# Medicine®

## OBSERVATIONAL STUDY

# Analysis of 24-Hour Ambulatory Blood Pressure Monitoring in Children With Obstructive Sleep Apnea

## A Hospital-Based Study

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**Abstract:** In the present study, we aimed to verify associations between ambulatory blood pressure (ABP) and pediatric obstructive sleep apnea (OSA) in a hospital-based population.

This was a cross-sectional observational study on children aged 4 to 16 years with OSA-related symptoms from a tertiary referral medical center. All children received overnight polysomnography and 24-hour recording of ABP. Severity of the disease was classified as primary snoring (apnea-hypopnea index, AHI <1), mild OSA (AHI 1−5), and moderate-to-severe OSA (AHI >5).

For 195 children enrolled in this study (mean age, $7.8 \pm 3.4$ years; 69% boy), ABP increased as severity of OSA increased. During daytime, children with moderate-to-severe OSA had significantly higher systolic blood pressure (BP) ($117.0 \pm 12.7$ vs $110.5 \pm 9.3$ mmHg), mean arterial pressure (MAP) ($85.6 \pm 8.1$ vs $81.6 \pm 6.8$ mmHg), and diastolic BP load ($12.0 \pm 9.6$ vs $8.4 \pm 10.9$ mmHg) compared with children with primary snoring. During nighttime, children with moderate-to-severe OSA had significantly higher systolic BP ($108.6 \pm 15.0$ vs $100.0 \pm 9.4$ mmHg), MAP ($75.9 \pm 9.6$ vs $71.1 \pm 7.0$ mmHg), systolic BP load ($44.0 \pm 32.6$ vs $26.8 \pm 24.5$ mmHg), systolic BP index ($0.5 \pm 13.1$ vs $-6.8 \pm 8.5$ mmHg), and higher prevalence of systolic hypertension (47.6% vs 14.7 %) compared with children with primary snoring. Multiple linear regression analyses revealed an independent association between AHI and nighttime systolic BP and MAP after adjusting for adiposity variables.

This large hospital-based study showed that children with moderate-to-severe OSA had a higher ABP compared with children who were primary snorers. As elevated BP in childhood predicts future cardiovascular risks, children with severe OSA should be treated properly to prevent further adverse cardiovascular outcomes.

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**Abbreviations**: ABP = ambulatory blood pressure, AHI = apnea-hypopnea index, BMI = body mass index, BP = blood pressure, MAP = mean arterial pressure, OSA = obstructive sleep apnea, PSG = polysomnography.

## INTRODUCTION

Sleep-disordered breathing includes a spectrum of upper airway disorders ranging from primary snoring to obstructive sleep apnea (OSA).[1,2] In adults, untreated OSA is associated with hypertension[3,4] and other cardiovascular morbidities.[5,6] In children, Guilleminault et al[7] first described high blood pressure (BP) with OSA in 1976. Since then, several studies have linked OSA with BP in a pediatric population.[7−12] Although some studies have reported a trend of elevated BP in children with OSA,[7−12] a recent meta-analysis by Zintzaras et al[13] reported that evidence of an association between moderate-to-severe childhood OSA and hypertension is insufficient. As the literature show inconsistent results regarding associations between BP and pediatric OSA, further studies are needed to clarify this clinical relevant issue.

Ambulatory blood pressure (ABP) monitoring is a standard diagnostic tool for BP measurements because of high reliability and reproducibility. Monitoring ABP provides an estimate of mean BP level, the diurnal rhythm of BP, and BP variability. Several studies have shown that ABP monitoring, when compared with clinic casual BP measurements, is superior in predicting target organ damage, morbid events, or cardiovascular risk.[14] However, few studies have used ABP monitoring to elucidate associations between BP and OSA in children.[8−12] Furthermore, obesity increases risk of pediatric OSA.[2,15] Obesity in children can also cause hypertension, which ultimately increases cardiovascular risks.[16,17] Previous studies have used a small sample size and have not considered the independent role of childhood OSA on BP.[8−12,18]

The aims of this study were to compare ABP level in children with OSA of varying severity (ie, primary snoring, mild, moderate-to-severe OSA) and to investigate the association between childhood OSA and BP parameters.

## MATERIALS AND METHODS

### Study Population

Children aged 4 to 16 years with symptoms suggestive of OSA, including snoring, excessive daytime sleepiness, or breathing pauses reported by parents who were referred to the National Taiwan University Hospital, were recruited from the respiratory (P-LL), pediatric (W-CC), and otolaryngologic clinics (W-CH) between September 2012 and March 2015.[19] Approval for this study was obtained from the Ethics Committee of National Taiwan University Hospital, and written informed consent was obtained from each participant or their parents. Basic data, clinical history, and physical examination data were obtained by the corresponding author.

The weight and height of each child were measured. Age- and sex-corrected body mass index (BMI) was applied using established guidelines to convert BMI into BMI percentile.[20] Obesity was defined as age- and sex-corrected BMI >95th percentile.[2,20] Exclusion criteria included the following: children with craniofacial abnormalies, genetic disorders, or neuromuscular diseases; children who had previously received tonsillectomy or adenoidectomy; significant medical illnesses such as respiratory or cardiac disease.

### Polysomnography

Overnight polysomnography (PSG) study (Embla N7000, Reykjavik, Iceland) was performed in the sleep center of the National Taiwan University Hospital.[2,15,18,19,21–26] The sleep parameters were scored according to the 2007 American Academy of Sleep Medicine standards.[27] Briefly, apnea was defined as ≥90% decrease in airflow and hypopnea was a ≥50% decrease in airflow associated with reduced arterial oxygen saturation in ≥3% or an arousal for duration of ≥2 breaths. Disease severity in children was further characterized as primary snoring (apnea-hypopnea index, AHI <1/h), mild OSA (AHI 1–5/h), and moderate-to-severe OSA (AHI ≥5/h).[21–26]

### 24-Hour ABP

All participants received 24-hour ABP monitoring using the Oscar 2 oscillometric monitor (SunTech Medical, Model 222, Morrisville, NC), which has been validated by the International Protocol of the European Society of Hypertension and British Hypertension Society.[28,29] Measurements were obtained with the appropriately sized cuff on the nondominant arm. Participants were asked to maintain their usual activity but to remain still during daytime measurements. The monitors were programmed for daytime (7 AM to 10 PM) measurements of BP at 15-minute intervals and for nighttime (10 PM to 7 AM) measurements of BP at 30-minute intervals. The cutoff for daytime and nighttime was defined according to the sleep diary derived from parents or children. Extreme outlier BP readings were assumed to be invalid and were discarded as artifacts. Therefore, visual inspections of grossly inconsistent readings were made before interpretation, and only measurements with systolic BP <240 and >70 mmHg, diastolic BP <140 and >40 mmHg, heart rate <125 beats/min, and pulse pressure >40 but <100 mmHg with a diastolic BP <systolic BP were considered valid.[30]

Mean systolic, diastolic BP, and mean arterial pressure (MAP) were calculated for daytime and nighttime. The BP load was defined as percentage of valid BP measurements >95th percentile of BP for age and sex. Different age and sex groups were compared by applying the BP index.[30] The BP index was calculated by using the following formula: BP index = (measured BP – 95th percentile)/95th percentile × 100.[12] Hypertension was defined as mean SBP or DBP values > 95th percentile of the ABP norm.[30]

Nocturnal dipping of systolic and diastolic BP was calculated as the difference between mean daytime and nighttime BP and expressed as a percentage of dipping. Subjects with nocturnal BP dipping <10% were defined as nondippers.

### Statistical Analysis

Data were analyzed using IBM SPSS Statistics 20.0 (IBM Corporation, New York, NY). Continuous data were expressed as the mean and standard deviation, and categorical data were expressed as the number and percentage. The distribution of continuous variables among the disease severity groups was compared by Kruskal-Wallis test with Bonferroni post hoc multiple comparison when the overall test was significant. Differences in proportions between groups were examined with the Fisher exact test. The association between the sleep parameter (ie, AHI) and BP measurements was investigated by multivariable linear regression analysis. The AHI values were logarithmically transformed (natural log [x +0.1]) because its distribution was skewed and contained zero. Several models were constructed to evaluate associations between AHI and BP parameters with adjustment for age, sex, height, and adiposity (ie, BMI, BMI percentile, and obesity). A $P$ value <0.05 was considered statistically significant.

## RESULTS

### Demographics

From September 2012 to March 2015, children with symptoms suggestive of OSA were invited to participate. The final analysis included 195 subjects who had overnight PSG data and ABP measurements. Mean age was 7.8 ± 3.4 years; 69.0% (134/195) were male. For OSA groups, 17.4% (34/195) had primary snoring (AHI <1), 39.5% (77/195) had mild OSA (5 ≥ AHI >1), and 43.1% (84/195) had moderate-to-severe OSA (AHI >5). Table 1 compares the basic characteristics and PSG data among children with different disease severity. Children with moderate-to severe OSA had a higher BMI, BMI percentile, and the prevalence of obesity than that of primary snoring children. Children with mild OSA had a higher BMI percentile than that of primary snorers. Not surprisingly, children with higher AHI levels also had a significantly higher oxygen desaturation index, arousal index, and lower mean and minimum oxygen saturation.

### BP During Daytime

During daytime, children with moderate-to-severe OSA had significantly higher systolic BP (117.0 ± 12.7 vs 110.5 ± 9.3 mmHg), mean arterial pressure (MAP) (85.6 ± 8.1 vs. 81.6 ± 6.8 mmHg), and diastolic BP load (12.0 ± 9.6 vs. 8.4 ± 10.9 mmHg) compared with children with primary snoring. However, systolic and diastolic BP index did not significantly differ among OSA subgroups. Additionally, the daytime prevalence of systolic and diastolic hypertension did not significantly differ among the OSA subgroups (Table 2) (Figures 1–3).

### Blood Pressure During Nighttime

During nighttime, children with moderate-to-severe OSA had significantly higher systolic BP (108.6 ± 15.0 vs

**TABLE 1.** Demographic, Anthropometric, and Polysomnographic Data

| Variable | Whole cohort (N = 195) | AHI <1 (n = 34) | 1 ≦ AHI <5 (n = 77) | AHI ≧5 (n = 84) | P* |
|---|---|---|---|---|---|
| Age, y | 7.8 (3.4) | 7.4 (2.7) | 7.6 (3.2) | 8.1 (3.8) | 0.781 |
| Male sex | 134 (68.7) | 17 (50.0) | 55 (71.4) | 62 (73.8) | 0.039[†] |
| Weight, kg | 33.3 (20.0) | 26.1 (10.0) | 33.0 (18.7) | 36.4 (23.3) | 0.236 |
| Height, cm | 125.7 (21.7) | 122.4 (15.6) | 125.2 (21.9) | 127.5 (23.7) | 0.580 |
| BMI, kg/m$^2$ | 19.2 (5.2) | 16.7 (2.7) | 19.3 (4.8) | 20.2 (6.0)[‡] | 0.019 |
| BMI percentile | 65.8 (31.0) | 53.3 (27.7) | 68.7 (31.6)[‡] | 68.2 (30.8)[‡] | 0.016 |
| Obese, n % | 57 (29.2) | 3 (8.8) | 25 (32.5) | 29 (34.5) | 0.010[†] |
| Neck circum, cm | 27.3 (4.5) | 25.6 (2.5) | 27.4 (4.1) | 27.8 (5.3) | 0.157 |
| Waist circum, cm | 64.4 (15.3) | 57.5 (7.8) | 64.8 (14.8) | 66.6 (17.2) | 0.066 |
| Sleep time, min | 341.3 (44.6) | 343.1 (35.3) | 344.3 (47.2) | 337.9 (45.7) | 0.693 |
| Sleep efficiency, % | 85.8 (10.3) | 87.2 (8.3) | 86.5 (10.3) | 84.6 (10.9) | 0.280 |
| N1, % | 4.9 (4.5) | 4.4 (3.2) | 4.5 (3.5) | 5.5 (5.6) | 0.967 |
| N2, % | 38.3 (15.1) | 35.9 (14.0) | 36.8 (13.4) | 40.6 (16.7) | 0.218 |
| N3, % | 37.9 (15.0) | 41.4 (13.5) | 38.2 (13.8) | 36.2 (16.5) | 0.105 |
| REM, % | 18.9 (6.8) | 18.4 (6.2) | 20.5 (6.6) | 17.7 (6.9)[§] | 0.031 |
| AHI, /h | 10.7 (16.6) | 0.3 (0.2) | 2.2 (1.1)[‡] | 22.7 (19.6)[§,‡] | <0.001 |
| ODI, /h | 4.1 (9.3) | 0.1 (0.2) | 0.6 (0.8)[‡] | 8.8 (12.7)[§,‡] | <0.001 |
| MeanSaO$_2$, % | 97.2 (1.3) | 98.0 (0.6) | 97.4 (0.8)[‡] | 96.7 (1.6)[§,‡] | <0.001 |
| MinSaO$_2$, % | 86.8 (9.6) | 90.5 (16.3) | 90.1 (2.7)[‡] | 82.3 (8.3)[§,‡] | <0.001 |
| SaO$_2$ <90, % | 0.81 (2.76) | 0.00 (0.02) | 0.03 (0.11) | 1.84 (3.98)[§,‡] | <0.001 |
| Arousal index, /h | 6.5 (6.3) | 4.0 (1.7) | 4.7 (2.3) | 9.2 (8.6)[§,‡] | <0.001 |
| PLM arousal, /h | 0.10 (0.35) | 0.22 (0.57) | 0.08 (0.30) | 0.06 (0.26) | 0.082 |

Continuous data are expressed as mean ± SD, and categorical data as N (%). AHI = apnea-hypopnea index, BMI = body mass index, MeanSaO$_2$ = mean oxygen saturation, MinSaO$_2$ = minimum oxygen saturation, ODI = oxygen desaturation index, PLM = periodic limb movement, REM = rapid eye movement.
* Kruskal-Wallis test.
† Fisher exact test.
‡ $P < 0.05$ vs AHI <1 for the Bonferroni post hoc comparison.
§ $P < 0.05$ vs 1 ≦ AHI <5 for the Bonferroni post hoc comparison.

100.0 ± 9.4 mmHg), MAP (75.9 ± 9.6 vs 71.1 ± 7.0 mmHg), pulse pressure (49.2 ± 10.4 vs 43.4 ± 6.1 mmHg), systolic BP load (44.0 ± 32.6 vs 26.8 ± 24.5 mmHg), systolic BP index (0.5 ± 13.1 vs −6.8 ± 8.5 mmHg), and prevalence of systolic hypertension (47.6 vs. 14.7 %) compared to primary snoring children. Table 2 shows that children with moderate-to-severe OSA had significantly higher systolic BP (108.6 ± 15.0 vs 103.0 ± 13.3 mmHg), MAP (75.9 ± 9.6 vs 72.3 ± 9.2 mmHg), systolic BP load (44.0 ± 32.6 vs 30.6 ± 29.3 mmHg), and systolic BP index (0.5 ± 13.1 vs −4.2 ± 12.4 mmHg) compared with children with mild OSA (Figures 1−3).

### Nocturnal BP Dipping

Compared with those with mild OSA, children with moderate-to-severe OSA had significantly lower systolic nocturnal BP dipping (7.1 ± 8.6 vs 10.0 ± 7.8 mmHg). However, diastolic nocturnal BP and the proportion of nondippers did not significantly differ among the 3 OSA subgroups (Table 2).

### Multivariable Linear Regression Analyses

Multiple linear regression models were used to explore associations between AHI and BP parameters after adjusting for possible confounders (ie, age, sex, height, and adiposity variables) (Table 3). Unadjusted analysis showed the AHI was positively correlated with daytime systolic BP, daytime MAP, nighttime systolic BP, nighttime diastolic BP, and nighttime MAP. Associations between AHI and daytime BP parameters were partially affected by adiposity, whereas AHI correlated positively with nighttime systolic BP and MAP after adjusting possible confounders.

### DISCUSSION

This large, hospital-based study elaborates associations between ABP and OSA in children. Analytical results show that children with OSA had elevated ABP with increased disease severity. In this study cohort, children with moderate-to-severe OSA had significantly higher daytime systolic BP and diastolic BP load compared with primary snorers. In children with moderate-to-severe OSA, nighttime systolic BP, systolic BP load, and systolic BP index were also significantly higher compared with primary snorers. The effect of OSA on nighttime BP is independent of obesity. These findings provide robust evidence that, in children with OSA, BP is higher in those who are primary snorers. From a clinical perspective, elevated BP in childhood predicts future cardiovascular risks.[31–32] Therefore, children with severe OSA require monitoring of cardiovascular parameters and proper treatment to prevent further adverse cardiovascular consequences.

In children, Guilleminault et al[7] first linked OSA to elevated BP.[7–12] However, a meta-analysis by Zintzaras et al[13] offered insufficient evidence that moderate-to-severe OSA was associated with hypertension, a finding that may be confounded by a small sample size and large heterogeneity among studies. In contrast, many recent studies demonstrated a

**TABLE 2.** 24-Hour Ambulatory Blood Pressure in OSA Subgroups

| Parameter | Whole cohort (N = 195) | AHI <1 (n = 34) | 1 ≦ AHI <5 (n = 77) | AHI ≧5 (n = 84) | P* |
|---|---|---|---|---|---|
| Daytime | | | | | |
| Systolic BP | 114.9 (11.6) | 110.5 (9.3) | 114.5 (10.7) | 117.0 (12.7)[†] | 0.037 |
| Diastolic BP | 68.9 (6.2) | 67.1 (6.4) | 68.5 (5.5) | 69.9 (6.5) | 0.048 (ns) |
| Heart Rate | 94.9 (11.0) | 94.0 (9.1) | 95.3 (10.2) | 94.9 (12.5) | 0.855 |
| Mean arterial pressure | 84.2 (7.5) | 81.6 (6.8) | 83.9 (6.8) | 85.6 (8.1)[†] | 0.034 |
| Pulse Pressure | 46.0 (8.0) | 43.4 (6.7) | 45.9 (7.8) | 47.2 (8.4) | 0.087 |
| Systolic BP load | 24.0 (23.5) | 17.2 (19.5) | 23.4 (22.2) | 27.3 (25.7) | 0.107 |
| Diastolic BP load | 10.5 (9.9) | 8.4 (10.9) | 9.7 (9.5) | 12.0 (9.6)[†] | 0.020 |
| Systolic BP index | −7.8 (8.6) | −10.3 (7.5) | −8.2 (8.3) | −6.5 (9.1) | 0.130 |
| Diastolic BP index | −18.8 (7.4) | −20.6 (7.7) | −19.3 (6.6) | −17.6 (7.8) | 0.061 |
| Systolic hypertension, n (%) | 40 (20.5) | 3 (8.8) | 16 (20.8) | 21 (25.0) | 0.132[‡] |
| Diastolic hypertension, n (%) | 2 (1.0) | 1 (2.9) | 1 (1.3) | 0 (0.0) | 0.168[‡] |
| Nighttime | | | | | |
| Systolic BP | 104.9 (13.9) | 100.0 (9.4) | 103.0 (13.3) | 108.6 (15.0)[†,§] | 0.005 |
| Diastolic BP | 57.9 (7.7) | 56.7 (6.6) | 56.9 (7.8) | 59.4 (7.9) | 0.074 |
| Heart Rate | 78.4 (11.8) | 75.7 (8.3) | 77.5 (11.4) | 80.3 (13.2) | 0.173 |
| Mean arterial pressure | 73.6 (9.2) | 71.1 (7.0) | 72.3 (9.2) | 75.9 (9.6)[†,§] | 0.011 |
| Pulse Pressure | 46.9 (9.3) | 43.4 (6.1) | 46.1 (8.6) | 49.2 (10.4)[†] | 0.006 |
| Systolic BP load | 35.7 (30.7) | 26.8 (24.5) | 30.6 (29.3) | 44.0 (32.6)[†,§] | 0.009 |
| Diastolic BP load | 21.4 (21.9) | 15.5 (17.8) | 19.4 (20.9) | 25.6 (23.7) | 0.045 (ns) |
| Systolic BP index | −2.5 (12.4) | −6.2 (8.5) | −4.2 (12.4) | 0.5 (13.1)[†,§] | 0.009 |
| Diastolic BP index | −11.0 (12.2) | −12.4 (10.0) | −12.7 (12.6) | −8.9 (12.4) | 0.082 |
| Systolic hypertension, n (%) | 70 (35.9) | 5 (14.7) | 25 (32.5) | 40 (47.6) | 0.002[‡] |
| Diastolic hypertension, n (%) | 38 (19.5) | 5 (14.7) | 14 (18.2) | 19 (22.6) | 0.602[‡] |
| Dipping | | | | | |
| Systolic dipping | 8.7 (7.8) | 9.4 (4.6) | 10.0 (7.8) | 7.1 (8.6)[§] | 0.022 |
| Diastolic dipping | 15.8 (8.5) | 15.5 (6.9) | 16.9 (8.9) | 14.8 (8.7) | 0.183 |
| Systolic nondipper, n (%) | 100 (51.3) | 17 (50.0) | 33 (42.9) | 50 (59.5) | 0.106[‡] |
| Diastolic nondipper, n (%) | 44 (22.6) | 10 (29.4) | 14 (18.2) | 20 (23.8) | 0.390[‡] |

Continuous data are expressed as mean ± SD, and categorical data as N (%). AHI = apnea-hypopnea index, BP = blood pressure, OSA = obstruc-obstructive sleep apnea.

\* Kruskal-Wallis test.

[†] $P < 0.05$ vs AHI <1 for the Bonferroni post hoc comparison.

[‡] Fisher exact test; ns indicates no significance, post hoc comparisons were found.

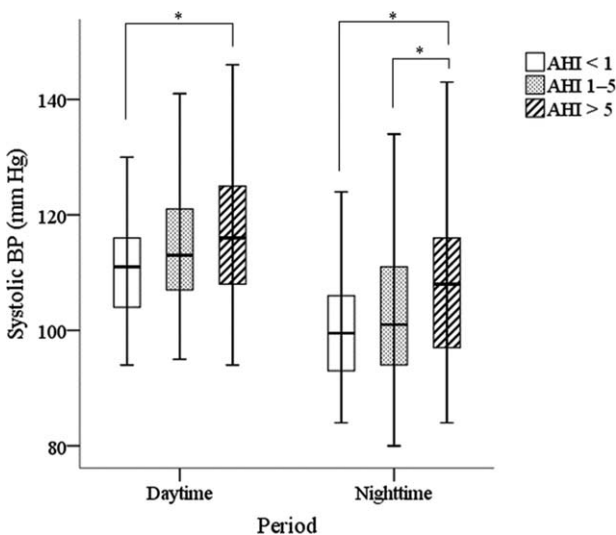[§] $P < 0.05$ vs 1 ≦ AHI <5 for the Bonferroni post hoc comparison.



**FIGURE 1.** Systolic BP during different periods for OSA subgroups. *Post hoc test, $P < 0.05$. BP = blood pressure, OSA = obstructive sleep apnea.
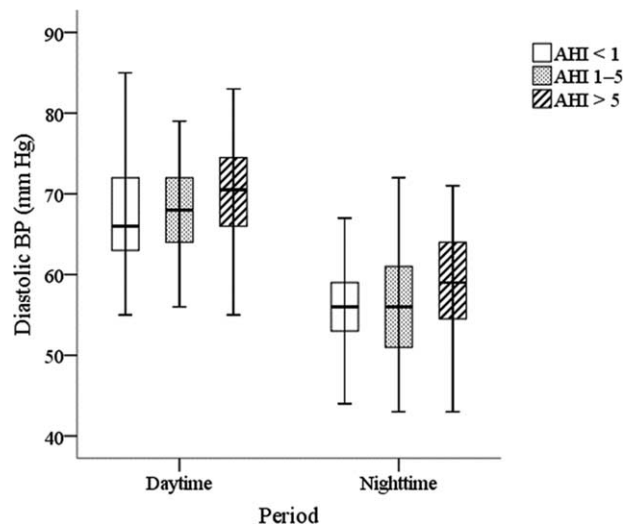


**FIGURE 2.** Diastolic BP during different periods for OSA subgroups. *Post hoc test, $P < 0.05$. BP = blood pressure, OSA = obstructive sleep apnea.
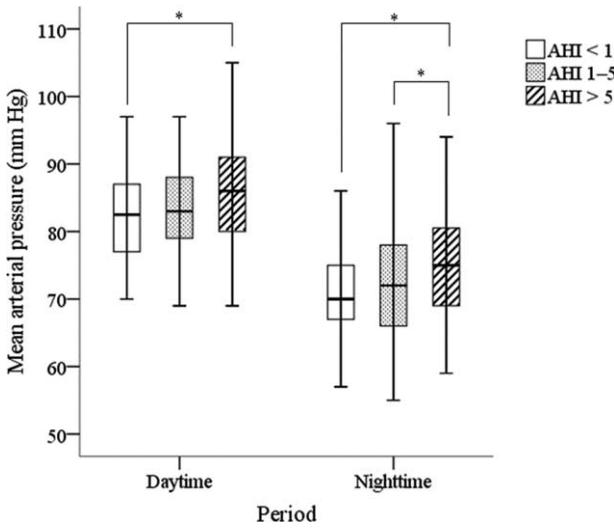
**FIGURE 3.** Mean arterial pressure during different periods for OSA subgroups. *Post hoc test, $P < 0.05$. BP = blood pressure, OSA = obstructive sleep apnea.

trend of elevated BP in children with OSA.[8-12,33-35] Li et al[8] and Xu et al[12] reported a dose-response relationship between OSA and BP in children. Horne et al[35] indicated that pediatric OSA increased BP during sleep more than in control, regardless of OSA severity. Weber et al[11] showed that children with OSA had higher diastolic and mean BP compared with those of primary snoring. The present study found that moderate-to-severe OSA was associated with elevated BP during daytime and nighttime. In children, OSA also had a strong correlation with systolic BP during nighttime, and the correlation was independent of obesity. These findings indicate that childhood OSA is associated with elevated BP. Interestingly, the

prevalence of non-dippers in this study did not significantly differ among OSA subgroups. However, children with moderate-to-severe OSA had less systolic nocturnal dipping. Previously, Li et al, Horne et al, and Nisbet et al observed that nocturnal dipping is preserved in young children with OSA.[8,36,37] But Weber et al[11] demonstrated children with OSA had decreased degree of nocturnal dipping. In these children, exposure to the effects of OSA was sufficiently long or the disease severity was sufficiently high to affect nocturnal dipping profiles. Therefore, OSA and cardiovascular effects may be resolved when appropriate treatment is administrated.

Impacts of pediatric OSA on cardiovascular effects have received increasing attention.[31-43] Evidence of autonomic dysfunction was found in children with OSA during both wakefulness and sleep.[33-35] Moreover, BP dysregulation, elevated sympathetic activity and impairment of autonomic reflexes occur in school-aged children and adolescents with OSA.[39-42] Notably, cardiovascular consequences of OSA occur not only in children with OSA, but also in children with primary snoring.[43] This hospital-based study recruited participants with clinical symptoms. Therefore, cardiovascular comparisons between children with OSA and normal controls were not obtained.

High BP in adulthood is a well-established risk factor for early disability and death.[44] Although BP levels in childhood cannot be directly linked with cardiovascular events in adults, a number of studies have reported that intermediate markers of target organ damage, including left ventricular hypertrophy, atherosclerosis, or cognitive dysfunction, are detectable in children and adolescents with elevated BP.[31,32,45-47] Importantly, elevated BP in children continues into adulthood and is often associated with modifiable risk factors such as lack of physical activity, obesity, and sleep problems.[31,32,48,49] If the diagnosis of high BP is not made, associated risk factors are unlikely to decrease. This study highlighted correlations between increased BP and OSA in children, implying early awareness and proper treatment should be considered in

**TABLE 3.** Multivariable Linear Regression Analyses of the Association Between AHI (in Log Scale) and BP Parameters After Adjustment of Possible Confounders

| Time/Model | SBP | | DBP | | MAP | |
|---|---|---|---|---|---|---|
| | β (95% CI) | *P* | β (95% CI) | *P* | β (95% CI) | *P* |
| Daytime | | | | | | |
| Model 1 | 1.6 (0.6, 2.7) | 0.002 | 0.5 (−0.001, 1.1) | 0.051 | 0.9 (0.3, 1.6) | 0.007 |
| Model 2 | 1.2 (0.4, 2.0) | 0.005 | 0.5 (−0.1, 1.0) | 0.094 | 0.7 (0.1, 1.3) | 0.019 |
| Model 3 | 0.8 (−0.01, 1.6) | 0.053 | 0.3 (−0.3, 0.8) | 0.297 | 0.5 (−0.1, 1.1) | 0.120 |
| Model 4 | 1.0 (0.2, 1.8) | 0.015 | 0.4 (−0.2, 0.9) | 0.162 | 0.6 (0.01, 1.2) | 0.046 |
| Model 5 | 1.0 (0.2, 1.8) | 0.019 | 0.3 (−0.2, 0.9) | 0.205 | 0.6 (−0.02, 1.1) | 0.059 |
| | | | | | | |
| Nighttime | | | | | | |
| Model 1 | 2.6 (1.4, 3.8) | <0.001 | 0.8 (0.1, 1.5) | 0.025 | 1.4 (0.6, 2.2) | 0.001 |
| Model 2 | 2.3 (1.2, 3.4) | <0.001 | 0.8 (0.1, 1.5) | 0.027 | 1.3 (0.5, 2.1) | 0.001 |
| Model 3 | 1.9 (0.8, 3.1) | 0.001 | 0.6 (−0.1, 1.3) | 0.079 | 1.1 (0.3, 1.9) | 0.009 |
| Model 4 | 2.1 (1.0, 3.2) | <0.001 | 0.7 (−0.004, 1.4) | 0.051 | 1.2 (0.4, 2.0) | 0.004 |
| Model 5 | 2.1 (1.0, 3.2) | <0.001 | 0.6 (−0.04, 1.3) | 0.065 | 1.1 (0.4, 1.9) | 0.005 |

Model 1: unadjusted analysis; Model 2: adjusted for age, sex, height; Model 3: adjusted for age, sex, height, and BMI; Model 4: adjusted for age, sex, height, and BMI percentile; Model 5: adjusted for age, sex, height, and obesity; AHI = apnea-hypopnea index, BMI = body mass index, CI = confidence interval, DBP = diastolic blood pressure, MAP = mean arterial pressure, SBP = systolic blood pressure.

this category of patients to prevent further cardiovascular complications.

This study has certain limitations. First, this study lacked a normal control group.[50] In this hospital-based study, primary snorers were the comparison group. However, children who are primary snorers are not considered innocent anymore,[51] and evidence exists that children with primary snoring had higher BP levels compared with normal controls.[43] Second, this study was a cross-sectional study. Further longitudinal, prospective studies are needed to elucidate long-term changes of OSA and its association with BP.[52–55] Third, this study did not link BP with other cardiovascular measurements, and evidence of early target-organ damage in children with OSA was not obtained.[56] Finally, because of the lack of normal BP reference in Taiwanese children, this study used normative data for the US population.[30] Future studies should examine effect of treatment on BP in children with sleep disturbances.[18,57–60]

## CONCLUSIONS

This large, hospital-based study shows that daytime and nighttime BP levels elevated with increased severity of OSA in children. Compared with children with primary snoring, those with moderate-to-severe OSA had a significantly higher BP. This study identified a correlation between OSA severity and nighttime BP, and the correlation was independent of obesity. Since elevated BP in childhood predicts future cardiovascular risks, children with OSA and hypertension should receive proper treatment to prevent further adverse cardiovascular consequences. Further studies are needed to elucidate the impacts of surgery or other treatment strategies on BP parameters in children with obstructive sleep disorders.

## ACKNOWLEDGMENTS

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