Clinical Evaluation in Predicting Childhood Obstructive Sleep Apnea*

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Objective: **To determine whether parents' observation, clinical examination, and lateral upper airway radiograph are useful in detecting clinically significant obstructive sleep apnea (OSA) in children.**

Method: **We retrospectively reviewed data of 50 children aged 4 to 18 years who were consecutively referred to a sleep clinic for suspected OSA. All subjects underwent clinical assessments including standardized history collection, physical examination, and lateral neck radiograph for measurement of postnasal space. Each child underwent overnight polysomnography on the night of clinical assessments. Patients with clinically significant OSA, defined as apnea-hypopnea index (AHI) > 5, were compared with primary snorers, defined as AHI ≤ 5.**

Results: **Thirty-one children had clinically significant OSA, and 19 children were primary snorers. The prevalence of risk factors including allergic rhinitis, obesity, and craniofacial anomaly was similar between the two groups. Observable apnea during sleep, nocturnal enuresis, intrusive naps, mouth breathing, enlarged tonsils, and radiologic features of upper airway narrowing due to adenoid hypertrophy were found to be predictors for clinically significant OSA. Combining upper airway narrowing and mouth breathing or nocturnal enuresis had a sensitivity of 90.3%, and combining all six predictors had a sensitivity of 93.5% of detecting OSA.**

Conclusion: **Combining clinical and radiologic findings might be helpful to screen for children with clinically significant OSA who need earlier investigation and intervention.**

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Key words: Chinese children; clinical predictors; sleep apnea, obstructive

Abbreviations: AHI = apnea-hypopnea index; ANR = adenoidal-nasopharyngeal ratio; BMI = body mass index; EDS = excessive daytime sleepiness; NPV = negative predictive value; OR = odds ratio; OSA = obstructive sleep apnea; PPV = positive predictive value

The prevalence of obstructive sleep apnea (OSA) is approximately 2% in children, but primary snoring is reported to be more common, ranging from 3 to 12%.[1] OSA can result in severe complications if left untreated, while primary snoring, defined as snoring without altered sleep architecture, alveolar ventilation, or oxygenation, is considered to be a clinically benign condition. At present, the "gold standard" for diagnosis of OSA is polysomnography performed in a sleep laboratory. However, polysomnography is expensive, time and labor consuming, and not widely available.[1] A simple screening tool to identify children who require early referral for polysomnography is highly desirable. Despite extensive

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research including the use of scoring of historical and physical findings,[2,3] nocturnal oximetry monitoring,[4] and adenoid size measurement on plain lateral neck radiography,[5,6] none of the methods have sufficient sensitivity to detect OSA in children with snoring. Nevertheless, there was no study on the diagnostic performance of a combination of these screening tests. Hence, we aimed to determine whether a combination of parents' observation, physicians' clinical findings, and radiologic assessment of upper airway narrowing is useful in screening for clinically significant OSA in Chinese children.

Materials and Methods

Study Design and Subjects

Children aged 4 to 18 years with suspected OSA consecutively referred to our sleep clinic from April 1999 to March 2003 were included in this retrospective study. Patients with known history of chronic pulmonary diseases and neuromuscular diseases were excluded. Each subject underwent clinical evaluation including a standardized clinical data sheet, physical examination, radiograph of postnasal space for upper airway assessment, and overnight polysomnography. Informed consent for radiography and polysomnography was obtained from parents. The retrieval and analysis of all the collected clinical information and radiography and polysomnography results were approved by the Institutional Review Board of the University of Hong Kong.

Historical Data

A standardized clinical data sheet consisted of questions regarding the child's snoring patterns, nighttime and daytime symptoms, as well as other symptoms associated with OSA. We asked the parents if their children sleep during sedentary activities in the daytime, *eg*, falling asleep during car travel, doing homework, or watching television, to evaluate the child's daytime sleepiness, and we used intrusive naps to define these phenomena. The questionnaires were administered by the pediatrician admitting the patient with an accompanying parent on the day of sleep study. All data collected were verified by the third author during the follow-up visit at a sleep clinic.

Physical and Radiologic Findings

Body weight and height were measured, and body mass index (BMI) was calculated. Physical examination was performed by a pediatrician with special attention to craniofacial abnormalities, swollen nasal turbinates, mouth breathing, and tonsillar enlargement. Obesity was defined as BMI $>$ 90th percentile of the age- and sex-standardized BMI chart developed for Chinese children.[7] The tonsil size was graded by direct visualization: 0, not visible; 1, extending to the pillars; 2, enlarged beyond the pillars but not meeting uvula; 3, meeting the uvula; and 4, "kissing" at the midline. Moderate-to-severe tonsillar hypertrophy was defined as grade 3 or above. The extent of upper airway narrowing due to adenoidal hypertrophy was assessed by one pediatric radiologist blinded to the polysomnography results. The radiologist categorized adenoid enlargement with adenoidal-nasopharyngeal ratio (ANR) $<$ 0.5 as normal or mild and adenoid enlargement with ANR $>$ 0.5 as moderately or severely enlarged adenoid.[8,9] For the sake of reliability and reproducibility, upper airway narrowing due to adenoidal hypertrophy was simply analyzed as a binary variable, *ie*, presence or absence of upper airway narrowing using ANR $<$ 0.5 as cut-off.

Polysomnography

Standard overnight polysomnography was performed (Alice 4; Respironics; Murrysville, PA), with the accompanying parent sleeping in the same room with the child. The following parameters were measured: four-channel EEG with bilateral central and occipital leads, electrooculography to measure vertical and horizontal eye movements, electromyography with submental electrodes, ECG, airflow measurement through nose and mouth by a thermistor, thoracic and abdominal plethysmograph to measure respiratory effort, pulse oximetry, and tracheal sound recording by a microphone secured to the neck. Polysomnography was interpreted by a pediatrician trained in sleep medicine who was unaware of the clinical and radiographic findings. Respiratory events were defined by standard criteria.[10] An obstructive apnea event was defined as cessation of airflow with increase in irregular respiratory and abdominal movement for $>$ 10 s. Central apnea was defined as cessation of breathing with no respiratory effort for $>$ 10 s. Hypopnea was defined as a reduction in oronasal flow by $>$ 50% associated with arousals and /or desaturations. Desaturation was defined as a drop of oxygen saturation by 4% or more from the baseline. Stage of sleep and arousal were characterized according to standard criteria with the help of EEG, electrooculography, and electromyography. Apnea-hypopnea index (AHI) was defined as the average number of apneas plus hypopneas per hour of sleep. The child was considered to have clinically significant OSA if the AHI was $>$ 5. An AHI $\leq$ 5 was considered to be clinically nonsignificant, and these children were classified as primary snorers.

Statistical Analysis

Twenty-five historical items from the clinical data sheet, 13 physical items, and 1 radiologic item were put into the analysis. The sensitivity, specificity, positive predictive value (PPV), and negative predictive value (NPV) of all these 39 clinical findings were calculated by standard formulae. In univariate analysis, categorical variables were compared between the OSA and the primary snoring groups by χ^2 tests or Fisher exact tests where appropriate, and crude odds ratios (ORs) were calculated for each item. Continuous variables such as age and BMI were compared between groups by two-sample t test.

Items that had statistically significant crude ORs were combined in an attempt to increase the sensitivity of OSA prediction. We regarded either one or more of these items being present as a positive test result and then compared the results between the two groups using a χ^2 test. The sensitivity, specificity, PPV, and NPV were calculated to determine which combinations of predictors of OSA had the best diagnostic performance.

All statistical analyses were performed using statistical software (SPSS version 10.0; SPSS; Chicago, IL). All statistical tests were two tailed with 0.05 as the threshold level of significance unless otherwise stated.

Results

Descriptive Statistics

Fifty-one patients were recruited, but 1 patient did not have the standardized clinical data sheet

completed and was excluded. Thirty-one patients were classified as having clinically significant OSA, and 19 patients were classified as primary snorers by the AHI criteria detailed above. There was no significant difference between the two groups with respect to background demographic characteristics, including age, sex, and BMI (Table 1). The sensitivity, specificity, PPV, and NPV values of each selected questionnaire item and clinical and radiologic findings are shown in Table 2.

Univariate Analysis

The occurrence of observable apnea during sleep, nocturnal enuresis, and intrusive naps were found to be significantly different between the OSA group and the primary snoring group ($p = 0.018$, 0.016, and 0.013, respectively), but none of the items had a high sensitivity or specificity for predicting OSA. Snoring was more frequently reported in the OSA group (96.8% vs 78.9%), and the snoring was more often described as disturbing to others (67.7% vs 42.1%). However, the difference did not reach statistical significance. Difficulty in breathing was uncommon in either group, and none of the parents reported detection of cyanosis when their child was sleeping.

Among the physical findings, detection of mouth breathing and presence of moderate-to-severe tonsillar hypertrophy could effectively differentiate between the two groups. Obesity, swollen nasal turbinates, and craniofacial abnormalities were not significantly different. A significantly higher proportion of OSA patients had adenoid enlargement shown on radiography.

Combination of Predictors

The six items including observable apnea during sleep, nocturnal enuresis, intrusive naps, mouth breathing, moderate-to-severe tonsillar hypertrophy, and upper airway narrowing that were found to be significant predictors in univariate analysis were combined and assessed. The presence of mouth breathing had 100% specificity, meaning that if OSA was not present, the child almost certainly would not have mouth breathing. However, none of the predictors had sufficiently high sensitivity in screening for children with OSA. So we tested different combinations of predictors to see which combination had the highest sensitivity, the presence of which assured us not to miss a significant number of potential OSA cases. We found that the presence of upper airway narrowing or mouth breathing, or the presence of upper airway narrowing or nocturnal enuresis had the highest sensitivity in detecting children with OSA, and both had a sensitivity of 90.3%. If we combined all the six significant predictors, the sensitivity could reach 93.5%.

The PPV of mouth breathing was 100%, meaning that if a child had mouth breathing, he or she almost certainly had OSA. However, none of the predictors had a sufficiently high NPV for us to rule out OSA. When we combined two predictors, the NPV increased. If a child did not have upper airway narrowing and mouth breathing, or if a child did not have upper airway narrowing and nocturnal enuresis, there was a 78.6% chance that OSA was not present. If the child had none of the six predictors, there was an 80% chance that OSA was not present. The presence of a combination of two predictive items that had the highest sensitivity and NPV and a combination of all predictors for predicting clinically significant OSA were calculated and presented in Table 3.

Table 1—*Demographic Data

Variables	OSA (n = 31)	Primary Snoring (n = 19)	p Value
Age, yr	7.8 ± 3.2	8.1 ± 3.7	0.82†
Male/female gender ratio	2.88:1	3.75:1	1.00‡
BMI, kg/m^2	18.6 ± 4.7	20.4 ± 6.9	0.37†

*Data are presented as mean ± SD unless otherwise indicated.
†Two-sample *t* test.
‡Fisher exact test.

Discussion

With heightened awareness of the public and primary care physicians, there is increasing referral of children with snoring or symptoms of OSA syndrome, or both, to a specialist setting. Polysomnography is considered to be the "gold standard" for diagnosing OSA syndrome, but it is resource demanding. Numerous studies have been conducted to find a screening tool to effectively identify patients with clinically significant OSA so as to prioritize polysomnography and allow prompt treatment. A systematic review[11] concluded that clinical findings are generally not reliable for diagnosing OSA in children. Neither were radiologic parameters useful in predicting OSA in children with clinically enlarged tonsils and adenoids.[6,12,13] Instead of relying on either clinical or radiologic parameters, we showed the potential usefulness of a combination of parental observation, physician evaluation, and radiologic findings to screen out those with clinically significant OSA for earlier polysomnography among children who were referred to a pediatric sleep clinic for

Table 2—*Distribution and Diagnostic Performance of Historical, Physical, and Radiologic Findings*

Predictors	OSA (n = 31), %	Primary Snoring (n = 19), %	p Value	Sensitivity, %	Specificity, %	PPV, %	NPV, %	OR (95% CI) of Difference
History								
Snoring > 1 yr	77.4	73.7	0.76	77.4	26.3	63.2	41.7	1.22 (0.33–4.60)
Snoring > 3 nights/wk	96.8	78.9	0.12	96.8	21.1	66.7	80	8 (0.82–78)
Snoring disturb others	67.7	42.1	0.14	67.7	57.9	72.4	52.4	2.88 (0.89–9.42)
Respiratory distress	22.6	5.3	0.22	22.6	94.7	87.5	42.9	5.25 (0.59–6.59)
Difficulty breathing	16.1	15.8	0.97	16.1	84.2	62.5	38.1	1.03 (0.22–4.89)
Chest retraction	22.6	5.3	0.22	22.6	94.7	87.5	42.9	5.25 (0.59–6.59)
Paradoxical chest movement	6.5	0	0.7	6.5	100	100	39.6	3.31 (0.15–2.68)
Cyanosis	0	0	1	0	100	0	38	
Observable apnea	35.5	5.3	0.018	35.5	94.7	91.7	47.4	9.90 (1.16–4.52)
Profound sweating	25.8	36.8	0.61	25.8	63.2	53.3	34.3	0.60 (0.17–2.04)
Restlessness	48.4	31.6	0.38	48.4	68.4	71.4	44.8	2.0 (0.61–6.72)
Nocturnal enuresis	51.6	15.8	0.016	51.6	84.2	84.2	51.6	5.69 (1.37–3.55)
Peculiar sleep position	38.7	57.9	0.24	38.7	42.1	52.2	29.6	0.45 (0.14–1.47)
Poor sleep quality	12.9	26.3	0.27	12.9	73.7	44.4	34.1	0.41 (0.28–9.41)
Difficult to wake up in the morning	16.1	10.5	0.69	16.1	89.5	71.4	39.5	1.64 (0.28–9.41)
Morning headache	6.5	15.8	0.36	6.5	84.2	40	35.6	3.16 (0.06–2.44)
Daytime somnolence	45.2	42.1	1	45.2	57.9	63.6	39.3	1.13 (0.36–3.58)
Abnormal behaviour	3.2	5.3	0.72	3.2	94.7	50	37.5	0.6 (0.04–10.2)
Poor school performance	35.5	26.3	0.55	35.5	73.7	68.8	41.2	1.54 (0.44–5.42)
Daytime naps > 1 h/d	38.7	21.1	0.28	38.7	78.9	75	44.1	2.37 (0.63–8.86)
Intrusive naps	45.2	10.5	0.013	45.2	89.5	87.5	50	7.00 (1.38–5.63)
Nasal obstruction/congestion	67.7	68.4	0.96	67.7	31.6	61.8	37.5	0.97 (0.28–3.30)
Mouth breathing during sleep	67.7	57.9	0.69	67.7	42.1	65.6	44.4	1.53 (0.47–4.98)
Mouth breathing during daytime	25.8	21.1	0.97	25.8	78.9	66.7	39.5	1.30 (0.33–5.11)
Frequent sore throat/dry mouth	29	10.5	0.24	29	89.5	81.8	43.6	3.48 (0.66–8.25)
Physical examination								
Obesity (BMI >90th percentile)	38.4	31.6	0.77	38.4	68.4	66.7	39.4	1.37 (0.41–4.58)
Dysmorphic facial features	19.4	10.5	0.48	19.4	89.5	75	39.5	2.04 (0.37–1.34)
Long adenoid facies	9.7	0	0.43	9.7	100	100	39.6	4.79 (0.23–8.08)
Swollen nasal turbinate	51.6	52.6	0.77	51.6	47.4	61.5	36	0.96 (0.31–3.01)
Midface hypoplasia	16.1	0	0.43	16.1	100	100	41.3	8.09 (0.42–155)
Micronagnathia/retrognathia	12.9	5.3	0.7	12.9	94.7	80	39.1	2.67 (0.28–5.85)
Nasal polyps	0	5.3	0.8	0	94.7	0	36	0.20 (0.01–5.06)
High-arched palate	3.2	0	0.43	3.2	100	100	38	1.92 (0.07–9.55)
Abnormal tongue	0	0	1	0	100	0	37.3	
Abnormal gag reflex	0	0	1	0	100	0	37.3	
Mouth breathing observed	38.7	0	0.0015	38.7	100	100	50	25.0 (1.38–452.66)
Abnormal tongue control	0	0	1	0	100	0	37.3	
Tonsillar size = grade 3	48.4	15.8	0.0326	48.4	84.2	83.3	48.5	5.00 (1.21–0.70)
Upper airway narrowing on radiography	80.6	42.1	0.0125	80.6	57.9	75.8	64.7	5.73 (1.60–0.50)

snoring. However, this composite score should not be considered as surrogate of polysomnography to diagnose OSA or identify children who might be at risk for postoperative complications due to severe OSA, and therefore positive screens should not be used *en lieu* of polysomnography.

In our study, observable apnea during sleep, nocturnal enuresis, intrusive naps, physician-detected

Table 3—*Combination of Predictors With the Highest Sensitivity and NPV*

Predictors	AHI > 5 (n = 31)		AHI = 5 (n = 19)		p Value	Sensitivity, %	Specificity, %	PPV, %	NPV, %
	Positive	Negative	Positive	Negative					
Upper airway narrowing or mouth breathing	28	3	8	11	0.0007	90.3	57.9	77.8	78.6
Upper airway narrowing or enuresis	28	3	8	11	0.0007	90.3	57.9	77.8	78.6
All six predictors	29	2	11	8	0.0037	93.5	42.2	72.5	80.0

mouth breathing, moderate-to-severe tonsillar hypertrophy, and radiologic evidence of adenoid hyperplasia were significantly more common in the OSA group than the primary snoring group. In the literature, observable apnea has a sensitivity of 47 to 74% and specificity of 17 to 90% in diagnosing OSA in children.[3,14–18] The high variability in diagnostic performance of this feature reflects the variable diagnostic criteria used and the different composition of patient samples.[11] Although the sensitivity and specificity of observable apnea are not very good, it gives certain help to clinicians deciding on further investigation.

Many studies[19,20] have reported nocturnal enuresis as a common manifestation of OSA in children. Wang et al[16] reported that enuresis had the highest predictive accuracy among several clinical features examined in 82 children. This was supported by a larger study[21] that showed a significantly higher prevalence of nocturnal enuresis in OSA patients (47%) compared to non-OSA children (17%). The prevalence of primary nocturnal enuresis was 7.3% in 7-year-old children in Sweden[22] and 10.9% of boys and 9.4% of girls in our locality.[23] The frequency of nocturnal enuresis appeared unusually high in our children with OSA (51.6%) compared to 15.8% in primary snorers, which did not differ significantly from normal children. Our study added supporting evidence that OSA symptoms should be properly questioned when a child presented with nocturnal enuresis.

Excessive daytime sleepiness (EDS) is considered as a key feature of OSA in adults, but it remains unclear whether it also occurs frequently in children with OSA. Gozal et al[24] reported that parental report may not reflect EDS accurately. However, the study showed that the reduction in sleep latencies in children with OSA reached statistical significance compared to the primary snorers and control groups. In our study, we included questions on daytime sleepiness, need for afternoon nap >1 h/d, and intrusive naps. Only intrusive naps turned out to be a significant predictor, being much more common in children with clinically significant OSA (45.2% vs 10.5%), suggesting this to be a more sensitive indicator of EDS in children with OSA.

In the literature,[3,15,16,18] mouth breathing was found to have a sensitivity of 29 to 78% and a specificity of 27 to 56% in diagnosing OSA. We evaluated both parents' report and physician's observation of mouth breathing in our cohort and found that only the latter was predictive of OSA. More severe symptoms with persistence of mouth breathing at the time of consultation that could be detected by physician may be the explanation for the difference observed, analogous to the detection of wheezing during consultation reflecting poor control asthma.

Although enlarged adenoid and tonsils is believed to be one of the most common causes for OSAS in children, previous studies failed to show that either enlarged adenoid or tonsils were good predictors of OSA. However, most of these studies included either clinical assessment of tonsils only[14,16,17] or radiologic assessment of adenoid[6] or tonsillar size only.[5] Our study showed that the inclusion of both clinical assessment of tonsils and radiologic assessment of upper airway narrowing could satisfactorily predict OSA. We used a crude objective criteria in defining presence or absence of upper airway narrowing due to enlarged adenoids because a previous study[25] showed that subjective judgment of adenoid size correlated well with ANR. Our findings suggest an additive effect of enlarged tonsils and adenoid in causing OSA in children, and that clinical tonsil size and radiologic evaluation at the level of adenoid can provide simple but sensitive assessment of upper airway patency in children with suspected OSA.

Obesity is associated with OSA in adults, but most children with OSA are not obese.[3,17] Our study also found that obesity was not more prevalent in children with OSA. The role of allergic rhinitis in childhood OSA is also controversial.[26,27] We did not find any relationship between allergic rhinitis and OSA. Craniofacial abnormality is also not helpful to distinguish OSA from primary snoring in our cohort either.

Our study suggests that the combination of various clinical predictors can potentially help in determining further investigation and management. The combination of six significant predictors had a high NPV. OSA with an AHI >5 is unlikely without the combination of snoring and the six predictors. Although surgical intervention like tonsillectomy and adenoidectomy may still be indicated in patients with OSA and a milder AHI index, this tool might allow for medical therapy such as combination of nasal steroids and leukotriene antagonists for mild OSA or primary snoring until polysomnography is available.[28] In addition, children with negative predictors should be followed up cautiously, as 20% of children with significant OSA are missed. However, as the specificity and PPV of mouth breathing approached 100%, children with snoring and mouth breathing warrant earlier polysomnography for confirmation of OSA so as to facilitate further clinical decision making.

There were several limitations in our study. First, the cut-off value of AHI for diagnosing OSA in children is controversial. Based on normative data, an obstructive apnea index of 1[1] or an AHI of 1.5[29]

are considered to be statistically significant in children. However, it is not known which level is clinically significant and requires treatment, and there are no studies correlating polysomnographic parameters with clinical outcome.[30] An apnea index of 10 is usually considered to be severe by most pediatric pulmonologists, but this does not give an accurate picture of breathing disturbance in many children with obstructive hypoventilation.[31] Thus, we decided to use an AHI > 5 as a cut-off, and this has also been adopted in other studies.[16,32] In either study, adenoidectomy and tonsillectomy would be considered if AHI was > 5. Second, our high baseline prevalence of clinically significant OSA (62%) suggested that our sample might be highly skewed toward clinically significant OSA. Since all the patients recruited were referred for suspected OSA, our study results could not be readily applied to the general population. Yet, they might be useful in increasing the posttest probability of clinically significant OSA in selected population of children who presented with snoring. Third, our sample size was small, which might limit the power to detect a significant difference between the two groups with respect to certain clinical predictors of OSA, such as frequent and disturbing snoring or obesity. The confidence intervals of ORs of significant predictors were quite wide, reflecting the imprecision of the magnitude of risk estimate associated with a certain predictor. Further studies involving larger samples in different populations are required to verify our results and provide a more precise estimate of the predictive values of various indicators of significant OSA. Despite these limitations, our study did suggest that a combination of clinical symptoms including observable apnea during sleep, nocturnal enuresis, intrusive naps in the daytime, presence of mouth breathing and enlarged tonsils on physical examination, and radiographic evidence of adenoid hypertrophy might be helpful to screen out patients with clinically significant OSA from children with snoring for earlier polysomnography and possible intervention. This is particularly useful in places with limited health-care resources.

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