Sleep Quality and Elevated Blood Pressure in Adolescents

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Background—We assessed whether insufficient sleep is associated with prehypertension in healthy adolescents.

Methods and Results—We undertook a cross-sectional analysis of 238 adolescents, all without sleep apnea or severe comorbidities. Participants underwent multiple-day wrist actigraphy at home to provide objective estimates of sleep patterns. In a clinical research facility, overnight polysomnography, anthropometry, and 9 blood pressure measurements over 2 days were made. Exposures were actigraphy-defined low weekday sleep efficiency, an objective measure of sleep quality (low sleep efficiency \( \leq 85\% \)), and short sleep duration (\( \leq 6.5 \) hours). The main outcome was prehypertension (\( \geq 90\% \) percentile for age, sex, and height), with systolic and diastolic blood pressures as continuous measures as secondary outcomes. Prehypertension, low sleep efficiency, and short sleep duration occurred in adolescents with low sleep efficiency and 2.8-fold (95% CI, 1.1 to 7.3) in those with short sleep. In analyses adjusted for sex, body mass index percentile, and socioeconomic status, the odds of prehypertension increased 3.5-fold (95% CI, 1.5, 8.0) for low sleep efficiency and 2.5-fold (95% CI, 0.9 to 6.9) for short sleep. Adjusted analyses showed that adolescents with low sleep efficiency had on average a 4.0 ± 1.2-mm Hg higher systolic blood pressure than other children (P<0.01).

Conclusions—Poor sleep quality is associated with prehypertension in healthy adolescents. Associations are not explained by socioeconomic status, obesity, sleep apnea, or known comorbidities, suggesting that inadequate sleep quality is associated with elevated blood pressure. (Circulation. 2008;118:1034-1040.)

Key Words: blood pressure ■ epidemiology ■ hypertension ■ pediatrics ■ sleep

Hypertension is an increasingly prevalent health problem in adults and adolescents alike. Between 1988 and 1999, prehypertension (ie, a blood pressure [BP] \( \geq 90\% \) percentile for height, age, and sex) and hypertension were estimated to increase in children by 2.3% and 1%, respectively.\(^1\) Childhood hypertension is associated with hypertension in adulthood, a risk factor for cardiovascular disease incidence and death.\(^2,5\) It also is associated with end-organ damage, notably left ventricular hypertrophy, in both children and adults.\(^6,7\)

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Several studies have implicated insufficient sleep as a risk factor for hypertension in adults.\(^8–12\) Although the cause is unclear, experimental studies indicate that shorter sleep results in metabolic and endocrine dysfunction, which may contribute to cardiovascular disease.\(^13–17\) Studies in both adult and pediatric populations also have reported associations of shorter sleep duration with obesity and impaired glucose tolerance.\(^14,18,19\) These findings have a potentially large public impact given the frequency of sleep curtailment.\(^20\)

Few studies have addressed the relationship between sleep and hypertension in children. A higher level of diastolic but not systolic BP was reported in children with obstructive sleep apnea compared with primary snorers.\(^21\) The Tucson’s Children’s Assessment of Sleep Apnea Study found that elevations in systolic and diastolic BPs were independently associated with sleep efficiency, respiratory disturbance index (a measure of sleep apnea), and obesity in 230 children 6 to 11 years of age.\(^22\) To the best of our knowledge, no studies have examined the association between insufficient sleep and BP in adolescents free of sleep apnea. In this report, we examine the relationship between prehypertension and systolic and diastolic BP levels and objective measures of sleep quality and duration in a community-based cohort of adolescents. First, we hypothesize that adolescents with poor sleep quality or short sleep duration will be at increased odds of prehypertension. Second, we posit that adolescents with short sleep duration or poor sleep quality will have higher systolic and diastolic BP readings on average compared with adolescents with better-quality sleep. We excluded adolescents with clinically significant levels of sleep apnea to minimize the influence of this exposure on BP and sleep duration measurement.

Methods

Study Population
The sample was derived from the Cleveland Children’s Sleep and Health Study, a longitudinal cohort study. Data for this analysis are from...
238 adolescents free of severe illnesses who participated in an examination performed between 2002 and 2006 aimed at participants 13 to 16 years of age. Details of the study population have been reported elsewhere and are reviewed in the online Data Supplement.

**Study Protocol**
Adolescents underwent 5-7 day wrist actigraphy and completed a daily sleep log at home during the week before a clinical research center examination and when free of acute illness. After this period of in-home monitoring, participants were studied in a dedicated clinical research center where overnight polysomnography and physiological and anthropometric assessments were performed using a standardized protocol. Examinations at the research center began at approximately 5 PM and ended the following day at 11 AM. Informed consent was obtained from the child’s legal guardian, and written assent was obtained from the child. The study was approved by the governing institutional review board.

**Measurements**

**Actigraphy**
Sleep-wake estimation was made with wrist actigraphy (Octagonal Sleep Watch 2.01, Ambulatory Monitoring Inc, Ardsley, NY) analyzed with the Action-W software and the time-above-threshold algorithm. Using weekday data (minimum, 3 days), we calculated mean sleep duration and mean sleep efficiency, an objective measure of sleep continuity and quality defined as the percentage of time in bed estimated to be asleep (ie, total time estimated to be asleep divided by the total time in bed for the major sleep period times 100). Adolescents with a sleep efficiency ≤85% were considered to have low sleep efficiency. Given the lack of data on cutoffs for defining short sleep duration in this age, we used the lowest decile of mean sleep duration on weekdays to define short sleep duration, which approximated 6.5 hours.

**Blood Pressure**
Three BP readings were obtained at each of 3 times (9 PM [supine] the night of the polysomnography and 8 AM [supine] and 9:30 AM [sitting] the following morning) following published guidelines. After a 10-minute rest period, BP was obtained by trained nurses [sitting] the following morning) following published guidelines. Sixty-one adolescents (26%) had low sleep efficiency. Aver-

**Other Measurements**
A rigid stadiometer was used to measure height, and a calibrated digital scale was used to measure weight. Body mass index (BMI) was calculated by dividing the weight in kilograms by height in meters squared and converted into age- and sex-adjusted percentiles (http://www.cdc.gov/growthcharts/). Overweight was defined as BMI ≥95th percentile. Adolescents who were reported to snore loudly at least 1 to 2 times per week during the past month were categorized as snorers. The apnea–hypopnea index was defined as the percentage of time in bed estimated to be apneic or hypopneic during the total time in bed for the major sleep period times 100. Adolescents with a sleep apnea–hypopnea index ≥5 were considered to have low sleep efficiency. Given the lack of data on cutoffs for defining short sleep duration in this age, we used the lowest decile of mean sleep duration on weekdays to define short sleep duration, which approximated 6.5 hours.

**Statistical Analysis**
Between-group differences for the binary outcome, prehypertension, were assessed with the Pearson 2 test for categorical variables, the 2-sample t test for normally distributed variables, and the Wilcoxon rank-sum test for non-normally distributed measures. To assess confounding, associations between the primary exposures, low sleep efficiency (≤85%) and short sleep duration (<6.5 hours), and sociodemographic characteristics also were examined. Spearman and Pearson correlations assessed the strength of the linear relationship between sleep characteristics obtained from polysomnography and actigraphy. Logistic regression analyses were used to examine whether adolescents with short sleep duration or low sleep efficiency were at increased odds of prehypertension. Given the relatively small number of adolescents with prehypertension, covariate adjustment was limited to the SES z score and the 2 variables most strongly associated with prehypertension: sex and BMI percentile. Multiple linear regression, adjusted for age, sex, race, preterm status, BMI percentile, and SES z score, was used to examine the linear associations between sleep duration or sleep efficiency with continuously measured systolic and diastolic BP levels. Additional analyses included low sleep efficiency from the polysomnography as the exposure. Residual confounding by snoring or the apnea–hypopnea index also was assessed by including these measures as covariates in the adjusted analyses.

The authors had full access to the data and take full responsibility for the integrity of the data. All authors have read and agree to the manuscript as written.

**Results**
Characteristics of the analytic sample are shown in Table 1. The average participant was 13.7±0.7 years of age. As designed, the sample had an ≈50% representation of boys, blacks, and children born prematurely. One fifth of the sample was overweight. Approximately one-fourth reported their household income as less than $20,000 per year. Sixty-one adolescents (26%) had low sleep efficiency. Average weekday sleep duration was 7.71 hours, and 11% of the sample slept ≤6.5 hours.

Sample characteristics stratified by prehypertension also are shown in Table 1. Overall, 33 children (14%) met the criteria for prehypertension, including 19 who had prehypertension and 14 who were hypertensive. Compared with normotensive adolescents, those with prehypertension tended to more often be male, tended to have a higher BMI, and were more frequently from neighborhoods with a lower median income (P=0.05 to 0.10). Both low sleep efficiency (P<0.0001) and short sleep duration (P=0.06) were >2-fold more prevalent in those with prehypertension compared with normotensive adolescents.

The distribution of various BP measures is further detailed in Table 2. Using the mean of 9 BP readings, we classified ≈11% of the sample as having elevated systolic BP and 5% as having elevated diastolic BP. All measures of systolic BP were significantly higher among the adolescents with low sleep efficiency compared with those with higher sleep efficiency. Adolescents with low sleep efficiency also had a higher prevalence of elevated diastolic BP and had higher 8 AM diastolic BP values. Adolescents with short sleep duration did not differ from those with longer sleep duration in regard to systolic BP but had a higher average diastolic BP and higher prevalence of elevated diastolic BP (24.0% versus 2.4%; P<0.001).
To assess confounding, associations among the sleep exposures and sociodemographic characteristics were examined (see the online Data Supplement). Adolescents with low sleep efficiency had a higher BMI, were more often male, and were from households with lower incomes and lower levels of caregiver education. These characteristics were not significantly associated with short sleep duration. Approximately two thirds (68.0%) of adolescents with short sleep duration also had low sleep efficiency, whereas 27.9% of adolescents with low sleep efficiency also had short sleep duration. The correlation between mean weekday sleep efficiency and sleep efficiency from the night of the polysomnography was low ($r=0.13$, $P=0.04$), as was the correlation between mean weekday sleep duration and sleep duration from the night of the polysomnography ($r=-0.06$, $P=0.37$). Approximately one third (32.8%) of adolescents with low sleep efficiency as assessed on actigraphy also had low sleep efficiency from the polysomnography.

Results of the logistic regression models of the association between each sleep measure and the odds of prehypertension are shown in Tables 3 and 4. After adjustment for sex, BMI percentile, and SES $z$ score, those with low sleep efficiency had 3.5 times the odds of prehypertension compared with those without low sleep efficiency (95% CI, 1.54 to 7.96).
Weaker associations were observed between 0.40-mm Hg decrease in systolic BP and 0.06-mm Hg decrease in diastolic BP (P=0.001). Weaker associations were observed between 0.20 mm Hg increase in systolic BP and 0.20 mm Hg increase in diastolic BP (P=0.06).

The unadjusted and adjusted associations between continuously measured systolic and diastolic BP levels with sleep efficiency are shown in Table 5. After adjustment for age, sex, race, term status, BMI percentile, and SES z score, the model predicts that each 5% increase in sleep efficiency was associated with a 1.24-mm Hg increase in systolic BP (P=0.002) and with a 0.20 mm Hg increase in diastolic BP (P=0.4726). Similarly, those with low polysomnography sleep efficiency had systolic BP that was 3.26 mm Hg higher compared with those with higher sleep efficiency (P=0.0001). When low sleep efficiency was modeled as a dichotomous exposure, the adjusted model estimates that adolescents with low sleep efficiency had a mean systolic BP that was on average 3.99 ± 1.24 mm Hg higher compared with those with higher sleep efficiency (P=0.0002). Including sleep duration as a continuously measured covariate did not alter the primary associations of sleep efficiency and BP (data not shown).

Similar to the models of prehypertension, sleep duration was more weakly associated with continuously measured systolic and diastolic BPs compared with sleep efficiency (Table 6).

Additional Analyses

The primary analyses also were repeated with low sleep efficiency ascertained via polysomnography as the exposure. Consistent with the results of the primary analysis, after adjustment for sex, BMI percentile, and SES z score, those with polysomnography sleep efficiency ≤85% had nearly 3 times the odds of prehypertension as those with better sleep (OR, 2.83; 95% CI, 1.28, 6.24). Also consistent with the results of the actigraphy-defined sleep exposures, in adjusted analyses, each 1% increase in sleep efficiency was associated with a 0.20 ± 0.06-mm Hg decrease in systolic BP (P<0.001). Similarly, those with low polysomnography sleep efficiency had systolic BP that was 3.26 ± 1.25 mm Hg higher on average compared with those with better sleep (P=0.01).

Although analyses were restricted to children without clinically significant sleep apnea, additional analyses assessed potential residual confounding by snoring or the apnea–hypopnea index (ie, in an apnea–hypopnea index range of 0 to 4.9). The results show that loud snoring was not significantly associated with prehypertension, systolic BP, or diastolic BP. In contrast, although the apnea–hypopnea index

<table>
<thead>
<tr>
<th>Table 2. BP in Subgroups Defined by Sleep Quality</th>
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<tr>
<td>Sleep Efficiency &gt;85% (n=177)</td>
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<tr>
<td><strong>Systolic BP</strong></td>
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<tr>
<td>Systolic BP percentile</td>
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<tr>
<td>Elevated systolic BP (≥90th percentile), n (%)</td>
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<tr>
<td>Mean systolic BP, mm Hg</td>
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<tr>
<td>9 PM</td>
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<td>Mean diastolic BP, mm Hg</td>
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<td>8 AM</td>
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<td>9:30 AM</td>
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<tr>
<td><strong>Diastolic BP</strong></td>
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<td>Diastolic BP percentile</td>
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<td>Elevated diastolic BP (≥90th percentile), n (%)</td>
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<tr>
<td>9 PM</td>
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<td>Mean diastolic BP, mm Hg</td>
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<tr>
<td>8 AM</td>
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<tr>
<td>9:30 AM</td>
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<tr>
<td>Prehypertension, n (%)</td>
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<td>Hypertension, n (%)</td>
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<tr>
<td><strong>Values are mean±SD or median (interquartile values) as appropriate.</strong></td>
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</table>

Short sleep duration was associated with a 2.8-fold increased odds of prehypertension in unadjusted analyses (95% CI, 1.07 to 7.34), but this association was modestly attenuated after adjustment for sex, BMI percentile, and SES z score (odds ratio [OR], 2.54; 95% CI, 0.93, 6.90).

The unadjusted and adjusted associations between continuously measured systolic and diastolic BP levels with sleep efficiency are shown in Table 5. After adjustment for age, sex, race, term status, BMI percentile, and SES z score, the model predicts that each 5% increase in sleep efficiency was associated with a 1.5 ± 0.40-mm Hg decrease in systolic BP (P<0.001). Weaker associations were observed between sleep efficiency and diastolic BP; ie, each 5% increase in sleep efficiency was associated with a 0.65 ± 0.35-mm Hg decrease in diastolic BP (P=0.05). When low sleep efficiency was modeled as a dichotomous exposure, the adjusted model estimates that adolescents with low sleep efficiency had a mean systolic BP that was on average 3.99 ± 1.24 mm Hg higher compared with those with higher sleep efficiency (P=0.0002). Including sleep duration as a continuously measured covariate did not alter the primary associations of sleep efficiency and BP (data not shown).

Similar to the models of prehypertension, sleep duration was more weakly associated with continuously measured systolic and diastolic BPs compared with sleep efficiency (Table 6).

<table>
<thead>
<tr>
<th>Table 3. Association Between Low Sleep Efficiency and Odds of Prehypertension</th>
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<tbody>
<tr>
<td>Unadjusted OR (95% CI)</td>
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<td>-----------------------------------------------</td>
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<tr>
<td>Low sleep efficiency (≤85%)</td>
</tr>
<tr>
<td>Male</td>
</tr>
<tr>
<td>BMI percentile (per 10-unit increase)</td>
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<tr>
<td>SES z score</td>
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</tbody>
</table>
does not confound the association between the outcomes and the sleep exposures, it was associated with increased odds of prehypertension after adjustment for low sleep efficiency, sex, and BMI percentile; ie, for each 1-unit increase in the apnea–hypopnea index, the odds of prehypertension increased by 47% (OR, 1.47; 95% CI, 1.00 to 2.17). Similarly, the apnea–hypopnea index was significantly associated with systolic BP in adjusted models; after adjustment for subject characteristics and low sleep efficiency, for each 1-unit increase in the apnea–hypopnea index, mean systolic BP increases by 1.65 mm Hg on average ($P=0.009$).

**Discussion**

To the best of our knowledge, this is the first reported association between low sleep efficiency and short sleep duration objectively measured in the child’s usual sleep environment with elevated BP (prehypertension or hypertension) in adolescents without clinically significant levels of sleep apnea. Specifically, adolescents with poor sleep quality, as measured by a sleep efficiency of $\approx 85\%$, were at 3.5-fold increased odds of being prehypertensive or hypertensive. Similar findings were observed when single-night polysomnography was used to quantify sleep efficiency. The association between low sleep efficiency and prehypertension persisted even after adjustment for sex, SES, and adiposity. The results did not change appreciably after adjustment for snoring or the apnea–hypopnea index. Short sleep duration also was associated with a 2.5-fold increase in the odds of prehypertension or hypertension. However, it was not clear whether this association was attributable to the low sleep efficiency found in a majority of the adolescents with short sleep duration.

In adults, poor sleep quality identified by questionnaires has been reported in association with an increased prevalence of hypertension, and an increased rate of “nondipper hypertension.” However, poor sleep quality in adults often occurs in the presence of primary sleep disorders such as sleep apnea or insomnia or secondary to numerous comorbidities. Therefore, adult studies reporting associations with disturbed or reduced sleep and hypertension have been cautiously interpreted because of concerns about residual confounding.

One large prospective study reported associations of short sleep duration in women but not men; another study showed no association of hypertension and sleep duration in the elderly, a group with a high prevalence of morbidities. Because adolescents with major comorbidities, including those with clinically significant levels of sleep apnea, were excluded from our analyses (to minimize confounding and to reduce measurement error), it is unlikely that major confounding resulting from medical illnesses, medications, or sleep-related hypoxemia explains the strong association between low sleep efficiency and elevated BP. Given that the association between BP and low sleep efficiency persisted even after adjustment for average sleep duration, our findings also suggest that recurrent arousals or awakenings from sleep (which reduce sleep efficiency) are associated with elevated BP. Our findings are consistent with a report from a sample of preadolescent children studied with single-night polysomnography that demonstrated an association between low sleep efficiency and elevated BP after adjustment for the apnea–hypopnea index.

The 3.5-fold increased odds of prehypertension or hypertension in children with low sleep efficiency, if causal, suggests associations with a potential large public health impact. Although the overall prevalence of low sleep efficiency in general pediatric samples is unknown, our prevalence of 26% is likely an underestimate given the exclusion of children with sleep disorders and significant comorbidities. Our finding of an increased prevalence of low sleep efficiency among vulnerable population subgroups such as poor children and those of minority ethnicity may be of special concern because these groups are known to be at risk for hypertension and other adverse health outcomes.

Low sleep efficiency was associated with an average adjusted increase in systolic BP of 4 mm Hg. Although limited data are available in children to interpret the clinical significance of this absolute elevation, large cohort studies suggest a log-linear increase in morbidity in association with incremental changes in systolic BP.

Short sleep duration was associated with a 2.5-fold increased odds of prehypertension, an association attributable partly to low sleep efficiency. Short sleep duration has been increasing in all ages and is associated with an increased risk for obesity. Thus, efforts to optimize sleep in childhood may improve the BP profile of children through obesity-dependent and independent mechanisms.

**Table 5. Association Between Actigraphy Sleep Efficiency (per 1% Increase) and Continuously Measured BP**

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted OR (95% CI)</th>
<th>$P$</th>
<th>Adjusted OR (95% CI)</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td>**Short sleep ($\leq 6.5$ h)</td>
<td>2.79 (1.07–7.34)</td>
<td>0.0366</td>
<td>2.54 (0.93–6.90)</td>
<td>0.0679</td>
</tr>
<tr>
<td>Male</td>
<td>...</td>
<td>...</td>
<td>2.20 (0.99–4.88)</td>
<td>0.0523</td>
</tr>
<tr>
<td>BMI percentile (per 10-unit increase)</td>
<td>...</td>
<td>...</td>
<td>1.13 (0.98–1.31)</td>
<td>0.1042</td>
</tr>
<tr>
<td>SES z score</td>
<td>...</td>
<td>...</td>
<td>0.75 (0.46–1.23)</td>
<td>0.2526</td>
</tr>
</tbody>
</table>

*Adjusted for age, sex, race, term status, BMI percentile, and SES z score.

**Table 6. Association Between Actigraphy Sleep Duration (per 1-Hour Increase) and Continuously Measured BP**

<table>
<thead>
<tr>
<th></th>
<th>Unadjusted</th>
<th>$\beta\pm SE$</th>
<th>$P$</th>
<th>Adjusted</th>
<th>$\beta\pm SE$</th>
<th>$P$</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Systolic BP</strong></td>
<td>–1.74±0.53</td>
<td>0.0012</td>
<td>–0.98±0.52</td>
<td>0.06</td>
<td></td>
<td></td>
</tr>
<tr>
<td><strong>Diastolic BP</strong></td>
<td>–0.60±0.41</td>
<td>0.15</td>
<td>–0.41±0.44</td>
<td>0.34</td>
<td></td>
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</tbody>
</table>

*Adjusted for age, sex, race, term status, BMI percentile, and SES z score.
pathways. Further work is needed to dissect the relative influences of sleep curtailment from sleep disruption on health outcomes, which will be important in determining whether future interventions would be best directed at improving sleep time, sleep consolidation, or both.

The cause of low sleep efficiency in healthy adolescents is unclear. Sensitivity analyses did not indicate an association between low sleep efficiency and common childhood disorders such as asthma or ADHD or caffeine or tobacco use, nor were these variables confounders in the association between sleep efficiency and BP (data not shown). It is possible that unknown psychological disorders may have confounded our results, but this seems unlikely given the strong associations and community sampling design.

Although children with significant sleep apnea were excluded from our analyses, the apnea–hypopnea index (in a range of 0 to 4.9) was significantly associated with prehypertension and systolic BP after adjustment for sleep efficiency. This suggests that even mild sleep-disordered breathing may contribute to abnormal BP levels, a result consistent with reports of more severely affected children from sleep clinic samples.

Strengths of this report are the inclusion of a community-based sample of children, minimizing referral biases, and the use of objective measures of sleep duration and multiple measures of BP, minimizing measurement error and reporting biases. By characterizing numerous risk factors and comorbidities, we were able to restrict the analytical sample to children without disorders likely to confound associations with sleep quality. Although former preterm children were overrepresented by design, there was no evidence of any differences in the exposures, responses, or associations between preterm and full-term children, suggesting that our results should be generalizable to other pediatric samples.

There are no established cutoffs to define thresholds of sleep duration or sleep efficiency that increase morbidity in adolescents. In adults, sleep durations of <6 hours have been associated with a variety of adverse health outcomes, and sleep efficiencies of <85% are considered low. Our choice for defining short sleep duration as <6.5 hours was to approximate the cutoff associated with hypertension risk in adults, which, in our sample, represented the lowest decile. However, examination of a larger sample may permit a more comprehensive assessment of dose response and threshold levels for each sleep exposure.

A limitation of this cross-sectional study is that BP status was determined from measurements made on 2 consecutive days. Because BP may vary from day to day, repeated measurements over time are needed to identify children with persistent elevations in BP. Another limitation is that the reported associations do not provide proof of causality. We also cannot exclude the possibility that elevated BP operates as a risk factor for poor sleep. It is important, however, to interpret our findings in light of the biological plausibility of the observed associations and experimental data that show acute effects of sleep disruption on BP. Mechanisms linking poor sleep efficiency or sleep deprivation with hypertension may be through disruptions in cortisol secretion, stimulation of the renin-angiotensin system and sympathetic nervous system as measured by increased secretion of catecholamines and abnormalities in sympathovagal balance, and abnormal secretion of vasoactive hormones, including endothelin, vasopressin, and aldosterone. Experimental sleep disruption has been associated with elevated BP in sleep in normal subjects. Although some experimental models suggest that sustained elevations in BP require sleep fragmentation to occur in a background of intermittent hypoxemia (as occurs with sleep apnea), sleep fragmentation may be associated with elevated BP even in adults with a low apnea–hypopnea index or with simple snoring. Prospective and interventional studies are needed to provide further evidence of causality and to address whether improving sleep quality and duration reduces BP and risk of hypertension.

Conclusions

Extensive analyses using objective measures of sleep quality and duration and multiple measures of BP provide evidence for a strong association of low sleep efficiency with increased risk of prehypertension and hypertension in a healthy sample of adolescents. Our data suggest that low sleep efficiency may be associated more consistently with prehypertension than short sleep duration. Future research is needed to address whether prevention of hypertension in children should include not only weight management and exercise but also optimization of sleep. Our data underscore the need to monitor the quantity and quality of sleep as part of health supervision in children.

Sources of Funding

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Disclosures

Dr Rosen has received a subcontract from Advanced Brain Monitoring Inc to provide clinical research services funded through NIH Small Business Innovative Research and has received honoraria for society-sponsored educational talks. Dr Redline has received a subcontract from Cleveland Medical Devices Inc to provide clinical research services as part of NIH Small Business Innovative Research funding. The other authors report no conflicts.

References


CLINICAL PERSPECTIVE
Childhood hypertension is a risk factor for adult hypertension and for target-organ damage. Early recognition and intervention of childhood hypertension are believed to be important in reducing the risk of cardiovascular morbidity in adulthood. Traditional approaches for intervention focus on the role of overweight as a contributing cause of hypertension and include weight reduction, increased physical activity, and nutritional changes. The present report identifies a significant association between increased blood pressure and poor sleep quality (ie, increased wake time during the sleep period), found in 26% of a community sample of adolescents. Independent of obesity, sex, and socioeconomic status and unrelated to sleep apnea, adolescents with poor sleep had a 3.5-fold increased risk of prehypertension or hypertension. This finding suggests that approaches for optimizing sleep quality and duration in children may complement other behavioral approaches for preventing or treating pediatric hypertension. Monitoring sleep quality and duration in children as part of their health supervision may help to identify children who are at risk for both sleep problems and hypertension and who would benefit from behavioral interventions aimed at improving sleep.