in its entirety. If the variables were unable to be collected that particular patient was excluded from the study. Often this information was gleaned from a direct referral form previously completed by a primary care physician or a specialist such as an Otolaryngologist. The referring doctor would complete the Patient History section, in which they could check such parameters as excessive daytime sleepiness, loud snoring, neuromuscular disease, obesity, observed apneas, wakes with choking/gasping. Referring doctors could also write in other diagnoses that were not listed. This direct referral form was used in lieu of a sleep questionnaire if all parameters were answered and if it was evident the patient was nonverbal and could not complete the sleep questionnaire. Otherwise the patient was excluded due to lack of information. A total of 153 subjects with usable data were

analyzed. 54% were male and the predominant race groups were whites (45%) and blacks (43%, See Table 1). Neither gender (P = 0.4455) nor race (P = 0.1368) appeared related to the AHI scores. Subjects ranged in age from 38 months to 17.5 years (mean = 10.6, SD = 4.1). Subjects ranged in height from less than a meter (3 feet) to 1.88M tall (6 foot 2 inches). One height was not available for a subject. Subjects ranged in weight from 12.7kg (28 pounds) to 189kg (416 pounds). Height was roughly normally distributed, but weight was skewed, as is indicated by the fact that the median weight (42kg) was considerably lower than the mean weight (50kg). BMI was calculated from height and weight and averaged 23.32kg/m^2 (SD = 8.9). Since the amount of body fat changes with age and differs between boys and girls, the BMI-for-age percentiles are used for comparisons. BMI-for-age is strongly skewed with a large number of percentiles at 95% or above. There were 60 subjects (39%) who were described as obese since they were above the 95^{th} percentile for age and gender. Another 27 subjects (18%) were overweight (between the 85^{th} and 95^{th} percentile). Only 54 subjects (36%) had what is considered a healthy weight (between the 5^{th} and 85^{th} percentile); 11 subjects were underweight (7%).

Scales

There were two scales tested in this study and the components for each are summarized in Table 2. Over 59% of all subjects had a positive indication in the medical record for snoring (n = 91) but 11 subjects lacked any reference in the record regarding snoring (7%). For the purposes of the scale score, a “yes” was counted as positive and both “no” and “unknown” were not counted as positive (and so they are effectively counted as negative). Thus the prevalence of each of these components ranged from a high of 59% (snoring) to a low of 14% for both neuromuscular disorders and for genetic/congenital disorders.

Note that for the purposes of the scales, daytime neurobehavioral symptoms and excessive tiredness during daytime were combined such that if either was a "yes," the combination was scored as a positive. There were 13 "yes" for both, 13 "yes" on daytime neurobehavioral symptoms only, and 66 "yes" on excessive tiredness. And so there were 92 yeses on this combined indication (60%), 60 no (39%), and only 1 unknown (1%).

The primary outcome variable was observed apneas and hypopneas, as indicated by AHI. The raw AHI values ranged from 0 to 85.7, with a median value of 0.8. The strongly skewed values yielded a mean of 4.08 (SD = 9.53). Table 3 indicates that 82% (96=none, and 29=mild) were considered negative for apnea and therefore that 18% (16 moderate and 12 severe) were considered positive.

For the S(T1)OPBANG scale (snoring, tonsillar hypertrophy, sleep obstruction, daytime neurobehavioral symptoms/tiredness, BMI, age, neuromuscular and genetic), the scores ranged from 0 to 6 (Mean = 2.76, SD = 1.34) and for the S(T2)OPBANG scale (snoring, Epworth, sleep obstruction, daytime neurobehavioral symptoms/tiredness, BMI, age, neuromuscular, and genetic), the scores ranged from 0 to 6 (Mean = 2.84, SD = 1.42, see Table 4). Since they share components, it is not surprising that the two scores are strongly correlated ($r = 0.90$, $P < .0001$).

Logistic regression was used to test for a relationship between the S(T1)OPBANG scale and apnea. There was a statistically significant relationship (likelihood ratio chi-square = 11.5, P = 0.0007). Table 5 shows the relationship between each scale value and the sensitivity and specificity. For instance, if S(T1)OPBANG≥6 is used as a cutoff, then 2 subjects are predicted to be positive. Of the 28 actual positives one has a cutoff 6 or greater and so the sensitivity is 4% (1/28). Of the 125 actual negatives, all but one has a cutoff below 6 and so the specificity is 99% (124/125). As the cutoff decreases, sensitivity must go up and specificity must go down. If the

risk of false positives and false negatives were equal, then the cutoff yielding the largest sensitivity + specificity would be the optimal cutoff. In the case of S(T1)OPBANG, this cutoff is 4 (sensitivity=57%, specificity=78%).

For the S(T2)OPBANG scale there was also a statistically significant relationship (likelihood ratio chi-square = 8.27, P = 0.0040). Table 6 shows the relationship between the sensitivity and specificity and each scale value. In the case of S(T2)OPBANG, the cutoff yielding the largest sensitivity + specificity is 5 (sensitivity =36%, specificity = 90%).

One way to determine which of the two scales might be preferred is to use both as predictors in a multiple logistic regression. This indicates that when S(T1)OPBANG is used as a predictor, S(T2)OPBANG provides no additional predictive value (P = 0.70). Conversely, if S(T2)OPBANG is used as a predictor, then S(T1)OPBANG provides some additional predictive value (P = 0.0644).

Analysis of the components

Each of the individual components was first screened using a chi-square test (Table 7). The results indicate that the only statistically significant risk factor was sleep obstruction (P=0.001). However all components had a relative risk value greater than 1 except for neuropsych-behavioral symptoms/tiredness (RR=0.77). A multiple logistic regression analysis was used to test the significance of each of the components of the scales and shows the results for the components of S(T1)OPBANG (Table 8). Although the test that all eight components provided predictive value was significant (P = 0.0024), only one component was individually significant. The presence of an obstruction was positively related to apnea (P = 0.0010). Most of the other components had an odds-ratio larger than one (indicating a nominally positive relationship). However, two components—snoring and neurobehavioral symptoms/daytime tiredness had odds

ratios below one, which indicates that the presence of the component is negatively related to apnea. Similar results were found for the components of S(T2)OPBANG (Table 9). That is, although 23% of those who snore were OSA positive and 11% of those who did not snore (RR=2.04), after all of the other risk factors were taken into account, snoring was not a significant predictor of OSA (OR=0.74, P>0.6). Similarly 26% of children with tonsillar hypertrophy had OSA and16% of those who did not (RR=1.57). But tonsillar hypertrophy was not significant after others were accounted for (OR=1.96, P>0.2). There was no evidence that an Epworth score greater than 10 was related to OSA since those who were above the cutoff had 19% OSA, as compared to 18% of those below the cutoff. On the other hand of those with a sleep obstruction 34% were positive for OSA and only 9% of those without a sleep obstruction were positive to OSA (RR=3.98). This remained a significant predictor of OSA (OR=7.56, P=0.0010). Being positive for either neurobehavioral daytime symptoms or excessive daytime tiredness/irritability was actually nominally protective against OSA since there were fewer OSA case in those who were positive than in those who were negative (16% vs 215). Obese children were OSA positive 25% of the time and non-obese children 14% (RR=1.79) but the adjusted analysis did not indicate that it was a significant predictor (OR=1.90, P>0.2). Those in the age risk categories were OSA positive 23% of the time whereas those who were in the middle ages were positive 16% of the time (RR=1.42) but age was not statistically significant in the adjusted analysis (OR=2.42, P=0.1004). This result is suggestive, however. Those with neuromuscular diagnoses were as likely to be OSA positive than those without these diagnoses (19% vs 18) and the adjusted analysis also did not support a significant relationship (OR=3.06, P=0.1482). Again, this is a suggestive result. And those with genetic/congenital defects were more likely to be OSA

positive than those without these conditions (24% vs 17%, RR=1.37). Moreover, the adjusted analysis advocate some potential for a relationship (OR=3.71, P=0.0648).

Discussion

In this retrospective chart review, specific variables were compared with AHI scores in order to develop a screening tool with a high sensitivity and also a high specificity for pediatric obstructive sleep apnea. Less than half of children with OSA symptoms actually have the syndrome.[29] As a result screening for OSA is very complicated and causes many children to go undiagnosed. Presently, pediatric OSA is under diagnosed and as a result undertreated because of the high cost to test for OSA and the limited number of pediatric sleep laboratories. Consequently screening for OSA has become essential.[30] A recently published systematic review and meta-analysis by Canto et al (2014) explores the diagnostic value of alternative methods such as clinical history and physical examination to identify pediatric OSA, and also validates the role dentists play in screening patients.[31] In the following discussion, the findings of the current study will be compared to the results of Canto's systematic review where applicable. The results of the current study found a clinically significant correlation between each proposed scale and AHI, however only one individual component was strongly related to AHI. Suggesting that certain variables that present together in a single individual may predispose that person to OSA, more so than individual parameters. Below each variable evaluated in this study is dissected along with present findings and suggestions for a revised screening tool based on these results.

In a review on sleep disordered breathing in children, Padmanabhan et al. ascertained that snoring, apnea, and difficulty in breathing were the three main symptoms of OSA in children and

infants. Snoring occurs in almost all children with a sleep disorder; often it is the catalyst for parents to believe there is a problem and to pursue a medical consult.[32] Parents who hear their children snore perceive them struggling to breathe and often are anxious about their nighttime breathing habits.[10] Furthermore, snoring remains the most common complaint in sleep-disordered breathing for children under five years old.[26] Interestingly enough, only a fraction of children who snore have OSA,[32,33] and the presence of snoring alone cannot accurately predict OSA.[10] Habitual snoring is a hallmark of sleep-disordered breathing and denotes loud snoring at least three nights per week.[34] A great number of children who habitually snore have primary snoring, which is habitual snoring without changes in ventilation or oxygenation.[30] The correlation between snoring and AHI in our study overall had a very weak relationship both individually (P=0.0642) and once all values were adjusted for every component (P=0.6767). These results reflect our sample of patients as all subjects presented to the sleep center citing sleep difficulty. Our study suggests a correlation that is not necessarily predictive, reinforcing the idea that the presence of snoring does not automatically ascertain that the child has OSA.[10] Young et al determined 10-14% of children snore every night or every other night, and found a prevalence of OSA in 10 to 20% of habitual snorers.[35] Our results are similar- 21 out of 91 subjects who reported snoring exhibited moderate or severe OSA. Canto et al evaluated three snoring characteristics and reported the sensitivity and specificity of each: snoring disturbs others (sensitivity = 68% and specificity = 58%), snoring every night (sensitivity = 91% and specificity = 75%) and snoring extremely loudly (sensitivity = 52% and specificity = 78%). Snoring every night had the highest sensitivity with fair specificity.[31] Unfortunately snoring was not quantified in this retrospective chart review, and so parents may have reported their child snored even when it was infrequent. Moreover, our study was limited in that data relied on reporting of parents who

likely have varying subjective standards for what they consider "snoring" and also vary in their opportunity to observe the behavior. Snoring alone is not a sensitive indicator of OSA, but because it is a prevalent symptom of OSA it remains a useful variable in our screening tool.

As mentioned above, sleep obstruction is another common symptom of children with OSA and represented the O in our study. Obstructive apnea occurs when there is respiratory effort and lack of airflow.[36] In adults obstructive events need to be at least 10 seconds long in order to be scored. However, in children the obstructive events need to occur over 2 breaths or more.[4] A different standard is necessary as children have a faster respiratory rate and lower functional residual capacity. Moreover, shorter respiratory events in children have more physiological consequences.[10] Our results exemplified a strong ordinal relationship with AHI and obstruction (P=0.0010), with a sensitivity of 71% and a specificity of 69%. It is surmised that there is a strong correlation for two reasons, first the obstruction that parents report most likely represent the apnea and hypopnea events significant for OSA, and second, choking and gasping during sleep is a very distinct sound that may be definitively distinguished from that of snoring. Consequently while other symptoms reported by parents may not always be accurate, it is difficult to misinterpret pauses in breathing exemplified by "choking and gasping sounds". In Canto et al, evaluation of whether pauses in breathing were diagnostic were not as definitive. Observable apnea only showed a sensitivity = 36% with a strong specificity = 95%. Observed obstructive apneas had almost equal sensitivity and specificity, with 61 and 65%, respectively.[31] In this study obstruction represents the variable with the strongest correlation to AHI and thus remains in our revised screening tool.

For the T component of our modified STOPBANG we utilized two different variables- tonsillar hypertrophy and tiredness reflected through the value of the Epworth scale. Neither of

these variables showed a significant correlation with AHI in this study (P=0.2449 and P=0.4831, respectively). Presently the most common identified risk factor in childhood OSA is adenotonsillar hypertrophy.[2,37,38] Lingual and pharyngeal tonsils can be visualized intraorally whereas adenoids cannot. Tonsillar size can range from Type 0 where the tonsils are absent to Type IV where the right and left tonsillar tissue approaches the midline. Based on Scammon's growth curve, lymphatic tissue begins to shrink after the age of six but the presence of large tonsillar and adenoid tissue may negate this normal reduction and obstruction may persist.[8] The primary treatment for OSA in children is adenotonsillectomy.[2,37,38] Removal of the adenoids and tonsils relieves crowding in the airway and permits air to flow more freely through the nasal and oral passages.[2] In The Childhood Adenotonsillectomy Trial (CHAT) by Marcus et al., watchful waiting was compared to the outcomes of removing the tonsils in school-age children. This study found that patient's symptoms overall improved as well as quality of life and polysomnography findings. However surgical treatment did not improve attention or function evaluated through neuropsychological testing.[37] Like snoring, the presence of large tonsils does not automatically give the patient OSA. Several studies have reported that no relationship exists between the size of the tonsil and adenoids and the presence of OSA.[10,39,40] Canto et al systematic review found overall weak results concerning tonsils: with sensitivity = 69% and specificity = 53% for tonsillar hypertrophy and sensitivity = 81% and specificity = 58% for Grade 3 tonsil size.[31] There was not a significant correlation in our study to size of tonsils and AHI (P=0.2449). However this study was limited in that tonsillar hypertrophy was not routinely recorded. If the patient did not have a direct referral there would be an initial visit where size of tonsils was often noted. Frequently patients would also seek out an otolaryngologist around the same time frame where the size of the tonsils was noted. Tonsillar size was gleaned from these two documents. A

little less than half (72 out of 153) of the subjects' tonsil size was unable to be reported. These unknowns were recorded as "no" in the data analysis. These limitations likely cause the data in this study to underreport tonsillar hypertrophy. Despite these results and the lack of literature ascertaining tonsillar hypertrophy to predict OSA, it continues to a major cause of OSA[2] and consequently will remain in the revised screening tool.

The Epworth Sleepiness Scale (ESS) measures a person's general level of daytime sleepiness. It contains 8 questions that explore a person's propensity to fall asleep during the day. Scores range from 0 to 24. In adults, a score of greater than 10 suggests heightened daytime sleepiness. In a study by Melendres et al., researchers modified the ESS to be more applicable to children. The suggestion of alcohol was deleted in question 7 and driving a car in question 8 was changed to being a passenger in a car. Melendres' study found that the ESS score of patients with OSA was not statistically different from those with primary snoring. Their results did not illustrate a difference between the ESS score of patients with mild, moderate, and severe OSA.[41] These results are similar to those in the present study where the ESS had no relationship to AHI ($P = 0.4831$). Parents and patients were asked to complete a typical 8 question ESS that was not modified for children (Appendix 3). Scores above 10 signified excessive sleepiness. These results, similar to the Melendres study, suggest that ESS holds little value in predicting OSAS and will be omitted in the revised screening scale.

The P in our modified STOPBANG screening tool represents neuropsych-behavioral symptoms in which excessive tiredness and irritability during the daytime was combined with daytime neurobehavioral symptoms. Positive scores of neurobehavioral symptoms required a diagnosis from a medical professional of either attention deficit disorder with hyperactivity (ADHD), attention deficit disorder without mention of hyperactivity (ADD) or oppositional

defiant disorder (ODD). This variable was analyzed because daytime hyperactivity and inattention have been shown to be associated with restless sleep. Moreover, improved sleep patterns have led to positive changes in behavior.[23,42,43] Sleep fragmentation, which is prevalent in pediatric OSA, may result in impaired daytime functioning.[23,44,45] Relationships between OSA, hyperactivity, and inattentive behavior have been documented.[23,46-51] Yet excessive tiredness, irritability, and hyperactivity are widely prevalent in children without OSA.[23,41,52-56] Unlike adults, children do not have the degree of daytime tiredness with sleep disordered breathing because their nighttime obstructive spells are more transitory and the periods of arousal are less discernable.[32] The occurrence of excessive daytime sleepiness in children with OSA is vague as it depends on the perception of the parents or caretakers. Young children most likely will not report tiredness.[23] In this retrospective chart review, parents completed the sleep questionnaire for the majority of subjects under the age of 12, their answers seemingly subjective and naturally influenced by their own thoughts, feelings, and attitudes on tiredness and their child's irritability. The results of this study indicated no relationship to AHI score and the reporting of excessive tiredness/irritability (P=0.3191). This study found that higher AHI scores were apparent in those with neurobehavioral daytime symptoms however it was not significant. Literature on neurobehavioral symptoms exemplifies a wide range of results. In a study by O'Brien et al., 26% of children with mild symptoms of ADHD were shown to have OSA via a polysomnograph.[14,57] A more recent study found that in children 6 to 14 years old with ADHD, OSA was not a common underlying disorder or etiologic factor.[14,58] Yet there is evidence to show persistent sleep disturbance can affect cognition, mood, behavior, and family function.[14,59] As mentioned previously, the CHAT study conducted by Marcus et al. ascertained that surgical treatment for OSA in school-age children, while improving symptoms and quality of life, did not improve

attention or executive function.[37] Canto's review showed attention deficit/hyperactivity disorder to have a sensitivity = 52% and a specificity = 67%, not an overall strong correlation.[31] Based on the lack of evidence that psycho-behavioral symptoms and reports of tiredness/irritability have significant predictive value, this variable will be omitted in the revised scale.

The B in this retrospective chart review denotes body mass index (BMI) percentile for a given age. Due to varying changes in height and weight during a child's growth and development, BMI percentiles specific for age and sex are the best way to depict childhood weight status. BMI $\geq$ 95% indicates an obese child. BMI $\geq$ 85-94% represents children who are overweight. Underweight children are in the BMI $\leq$ 5% category.[60] It was proposed that BMI percentiles above 95% would place a patient at risk for OSA. Obesity has been found to predispose patients to OSA due to the mass loading of upper airway and respiratory muscles, in addition to impairment of ventilation. OSA in obese children ranges from 13 to 36%, based on the severity of obesity.[61,62] A review of the scatterplot (Figure 4) indicates that in this study not only are patients above the 85th percentile at risk for OSA, but also patients below the 10th percentile for BMI. These differences in BMI appear to account for the two types of pediatric OSA: Type I: non obese with adenotonsillar hypertrophy and Type II: obese with mild upper airway lymphadenoid hyperplasia[23] that were proposed earlier. With obesity on the rise, it is easy to forget that traditionally, OSA children were non-obese.[63,64] Case series have suggested that growth (especially weight gain) accelerates after surgery for OSA, proposing that OSA may inhibit growth.[65-68] These results agree with the findings in this study, that not only are overweight and obese children predisposed to OSA, but underweight children may also indicate OSA. Furthermore, BMI percentiles $\geq$85% and $\leq$10% appear to be a risk factor for OSA and these parameters will reside in the revised screening tool.

In our study age presents the A in STOPBANG and was defined as a risk factor for patients younger than 3 or older than thirteen. Evidence of systematic variability with age in pediatric OSA is lacking.[69] Pediatric OSA can occur at any age from birth to adolescence, however it is most common in the preschool age according to the International Classification of Sleep Disorders Diagnostic and Coding Manual.[12] It has been reported that the peak incidence of OSA is between 2 and 6 years of age,[9] while other studies cite a high prevalence around 2-8 years with a subsequent decline in frequency.[70] Our original age parameters are based on the theory that children younger than 3 may have underdeveloped airways and patients older than 13 are nearing their full growth potential and may start to develop adult risk factors for OSA such as obesity and high blood pressure. After analysis of the data, a reconsideration of age cutoffs is proposed, as it appears in this study that children younger than 4 and older than 16 are at most risk for OSA, yielding specificity as high as 88% and sensitivity as high as 61% (Figure 4). Thus these changes are taken into account in our revised scale.

The N in the modified STOPBANG screening tool represents neuromuscular disorders. Neuromuscular disorders related to abnormalities of muscle tone, hypotonia, and spasticity influence a child to have OSA.[9] The results of this study show a weakly positive relationship to AHI (P=0.1482). This study was limited in that there was a very low sample size of patients who had a neuromuscular disorder- only 21 patients out of 153. This low sample size may have prevented a predictive value with AHI. Some of the neuromuscular disorders of subjects in this study were muscular dystrophy and cerebral palsy. Neuromuscular deficits, along with craniofacial abnormalities and soft tissue hypertrophy, are frequently the origin of airway narrowing.[69] Patency of the upper airway is a reflection of the muscle activities. In neuromuscular disorders the decrease in muscle tone can greatly alter the airway. In disorders

such as duchenne muscular dystrophy and cerebral palsy, the presence of sleep disordered breathing arises because the weakened dilator and respiratory muscles are further weakened during sleep.[61] Although neuromuscular disorders did not show a strong correlation to AHI in this study, it remains in the revised scale as it is cited as one of the main causes of OSA.[32,35]

The G in the modified STOPBANG represents genetic disorders and congenital disorders and like neuromuscular disorders, did not show a strong correlation to AHI in the current study (P=0.0648). Many of these disorders are the underlying etiology of upper airway obstruction as a result of craniofacial malformation.[71,72] A wide range of these disorders results in variable expression in each patient with individualized respiratory compromise. Most often congenital disorders may result in the mandible failing to grow in utero.[71,72] Children with Down syndrome fall into the genetic disorder category and it has been shown that 31 to 45% of Down syndrome children are affected by OSA because of characteristics such as midfacial hypoplasia, micrognathia, and muscular hypotonia that reduce the size of the upper airway.[61,73] Other syndromes with craniofacial abnormalities such as Apert's and Crouzon's have been shown to be disposed to OSA.[61,74] In the systematic review and meta-analysis by Canto micrognathia/retrognathia had a sensitivity=0% with a specificity=95%. Furthermore, midface hypoplasia overall had a sensitivity = 16% and a specificity = 100%.[31] These results ascertain that craniofacial anomalies are not highly predictive of pediatric OSA. Like neuromuscular disorder patients, in our study there was a very small sample size from the data collected- only 21 out of 153 subjects had a genetic/congenital disorder. We propose to keep genetic/congenital disorders in the screening tool as craniofacial anomalies and syndromes were ascertained to be a cause of OSA.[32]

If the 7 revised components mentioned above were used to score the likelihood of OSA, a multiple logistic regression indicates that more components would have been statistically significant (see Table 10). Even though the p-values in the table are not entirely fair, as they are the result of post-hoc data mining, it does suggest that the additional factors of BMI risk, age risk, and instances of neuromuscular disorders or genetic/congenital disorders may be important indicators of higher OSA risk. Unfortunately, as snoring was measured in this study, this analysis did not give any statistical support for its continued inclusion.

There were several limitations in this retrospective chart review. The collection of data from the sleep questionnaire proved challenging because several questions pertained to the presence of a single variable and answers were varied. For instance, patients and parents had to answer several questions detailing the patient's tiredness throughout the day. Often times some of the questions would be answered denying the symptom and several more ascertaining the presence of the symptom. The majority was recorded, but since several questions represented one variable this could have skewed the results. Much of the information collected was limited to the parents. Many of the completed sleep questionnaires had inconsistent answers recorded. For instance, the patient would have a very low ESS value but parents would indicate that the patient did indeed have excessive sleepiness during the daytime in the actual questionnaire. There remained a lack of verification from the parents reporting and it was not clear whether the patient or parent had filled out the questionnaire. Expectation bias most certainly may have existed in this study as the Examiner was recording the PSG results and also the presence of specific variables that suggest OSA. In an attempt to avoid expectation bias, when able, variables were recorded first on the data sheet prior to accessing the PSG results. Since data was collected routinely prior to this study, often variables were not recorded and information was missing, this

restricted the amount of subjects able to be used. Prior to 2011, all sleep questionnaires and patient information was recorded in paper charts. These charts were unable to be accessed at the time of data collection; as a result the sample size was less than originally projected. The sample size does not represent an average population of children as all patients were believed to have a sleep disorder problem and as a result were seeking a diagnosis; thus this study may contain selection bias. It is possible that patients were unable to replicate their natural sleep during the polysomnography and did indeed have OSA. Patients whose ages ranged from 0–18 were included in the data collection. Patients over a certain age (12–14) often are almost fully grown and considered adults, consequently variables in the older age range could have skewed characteristics specific for children. Lastly, because this study was retrospective, researchers were limited in what variables could be used in the screening tool as to what information had been previously collected. For instance, tonsillar hypertrophy was considered as an important screening tool but had not always been recorded by the clinician.

There were several variables that this study did not focus on but may be relevant to pediatric OSA. It has been frequently mentioned that there is a genetic component to children with OSA. Future studies may want to include evaluation of whether the parents or siblings currently have a sleep disorder. The siblings of children who have been treated for sleep disorders are more likely have sleep disordered breathing.[11,75] In addition, children with a family history of OSA are four times more likely to have OSA compared to children from families with no OSA diagnosis.[76,77] It is also recommended to define snoring both quantitatively and qualitatively to omit those children whose snoring is infrequent and not really suggestive of OSA. Mouth breathing during the daytime (sensitivity = 26%, specificity = 79%) and during sleep (sensitivity = 68%, specificity = 42%) was evaluated for diagnostic quality in a previous

study[31]. Despite the mediocre results, mouth breathing is easily diagnosed by dentists and may be a variable useful for screening patients. Ethnicity may also play a role in screening at risk pediatric patients for OSA. Literature cites that being African American is a risk factor,[14,78-80] however this was not found in our study. Kheirandish-Gozal et al. found that the prevalence of OSA was increased in poorly controlled asthmatic children;[70] perhaps this variable should be included in future studies. Worthy of attention would be a prospective study in which variables typical of Pediatric OSA and commonly diagnosed clinically by dentists could be evaluated to determine a predictive value. Further evaluation is recommended to continue to strive and find a highly predictive screening tool for pediatric OSA.

Polysomnography studies have proven labor intensive and have shown to be inaccessive to children. Moreover Gozal ascertains that "development of simple, cheap, and reliable diagnostic tools that permit more expanded screening of at-risk populations, and enable accurate identification of the children with definitive disease or with definitive absence of disease would revolutionize the field and provide timely access to clinical care to a large sector of the pediatric population, thereby reducing the health burden of OSA".[30] This study attempted to further clarify which variables were strongly associated with childhood OSA, and thus could be used to develop a screening tool that would accurately predict the disorder in at risk children.

Conclusions

The purpose of the study was to develop a concise and easy-to-use questionnaire as a screening tool to aid in the diagnosis of OSA in pediatric patients. The screening scale proposed (S(T1)OPBANG) proved to be predictive of pediatric OSA. Based on the results of this study and the review of the literature the following components are recommended to remain in a revised screening tool: presence of snoring, sleep obstruction, and tonsillar hypertrophy; BMI, age, neuromuscular disorders and genetic/congenital disorders. Worthy of attention would be to explore ethnicity factors, presence of asthma, and family history of OSA in future studies.

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Table 1. Demographic Characteristics of Study Subjects (N=153)

Characteristic	N	Percent			
Gender					
Female	70	45.8			
Male	83	54.2			
Race/ethnicity					
Asian	4	2.6			
Black or African American	66	43.1			
Hispanic	5	3.3			
White	69	45.1			
Unknown / Not Reported	9	5.9			
	Mean	SD	Median	Range	
Age (years)	10.59	4.10	10.50	3.17	17.50
Age (months)	127.06	49.21	126.00	38.00	210.00
Height (M)	1.40	0.24	1.42	0.91	1.88
Weight (kg)	49.56	31.13	42.18	12.70	188.70
BMI (kg/mm^2) (n=152)	23.32	8.85	21.17	11.26	59.69
BMI percentile	72.73	33.52	91.42	0.00	100.00
Epworth scale (n=112)	8.57	6.29	8.00	0.00	24.00

Abbreviation: SD = standard deviation,

Table 2. Risk Factors

Risk Factor	Risk factor	N	Percent
Snore	No	51	33.3
	Yes	91	59.5
	Unknown	11	7.2
Tonsillar hypertrophy	No	50	32.7
	Yes	31	20.3
	Unknown	72	47.1
Epworth > 10	No	69	45.1
	Yes	43	28.1
	Unknown	41	26.8
Obstruction	No	80	52.3
	Yes	59	38.6
	Unknown	14	9.2
Daytime neurobehavioral symptoms	No	125	81.7
	Yes	26	17.0
	Unknown	2	1.3
Excessive tiredness/irritability during daytime	No	57	37.3
	Yes	79	51.6
	Unknown	17	11.1
Neuro/Muscular disorder	No	0	0.0
	Yes	21	13.7
	Unknown	132	86.3
Genetic/Congenital disorder	No	0	0.0
	Yes	21	13.7
	Unknown	132	86.3
BMI percent >= 95	No	92	60.1
	Yes	60	39.2
	Unknown	1	0.7
Age < 3 or > 13	No	105	68.6
	Yes	48	31.4
	Unknown	0	0.0

Table 3. Apnea-Hypopnea Index

Apnea-Hypopnea index	N	Percent
0-none	96	62.75
1-mild	29	18.95
2-moderate	16	10.46
3-severe	12	7.84

Table 4. Scales

	S(T1)OPBANG		S(T2)OPBANG	
Score	N	Percent	N	Percent
0	2	1.3	4	2.6
1	28	18.3	24	15.7
2	39	25.5	38	24.8
3	40	26.1	40	26.1
4	25	16.3	25	16.3
5	17	11.1	17	11.1
6	2	1.3	5	3.3

Table 5. S(T1)OPBANG Scale Results

S(T1)OPBANG	Specificity	Sensitivity	True Pos	True Neg	False Pos	False Neg
.	100%	0%	0	125	0	28
6	99%	4%	1	124	1	27
5	94%	39%	11	117	8	17
4	78%	57%	16	97	28	12
3	48%	68%	19	60	65	9
2	21%	86%	24	26	99	4
1	2%	100%	28	2	123	0
0	0%	100%	28	0	125	0
0	0%	100%	28	0	125	0

Logistic regression P = 0.0007, AUC = 67.7%

Table 6. S(T2)OPBANG Scale Results

S(T2)OPBANG	Specificity	Sensitivity	True Pos	True Neg	False Pos	False Neg
.	100%	0%	0	125	0	28
6	99%	14%	4	124	1	24
5	90%	36%	10	113	12	18
4	74%	50%	14	92	33	14
3	46%	68%	19	57	68	9
2	19%	86%	24	24	101	4
1	3%	100%	28	4	121	0
0	0%	100%	28	0	125	0
0	0%	100%	28	0	125	0

Logistic regression P = 0.0040, AUC = 64.3%

Table 7. Unadjusted Analysis Results

		OSA				
Risk indicator		Negative	Positive		RR	p-value
Snore	No	55	7	11%	2.04	0.0642
	Yes	70	21	23%		
Tonsillar hypertrophy	No	102	20	16%	1.57	0.2262
	Yes	23	8	26%		
Epworth > 10	No	90	20	18%	1.02	0.9515
	Yes	35	8	19%		
Sleep obstruction	No	86	8	9%	3.98	0.0001
	Yes	39	20	34%		
neuroPsych or tired	No	48	13	21%	0.77	0.4329
	Yes	77	15	16%		
BMI percent > 95	No	80	13	14%	1.79	0.0852
	Yes	45	15	25%		
Age < 3 or Age > 13	No	88	17	16%	1.42	0.3181
	Yes	37	11	23%		
Neuro/Muscular	No	108	24	18%	1.05	0.9241
	Yes	17	4	19%		
Genetic/Congenital	No	109	23	17%	1.37	0.4821
	Yes	16	5	24%		

Table 8. Components of S(T1)OPBANG

Component		Chi-square	P-value		OR	95% CI	
S	Snore=yes	0.17	0.6767		0.74	0.19	2.99
T	Tonsillar hypertrophy=yes	1.35	0.2449		1.96	0.63	6.05
O	Obstruction=yes	10.77	0.0010	*	7.56	2.26	25.27
P	Neurobehavioral=yes or Tiredness=yes	0.99	0.3191		0.61	0.24	1.60
B	BMI% > 95	1.30	0.2551		1.90	0.63	5.73
A	Age < 3 or Age > 13	2.70	0.1004		2.42	0.84	6.96
N	Neuromuscular=yes	2.09	0.1482		3.06	0.67	13.92
G	Genetic/congenital=yes	3.41	0.0648		3.71	0.92	14.90
	All 8 components	23.84	0.0024	*			

Table 9. Components of S(T2)OPBANG

Component		Chi-square	P-value		OR	95% CI	
S	Snore=yes	0.04	0.8441		0.87	0.23	3.34
T	Epworth	0.49	0.4831		1.52	0.47	4.86
O	Obstruction=yes	10.73	0.0011	*	7.26	2.22	23.76
P	Neurobehavioral=yes or Tiredness=yes	1.41	0.2344		0.52	0.18	1.53
B	BMI% > 95	1.19	0.2752		1.83	0.62	5.44
A	Age < 3 or Age > 13	2.20	0.1377		2.17	0.78	6.05
N	Neuromuscular=yes	1.81	0.1787		2.77	0.63	12.19
G	Genetic/congenital=yes	3.27	0.0704		3.68	0.90	15.09
	All 8 components	22.99	0.0034	*			

Table 10. Proposed Components

			OSA				Adjusted			
	Risk indicator		Negative	Positive		RR	OR	(95% CI)		p-value
S	Snore	No	55	7	11%	2.04	0.93	(0.21,	3.82)	0.9249
		Yes	70	21	23%					
T	Tonsillar hypertrophy	No	102	20	16%	1.57	2.11	(0.66,	6.72)	0.2057
		Yes	23	8	26%					
O	Sleep obstruction	No	86	8	9%	3.98	7.84	(2.41,	31.41)	0.0004 *
		Yes	39	20	34%					
B	BMI percent>85 or <10	No	45	4	8%	2.85	4.02	(1.28,	15.93)	0.0155 *
		Yes	79	24	23%					
A	Age younger than 4, older than 16	No	111	22	17%	1.81	4.33	(1.06,	18.34)	0.0410 *
		Yes	14	6	30%					
N	Neuro/Muscular disorder	No	108	24	18%	1.05	3.79	(0.76,	18.29)	0.1007
		Yes	17	4	19%					
G	Genetic/Congenital disorder	No	109	23	17%	1.37	5.03	(1.19,	21.22)	0.0289 *
		Yes	16	5	24%					

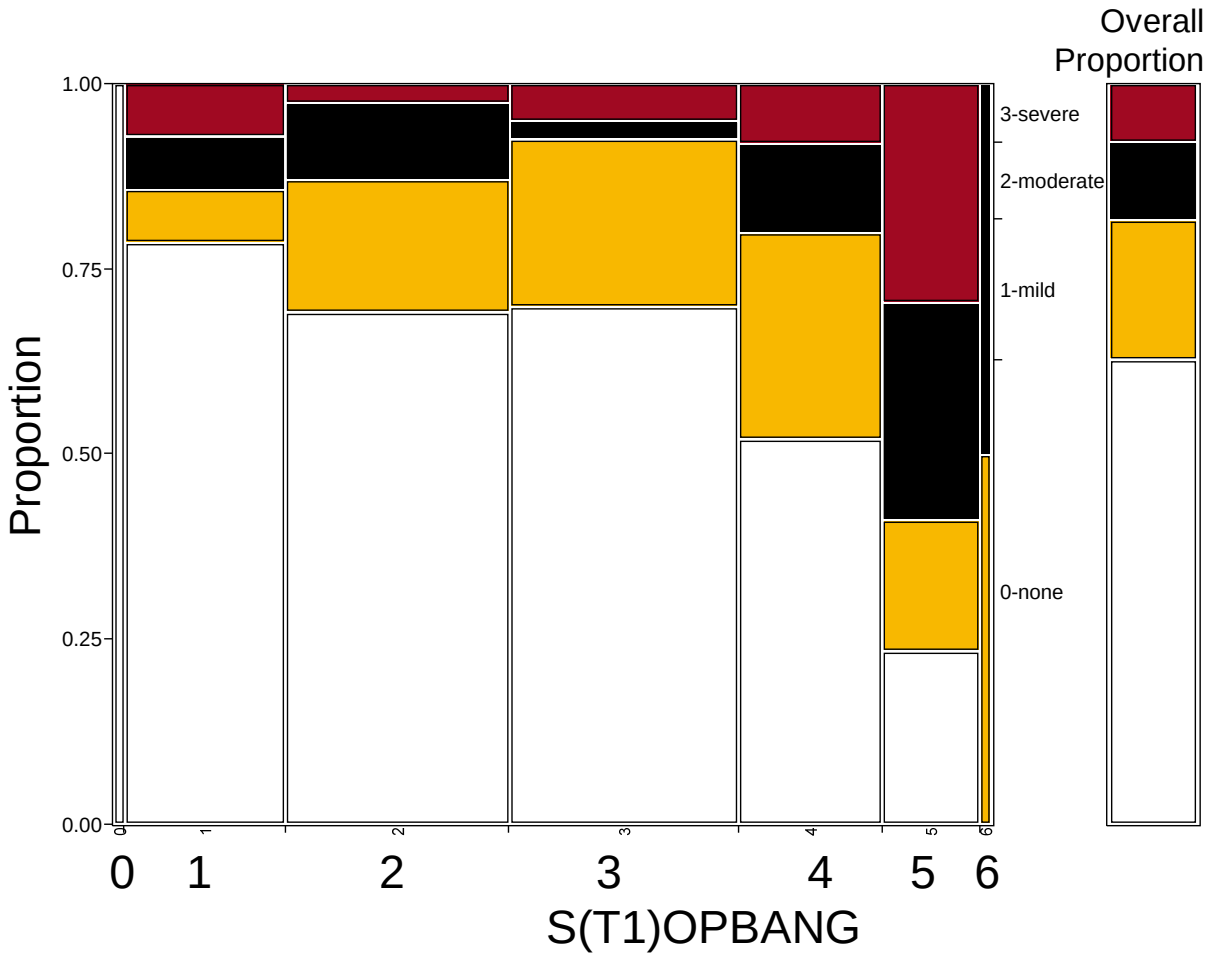

Figure 1. Results for S(T1)OPBANG

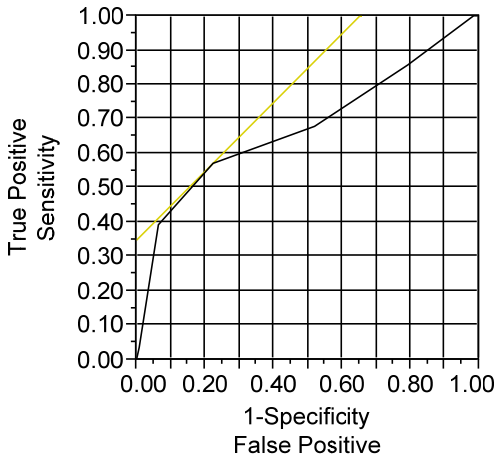

Figure 2. Receiver Operating Characteristic Curve for S(T1)OPBANG

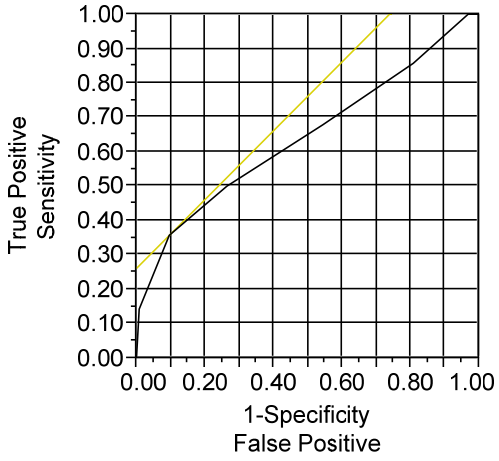

Figure 3. Receiver Operating Characteristic Curve for S(T2)OPBANG

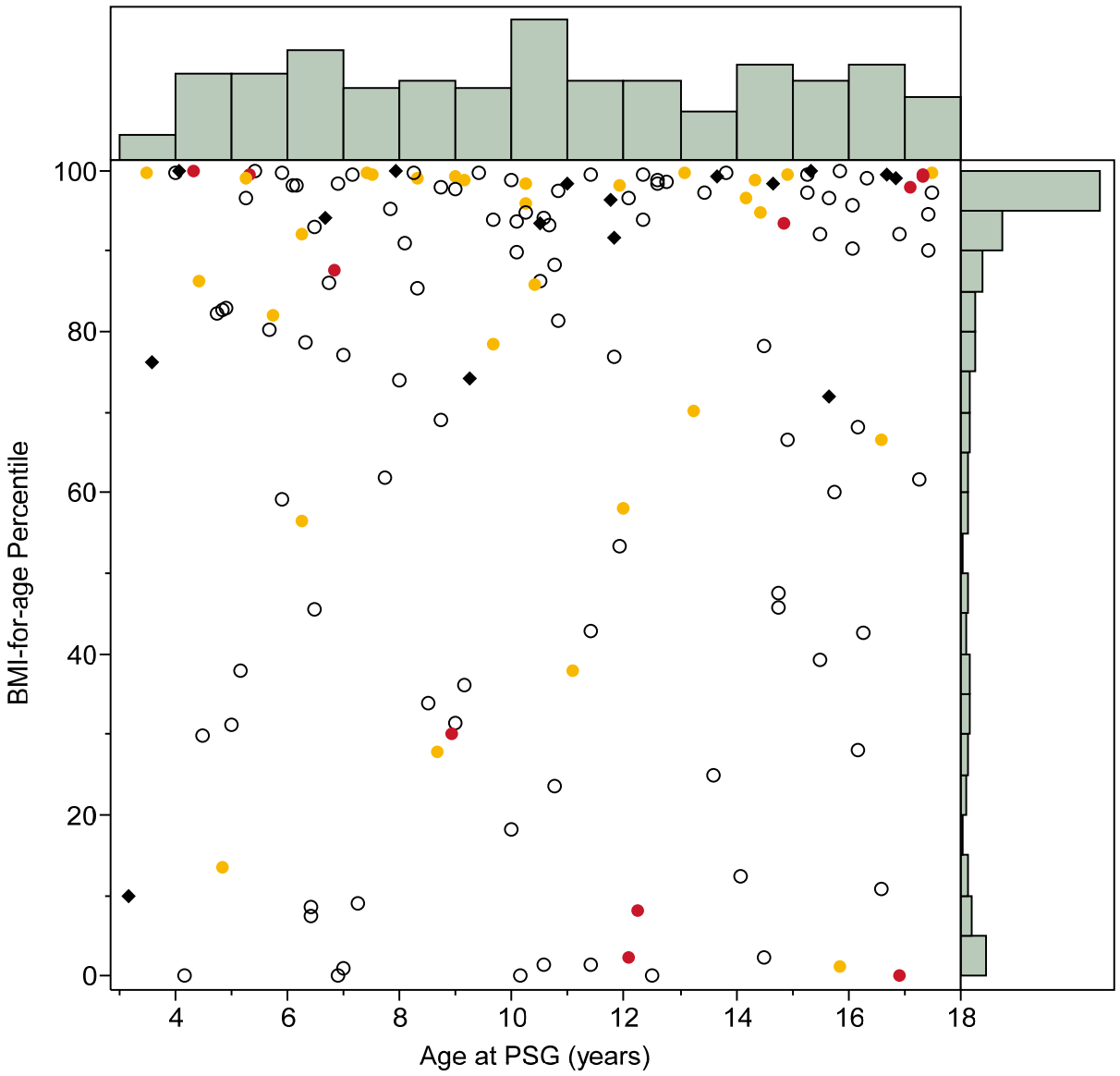

Figure 4. BMI Percentile for Age versus Age

Legend: AHI none ○, mild ● moderate ● and 12=severe ●

Appendix 1

Data Sheet

Obstructive Sleep Apnea in Children Study #_____

Demographics

1. Date of PSG _____
2. Age at time of PSG (__y__m) _____
   (<4 or >12 yrs= 1, otherwise 0)

3. Race (Choose one): Caucasian, African american, Asian, Hispanic other:_____
4. Gender (M or F) _____

Sleep Center Information

5. Snore (No, Yes, unknown) _____
6. Tonsillar hypertrophy (No, Yes, unknown) _____
7. Obstruction (No, Yes, unknown) _____
8. Daytime neurobehavioral symptoms (No, Yes, unknown)_____

a. ICD 314.01 Attention deficit disorder with Hyperactivity
b. ICD 314.00 Attention deficit disorder without mention of Hyperactivity
c. ODD oppositional defiant disorder
d. _____

9. Excessive tiredness/irritability during daytime (No, Yes, unknown) _____

10. Weight __lb/kg and Height: ____inches/cm
    BMI if provided in chart: _____
    BMI calculated by recorders:_____

11. Neuro/Muscular disorder(fill in):_____
12. Genetic/Congenital disorder (fill in):_____
13. Epworth scale _____

14. AHI score_____

Appendix 2

VCU Center for Sleep Medicine Questionnaire

Sleep Questions: Please respond to what extent a statement (item) has been applicable to you during the past 4 weeks. Score each item on a 4-point-scale:
1 (not at all) 2 (somewhat) 3 (rather much) 4 (very much)

Section 1: ____

1. I am told that I snore.	1 2 3 4
2. I sweat during the night.	1 2 3 4
3. I am told that I hold my breath when sleeping.	1 2 3 4
4. I am told that I wake up gasping for air.	1 2 3 4
5. I wake up with a dry mouth.	1 2 3 4
6. I wake up during the night while coughing or being short of breath.	1 2 3 4
7. I wake up with a sour taste in my mouth.	1 2 3 4
8. I wake up with a headache.	1 2 3 4

Section 2: ____

9. I have difficulty in falling asleep.	1 2 3 4
10. Thoughts go through my head and keep me awake.	1 2 3 4
11. I worry and find it hard to relax.	1 2 3 4
12. I wake up during the night.	1 2 3 4
13. After waking up during the night, I fall asleep slowly.	1 2 3 4
14. I wake up early and cannot get back to sleep.	1 2 3 4
15. I sleep lightly.	1 2 3 4
16. I sleep too little.	1 2 3 4

Section 3: ____

17. I see dreamlike images when falling asleep or waking up.	1 2 3 4
18. I sometimes fall asleep on a social occasion.	1 2 3 4
19. I have sleep attacks during the day.	1 2 3 4
20. With intense emotions, my muscles sometimes collapse during the day.	1 2 3 4
21. I sometimes cannot move when falling asleep or waking up.	1 2 3 4

Section 4: ____

22. I am told that I kick my legs when I sleep.	1 2 3 4
23. I have cramps or pain in my legs during the night.	1 2 3 4
24. I feel little shocks in my legs during the night.	1 2 3 4
25. I cannot keep my legs at rest when falling asleep.	1 2 3 4

Section 5: _____

26. I would rather go to bed at a different time.	1	2	3	4
27. I go to bed at very different times (more than 2 hr difference).	1	2	3	4
28. I do shift work.	1	2	3	4

Section 6: _____

29. I sometimes walk when I am sleeping.	1	2	3	4
30. I sometimes wake up in a different place than where I fell asleep.	1	2	3	4
31. I sometimes find evidence of having performed an action during the night I do not remember.	1	2	3	4

Section 7: _____

32. I have frightening dreams (if not, go to Item 37).	1	2	3	4
33. I wake up from these dreams.	1	2	3	4
34. I remember the content of these dreams.	1	2	3	4
35. I can orientate quickly after these dreams.	1	2	3	4
36. I have physical symptoms during or after these dreams (e.g., movements, sweating, heart palpitations, shortness of breath).	1	2	3	4

Section 8: _____

37. It is too light in my bedroom during the night.	1	2	3	4
38. It is too noisy in my bedroom during the night.	1	2	3	4
39. I drink alcoholic beverages during the evening.	1	2	3	4
40. I smoke during the evening.	1	2	3	4
41. I use other substances during the evening (e.g., sleep or other medication).	1	2	3	4
42. I feel sad.	1	2	3	4
43. I have no pleasure or interest in daily occupations.	1	2	3	4

Section 9: _____

44. I feel tired at getting up.	1	2	3	4
45. I feel sleepy during the day and struggle to remain alert.	1	2	3	4
46. I would like to have more energy during the day.	1	2	3	4
47. I am told that I am easily irritated.	1	2	3	4
48. I have difficulty in concentrating at work or school.	1	2	3	4
49. I worry whether I sleep enough.	1	2	3	4
50. Generally, I sleep badly.	1	2	3	4

Appendix 3

Epworth Scale

How sleepy have you been over the last 4 weeks? Situation	**Chance of Dozing or Sleeping** **Low** **(circle the most appropriate number)**			**High**
Sitting and reading	0	1	2	3
Watching TV	0	1	2	3
Sitting inactive in a public place	0	1	2	3
Being a passenger in a motor vehicle for an hour or more	0	1	2	3
Lying down in the afternoon	0	1	2	3
Sitting and talking to someone	0	1	2	3
Sitting quietly after lunch (no alcohol)	0	1	2	3
Stopped for a few minutes in traffic while driving	0	1	2	3
Total Score (add up the circled numbers)				

Vita

Jo Koontz Cronly was born on June 19, 1982 in Culpeper, Virginia. She received her Bachelor of Arts in biology and English from the University of Virginia in 2004. She completed her Masters in Science from Virginia Commonwealth University in 2008 and was accepted to Virginia Commonwealth University School of Dentistry, graduating magna cum laude with a Doctor of Dental Surgery in May 2012. Jo will complete her Pediatric Dentistry training at Virginia Commonwealth University in June 2014.