The Correlation Among Obesity, Apnea-Hypopnea Index, and Tonsil Size in Children*

Yuen-yu Lam, FHKAM(Paed); Eric Y.T. Chan, MRCPCH; Daniel K. Ng, FHKAM(Paed); Chung-hong Chan, BSc; Josephine M.Y. Cheung, RPSGT; Shuk-yu Leung; Pok-yu Chow, FHKAM(Paed); and Ka-li Kwok, FHKAM(Paed)

Background: **The correlation between obesity and severity of obstructive sleep apnea (OSA) is well established in adults, but data are inconsistent in children. We hypothesized that there is a significant correlation between the degree of obesity and the severity of OSA in children.**

Methods: **We retrospectively reviewed records of weight, height, history, and polysomnography of all 1- to 15- year-old children referred to our sleep laboratory. Children with known anomalies and repeated polysomnography were excluded from this study. Obesity was defined as body mass index z score (BMI Z score) > 1.96. The correlation between BMI Z score and apnea-hypopnea index (AHI) was assessed. Possible confounding factors, *ie*, age, gender, and tonsil size, were adjusted by multiple linear regression.**

Results: **Four hundred eighty-two children were included in this study. Obese children had a significantly higher AHI (median, 1.5; interquartile range [IQR], 0.2 to 7.0) than the AHI of nonobese children (median, 0.7; IQR, 0.0 to 2.5). BMI Z score was significantly correlated with log-transformed AHI (Ln[AHI]) [$r = 0.156$, $p = 0.003$]. BMI Z score and tonsil size were still correlated with Ln(AHI) even after adjusted for other confounding factors ($p = 0.001$).**

Conclusion: **Degree of obesity as measured by BMI Z score and tonsil size are significantly related to severity of OSA as reflected by the AHI, although the correlation is mild.**

(CHEST 2006; 130:1751–1756)

Key words: child; obesity; obstructive; polysomnography; sleep apnea

Abbreviations: AHI = apnea-hypopnea index; BMI = body mass index; BMI Z score = body mass index z score; IQR = interquartile range; Ln(age) = log-transformed age; Ln(AHI) = log-transformed apnea-hypopnea index; OSA = obstructive sleep apnea; OSAS = obstructive sleep apnea syndrome; SDB = sleep-disordered breathing

The prevalence of obstructive sleep apnea (OSA) in obese children was reported to be high.[1] In Hong Kong Chinese children, Wing et al[2] found that OSA, defined as OSA and apnea-hypopnea index (AHI) ≥ 5/h, was found in 32.6% of obese children vs 4.5% in a normal weight group. Severity of OSA was correlated with the degree of obesity in adults.[3] In children, this relationship was also demonstrated in some studies,[4,5] but other pediatric studies[6,7] could not demonstrate a significant correlation between degree of obesity and severity of OSA. In Chinese children, similar data were not available. The current study was carried out to determine the

*From the Department of Paediatrics, Kwong Wah Hospital, Hong Kong, SAR.

This project was supported by a research grant from the board of directors, Tung Wah Group of Hospitals, Hong Kong SAR, who had no involvement in the study design; collection, analysis, and interpretation of data; writing of the report; or the decision to submit the report for publication.
None of the authors have any conflicts of interest to disclose.
Manuscript received November 22, 2005; revision accepted June 19, 2006.

Correspondence to: Daniel K. Ng, FHKAM(Paed), Department of Paediatrics, 25 Waterloo Rd, Hong Kong SAR; e-mail: dkkng@ha.org.hk

DOI: 10.1378/chest.130.6.1751

dose-response relationship, if any, between the severity of OSA and the degree of obesity in Chinese children, as ethnic origin may affect the epidemiology and pathogenesis of both OSA and obesity.[8] Other confounding factors including gender, age, and tonsil size were also studied.

Materials and Methods

A retrospective review was conducted of all children aged 1 to 15 years and undergoing a first successful polysomnography from August 1995 to May 2003 in the sleep laboratory of the pediatric department of Kwong Wah Hospital, a public nonteaching general hospital with 150 pediatric beds. Polysomnography was done for suspected OSA as judged by one of the authors (D.K.N., P.Y.C., K.L.K.) on clinical grounds that usually included habitual snoring or witnessed apnea. There were no fixed criteria. Demographic data including age, sex, body weight, and height were analyzed. Body mass index (BMI) and BMI z score (BMI Z score) were calculated. The AHI, as defined below, would be used as an index of the severity of OSA. Allergic rhinitis or asthma as documented in the medical record were also included for analysis. Tonsil size in children was graded as suggested by Brodsky.[9] The grading was summarized as follows: 0 = no enlargement; 1 = tonsils occupy less than half of the transverse diameter of oropharynx; 2 = tonsils occupy half of the transverse diameter of oropharynx; 3 = tonsils occupy more than half of the transverse diameter of oropharynx; 4 = tonsils occupy whole of the transverse diameter of oropharynx, *ie*, kissing tonsils.

Patients with craniofacial abnormalities, chromosomal disorders, chronic lung disorders, and neurologic or neuromuscular diseases were excluded. The protocol of this study was approved by the ethics committee of Kwong Wah Hospital.

Polysomnography

Overnight polysomnography was done according to the American Thoracic Society standards[10] (Alice 3; Respironics; Murrysville, PA; or Seista; Compumedics; Abbotsford, Australia). No sedation or sleep deprivation were used. The following parameters were recorded during the study: (1) EEG; (2) right and left electrooculogram; (3) submental and tibial electromyogram; (4) ECG; (5) oronasal airflow thermistor or cannula; (6) oxygen saturation monitoring (Healthdyne Oximeter model 930; Respironics); (7) chest and abdominal wall motion by computer-assisted respiratory inductance plethysmograph; and (8) snoring microphone. Sleep stages were determined according to Rechtschaffen and Kales criteria.[11] Arousals were defined as recommended by the American Academy of Sleep Medicine.[12] Obstructive apnea was defined as the cessation of airflow despite breathing effort for more than two respiratory cycles. Obstructive hypopnea was defined as the decrease of airflow by > 50% but < 80% of baseline associated with desaturation of ≥ 4% or arousal despite breathing effort. The AHI, denoting the number of episodes of obstructive apnea and hypopnea per hour, was calculated.

OSA was defined as an AHI > 5/h in adults,[13] and patients with AHI ≤ 5 were classified as primary snorers, *ie*, snorers with normal polysomnography results. In children, an apnea index > 1 was statistically abnormal,[14] and the upper limit of AHI was reported to be 1.5.[15] In the current study, AHI > 1.5 was used to define OSA.

Obesity

BMI was used to quantify the adiposity. In childhood, BMI is strongly influenced by gender and age and changes with age; an absolute cut-off value to define obesity in children of different ages is therefore not feasible. Although pediatric BMI percentile curves are available, their application in parametric statistical procedures is compromised by the skewed distribution. We transformed an individual child's BMI value to z score based on the gender and age-specific smoothed measure of skewness, median, and the coefficient of variation values of Chinese children[16] by using the equation suggested by Cole.[17] Obesity was defined as BMI Z score > 1.96 in our study, and 2.5% of normal children were expected to have such a BMI Z score.

Statistical Analysis

Data from patients were transferred to a computer for statistical analysis (SPSS 11.0, Macintosh Version; SPSS; Chicago, IL). Descriptive statistics were done. The distribution of data was assessed by Kolmogorov-Smirnov test with Liliefors significance correction. Data were logarithm transformed before the multivariate analysis if the data were not normally distributed. Patients were stratified into obese and nonobese groups to compare the AHI by Mann-Whitney *U* test. The prevalence of OSA, asthma, and allergic rhinitis between the two groups was compared by Fisher Exact Test. The association of prevalence of OSA and tonsil size was assessed by χ^2 test for trend.

Correlation between AHI and BMI Z score was assessed by Pearson correlation. A correlation coefficient > 0.1 was considered a positive correlation. Multiple linear regression with univariate analysis of covariance was done to identify predictors of AHI. BMI Z score, tonsil size, age, and gender were entered as independent variables. The percentage of variance of AHI independently explained by the predictors was derived from Eta^2 (partial R^2).

The null hypothesis of our study was absence of correlation between BMI Z score and AHI ($r \leq 0.1$). An alternative hypothesis is $r > 0.1$ with $\alpha = 0.05$ and $\beta = 0.8$. The minimum sample size required for our study is a total number of 85.

Results

A total of 705 polysomnography reports were reviewed. Two hundred twenty-three reports were excluded by the exclusion criteria: 21 reports were children with known underlying diseases, 192 with repeated polysomnography and 10 outside the age range of 1 to 15 years. Four hundred eighty-two children (median age, 6 years; interquartile range [IQR]4 to 9 years; range, 1 to 15 years) were included in this study. There were more boys (n = 335, 69.5%) than girls (n = 147, 30.5%). One hundred eleven children (23.0%) were obese (BMI Z score > 1.96). Table 1 summarized the demographic characteristics, and anthropometric and polysomnographic characteristics of the subjects as stratified by obesity.

The AHI in the obese group was significantly higher than the nonobese group. The odds ratio of obese children to have an AHI > 1.5 compared with nonobese children was 2.03 (95% confidence inter-

Table 1—*Demographic, Anthropometric, and Polysomnographic Characteristics of the Subjects as Stratified by Obesity*

Characteristics	Obese Group (n = 111)	Nonobese Group (n = 371)	p Value
Median age (IQR), yr	8 (5 to 10)	6 (4 to 9)	0.002
Male/female gender, No.	79/32	256/115	0.725
Children with allergic rhinitis, No. (% of group under discussion)	64 (57.7)	248 (66.8)	0.089
Children with asthma, No. (% of group under discussion)	29 (26.1)	96 (25.9)	1.000
Median body weight (IQR), kg	45.6 (32.0–55.3)	21.6 (16.6–31.0)	<0.001
Median body height (IQR), cm	129.0 (119–144.0)	118.4 (105.0–133.0)	<0.001
Median BMI (IQR)	24.53 (22.72–29.41)	16.05 (14.72–17.71)	<0.001
Median BMI Z score (IQR)	2.56 (2.23–2.96)	0.30 (−0.52–1.04)	<0.001
Tonsil size, No. (%)			0.249
Grade 0	5 (4.5)	10 (2.7)	
Grade 1	26 (23.4)	88 (23.7)	
Grade 2	46 (41.4)	176 (47.4)	
Grade 3	27 (24.3)	64 (22.1)	
Grade 4	7 (6.3)	15 (4.0)	
Median AHI (IQR)	1.5 (0.2–7.0)	0.7 (0.0–2.5)	0.005
AHI > 1.5, No. (%)	55 (49.5)	121 (32.6)	0.002

val, 1.32 to 3.12). There was no significant difference of tonsil size between obese and nonobese groups.

Significant difference in mean AHI was found in subjects with different tonsil size grading (one-way analysis of variance, $F = 19.202$; degrees of freedom = 4; $p < 0.001$) [Table 2]. Subjects with tonsil size graded as 4 had a significantly higher AHI than those with other tonsil grading. Moreover, a significant trend of prevalence of OSA was found in subjects with increasing tonsil size (χ^2 test for trend, $\chi^2 = 16.54$; degrees of freedom = 1; $p < 0.001$). As the distributions of AHI and age deviated significantly from normal distribution (one-sample Kolmogorov-Smirnov test for AHI and age, $p < 0.001$), they were logarithmic transformed in further bivariate/multivariate analysis as log-transformed AHI (Ln[AHI]) and log-transformed age (Ln[age]).

Significant correlation was found between BMI Z score and Ln(AHI) as illustrated in Figure 1 ($r = 0.156$, $p = 0.003$). BMI Z score, Ln(age), gender, and tonsil size were entered into a regression model with Ln(AHI) as the dependent variable (Table 3). BMI Z score ($p = 0.001$) and tonsil size ($p = 0.007$) were the only predictors of Ln(AHI), and 3.2% and 2.0% of variance of Ln(AHI) could be accounted for by BMI Z score and tonsils grading, respectively. No significant correlation was found between Ln(AHI) and Ln(age) [$r = -0.009$, $p = 0.863$].

Table 2—*Percentage of OSA in Different Tonsil Size Groups*

Tonsil Size	Children With AHI > 1.5/ Total No. of Children (%)	Mean AHI (SD)
Grade 0	4/15 (26.7)	6.82 (21.65)
Grade 1	33/114 (28.9)	1.51 (2.36)
Grade 2	74/222 (33.3)	3.04 (7.38)
Grade 3	48/109 (44.0)	4.56 (8.48)
Grade 4	17/22 (77.3)	19.27 (23.25)*

*AHI of children with tonsil size = 4 was significantly higher ($p < 0.001$) when compared with tonsil grades 0–3.

Discussion

In our study, we demonstrated a significant, though mild, correlation between OSA as measured by AHI and the degree of obesity as reflected by the BMI Z score. We also demonstrated that in children, tonsillar hypertrophy is an important risk factor for OSA. The pathogenesis of OSA in children included a complex interplay of adenotonsillar hypertrophy, neuromuscular tone, anatomy of the airway, and obesity.[8] Hence, it should not be surprising that obesity was found to have only a mild correlation with AHI in the current study.

Available literature on the correlation between severity of childhood sleep-disordered breathing (SDB) and degree of obesity was inconsistent. Marcus et al[4] demonstrated a significant correlation between percentage of ideal weight for height with apnea index in a group of obese children and adolescents. However, the studies by Mallory et al[6] and Silvestri et al[7] demonstrated no correlation. The inconsistency was possibly due to the fact that only obese children were studied in the above three studies. Brooks et al[5] addressed this issue by including obese children as well as nonobese counterpart, and a significant correlation between percentage of

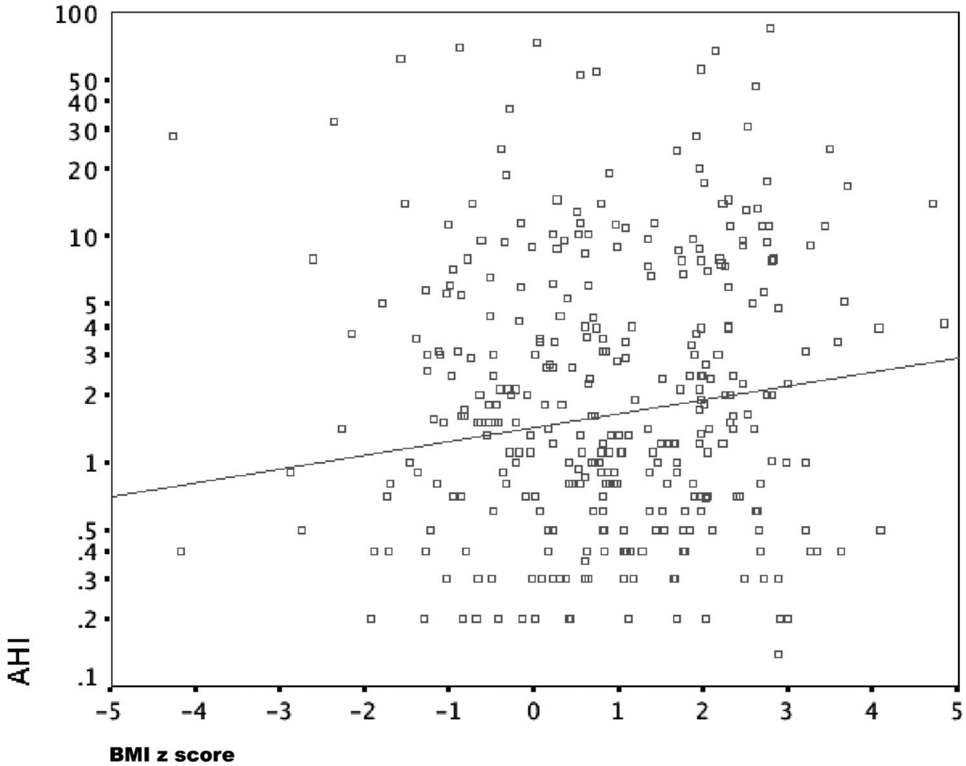

FIGURE 1. Correlation between AHI and BMI Z score (Y axis is in log scale).

ideal weight for height and respiratory disturbance index (or AHI) was found ($r = 0.49$, $p < 0.001$). The above four studies, however, were limited by the small sample size (< 50 patients each). The current series addressed the issue with a large sample size.

Another reason for the inconsistent result in previous studies could be the skewed distribution of variables, *eg*, body weight, apnea index, and AHI. The skewed distribution of these variables may violate the basic assumption of parametric analysis used in the studies, *ie*, Pearson correlation and multiple linear regression. This was addressed by the current study with the use of BMI Z score and Ln(AHI) to transform the skewed distribution to Gaussian distribution, satisfying the requirement for parametric analysis.

Compared with the current study, Brooks et al[5] demonstrated a much stronger positive correlation between AHI and percentage of ideal weight for height (percentage of ideal body weight) [$r = 0.49$, $p < 0.001$], compared with the correlation ($r = 0.156$, $p = 0.003$) between Ln(AHI) and BMI Z score in the current study. This was most likely due to the difference in the two studied populations. Our patients were older, a median age of 6 years vs mean age of 4.5 to 5 years in the study by Brooks et al,[5] in which the SD of percentage of ideal body weight for height was 12 in the low-AHI group, while the SD was 57.2 in the high-AHI group. In contrast, the BMI Z score distribution between non-OSA and OSA groups was similar in the current study (SD = 1.82 in the non-OSA group vs SD = 1.61 in the OSA group) [data not shown in results]. In addition, the discrepancy might be attributed to the fact that all the current study children were Chinese. Redline et al[18] reported ethnic difference in the prevalence of SDB, with African Americans being more prone to have SDB. A recent study by Kumar et al[19] reports that the distribution of fat in obese adults was found to be ethnic and gender dependent.

Table 3—*Regression Model With Ln(AHI) as the Dependent Variable**

Parameters	Regression Coefficient (SE)	p Value	Partial Eta^2, %
Intercept	0.258 (0.252)	0.306	0.3
Ln(age)	0.007 (0.060)	0.193	0.5
Male gender	0.111 (0.076)	0.145	0.6
BMI Z score	0.069 (0.021)	0.001	3.2
Tonsil size grade 4†	1.582 (0.587)	0.007	2.0
Tonsil size grade 3†	− 0.331 (0.518)	0.548	0.1
Tonsil size grade 2†	− 0.258 (0.502)	0.608	0.1
Tonsil size grade 1†	0.003 (0.587)	0.957	< 0.1

*Total R^2 of this regression model is 10.9%
†Compared with tonsil size grade 0.

Further studies should be undertaken to evaluate the role of ethnic difference in obesity and the pathogenesis of SDB.

The current study only demonstrated a mild though significant correlation between obesity and sleep apnea. This again illustrates that the pathogenesis of OSA in children is complex, with other factors playing important roles *eg*, the amount of fat around neck, and the size of tonsils and adenoid.[20] Horner et al[21] showed that more fat was present in the area surrounding the collapsible segment of the pharynx in adults with OSA, compared to equally obese control subjects without OSA. Mortimore et al[22] found that even relatively nonobese adults with OSA had excess fat deposition, especially anterolateral to the upper airway when compared with control subjects with the same level of obesity as defined by BMI and neck circumference. However, parapharyngeal fat as a contributing factor to OSA in obese children and adolescents has not been substantiated. Moreover, using BMI Z score as an index of obesity may not reflect the distribution of fat around the pharynx that determines the upper-airway patency during sleep. In children, measurements of neck and waist circumference were not a standard practice and no reference values were available. These measures would be helpful data in future studies of this area to confirm the role of parapharyngeal fat in childhood OSA.[20]

We evaluated the impact of tonsil size on the severity of OSA. Some of the previous studies[5,23–25] failed to establish a correlation between tonsil size by clinical inspection and different parameters of OSA. In other studies[5,26] using radiologic assessment of adenoidal-nasopharyngeal ratio, correlation with OSA indexes was also not established. Nevertheless, the study by Li et al[25] found a strong correlation between radiologic assessment of the tonsillar-pharyngeal ratio and the AHI in children with OSA syndrome (OSAS). Arens et al[27] used MRI to analyze upper-airway size of young children with OSAS (mean AHI, 8.4 ± 9.5) and matching control subjects. They found that children with OSAS had a significantly decreased cross-sectional area of airway adjacent to adenoid and tonsils. In the current study, we demonstrated that simple clinical staging of the tonsil size was correlated with the AHI; and previous negative study findings were most likely due to type II error, as the population of the current study was bigger than previous studies[5,23–25] that recruited from 22 to 50 children only.

Similar to previous pediatric studies,[4–7] the current study population was not recruited from the general population. All subjects were referred to our sleep laboratory for suspected OSA. This clinic population may be dissimilar to the general population, and the conclusion of this study may not be applicable to the general population. Further studies should also include subjects from the general population.

In conclusion, our study represents the largest series to date of children who underwent polysomnography. A positive dose-response correlation between AHI (a marker of OSA) and BMI Z score (a marker of obesity) is established. We also found that clinical staging of tonsil size was a useful measure to predict OSA. With the increased prevalence of obesity in children worldwide,[28] the increase in prevalence of associated morbidity like OSA should be anticipated. The combined effect of obesity and OSAS on the cardiovascular system is worrying,[29] and active education of both medical doctors and the general public is important to contain the impacts.

References

1 Ebbeling CB, Pawlak DB, Ludwig DS. Childhood obesity: public-health crisis, common sense cure. Lancet 2002; 360: 473–482

2 Wing YK, Hui HS, Pak WM, et al. A controlled study of sleep related disordered breathing in obese children. Arch Dis Child 2003; 88:1043–1047

3 Millman RP, Carlisle CC, McGarvey ST, et al. Body fat distribution and sleep apnea severity in women. Chest 1995; 107:362–366

4 Marcus CL, Curtis S, Koerner CB, et al. Evaluation of pulmonary function and polysomnography in obese children and adolescents. Pediatr Pulmonol 1996; 21:176–183

5 Brooks LJ, Stephens BM, Bacevice AM. Adenoid size is related to severity but not the number of episodes of obstructive apnea in children. J Pediatr 1998; 132:682–686

6 Mallory GB Jr, Fiser DH, Jackson R. Sleep-associated breathing disorders in morbidly obese children and adolescents. J Pediatr 1989; 115:892–897

7 Silvestri JM, Weese-Mayer DE, Bass MT, et al. Polysomnography in obese children with a history of sleep-associated breathing disorders. Pediatr Pulmonol 1993; 16:124–129

8 Schechter MS. Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome. Technical report: diagnosis and management of childhood obstructive sleep apnea syndrome. Pediatrics 2002; 109:e69

9 Brodsky L. Modern assessment of tonsil and adenoid. Pediatr Clin North Am 1989; 36:1551–1569

10 American Thoracic Society. Standards and indications for cardiopulmonary sleep studies in children. Am J Respir Crit Care Med 1996; 153:866–878

11 Rechtschaffen A, Kales A. A manual of standardized terminology: techniques and scoring systems for sleep stages of human subjects. Los Angeles, CA: UCLA Brain Information Service/Brain Research Institute, 1968; NIH publication No. 2040

12 EEG arousals, scoring rules and examples: a preliminary report from the Sleep Disorders Atlas Task Force. Sleep 1992; 15:173–184

13 American Academy of Sleep Medicine Task Force: sleep related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. Sleep 1999; 22:667–689

14 Marcus CL, Omlin KJ, Basinki DJ, et al. Normal polysomnographic values for children and adolescents. Am Rev Respir Dis 1992; 146:1235–1239

15 Witman MB, Keens TG, Davidson Ward SL, et al. Obstructive hyponoeas in children and adolescents: normal values [letter]. Am J Respir Crit Care Med 2003; 168:1540
16 Leung SS, Cole TJ, Tse LY, et al. Body mass index curves for Chinese children. Ann Hum Biol 1998; 25:169–174
17 Cole TJ. The LMS method for constructing normalized growth standards. Eur J Clin Nutr 1990; 44:45–60
18 Redline S, Tishler PV, Schluchter M, et al. Risk factors for sleep-disordered breathing in children: associations with obesity, race, and respiratory problems. Am J Respir Crit Care Med 1999; 159:1527–1532
19 Kumar BN, Meyer HE, Wandel M, et al. Ethnic differences in obesity among immigrants from developing countries, in Oslo, Norway. Int J Obes (Lond) 2006; 30:684–690
20 Schafer H, Pauleit D, Sudhop T, et al. Body fat distribution, serum leptin, and cardiovascular risk factors in men with obstructive sleep apnea. Chest 2002; 122:829–839
21 Horner RL, Mohiaddin RH, Lowell DG, et al. Sites and sizes of fat deposits around the pharynx in obese patients with obstructive sleep apnoea and weight matched controls. Eur Respir J 1989; 2:613–622
22 Mortimore IL, Marshall I, Wraith PK, et al. Neck and total body fat deposition in nonobese and obese patients with sleep apnea compared with that in control subjects. Am J Respir Crit Care Med 1998; 157:280–283
23 Ahlquist-Rastad J, Hultcrant Z, Svanholm H. Children with tonsillar obstruction: indications for and efficacy of tonsillectomy. Acta Paediatr Scand 1988; 77:831–835
24 Croft CB, Brockbank MJ, Wright A, et al. Obstructive sleep apnoea in children undergoing routine tonsillectomy and adenoidectomy. Clin Otolaryngol Allied Sci 1990; 15:307–314
25 Li AM, Wong E, Kew J, et al. Use of tonsil size in the evaluation of obstructive sleep apnoea. Arch Dis child 2002; 87:156–159
26 Chay OM, Goh A, Abisheganaden J, et al. Obstructive sleep apnea syndrome in Singapore children. Pediatr Pulmonol 2000; 29:284–290
27 Arens R, McDonough JM, Corbin AM, et al. Upper airway size analysis by magnetic resonance imaging of children with obstructive sleep apnea syndrome. Am J Respir Crit Care Med 2003; 167:65–70
28 Deckelbaum RJ, Williams CL. Childhood obesity: the health issue. Obes Res 2001; 9(suppl 4):239S–243S
29 Wilcox I, McNamara SG, Collins FL, et al. "Syndrome Z": the interaction of sleep apnoea, vascular risk factors and heart disease. Thorax 1998; 53(suppl 3):S25–S28