Twenty-Four–Hour Ambulatory BP in Snoring Children With Obstructive Sleep Apnea Syndrome*

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Introduction: **Obstructive sleep apnea syndrome (OSAS) is a known risk factor for hypertension in adults. This relationship is less clear in childhood OSAS.**
Objective: **This study examined the relationship between OSAS and 24-h ambulatory BP (ABP), a more accurate assessment than casual BP, in children with snoring.**
Methods: **Snoring children aged 6 to 15 years who underwent polysomnography in the sleep laboratory were recruited.**
Measurement: **Twenty-four–hour ABP monitoring was initiated a few hours before polysomnography. The children were classified into two groups: a high apnea-hypopnea index (AHI) group (obstructive AHI > 5/h), and a low-AHI group (AHI ≤ 5/h). Mean sleep, wake, and 24-h systolic BP (SBP) and diastolic BP (DBP) were recorded. A child was considered a "nondipper" if his or her mean SBP and DBP did not decrease by ≥ 10% during sleep.**
Results: **Ninety-six children (mean age ± SD, 9.4 ± 2.8 years) were recruited. Forty-one children were obese. When awake, the high-AHI group children had a significantly higher SBP. When asleep, both SBP and DBP were higher in the high-AHI group. Age, body mass index (BMI) z score, and desaturation index (DI) were significant predictors for elevated sleep DBP. BMI z score was the only significant predictor for wake and sleep SBP. Sixteen children (17%) had hypertension, and all were nondippers. Obese children in the high-AHI group had a significantly higher prevalence of hypertension than obese children in the low-AHI group. This relationship was not found in nonobese children.**
Conclusion: **The current study shows that increased DI contributed to the elevation of sleep DBP elevation.** ***(CHEST 2006; 130:1009–1017)***

Key words: BP monitoring, ambulatory; child; hypertension; sleep apnea, obstructive

Abbreviations: ABP = ambulatory BP; AHI = apnea-hyponea index; BMI = body mass index; BPI = BP index; CI = confidence interval; DBP = diastolic BP; DI = desaturation index; OR = odds ratio; OSAS = obstructive sleep apnea syndrome; SBP = systolic BP

There is now strong epidemiologic evidence that obstructive sleep apnea syndrome (OSAS) is an independent risk factor for hypertension in adults, including essential hypertension.[1] A number of adult cross-sectional and prospective population studies[2,3] demonstrated a modest but definite association between OSAS and hypertension, independent of confounding factors. The prevalence for childhood OSAS was estimated to affect 1 to 3% of preschool children.[4] As childhood OSAS differs from adult in its definition, etiologies, clinical manifestations, and sequelae, the association between OSAS and hypertension observed in adults may not apply to children.[5]

A review of the literature yielded four studies[6–9] of BP in children with OSAS. Their results were inconsistent, ranging from only subtle BP regulation abnormalities[6] to a higher sleep diastolic BP (DBP).[8] Ambulatory BP (ABP) monitoring by taking multiple readings was shown to be more reproducible and more accurate than casual BP.[10–12] Studies[13,14] of age- and distribution-adjusted pediatric ABP normal

values allow a more precise definition of hypertension in children. In this study, we aimed to study the difference, if any, in ABP parameters and prevalence of hypertension, as defined by ABP, between snoring children with a high and low apnea-hypopnea index (AHI), and to identify significant risk factors for the difference. We hypothesized that children with a higher AHI should have a higher BP compared to their low-AHI counterparts. Moreover, the number of apneic/hypopneic episodes during sleep should be a predictor of BP, independent of the effect of obesity.

Materials and Methods

Subjects

All children aged 6 to 15 years with symptoms of sleep-disordered breathing (snoring with or without witnessed apnea or excessive daytime sleepiness) and who underwent polysomnography in our pediatric sleep laboratory from March 1999 to September 2002 were recruited. The lower age limit was chosen, as normative ABP data are only available for children ≥ 6 years old.[13,14] A detailed history with special emphasis on sleep-related symptoms was obtained, followed by physical examination and urinalysis. Urinalysis was performed to exclude children with secondary hypertension. Children with cardiac, renal, chromosomal, and neuromuscular diseases were excluded. Children receiving medications that could affect BP were also excluded. Body mass index (BMI) was converted into a BMI z score according to the normal values of Hong Kong Chinese children.[15] Children were considered obese if their BMI z score was > 1.96 (corresponding to 95th percentile). Informed consent was obtained from the parents or legal guardian of each child, and assent was obtained from children > 11 years old. The Institutional Review Board of Kwong Wah Hospital approved the study.

Polysomnography

Overnight polysomnography was done according to American Thoracic Society standards[16] using the Alice 3 system (Respironics; Murrysville, PA). No sedation was used. Polysomnographic channels used are listed in Appendix 1. Sleep stages were determined according to Rechtschaffen and Kales.[17] Arousals were defined as recommended by the American Academy of Sleep Medicine.[18] For the polysomnographic parameters measured and their definitions, see Appendix 1. For our current study, we arbitrarily classified the subjects into two groups: a high-AHI group (AHI > 5/h) and a low-AHI group (AHI ≤ 5/h).

Twenty-Four–Hour ABP

ABP was measured by an oscillometric monitor (Spacelabs 90207; Spacelabs Medical; Redmond, WA) that was validated for children.[14,19,20] The child was admitted to the ward in the afternoon of the polysomnographic appointment, when the ABP monitor was attached to the nondominant arm using an appropriate-sized cuff. The monitor was programmed to measure BP every 15 min during the day and every 30 min during the night as previously described.[21] Sleep period was set as the time from sleep onset defined by EEG to sustained wakefulness. ABP was measured for 24 h. The ABP recording was considered acceptable when a minimum of one reading per hour for a minimum of 20 h (including six readings during sleep) was available. For the ABP parameters measured and their definitions, see Appendix 2. Systolic BP (SBP) and DBP indices (BP index [BPI]) for the wake, sleep, and entire 24-h period were used to take into account the fact that normal BP levels vary with age and height. We normalized this variation by deriving a BPI as suggested by Sorof et al.[22] This was summarized by the following formula:

$$\text{BPI} = \text{measured BP}/95\text{th percentile BP}$$

Thus, a BPI > 1.1 indicates a BP value that is 10% greater than the 95th percentile ABP, according to German norms published by Wuhl et al[13] and Soergel et al,[13,14] indicating the relative BP level compared to children of similar height. Hypertension was defined when the mean wake or sleep or 24-h systolic and/or diastolic ABPs were greater than the 95th percentile of ABP norms of Soergel et al.[14]

Statistical Analysis

A previous study[6] found that the mean difference in sleep DBP between subjects with or without OSAS was 8 ± 8 mm Hg. From this calculation, the required sample size for the current study was 17 for each group in order to have a power of 80%. Statistical software (SPSS 11.0 for Macintosh; SPSS; Chicago, IL) was used for statistical analysis. All results were expressed as mean ± SD. Demographic, polysomnographic, and ABP differences between the high-ABP and low-AHI groups were compared using the unpaired *t* test for continuous variables and Fisher exact test for categorical variables. To identify independent variables that might predict BP, we applied multiple linear regression models to the wake and sleep SBP and DBP parameters. Variables were initially screened for correlation with ABP parameters by Pearson correlation. Those with significant correlation were entered into a multiple linear regression model to evaluate for their independent effect. Log transformation was done for continuous variables that were not of normal distribution to reduce the influence of outlying data.

Secondly, Fisher exact test was used to compare the prevalence of hypertension between the two groups. Logistic regression was used to determine the relative contributions of different variables to predict hypertension. Finally, a subset analysis was conducted to evaluate the association of hypertension and AHI in obese children; $p < 0.05$ were considered statistically significant.

Results

Subject Characteristics

One hundred twenty-two children underwent polysomnography. Sixteen children were excluded

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because they had neuromuscular or chromosomal disorders, and 106 eligible children were recruited. All children completed the polysomnography and ABP recordings. Nine children were excluded because their ABP readings did not satisfy our criteria of success, and one child was excluded because the child did not sleep well during polysomnography (sleep efficiency, 47%). Of the remaining 96 children, mean age was 9.4 ± 2.8 years and male/female ratio was 3.2:1. This was consistent with the male preponderance of children undergoing polysomnography in our unit.[23] Forty-one children (43%) were obese, and 55 were not obese. Characteristics of the 96 children were shown on Table 1.

There were no significant differences in gender, age, height, and family history of hypertension between the two groups. However, the high-AHI group had significantly higher BMI z score than the low-AHI group (p = 0.009). This is expected because obesity is a known risk factor for OSAS.[24] Among the obese children in our cohort, 9 of 22 children (40.1%) belonged to the high-AHI group, whereas 8 of 74 nonobese children (10.8%) belonged to the high-AHI group. Hence, obese children were significantly more likely to have a higher AHI than nonobese children (odds ratio [OR], 5.71; 95% confidence interval [CI], 1.86 to 17.6; p = 0.003). Compared to the normal values,[25] sleep efficiency was slightly lower in our subjects, approximately 80%. Nonetheless, the mean arousal index and sleep efficiency were similar between the high-AHI and low-AHI groups.

Difference in ABP Parameters in High-AHI and Low-AHI Groups

While awake, the high-AHI group had significantly higher SBP index than the low-AHI group (Table 2). While asleep, both SBP and DBP indexes were significantly higher for the high-AHI group. The proportion of nondippers was similar for both groups.

Relationship Between ABP Parameters and Polysomnographic Variables

In all 96 studied children, ABP parameters were correlated with log AHI, log desaturation index (DI), arousal index, BMI z score, and age by Pearson correlation. These variables were entered into a multiple linear regression model (Table 3).

Gender was forced into this model. BMI z score was found to be the significant positive predictor for wake SBP index, sleep SBP index, and sleep DBP index, accounting for 9.9%, 13.0%, and 8.2% of variance of BP, respectively. For sleep DBP index, age and DI were the other significant predictors, accounting for 14.6% and 11.4% of variance, respectively.

OSAS and Hypertension

Sixteen of the 96 children (16.7%) studied were found to have systolic or diastolic hypertension during sleep or wake periods. The high-AHI group had a higher prevalence of hypertension than the low-AHI group (p = 0.0071; OR, 3.20; 95% CI, 1.04 to 9.83). The pattern of hypertension was listed in Table 4.

Of the 16 children with hypertension, 11 children had nocturnal hypertension only and the rest had hypertension during the entire 24-h period. None had daytime hypertension only. Six of these 16 children had systolic hypertension only, 5 children had diastolic hypertension only, and 5 children had both systolic and diastolic hypertension. All of these

Table 1—*Demographic, Anthropometric, and Polysomnographic Data of 96 Children**

Variables	High-AHI Group (AHI > 5/h; n = 17)	Low-AHI Group (AHI ≤ 5/h; n = 79)	p Value
Age, yr	9.7 ± 3.3	9.3 ± 2.7	0.60
Male gender	13 (76.5)	53 (76.0)	1.0
Height, cm	138.1 ± 16.0	135.9 ± 15.1	0.58
BMI, kg/m^2	24.14 ± 7.70	19.09 ± 4.72	0.018†
BMI z score	1.78 ± 0.93	0.75 ± 1.23	0.002†
Family history of hypertension	2 (11.8)	6 (7.6)	0.63
AHI, /h	23.1 ± 23.4	1.11 ± 1.22	0.001†
DI, /h	16.4 ± 17.8	1.86 ± 2.54	0.004†
Spo_2 nadir	73.9 ± 14.1	82.5 ± 22.7	0.14
TST90%	8.80 ± 9.86	0.924 ± 2.20	0.005†
Arousal index, /h	27.0 ± 16.3	15.8 ± 12.0	0.62
Sleep efficiency, %	82.8 ± 8.3	78.8 ± 10.7	0.151

*Data are presented as mean ± SD or No. of patients (%). Spo_2 = oxygen saturation by pulse oximetry; TST90% = total sleep time with arterial oxygen saturation < 90%.

†Significantly higher in the high-AHI group.

Table 2—*Twenty-Four–Hour Ambulatory SBP, DBP, and Number of Nondippers in 96 Children

Variables	High-AHI Group (AHI > 5/h; n = 17)	Low-AHI Group (AHI ≤ 5/h; n = 79)	p Value
Wake SBP index	0.90 ± 0.06	0.85 ± 0.07	0.015†
Wake SBP load, %	10.7 ± 15.1	6.0 ± 12.7	0.18
Wake DBP index	0.83 ± 0.05	0.81 ± 0.07	0.17
Wake DBP load, %	6.3 ± 10.6	7.1 ± 7.1	0.76
Sleep SBP index	0.98 ± 0.07	0.91 ± 0.07	< 0.001†
Sleep SBP load, %	37.7 ± 33.0	13.0 ± 20.6	0.008†
Sleep DBP index	0.95 ± 0.10	0.91 ± 0.08	0.044†
Sleep DBP load, %	30.6 ± 26.1	18.4 ± 20.2	0.034†
SBP nondipper	14 (82)	57 (72)	0.54
DBP nondipper	5 (29)	32 (40)	0.58

*Data are presented as mean ± SD or No. of patients (%).
†Significantly higher in the high-AHI group.

hypertensive children were nondippers or reverse dippers (sleep BP higher than wake BP). Logistic regression analysis failed to demonstrate any significant predictor of hypertension among these 96 children, although log DI approached significance (p = 0.056) [Table 1 of on-line Supplemental items].

Relationship Between OSAS and Hypertension in Obese Children

The distribution of hypertension in children with obesity and/or OSAS was shown in Figure 1. We conducted a subset analysis to evaluate the effect of OSAS and hypertension in obese children. Among obese children, the overall prevalence of hypertension was 9 of 22 children (40.1%). Of obese children in the high-AHI group, six children (66.7%) were hypertensive. Of obese children in the low-AHI group, three children (23.1%) were hypertensive. Hence, obese children in the high-AHI group had a higher risk of hypertension than obese children in the low-AHI group (OR, 6.67; 95% CI, 1.04 to 44.29). A similar risk was not demonstrated in nonobese children. Logistic regression analysis of data for the obese children could not identify any significant risk factor for nocturnal hypertension (Table 2 of on-line Supplemental items).

Discussion

This study demonstrated that snoring children with AHI > 5/h from a sleep laboratory cohort had higher 24-h SBP and sleep DBP than those with a lower AHI; however, mean BPs in children with AHI > 5/h did not exceed the 95th percentile of normal. DI independently predicted sleep DBP elevation. Obese children with an AHI > 5/h had a significantly higher risk of hypertension than obese children with a lower AHI. This suggested that OSAS *per se* may be related to higher nocturnal DBP, and that the impact of OSAS on BP may be higher in obese children. The high prevalence of nondippers in the current population suggests that there may be BP dysregulation in snoring children irrespective of AHI.

Recently, ABP monitoring has increasingly replaced casual BP in diagnosing hypertension. By taking multiple readings throughout the 24-h period, ABP monitoring was found to be more reproducible, more accurate, and of better prognostic value than casual BP.[10–12] Not only could ABP monitoring identify subtle diurnal abnormalities such as nocturnal hypertension and "nondipping" status,[26,27] it could also diagnose "true hypertension" more accu-

Table 3—*Predictors of ABP in 96 Children*

Predictors	Wake SBP		Wake DBP		Sleep SBP		Sleep DBP	
	β Coefficient	p Value	β Coefficient	p Value	β Coefficient	p Value	β Coefficient	p Value
Log AHI	2.313	0.300	1.619	0.323	− 0.053	0.981	− 1.575	0.315
Log arousal index	− 2.169	0.495	− 0.708	0.762	− 1.773	0.581	2.233	0.319
Log DI	− 0.692	0.511	− 0.237	0.759	1.243	0.245	1.922	0.012*
Age	0.203	0.618	− 0.158	0.598	− 0.508	0.220	− 0.855	0.004*
BMI z score	2.431	0.020*	0.729	0.331	2.877	0.007*	1.544	0.034*
Male gender	0.117	0.963	0.486	0.794	− 0.400	0.876	1.393	0.434
Intercept	− 18.396	< 0.001*	− 13.734	< 0.001*	− 4.311	0.392	− 3.453	0.325

*Statistically significant predictor of ABP parameters.

Table 4—*Patterns of Hypertension in Children With High AHI and Low AHI

Variables	High-AHI Group (AHI > 5/h; n = 17)		Low-AHI Group (AHI ≤ 5/h; n = 79)	
	24-h Hypertension	Nocturnal Hypertension	24-h Hypertension	Nocturnal Hypertension
Systolic	1	2	2	1
Diastolic	1	0	0	4
Systolic and diastolic	0	3	1	1
Total hypertension	2	5	3	6
Dipping status				
Nondipper	4		9	
Reverse	3		0	
Dipper	0		0	

*Data are presented as No. of patients. The high-AHI group had a higher incidence of hypertension than the low-AHI group (p = 0.0071; OR, 3.20; 95% CI, 1.04 to 9.83).

rately because it eliminated "white coat hypertension," which existed in up to 30% of children.[28] Thus, ABP should be a better tool to assess the specific nature of hypertension of OSAS.[29,30]

Studies by Marcus et al[6] and Kohyama et al[7] only measured serial BP during overnight polysomnography and found a significant correlation between AHI and BP. Enright et al[9] studied 239 children from the community and found obesity, sleep efficiency, and respiratory disturbance index as factors independently associated with casual SBP and DBP elevation. However, the accuracy of these unattended home studies remains to be determined, and their definition of hypopnea using 2% drop in pulse oximetry is not widely used in children. Moreover, their definition of elevated BP using readings from one sitting might not be representative.

A recent study by Amin et al[8] studied 24-h ABP in 60 snoring children and found significantly greater mean BP variability and smaller nocturnal BP dipping in OSAS subjects. Contrary to previous reports,[6,7,9] they found lower DBP during wake in the group with OSAS. They concluded that children with OSAS showed evidence of dysregulation of systemic BP. However, they used the casual BP norms for assessment of ABP values. In children, contrary to adults, the 95th percentile for mean systolic and diastolic ABP was shown to be significantly higher than the corresponding 95th percentile casual BP.[14,22] Using casual BP norms to interpret ABP

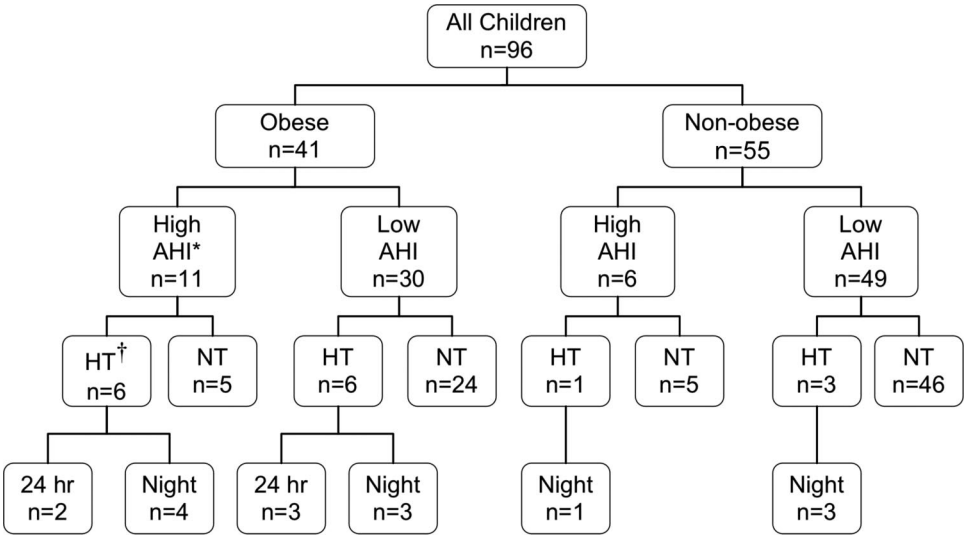

FIGURE 1. Relationship between OSAS and hypertension in 96 subjects completing 24-h ABP recording. HT = hypertension; NT = normotension; 24 hr = 24-h hypertension; Night = nocturnal hypertension.*The proportion of obese children in the high-AHI group was significantly higher than nonobese children (p = 0.0003; OR, 5.712; 95% CI, 1.858 to 17.554). †Among obese children, those in the high-AHI group had a higher incidence of hypertension than the low-AHI group (OR, 6.667; 95% CI, 1.004 to 44.284).

would overdiagnose daytime hypertension and underdiagnose nighttime hypertension.[14,22] In our study, we used the recently published distribution-adjusted pediatric ABP norms,[14] allowing more accurate ABP data interpretation. This may be the reason for the different findings between the current one and the study by Amin et al[8]: higher sleep DBP in children with AHI > 5/h in the current study vs lower sleep DBP in the study by Amin et al.

The predictors for sleep DBP in the current study were age, BMI z score, and DI, the latter being strongly correlated with AHI. The finding of these three predictors had two implications. Firstly, our finding of DI as an independent predictor for elevated sleep DBP is a novel finding. ABP studies in children demonstrated isolated systolic hypertension to be the typical pattern in obesity-related primary hypertension.[31,32] The elevated nocturnal diastolic ABP found in our cohort was, hence, unlikely to be primary hypertension, and we suggested the most likely explanation to be desaturation associated with obstructive sleep apnea, leading to sympathetic overdischarge resulting in hypertension during sleep.[33]

Secondly, similar to previous studies,[6–8] age was found to be a potent predictor of sleep DBP in the current study. It was suggested that an age- and gender-adjusted 24-h ABP indexing algorithm could be used to negate the age effect on ABP parameters.[13] We reanalyzed our data using this algorithm to index ABP parameters. The age effect on BP was lessened, but it still emerged as a significant predictor of sleep DBP. This suggested age to be a true predictor of sleep DBP. A recent study by Kaditis et al[34] shed some light on this issue; they found that age, gender, and BMI were the significant predictors of wake SBP in a heterogeneous group of habitual and nonhabitual snorers, while age was not a predictor of wake DBP. Sleep BP was not measured in that study.[34] Further studies need to be conducted to address this age-related increase in BP.

There may be a number of reasons why AHI was not identified as an independent factor in BP elevation. AHI may not be the ideal measure for sleep-disordered breathing in children. Rosen et al[35] observed that traditional criteria used for scoring obstructive apneas in adults often failed to identify significant obstructive events in children because children often did not have complete obstruction of the airway and were more likely to have intermittent or even sustained obstructive hypopnea instead of apnea.[36] The term *obstructive hypoventilation* has been used to describe the pattern of increased end-tidal carbon dioxide. Our lack of end-tidal carbon dioxide monitoring would lead to underestimation of the severity of sleep-disordered breathing. Indeed, subtle types of sleep-disordered breathing other than obstructive sleep apnea may lead to higher BP. Guilleminault et al[37] found repetitive increases in SBP and DBP in patients with upper airway resistance syndrome. Casual BP was noted to be higher even in primary snoring children (snorers with a normal AHI) than control subjects.[38]

The other reason for the absence of an association between AHI and BP elevation may be inadequate sample size. Adult population-based studies involving a much larger population suggested a causal, albeit not strong association between OSAS and hypertension. In a cross-sectional study of 6,132 men and women, Nieto et al[3] found that the adjusted OR of hypertension in severe OSAS category (AHI > 30/h) was 1.37 only. The prospective longitudinal analysis from the Wisconsin Sleep Cohort[2] followed up 709 participants over 4 years and found an adjusted OR of 2.9 (95% CI, 1.5 to 5.6) in new development of hypertension for an AHI of 15/h vs an AHI of zero. Using our results as the basis to calculate the required sample size (80% power and type 1 error of 0.05), a larger study involving at least 44 children with AHI > 5/h and the same number of children with AHI ≤5/h will be required.

In the current study, we defined hypertension using ABP, the most stringent criteria for defining hypertension because it eliminated white coat hypertension. Previous studies[6–9] did not demonstrate a higher prevalence of systemic hypertension in OSAS children. Our current study was the first to find a higher prevalence of hypertension, as defined by ABP norms, in obese OSAS children. The prevalence of hypertension, as defined by casual BP, was reported to be 5 to 22% in obese children.[39] However, the prevalence of hypertension as defined by ABP in obese children was not known though it might well be lower in the absence of white coat hypertension in ABP. In our group of obese OSAS children, the prevalence of daytime hypertension was 11.1% (one of nine children), which was similar to that of previous reports.[6–9] However, the prevalence rose to 54.5% (six of nine children) if nocturnal hypertension was included.

As to the type of hypertension, large screening studies[39,40] in school children reported a threefold higher prevalence of systolic than diastolic hypertension. However, in our study, diastolic hypertension was as common as systolic hypertension. In an ABP monitoring study trying to differentiate children with secondary hypertension from primary hypertension, Flynn[32] found that a pattern of daytime DBP elevation, and/or nighttime SBP and DBP elevation was more indicative of secondary hypertension. This suggested that the diastolic hypertension pattern found in the current study was likely to be of secondary cause, the most likely being OSAS.

Another important finding was that all our hypertensive children were nondippers and three were reverse dippers. Not only was the nondipping status found to be associated with secondary hypertension in adults[41] and children,[42] it was also shown to predict end-organ damage.[12,26] Hence, long-term follow-up is required for our hypertensive children to allow for early detection of end-organ damage.

There were deficient areas in the current study. The first was use of thermistor. Thermistors was reported to have suboptimal sensitivity in detecting apneas and hyponeas compared to nasal cannulae in children.[43] Future studies should employ nasal cannulae to detect airflow for more accurate quantification of the severity of sleep apnea. The second deficiency in the current study was selection bias. All recruited children were patients referred to our sleep laboratory for investigation of suspected OSAS. This highly biased population limits the generalization of our findings to normal children, as those with AHI <1/h in our series were all snorers. Guilliminault and Lee[44] commented that primary snoring could never be regarded as "benign," and our previous study[38] also showed that primary snorers had a higher BP than nonsnorers. Further studies should enroll nonsnoring children as the control group.

We used a standard ABP sampling frequency protocol to study our children.[21] Marrone et al[29] suggested that more frequent BP measurements during ABP were required for adults with OSAS due to the increased variability of BP in this group compared to the normal population. However, high BP sampling rate at night might cause sleep disruption.[30] Thus, we selected to use a less frequent sampling rate (30 min) than previous study (15 min).[8] Even though we minimized the possible sleep disruption by reducing the sampling rate, the mean arousal index in our subjects was quite high while sleep efficiency was lower, compared to normal subjects without overnight BP monitoring.[25] Arousal from sleep is a potent trigger for sympathetic activity and sleep disruption caused by ABP monitoring during overnight polysomnography may lead to higher BP measurement. However, this disruption should affect both the low-AHI and high-AHI groups and should not affect comparison between the two groups. Furthermore, the mean arousal index in the current study was similar to that of study by Amin et al[8] (low-AHI group, 12 ± 9/h vs 15.8 ± 12.0/h, $p = 0.183$; high-AHI group, 28 ± 27/h vs 27.0 ± 16.3/h, $p = 0.895$). Amin et al[8] also did a subanalysis to assess the effect of BP recording on arousals by measuring arousals within 30 s of BP measurement, and found that > 80% of BP measurements did not affect sleep and only a very small number of measurements led to full awakening.

We also suspected that the effect of obesity on BP may not be completely determined despite adjust-

Appendix 1

Polysomnographic Channels, Parameters Measured, and Definitions*

Polysomnographic Parameters	Definitions
Obstructive apnea	Cessation of airflow with respiratory effort for more than two respiratory cycles
Hypopnea	Decrease of airflow by > 50% but ≤ 80% of baseline associated with desaturation ≥ 4% or arousal despite breathing effort
AHI	No. of episodes of obstructive apnea and hypopnea per hour of sleep
SaO_2 nadir	Lowest arterial oxygen saturation recorded with good pulse wave signal
TST%90	Percentage of total sleep time with arterial oxygen saturation < 90%
DI	No. of episodes per hour of sleep with arterial oxygen saturation decreased by ≥ 4% from baseline
Arousal index	No. of EEG arousals per hour of sleep.

*The following channels were used: EEG; electro-oculography; submental and tibial electromyography; ECG; oronasal airflow thermistor; oxygen saturation monitoring with pulse-wave signal (Healthdyne Oximeter; Respironics); chest and abdominal wall motion by computer-assisted respiratory inductance plethysmography; and snoring microphone.

Appendix 2

ABP Parameters Measured and Definitions

Parameters	Definitions
Wake/sleep systolic/diastolic mean BPs	Average systolic and diastolic and mean BP for wake, sleep, and the entire 24-h periods.
SBP and DPB load	Percentage of each child's BP values that exceeded the pediatric ambulatory 95th percentile BP specific for that child's gender and height.
Nocturnal SBP and DBP dipping (BP load)	Calculated by subtracting the average sleep BP from the average wake BP and dividing the difference by the average wake BP. A child was considered a nondipper if by convention his or her mean SBP and DBP did not decrease by 10% during sleep.
Wake/sleep SBP/DPB index (BPI)	Calculated by the following formula: (measured mean BP)/95th percentile BP.

ment by multivariate analysis. Future studies comparing population matched for age, gender, and BMI are warranted.

In conclusion, children with an AHI > 5/h had higher wake SBP, sleep SBP, and sleep DBP. Increased DI contributed significantly to the elevation of sleep DBP elevation.

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