Morning Blood Pressure Is Associated with Sleep Quality in Obese Adolescents

Tamara S. Hannon, MD, MS[1], Wanzhu Tu, PhD[2], Sara E. Watson, MD[1], Hasnaa Jalou, MD[1], Sangeeta Chakravorty, MD[3], and Silva A. Arslanian, MD[3]

Objective To examine relationships among blood pressure (BP), adiposity, and sleep quality with the use of overnight polysomnography in obese adolescents.

Study design Overnight polysomnogram and morning BP measurements were performed in obese (body mass index [BMI] >95th percentile) nondiabetic adolescents (eligible age range 12-18 years, n = 49). Subjects were stratified into 2 groups, one with normal BP, and one with elevated BP, and demographic and clinical characteristics were compared between the groups. Multiple linear regression analysis was used to assess the effects of sleep quality on BP.

Results Participants (n = 27) had a normal morning BP, and 22 (44.9%) had elevated morning BP. There were no differences in age (P = .53), sex (P = .44), race (P = .58), or BMI (P = .56) between the 2 BP groups. The group with elevated BP spent shorter percentages of time in rapid eye movement (REM; P = .006) and slow-wave sleep (SWS; P = .024). Multiple linear regression analysis showed that a lower percentage of both REM and SWS was associated with increased morning BP after we adjusted for pubertal stage, sex, race, and BMI.

Conclusion Lack of deeper stages of sleep, REM sleep, and SWS is associated with greater morning BP in obese adolescents, independent of BMI. Poor sleep quality should be considered in the work-up of obese youth with hypertension. Intervention studies are needed to evaluate whether improving the quality of sleep will decrease BP elevation. *(J Pediatr 2014;164:313-7)*.

Increasing prevalence of both hypertension and obesity-related sleep disorders in youth are directly associated with the obesity epidemic.[1,2] Studies in adults have shown that poor sleep quality and hypertension are linked and that this relationship is independent of obesity.[3-7] Moreover, the association between blood pressure (BP) and sleep quality in adults is characterized by sleep fragmentation and a relative lack of restorative deeper sleep, particularly slow-wave sleep (SWS), during which sympathetic nervous system activity decreases and heart rate and BP decrease.[8,9]

In adolescents, whether there is an additional impact of poor sleep quality on BP above and beyond that associated with obesity is not understood. The objective of this study was to examine relationships between adiposity, BP, and objective measures of sleep quality measured by polysomnogram (PSG). We performed a secondary analysis of a study designed to evaluate insulin sensitivity and measures of sleep quality in obese adolescents in which morning BP measures also were collected.[10] The primary hypothesis was that morning BP would be greater among patients with poorer sleep quality characterized by less deep sleep (SWS), independent of body mass index (BMI).

Methods

The study was approved by the University of Pittsburgh and Indiana University Institutional Review Boards and performed in the Pediatric Clinical and Translational Research Centers at these institutions. This was a secondary analysis of a study powered to detect the association between insulin sensitivity and sleep-disordered breathing measured by use of the apnea-hypopnea index (AHI) as the primary outcome in obese black and white adolescents.[10] Data from this secondary analysis have not been previously reported.

Nondiabetic patients referred for the clinical evaluation of obesity who met study criteria were invited to participate in the original study. Participants

AHI	Apnea-hypopnea index
BMI	Body mass index
BP	Blood pressure
DBP	Diastolic blood pressure
PSG	Polysomnogram
REM	Rapid eye movement
SpO_2	Saturation level of oxygen in hemoglobin
SBP	Systolic blood pressure
SWS	Slow-wave sleep

From the Departments of [1]Pediatrics and [2]Biostatistics, Indiana University School of Medicine, Indianapolis, IN; and [3]Department of Pediatrics, Children's Hospital of Pittsburgh, University of Pittsburgh Medical Center, Pittsburgh, PA

Supported by the Indiana University Purdue University Signature Center Grant Initiative funds (to T.H. and W.T.), the National Institutes of Health (grants R03HD057532 to T.H.; T32 DK065549-08 to S.W.; and K12DK063704 to S.A.), the Clinical and Translational Science Award to University of Pittsburgh (UL1RR024153), and the Clinical and Translational Science Institute Clinical Research Center at Indiana University School of Medicine (TR000006).

 http://dx.doi.org/10.1016/j.jpeds.2013.10.011

were not previously identified as having hypertension or sleep disorders. After obtaining informed assent/consent, we studied 50 obese (BMI >95th percentile) nondiabetic adolescents (eligible age range 12-18 years). Exclusion criteria included airway disease, smoking, uncontrolled chronic disease or current use of chronic medications affecting glucose regulation or BP, and obesity associated with a syndrome, such as Prader-Willi syndrome.

Overnight PSG was performed, and we recorded data with either the Sensormedics Somnostar Pro version 7.2 software (Yorba Linda, California) or Sandman Elite 9.1 sleep diagnostic software (Embla, Buffalo, New York), applying the following electroencephalographic montage: F3M2, F4M1, C3M2, C4M1, O2M1, O1M2, L-EOG, R-EOG, chin electromyography, limb electromyography, and the following cardiorespiratory measurements: blood oxygen saturation and pulse, nasal pressure, airflow (nasal or oral thermistor), thoracic and abdominal excursion (uncalibrated respirator inductance plethysmography), pulse, and electrocardiogram. The PSG data were interpreted by 1 of 2 sleep medicine coinvestigators. The AHI, which is the total number of obstructive apnea and hypopnea events per 1 hour of sleep, was hand-scored with the use of pediatric criteria and calculated by following the American Academy of Sleep Medicine manual for scoring guidelines.[11] One participant's data were excluded from the analysis because very severe obstructive sleep apnea was apparent on the PSG (AHI = 50 events per hour).

After the PSG (6:00-7:00 a.m.) morning BP was measured in duplicate (at 10-minute intervals) by a trained research nurse with a calibrated Dinamap automated oscillometric device (GE Medical Systems Information Technologies, Inc, Milwaukee, Wisconsin) who used a cuff appropriate for arm size with the participant awake, seated, and at rest. The analysis was based on the average morning systolic BP (SBP) and diastolic BP (DBP). Elevated BP was identified if the SBP or DBP was either at least ≥90th percentile for age, sex, and height; if the SBP exceeded 120 mm Hg; or the DBP exceeded 80 mm Hg.[12]

Percent body fat was measured by dual-energy X-ray absorptiometry in all but 4 participants. In the remaining participants, dual-energy X-ray absorptiometry was not performed because the participants' weight exceeded the limits for the equipment. Pubertal stage was determined by physical examination.[13]

Statistical Analyses

Study subjects were stratified into 2 groups, one with normal BP, and one with elevated BP (as defined previously). Demographic and clinical characteristics of the subjects were summarized and compared between the groups. Categorical variables were compared using the χ^2 test, continuous variables were compared using the 2-sample t test and a nonparametric test (Mann-Whitney U statistic) was used for non-normally distributed measures. Scatter plots were used to graphically examine the relationships between sleep characteristics obtained from the PSG, BMI, waist circumference, and BP. Pearson correlation coefficients were used to quantify the magnitudes of the linear correlations. Multiple linear regression analysis was then conducted to assess the effects of sleep quality on BP, including the percent of sleep time spent in rapid eye movement (REM) sleep and SWS, and the AHI, while we controlled for the effects of age or pubertal stage, sex, race (as a dichotomous variable), and BMI. All analyses were implemented with SPSS software (version 20; Chicago, Illinois). $P < .05$ was considered statistically significant.

Results

The characteristics of the study participants are reported in Table I. The study included 49 participants; mean age 14.4 ± 1.7 (SD) years; 51.9% male; 57.1% white, 40.7% black, and 2.0% mixed-race (black/white). Participants (n = 27) had morning BP values in the normal range, and 22 (44.9%) had elevated morning BP. There were no significant differences in age, pubertal stage (Tanner 4 in

Table I. Characteristics of the study participants

	BP normal, n = 27	BP elevated, n = 22	*P* value
Morning BP, mm Hg			
SBP	109 ± 7 (111; 94-120)	134 ± 9 (132; 121-157)	<.001
DBP	61 ± 5 (60; 51-72)	72 ± 7 (70; 60-87)	<.001
Demographics			
Age, y	14.3 ± 1.7	14.6 ± 1.6	.53
Sex			.44
Male, n (%)	14 (51.9)	9 (40.9)	
Female, n (%)	13 (48.1)	13 (59.1)	
Race			.58
White, n (%)	16 (59.3)	12 (54.5)	
Black, n (%)	10 (37.0)	10 (45.5)	
Mixed-race, n (%)	1 (3.7)	0 (0.0)	
Adiposity measures			
BMI, kg/m^2	38.7 ± 8.9 (35; 28.4-61.6)	39.9 ± 8.5 (37.9; 28.7-57.1)	.56
BMI SDS	3.95 ± 1.65 (3.55; 1.80-8.31)	3.92 ± 1.41 (3.50; 2.04-6.31)	.94
Body fat, %, n = 41	46.2 ± 4.62 (46.4; 37.5-53.8)	47 ± 7.4 (46.3; 36.7-58.5)	.67
Waist, cm	114.8 ±17.7 (108; 91-155)	114.3 ± 19.6 (112; 78-149)	1.00
Sleep measures			
REM, %	20.9 ± 3.6 (20.6; 13.5-29.4)	17 ± 4.9 (17.2; 6.6-27.6)	.003
SWS, %	21.5 ± 6.9 (20.8; 8.2-32)	17.4 ± 6.9 (17; 5-31.7)	.04
PSG sleep time, min	421.2 ± 28.5 (417; 354-483)	396.7 ± 75.2 (395; 233-494)	.16
Sleep latency, min	13.9 ± 11.7 (12; 1-46.5)	18.3 ± 16.4 (15.2; 0.5-67)	.38
Time to REM, min	118.8 ± 44.3 (116.5; 50-221.5)	133.4 ± 70.7 (101.5; 42-276)	.82
AHI, events/h	2.9 ± 2.9 (1.7; 0.1-12.4)	4.6 ± 5.5 (1.2; 0.0-20)	.88
Arousal index, events/h	9.2 ± 4.8 (8.4; 4.1-25.3)	9.3 ± 5.2 (7.9; 2-22.2)	.98
Minimum SpO_2, %	89.9 ± 4.7 (92; 77-95)	89.3 ± 5.3 (90.5; 76-96)	.86

Data are expressed as mean ± SD (median; range) or mean (%).