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Nocturnal blood pressure patterns in pediatric obstructive sleep apnea: The Kids Trial study

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ABSTRACT

Obstructive sleep apnea (OSA) in children is associated with cardiovascular risk, but its impact on sleep blood pressure (BP) remains incompletely understood.
Objective: To assess the association between the severity of OSA, stratified by the obstructive apnea-hypopnea index (OAHI), and altered sleep BP patterns, specifically the non-dipping pattern (NDP), using ambulatory 24-h BP monitoring (ABPM).

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Methods: The Kids-Trial is a multicenter observational trial (NCT03696654) conducted in Spain, including children with suspected OSA that underwent polysomnography for its diagnosis. To estimate the impact of OAHI on the risk of NDP, a multivariable logistic regression model was constructed and odds ratio (OR) was calculated.
Results: A total of 267 children were included (median age of 6 years and 42.7 % girls). When BP was assessed through z-score, significant positive trends were observed according to OSA severity in 24-h mean BP, 24-h systolic BP, sleep mean BP, sleep systolic BP, sleep diastolic BP, and NDP. A negative trend was observed for the drop in sleep mean BP. The adjusted model showed that severe OSA was independently associated with a reduced drop in mean BP (−3.68 (−6.9; −0.47); p = 0.026). Adjusted multivariable analysis of NDP showed an increased but not significant risk of NDP in severe OSA vs. non-OSA (OR = 2.76 (0.92, 8.30)).
Conclusion: Severe pediatric OSA was independently associated with disrupted nocturnal BP regulation, including a reduced BP dipping. These findings underscore the importance of ABPM in pediatric sleep evaluations.
Clinical trial registration: clinicaltrials.gov NCT03696654.

1. Introduction

Obstructive sleep apnea (OSA) is characterized by repetitive episodes of upper airway obstruction during sleep which are associated with blood pressure (BP) rises due to enhanced sympathetic activation [1,2]. Pediatric OSA offers important advantages for study, as children are generally free of cardiovascular (CV) risk factors and its consequences may be reversible. Moreover, adenotonsillectomy is the most effective treatment for pediatric OSA, with efficacy that does not depend on patient adherence [3].

The CV risk associated with OSA in children has been extensively studied [4] with the strongest evidence associating OSA with BP rises. Persistent OSA in children is associated with an increased risk of future hypertension [5–7] and future coronary heart disease in adulthood [8]. Increases in both systolic (SBP) and diastolic (DBP) BP and BP variability have been reported [9] and tend to persist at follow-up [10,11]. Nighttime BP levels and non-dipping pattern (NDP) are considered CV risk factors, as they both were independently associated with the total future CV event rate [12]. Nevertheless, it is still controversial whether reduced nocturnal dipping occurs in children with OSA [13–17]. A few studies have been conducted with 24-h ambulatory BP monitoring (ABPM) [18–20], losing relevant information of circadian BP patterns. Despite the importance of the nocturnal pattern, few studies have examined the influence of the NDP in pediatric patients with OSA as a marker of future CV risk [13,19,21]. Therefore, CV health requires maintaining circadian BP regulation (including 24-h control, adequate circadian rhythm, and appropriate BP variability), rather than focusing solely on BP values [22].

The American Heart Association (AHA) advocates for the increased utilization of ABPM in pediatric populations, with an emphasis on those exhibiting elevated CV risk, including OSA [23]. Despite the high prevalence of OSA in preschool children, most clinical studies have focused on children older than five years, resulting in a paucity of research on younger children. In this context, the present study provides novel evidence addressing a key gap in the literature, contributing to a better understanding of early CV consequences, and helping to address the management of OSA in this vulnerable population.

For these reasons, accurate studies to evaluate BP fluctuation in pediatric patients with OSA are needed to understand the impact of the sleep disorder on BP profile and overall CV risk. We hypothesized that children with OSA would show a higher risk of a NDP and that this risk would increase in proportion to OSA severity. The aim was to assess whether the severity of OSA, stratified by the obstructive apnea-hypopnea index (OAHI), is associated with altered nocturnal BP patterns, specifically the NDP, using 24-h ABPM.

2. Materials and methods

The Kids Trial study (NCT03696654) is a multicenter, longitudinal, prospective study described previously [24]. Data were accrued between January 30, 2018, and August 28, 2023. Children aged 4 to 18 years who were referred to the sleep unit with suspected OSA based on parental report of habitual snoring and/or witnessed apnea were consecutively enrolled. Exclusion criteria were: 1) cardiovascular, cerebrovascular, or unstable severe or worsening respiratory disease, 2) genetic disease, 3) chronic insomnia and/or depressive syndrome, 4) previous upper airway surgery and/or continuous positive airway pressure (CPAP) treatment, and 5) contraindication to ABPM, including arrhythmias, latex allergy, or coagulation disorders.

2.1. Sleep study

In hospital full polysomnography (PSG) was performed and scored according to the guidelines of the American Academy of Sleep Medicine (AASM) 2017 [25] including electroencephalogram, electrooculogram, electromyogram and respiratory signals. Respiratory events were scored according to the pediatric scoring rules: apnea was defined as a flow decrease >90 % in two respiratory cycles for obstructive apnea and >20 s or two respiratory cycles accompanied by a 3 % oxygen desaturation or arousal in central apnea. Hypopnea was defined as a 90–30 % flow decrease in two respiratory cycles with a 3 % desaturation or microarousal. The PSG was valid if it had >300 min of study and >180 min of sleep. Apnea-hypopnea index (AHI) was established as the number of apneas plus hypopneas per hour of sleep. The OAHI was defined as the summatory of the number of obstructive apneas, obstructive hypopneas and mixed apneas divided by the total hours of sleep recorded during the PSG study. Subjects with OAHI greater than 1 per hour of sleep were classified as having OSA, as suggested by the European Respiratory Society (ERS) statement on pediatric OSA [26]. An OAHI <1/h was considered non-pathological. Severity of OSA was classified as mild when OAHI was from 1 to 4.9/h, moderate when OAHI was from 5 to 9.9/h and severe when OAHI was greater than 10/h. Other variables recorded included sleep efficiency, sleep latency, % of N1, N2, and N3, rapid eye movement (REM), arousal index, average peripheral oxygen saturation (SpO_2), nadir SpO_2, max end-tidal CO_2 (Et CO_2), average Et CO_2, and hypoventilation (%).

2.2. Blood pressure study

Within a month from the PSG, office BP was measured at the clinic with a pediatric sphygmomanometer (SVM-7160 Nihon Kohden) validated for pediatric age and was used in the non-dominant arm. The patient needed to be seated for at least 5 min before the BP measures and to remain seated with uncrossed legs and an empty bladder in a quiet environment. Three BP measurements were taken every 3 min, discarding the first one and averaging the last two.

The same day as the office BP (to be used for calibration of the ABPM), all children underwent ABPM (Spacelabs OnTrak) according to European guidelines [27]. All ABPM recordings in our study were analyzed centrally by a single core laboratory, using standardized procedures and predefined quality criteria. The monitors were transported in pouches positioned laterally to the patient using straps and/or belts.

Validated paediatric ABPM devices and cuffs were applied to the non-dominant arm. BP measurements were measured every 20 min until 10 p.m. and every 30 min during the night. The test was considered valid when at least one reading per hour was available, with 40 valid readings and 65 % of the programmed readings being required for its successful completion. Daytime and nighttime periods were established based on the parents-reported wake up and bedtimes. Monitor-recorded BP data were then downloaded for analysis by the central core lab. BP indices were calculated for both SBP and DBP, including 24-h BP, sleep BP, and wake BP. The dipping status was determined by calculating the percentage decline in mean arterial pressure (MAP) from wake to sleep values. A NDP was defined when the sleep BP reduction was less than 10% [27]. To account for age- and sex-related changes in ABPM during childhood, we calculated standardized deviation scores (SDS; equivalent to z-scores) for 24-h, wake, and sleep SBP, DBP, and MAP. SDS were derived using the sex-specific LMS reference values published by Wühl et al. for pediatric ABPM [28].

2.3. Demographic and clinical characteristics

Demographic data including sex and age were recorded. For clinically relevant characteristic assessment, parents completed the Paediatric Sleep Questionnaire (PSQ) [29]. The PSQ score was calculated by summing all "yes" [1] responses and dividing by the number of "yes" and "no" (0) responses, excluding non-answered questions. A cutoff score of 0.33 was established to be considered positive. Physical examination including anthropometric measurements (body mass index-BMI, neck, waist, and hip circumference), in office BP (SBP and DBP), and otorhinolaryngology examination (Mallampati, tonsil size, palate and mandible classification) were systematically performed in all patients. Data of micro-retrognathia (Yes/No), Mallampati (I, II, III, III/IV), tonsillar hypertrophy (I, II, III/IV), adenoid hypertrophy (Yes/No), ogival palate (Yes/No), and bite (III/II/open/asymmetric) were taken. Body mass index (BMI) was calculated as weight (kg) divided by height squared (m^2). BMI-for-age z-scores were computed using World Health Organization (WHO) growth references, applying the WHO Child Growth Standards for children <5 years and the WHO 2007 reference for ages 5–19 years, using sex and age in months [30,31].

The study was conducted in accordance with the Declaration of Helsinki and approved by the Ethics and Clinical Trials Committee of the Hospital Universitario de Guadalajara (P02/18) and the parents/children signed an informed consent form.

2.4. Statistical analysis

Data correspond to a secondary cross-sectional analysis of baseline assessments from the Kids Trial (NCT03696654) [24]; therefore, the original trial sample size calculation was not based on the objectives of the present study. No imputation of missing data was performed. Analyses were conducted in all eligible participants with valid PSG-derived OAHI data and valid ABPM measurements permitting calculation of day–night MAP decline; participants were not excluded based on dipping status. Missingness for other variables was reported when present, and multivariable models were fitted in the subset with complete data for all model covariates (no further case loss within models).

Continuous variables are summarized as median (P25–P75), and categorical variables as counts and percentages. OSA severity was analyzed using prespecified ordered categories of OAHI (<1, 1 to <5, 5 to <10, and ≥10 events/h), based on clinically used pediatric OSA severity thresholds. For the trend analyses, these OAHI categories were treated as an ordinal variable to assess whether BP outcomes changed progressively across increasing OSA severity, rather than to model the effect of OAHI as a continuous predictor. Trends across ordered OAHI categories were assessed using: (i) Spearman rank correlation test for continuous variables; (ii) Cochran–Armitage test for trend for dichotomous variables; and (iii) Mantel–Haenszel linear-by-linear association test for ordinal categorical variables.

The association between OAHI severity and a NDP was examined using multivariable logistic regression adjusting for sex, age, and BMI-for-age z-score. In addition, multivariable linear regression was used to model nocturnal MAP decline (percentage) and the standardized day–night MAP difference, using the same covariate set. Logistic regression results are reported as odds ratios (OR) with 95% confidence intervals (CI) based on Wald standard errors; linear regression results are reported as regression coefficients with 95% Wald CIs. All tests were two-sided, with a significance level of 0.05. Analyses were performed in R (R Foundation for Statistical Computing, Vienna, Austria).

3. Results

3.1. Patient characteristics

Of the 286 children in total included in the Kids Trial study, 267 were selected as evaluable for the present analysis. Children were excluded due to missing data from PSG and/or because they did not meet the established quality criteria for ABPM. The children presented a median (25th and 75th percentile) age of 6 [5,8] years, of whom 118 (42.7%) were girls. The anthropometric characteristics showed a BMI z-score of 0.5 (−0.3, 1.8). The PSQ was positive in 61% of children. In terms of symptoms, 59% of the participants snored more than half of the time, 45% had breathing problems, 55.3% had witnessed apneas, 29.3% had enuresis, and 7.77% had growth retardation.

The mean office SBP was 101.5 (94.8, 108.8) mmHg, and the mean office DBP was 65.0 (60.0, 70.5) mmHg. The most common otorhinolaryngology physical finding was tonsillar hypertrophy, with 46.6% of patients having a score of 3/4. The rest of the examination (palate, mandible, and bite) was normal in most patients (Table 1).

3.2. Polysomnographic variables and OSA severity groups

The median AHI was 6.2/h (3.3, 11.1) events per hour. Children with OSA were classified according to OAHI in four severity groups: non-OSA: 20 (7.49%), mild OSA: 111 (41.57%), moderate OSA: 75 (28.09%) and severe OSA: 61 (22.85%). Additional PSG variables, and their characteristics related to OSA severity are presented in Table 2.

3.3. 24-H ambulatory blood pressure parameters

Wake and sleep MAP, SBP and DBP parameters from ABPM are shown in Table 3.

Significant positive trends were observed in 24-h MAP, 24-h SBP, sleep SBP, sleep DBP and sleep MAP. A negative trend was observed in the drop in sleep MAP. A more frequent presence of NDP across OSA severity groups was noticed. No statistically significant trends were observed for wake BP values (Table 3 and Fig. 1). To account for age- and sex-related changes in ABPM, the same results were found when BP was assessed through SDS (z-score) (Table 3).

Multivariable linear regression model adjusted for sex, age and BMI showed that the presence of OSA was independently associated with a reduced drop in sleep MAP, with an adjusted coefficient of −3.68 (95% confidence interval [CI]: −6.9; −0.47, $p = 0.026$). Age was also independently associated with the outcome (0.42 (0.118; 0.722), $p = 0.007$) (Fig. 2A). A similar trend for a reduced drop in MAP SDS was observed. Multivariable logistic regression analysis adjusted for sex, age, and BMI, showed a non-significant increase in the odds of NDP for OAHI ≥10/h compared with OAHI <1/h (OR 2.76, 95% CI (0.916; 8.3)) (Fig. 2B).

4. Discussion

This secondary analysis of the Kids Trial study is among the first to systematically demonstrate that severe pediatric OSA is independently associated with reduced drop in sleep MAP using PSG and ABPM in a

Table 1
Patient characteristics.

	Total *N = 267*	OAHI <1/h N = 20	1 ≤ OAHI <5/h N = 111	5 ≤ OAHI <10/h N = 75	OAHI ≥10/h N = 61	p value
Demographic characteristics						
Age [years]	6 (5,8)	6 (6,8)	6 (5,8)	6 (5,9)	6 (4,8)	0.757
Sex [female]	114 (42.7%)	9 (45.0%)	52 (46.8%)	27 (36.0%)	26 (42.6%)	0.449
Anthropometric characteristics						
Weight [kg]	23.5 (19.3,34.5)	24.7 (20.3,36.3)	22.5 (19.2,29.7)	24.4 (20.1,35.0)	23.8 (18.7,35.6)	0.686
BMI z-score	0.5 (−0.3, 1.8)	0.7 (−0.3, 1.3)	0.3 (−0.5, 1.8)	0.7 (−0.3, 1.9)	0.9 (−0.2, 1.8)	0.225
Neck circumference [cm]	27.0 (25.5,29.0)	27.0 (26.4,28.5)	26.5 (25.5,28.5)	27.0 (26.0,30.0)	27.0 (25.0,30.0)	0.701
Waist circumference [cm]	58.0 (53.0,66.0)	57.3 (53.0,65.3)	57.0 (53.3,65.0)	58.5 (55.0,67.5)	58.0 (52.8,69.5)	0.692
Hip circumference [cm]	64.0 (59.0,75.0)	64.8 (59.4,76.5)	63.5 (58.3,70.0)	66.0 (60.5,77.0)	63.0 (58.5,78.5)	0.736
Office SBP [mmHg]	101.5 (94.8108.8)	100.3 (93.4109.0)	101.5 (94.8106.8)	100.0 (93.5110.3)	102.5 (97.5110.0)	0.160
Office DBP [mmHg]	65.0 (60.0,70.5)	62.3 (58.8,67.9)	65.5 (60.3,69.8)	64.5 (58.3,71.3)	66.5 (61.0,71.5)	0.270
PSQ positive (abnormal)	163 (61.0%)	11 (55.0%)	67 (60.4%)	45 (60.0%)	40 (65.6%)	0.413
Otorhinolaryngology physical examination						
Mallampati classification						0.838
Class 1	61 (23.6%)	5 (26.3%)	23 (21.3%)	17 (23.3%)	16 (27.6%)	
Class 2	139 (53.9%)	12 (63.2%)	59 (54.6%)	38 (52.1%)	30 (51.7%)	
Class 3	58 (22.5%)	2 (10.5%)	26 (24.1%)	18 (24.7%)	12 (20.7%)	
Class 4	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Missing	*9*	*1*	*3*	*2*	*3*	
Tonsil size						0.075
Grade 1 (0–25 %)	10 (3.8%)	0 (0.0%)	4 (3.7%)	6 (8.1%)	0 (0.0%)	
Grade 2 (26–50 %)	61 (23.1%)	6 (30.0%)	29 (26.6%)	16 (21.6%)	10 (16.4%)	
Grade 3 (51–75 %)	123 (46.6%)	8 (40.0%)	53 (48.6%)	32 (43.2%)	30 (49.2%)	
Grade 4 (76–100 %)	70 (26.5%)	6 (30.0%)	23 (21.1%)	20 (27.0%)	21 (34.4%)	
Missing	*3*	*0*	*2*	*1*	*0*	
Palate (ogival)	85 (32.4%)	4 (20.0%)	41 (38.0%)	23 (31.1%)	17 (28.3%)	0.618
	5	*0*	*3*	*1*	*1*	
Mandible (abnormal)	48 (18.1%)	6 (30.0%)	25 (22.7%)	9 (12.0%)	8 (13.3%)	0.026 *
Missing	*2*	*0*	*1*	*0*	*1*	
Bite						0.595
Class 3	17 (20.2%)	2 (28.6%)	9 (21.4%)	4 (18.2%)	2 (15.4%)	
Class 2	53 (63.1%)	4 (57.1%)	22 (52.4%)	17 (77.3%)	10 (76.9%)	
Open	14 (16.7%)	1 (14.3%)	11 (26.2%)	1 (4.5%)	1 (7.7%)	
Asymmetric	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	0 (0.0%)	
Missing	183	13	69	53	48	

Frequencies and percentages have been obtained for the categorical variables and median (P25 and P75 percentiles) for the continuous variables. Obstructive apnea-hypopnea index (OAHI), Body mass index (BMI), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Pediatric Sleep Questionnaire (PSQ).

large, multicenter cohort. The major impact of OSA in BP was seen for sleep BP parameters only whilst wake BP did not show significant associations. This study also showed a trend in increased risk of NDP related to severe OSA compared with non-OSA, although it was non-significant.

Previous cross-sectional studies focusing on the contributing effects of OSA on BP outcomes in children have demonstrated similar results. Kwok et al. [32] showed that pediatric OSA was associated with BP dysregulation, but not with other CV co-morbidities. A meta-analysis published in 2022 [9] summarized the current evidence and concluded that OSA was associated with elevated daytime and nighttime SBP. The findings of our study confirm and in part extend the results of this meta-analysis in that BP was affected exclusively at night, without differences in daytime values. Furthermore, this nocturnal alteration occurred not only for SBP but also affected DBP. These aspects enhance the concept that OSA can influence circadian BP regulation in the absence of other confounding CV risk factors. As adverse CV outcomes have been strongly associated with circadian BP patterns disruption [33], OSA may increase CV risk even at an early age. This greater impact on nocturnal BP reinforces the biological plausibility of the influence of OSA on the alterations. Respiratory events cause autonomic instability that increases nocturnal sympathetic activity determining BP and heart rate rises during sleep [21].

In agreement to our results, a recent study reported BP differences between previously diagnosed OSA children (n = 102) and controls (n = 117) [21]. Children with moderate to severe OSA had changes in circadian BP patterns, measured by earlier BP peaks and nadirs and time arrived at peak BP velocity than controls. Performing a sleep study was an inclusion criterion, but this was not standardized as it was not part of the study protocol, where only actigraphy was performed. Furthermore, BP was evaluated through BP timing of peak systolic and diastolic BP velocities. Accordingly, Amin et al., 2004 [13] showed a significant linear trend for the difference in MAP across severity groups. However, Horne et al., 2013 [16] described that nocturnal dipping was preserved in children with OSA regardless of its severity. Leung et al., 2006 [19] found a similar proportion of NDP in children with OSA and controls. In contrast to previous studies, our study adds novel evidence by showing that OSA severity is related with higher nocturnal BP levels, both systolic and diastolic, together with a higher frequency of NDP. These findings are particularly relevant because the assessment was performed using two gold-standard and clinically applicable tools: in-hospital PSG for OSA diagnosis and 24-h ABPM for BP evaluation.

Our results suggest that elevated OAHI may be associated with a higher prevalence of NDP, particularly in the severe group, as we observed a non-significant trend toward a higher prevalence of NDP across increasing OAHI categories. If confirmed in future studies, this would support the hypothesis that more severe OSA is associated with impaired BP regulation.

It has been suggested that the lack of BP dipping and increased BP values during childhood could be associated with high BP and increased CV risk in early adulthood [5,10,34]. Therefore, its detection would be crucial for preventing long-term complications. Supporting this future risk, Ai et al., 2022 [9], showed that moderate-to-severe childhood OSA presented a significantly higher SBP in late adolescence or young adulthood, but such differences were not found in those with mild OSA. More recently, in a secondary analysis of the Kids Trial study, higher hypoxic burden, area under the desaturation curve of each respiratory event, was independently associated with elevated nocturnal BP and a

Table 2
Polysomnographic variables.

	Total *N = 267*	OAHI <1/h *N = 20*	1 ≤ OAHI <5/h *N = 111*	5 ≤ OAHI <10/h *N = 75*	OAHI ≥10/h *N = 61*	p value
Polysomnographic variables						
TST [min]	430.5 (399.5, 463.9)	410.2 (394.4, 432.1)	431.8 (401.4, 459.6)	430.5 (401.7, 470.3)	433.0 (395.0, 481.5)	0.138
Sleep efficiency [% TST]	91.1 (85.6, 95.4)	89.1 (82.4, 94.6)	91.7 (86.5, 95.8)	90.5 (84.2, 94.2)	90.9 (86.2, 96.6)	0.706
NREM latency [min]	9.8 (3.6, 19.8)	9.8 (5.5, 14.8)	8.6 (2.8, 19.1)	10.3 (5.4, 22.5)	10.0 (1.8, 20.0)	0.663
REM latency [min]	106.5 (71.8, 149.8)	129.5 (90.3, 178.0)	98.0 (69.3, 137.8)	103.5 (71.0, 143.0)	109.2 (75.0, 154.8)	0.822
WASO [min]	27.0 (13.0, 48.1)	37.0 (15.4, 60.8)	24.8 (12.1, 44.8)	32.6 (17.6, 52.5)	22.5 (10.0, 42.0)	0.504
N1 [% TST]	2.4 (0.5, 5.5)	2.4 (0.8, 5.1)	2.0 (0.4, 5.4)	2.8 (1.0, 5.9)	2.0 (0.2, 5.4)	0.763
N2 [% TST]	33.4 (20.3, 42.2)	36.3 (20.5, 38.8)	31.4 (16.8, 41.4)	32.5 (22.7, 44.4)	33.6 (25.3, 41.0)	0.397
N3 [% TST]	43.3 (33.6, 54.6)	44.5 (35.7, 56.3)	44.6 (34.6, 58.8)	43.7 (31.8, 53.2)	42.2 (33.8, 47.8)	0.108
REM [% TST]	21.1 (16.4, 25.0)	19.1 (12.6, 24.3)	21.6 (17.0, 25.1)	20.4 (16.2, 25.0)	21.1 (16.5, 25.2)	0.660
Total arousal index	12.6 (8.8, 19.4)	8.3 (5.8, 12.4)	11.6 (8.9, 15.9)	12.9 (8.6, 21.1)	17.3 (10.6, 22.9)	<0.001*
Respiratory arousal index	2.0 (0.8, 4.8)	0.2 (0.1, 0.4)	1.5 (0.7, 2.6)	3.1 (1.2, 6.1)	5.7 (2.9, 12.6)	<0.001*
Spontaneous arousal index	5.2 (3.2, 8.9)	4.1 (3.2, 6.1)	5.5 (3.9, 8.3)	5.5 (2.2, 9.3)	4.9 (3.0, 9.8)	0.951
Arousal index by PLM	3.2 (1.6, 4.9)	3.1 (2.2, 4.9)	3.6 (2.1, 5.1)	3.0 (1.6, 4.6)	2.3 (1.3, 4.3)	0.052
AHI [events/h]	6.2 (3.3, 11.1)	1.4 (0.9, 2.1)	3.5 (2.6, 4.7)	8.0 (7.0, 9.4)	18.9 (13.2, 27.7)	<0.001*
Supine AHI [events/h]	6.8 (3.5, 13.1)	1.1 (0.1, 2.0)	4.2 (2.9, 6.1)	8.6 (6.9, 11.7)	25.7 (15.3, 39.5)	<0.001*
Missing	*10*	*2*	*2*	*3*	*3*	
Non-supine AHI [events/h]	4.9 (2.1, 9.6)	1.6 (0.9, 2.3)	2.9 (1.5, 4.8)	6.4 (4.8, 8.6)	14.8 (9.6, 24.2)	<0.001*
Missing	*8*	*1*	*2*	*4*	*1*	
Central AHI [events/h]	0.7 (0.3, 1.4)	0.7 (0.5, 1.4)	0.6 (0.2, 1.4)	0.8 (0.4, 1.3)	0.9 (0.4, 1.8)	<0.001*
Obstructive AHI [events/h]	5.1 (2.5, 9.4)	0.5 (0.3, 0.7)	2.9 (1.8, 3.8)	6.8 (6.0, 8.4)	18.5 (12.2, 26.2)	<0.001*
Baseline $PaCO_2$ [mmHg]	36.0 (33.3, 39.0)	34.2 (31.3, 36.5)	35.6 (33.4, 38.1)	37.6 (34.5, 41.0)	36.8 (34.0, 39.5)	0.004 *
Maximum $PaCO_2$ [mmHg]	45.0 (41.0, 49.0)	43.0 (38.5, 44.8)	45.0 (41.4, 47.2)	45.0 (40.8, 49.0)	46.7 (42.8, 51.0)	0.010 *
$PaCO_2$ > 50 mmHg [%]	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 0.0)	0.0 (0.0, 2.0)	0.001 *
Minimum SaO_2 [%]	89.0 (86.0, 91.0)	91.0 (89.0, 92.0)	90.0 (88.0, 92.0)	89.0 (86.5, 91.0)	86.0 (81.0, 89.0)	<0.001*
Mean SaO_2 [%]	96.0 (95.0, 97.0)	96.0 (95.0, 97.0)	96.0 (95.0, 97.0)	96.0 (95.0, 97.0)	95.0 (95.0, 96.0)	0.020 *
Desaturation index	3.6 (1.7, 8.1)	2.7 (0.9, 3.8)	2.7 (1.3, 4.7)	3.6 (1.6, 8.0)	15.0 (4.2, 20.2)	<0.001*

Frequencies and percentages have been obtained for the categorical variables and median (P25 and P75 percentiles) for the continuous variables. [1]Spearman rank correlation (trend) was performed. Obstructive apnea-hypopnea index (OAHI), Total sleep time (TST), Non-rapid eye movement (NREM), Rapid eye movement (REM), Wake after sleep onset (WASO), Periodic leg movement (PLM), apnea-hypopnea index (AHI), Partial pressure of carbon dioxide ($PaCO_2$), Oxygen saturation (SaO_2).

Table 3
24-hour ambulatory blood pressure parameters by obstructive sleep apnea severity groups.

ABPM variable [mmHg]	Total *N = 267*	OAHI <1/h N = 20	1 ≤ OAHI <5/h N = 111	5 ≤ OAHI <10/h N = 75	OAHI ≥10/h N = 61	p value
24-h SBP	98.0 (93.0, 105.0)	97.5 (91.5, 102.5)	98.0 (93.0, 103.0)	97.0 (93.5, 107.5)	100.0 (95.0,108.0)	0.012 *
SDS (z-score) 24-h SBP	−1.05 (−2.02, −0.29)	−1.51 (−2.26, 0.64)	−1.05 (−2.16, −0.48)	−1.20 (−2.10, −0.12)	−0.84 (−1.43, 0.01)	0.013*
24-h DBP	64.0 (61.0, 67.0)	63.5 (59.8, 67.0)	63.0 (60.5, 67.0)	64.0 (59.0, 68.0)	64.0 (62.0, 69.0)	0.073
SDS (z-score) 24-h DBP	−0.49 (−1.07, 0.28)	−0.56 (−1.25, 0.03)	−0.53 (−1.08, 0.13)	−0.44 (−1.41, 0.36)	−0.36 (−0.76, 0.51)	0.057
24-h MAP	75.0 (71.0, 79.0)	74.0 (70.0, 77.3)	74.0 (71.0, 78.0)	75.0 (71.0, 80.0)	76.0 (73.0, 81.0)	0.006 *
SDS (z-score) 24-h MAP	−0.63 (−1.29, 0.00)	−0.91 (−1.54, 0.41)	−0.70 (−1.30, −0.14)	−0.75 (−1.39, 0.08)	−0.35 (−0.86, 0.33)	0.003*
Wake SBP	102.0 (97.0, 109.0)	101.5 (97.3, 104.5)	102.0 (96.5, 107.0)	102.0 (97.0, 110.0)	103.0 (98.0,111.0)	0.080
SDS (z-score) Wake SBP	−1.15 (−2.04, −0.42)	−1.61 (−1.99, 0.65)	−1.15 (−2.13, −0.55)	−1.37 (−2.24, −0.29)	−0.97 (−1.68, 0.09)	0.073
Wake DBP	68.0 (63.0, 71.0)	68.5 (64.3, 70.3)	67.0 (63.0, 71.0)	67.0 (63.0, 71.0)	68.0 (64.0, 72.0)	0.454
SDS (z-score) Wake DBP	−0.77 (−1.49, −0.17)	−0.71 (−1.39, 0.32)	−0.88 (−1.50, −0.26)	−0.82 (−1.60, −0.22)	−0.77 (−1.45, 0.00)	0.412
Wake MAP	78.0 (74.0, 82.0)	77.0 (75.8, 80.3)	78.0 (74.0, 81.0)	78.0 (74.0, 82.5)	79.0 (75.0, 85.0)	0.093
SDS (z-score) Wake MAP	−0.91 (−1.60, −0.36)	−1.14 (−1.48, 0.57)	−0.93 (−1.67, −0.51)	−0.98 (−1.71, −0.29)	−0.71 (−1.53, 0.05)	0.069
Sleep SBP	92.0 (86.0, 99.4)	87.0 (83.0, 95.5)	91.0 (85.0, 95.5)	91.0 (86.0, 102.0)	96.0 (91.0,102.0)	<0.001*
SDS (z-score) Sleep SBP	−0.60 (−1.60, 0.22)	−1.50 (−2.02, 0.20)	−0.72 (−1.63, −0.05)	−0.82 (−1.72, 0.49)	−0.19 (−0.75, 0.66)	0.002*
Sleep DBP	57.0 (53.0, 61.0)	53.5 (52.0, 57.3)	56.0 (53.5, 60.0)	57.0 (52.0, 61.0)	59.0 (56.0, 63.0)	0.003 *
SDS (z-score) Sleep DBP	0.33 (−0.42, 0.82)	−0.34 (−0.70, 0.23)	0.19 (−0.35, 0.75)	0.28 (−0.65, 0.98)	0.61 (−0.06, 1.00)	0.003*
Sleep MAP	69.0 (65.0, 73.0)	66.0 (63.0, 70.0)	68.0 (65.0, 71.0)	69.0 (64.5, 74.0)	72.0 (68.0, 75.0)	<0.001*
SDS (z-score) Sleep MAP	0.06 (−0.62, 0.72)	−0.44 (−0.99, 0.07)	0.02 (−0.59, 0.55)	−0.03 (−0.72, 0.68)	0.55 (−0.16, 1.03)	<0.001*
Drop in sleep MAP	11.3 (7.6, 15.6)	15.1 (8.9, 17.7)	11.4 (8.4, 15.6)	11.5 (7.0, 15.4)	9.4 (6.3, 14.1)	0.013 *
Difference Wake - Sleep PAM in SDS	−1.08 (−1.62, −0.44)	−0.59 (−1.33, 0.27)	−1.04 (−1.47, −0.47)	−1.04 (−1.70, −0.32)	−1.20 (−1.85, 0.53)	0.076
Non-dipping pattern	114 (42.7%)	6 (30.0%)	42 (37.8%)	32 (42.7%)	34 (55.7%)	0.012*

Frequencies and percentages have been obtained for the categorical variables and median (P25 and P75 percentiles) for the continuous variables. Ambulatory blood pressure monitoring (ABPM), Obstructive apnea-hypopnea index (OAHI), Systolic blood pressure (SBP), Standardized deviation scores (SDS), Diastolic blood pressure (DBP), Mean arterial pressure (MAP).

higher risk of NDP—findings from our group that align with and contextualize the nocturnal BP dysregulation [35]. Early OSA detection and treatment may promote CV health in children and future adulthood. Fortunately, and considering that OSA is very prevalent at very early ages, identifying the presence of NDP does not require reference values (only available in >5 years old) and can be identified by assessing the drop (<10 %) in BP between day and night in the same patient.

Age was independently associated with drop in MAP and NDP. Previous studies established the prevalence of OSA peaks between 2 and 8 years of age, corresponding to a peak prevalence of adenotonsillar hypertrophy [36,37], whilst others correlated obesity as a persistent risk factor for OSA in middle childhood and late adolescence [38]. There are other pathophysiologic mechanisms proposed to explain the presence of OSA in children such as the upper airway (dependent on craniofacial and soft tissue structures), increases in upper airway resistance, retro positioning of the mandible, micrognathia, high arched palate,

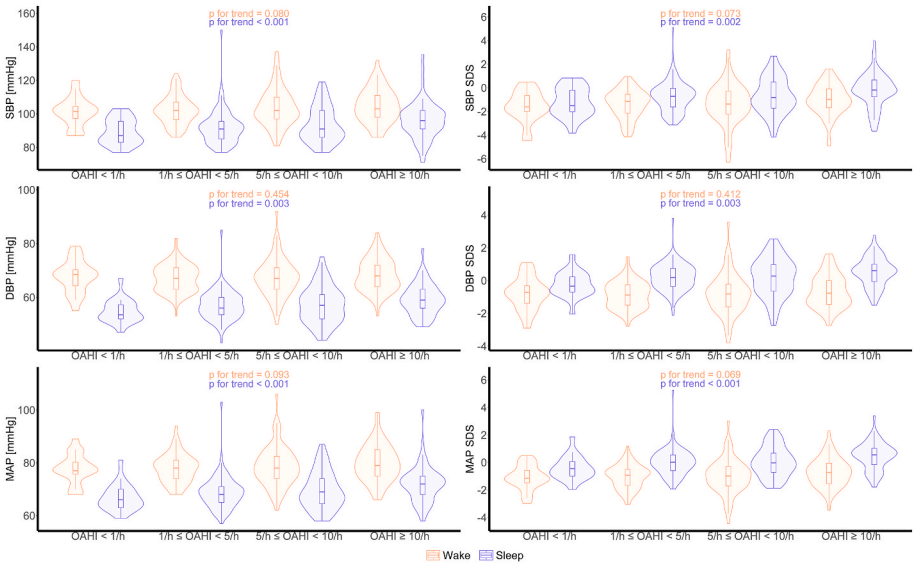

Fig. 1. Violin plots showing the distribution of systolic blood pressure (A), diastolic blood pressure (B) and mean arterial pressure (C) during wake and sleep according to the severity groups of obstructive sleep apnea. Right column represents SDS values, during wake and sleep according to the severity groups of obstructive sleep apnea. Abbreviations: Obstructive apnea-hypopnea index (OAHI), Systolic blood pressure (SBP), Diastolic blood pressure (DBP), Mean blood pressure (MAP).

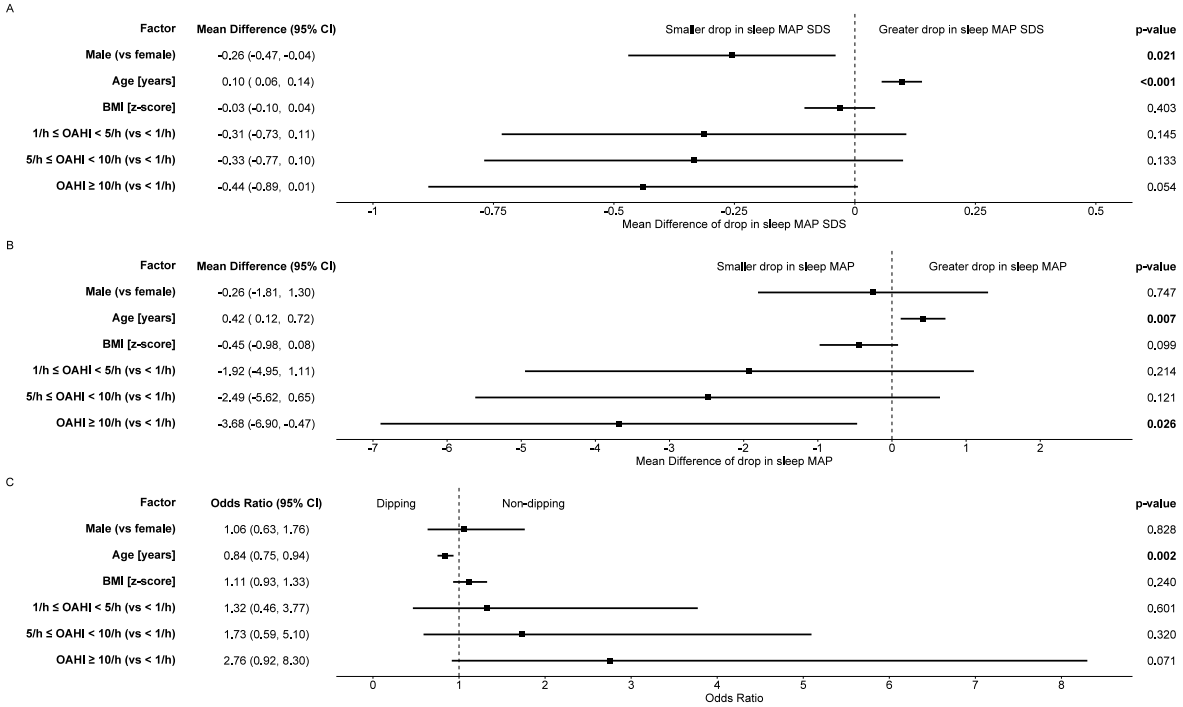

Fig. 2. Multivariable regression analyses for predictors of obstructive sleep apnea and blood pressure patterns. A: Multivariable linear regression model for drop in mean arterial pressure (day-night standardized deviation scores values). B: Multivariable linear regression model for drop in mean arterial pressure. C: Multivariable logistic regression analysis for non-dipping pattern. Abbreviations: Confidence interval (CI), body mass index (BMI), Obstructive apnea-hypopnea index (OAHI), Mean arterial blood pressure (MAP), standardized deviation scores (SDS).

neuromuscular activation, arousal threshold, and ventilatory control [36,39]. In our study, these results could be explained because the median age was 6 years, and consequently, this subpopulation is typically known for having more tendency for adenotonsillar hypertrophy and a less developed airway. Age is a critical factor influencing the maturation of the autonomic nervous system, which is responsible for regulating BP and CV responses during sleep. Younger children, whose autonomic function is still developing, may exhibit a more pronounced sympathetic response to intermittent hypoxia, leading to the disruption of the normal nocturnal dipping pattern. Including age as an independent factor accounts for the developmental changes in CV regulation and improves the understanding of how age interacts with OSA severity to influence BP patterns [40–44].

Some questions regarding the clinical management of these patients arise in view of these results. Firstly, if a BP study should be performed in pediatric patients with OSA. Considering the elevation of BP values, it can be posited that conducting an ABPM should be considered in children with suspected or confirmed OSA, especially severe cases.

Secondly, related to the type of BP study it seems that office BP measurements are clearly insufficient to detect nocturnal dysregulation and that such studies should be performed using ABPM. Thirdly, the relevance of performing ABPM, which could have important therapeutic implications, including the presence of NDP in the therapeutic algorithm, even in patients with lower OAHI. In this context, the NDP may serve as a practical, age-independent marker of CV risk.

Accordingly, the scientific update established by the AHA recognizes that there has been significant expansion of the evidence base for use of ABPM in the pediatric population, including new data linking ABPM levels with the development of BP–related target organ damage [23]. ABPM is a more powerful predictor of CV events, and much more reproducible than office BP [45].

At the same time, we fully agree with current guideline recommendations [23,26,45], which endorse the broader use of ABPM in pediatric populations at risk, including children with suspected or confirmed OSA across the severity spectrum. Therefore, our conclusion is that ABPM is especially informative in children with more severe OSA, but it should not be limited exclusively to them. These results could help in establishing causal associations and developing new treatment strategies, contributing to clinical guidelines for OSA management in the paediatric population.

The strengths of the study comprise, firstly, the large number of participants in a well-designed clinical study. All participating centers performed study procedures adopting the same methodology with variables collected systematically, thus reducing the bias. The sleep study was PSG, which is the gold standard for OSA diagnosis in children and ABPM was performed in all participants, identifying variability patterns undetectable by in-office measurements.

Some study limitations need to be commented on. As this was a multicenter study, a possible limitation could be the different criteria in the analysis of PSG studies. To minimize this potential bias, only sleep units with extensive experience in performing PSG in children were included in the study. Additionally, the use of the AASM 2017 guidelines was protocolized to standardize the definitions of the events when establishing the severity of OSA. Another limitation of our severity-stratified analyses was the small number of the non-OSA group (OAHI <1/h). This study did not include a control group of non-snoring children. This constrains statistical power for comparisons across OAHI strata and may affect the robustness of multivariable models, as reflected by wider confidence intervals.

Another limitation arises from the separation of the day of PSG recording from the ABPM performance. This was done to avoid any potential sleep disruptions caused by the ABPM device, as it may interfere with sleep patterns, and to account for possible inaccuracies in parental reports of bedtime and wake time. To further explore these findings, future longitudinal studies could investigate the reversibility of NDP and drop in MAP and explore other markers of preclinical organ damage after adenotonsillectomy. Previous studies suggest a potential impact of adenotonsillectomy on vascular health, including BP [46], endothelial function [47] and cardiac function and structure [48]. Further research could focus on identifying which patient characteristics, such as nocturnal BP, may predict significant CV benefits from surgery. Finally, validate ABPM-based algorithms for pediatric OSA management. Finally, to address the potential variability between centers, a concordance analysis was conducted., Centre-level clustering was generally low (ICCs mostly <0.15, several close to zero), suggesting that between-center differences were limited and unlikely to materially affect our findings; nonetheless, estimates were imprecise due to the small number of centers and unbalanced site sizes.

5. Conclusions

In this multicenter cohort, severe pediatric OSA was independently associated with disrupted nocturnal BP regulation, including reduced MAP dipping and a trend of increased risk of NDP. These findings underscore the importance of ABPM in pediatric sleep evaluations. Prospective studies are needed to determine whether treatment of OSA can normalize BP patterns and reduce long-term hypertension risk.

CRediT authorship contribution statement

Olga Mediano: Writing – review & editing, Writing – original draft, Validation, Supervision, Methodology, Investigation, Funding acquisition, Conceptualization. **María Castillo-García:** Writing – review & editing, Investigation. **Sofía Romero-Peralta:** Visualization, Validation, Supervision, Methodology, Investigation. **María Esther Viejo-Ayuso:** Methodology, Investigation. **Laura Silgado-Martínez:** Methodology, Investigation. **Leticia Álvarez-Balado:** Methodology, Investigation. **Carles Forné:** Formal analysis, Data curation. **Pilar Resano-Barrio:** Visualization, Validation, Methodology, Investigation. **Francisco García-Rio:** Visualization, Validation, Methodology, Investigation. **Irene Cano-Pumarega:** Methodology. **Manuel Sánchez-de-la-Torre:** Visualization, Validation, Methodology, Investigation. **Alfonso Ortigado:** Methodology. **Laura Fidalgo:** Methodology. **Ángel Rodríguez:** Methodology. **Sonia López-Monzoni:** Visualization, Methodology, Investigation. **Ainhoa Álvarez:** Methodology. **Carla Pía-Martínez:** Methodology. **Genoveva del Río:** Methodology. **Esther Bragado:** Methodology. **Alejandra Roncero:** Methodology. **Belén García-Mediano:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation. **Álvaro Mato-López:** Writing – review & editing. **Adriana Rodríguez-Perojo:** Writing – review & editing. **Martino F. Pengo:** Methodology, Investigation. **Esther Solano-Pérez:** Writing – review & editing, Writing – original draft, Visualization, Validation, Methodology, Investigation.

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: Olga Mediano reports a relationship with Eli Lilly and Company that includes: consulting or advisory. Olga Mediano reports a relationship with ResMed that includes: consulting or advisory. Olga Mediano reports a relationship with Linde Inc that includes: speaking and lecture fees and travel reimbursement. Olga Mediano reports a relationship with GSK that includes: speaking and lecture fees and travel reimbursement. Olga Mediano reports a relationship with Menarini Laboratories that includes: speaking and lecture fees and travel reimbursement. Olga Mediano reports a relationship with Philips that includes: speaking and lecture fees and travel reimbursement. Olga Mediano reports a relationship with Vivisol B that includes: speaking and lecture fees and travel reimbursement. Carles Forne reports a relationship with Servicio de Salud de Castilla-La Mancha that includes: non-financial support. Irene Cano-Pumarega reports a relationship with Nox Medical that includes: travel reimbursement. Irene Cano-Pumarega reports a relationship with Philips that includes: travel reimbursement. Irene Cano Pumarega reports a relationship with ResMed that includes: travel reimbursement. Irene Cano Pumarega reports a relationship with Eli Lilly and Company that includes: consulting or advisory. Irene Cano Pumarega reports a relationship with Bioprojet that includes: consulting or advisory. Manuel Sanchez de la Torre reports a relationship with ResMed that includes: speaking and lecture fees and travel

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References

[1] Barbé F, Durán-Cantolla J, Sánchez-de-la-Torre M, Martínez-Alonso M, Carmona C, Barceló A, et al. Effect of continuous positive airway pressure on the incidence of hypertension and cardiovascular events in nonsleepy patients with obstructive sleep apnea: a randomized controlled trial. JAMA 2012 May 23;307(20). https://doi.org/10.1001/jama.2012.4366.

[2] Martínez-García MA, Capote F, Campos-Rodríguez F, Lloberes P, Díaz De Atauri MJ, Somoza M, et al. Effect of CPAP on blood pressure in patients with obstructive sleep apnea and resistant hypertension: the HIPARCO randomized clinical trial. JAMA 2013 Dec 11;310(22):2407. https://doi.org/10.1001/jama.2013.281250.

[3] Solano-Pérez E, Coso C, Romero-Peralta S, Castillo-García M, López-Monzoni S, Ortigado A, et al. New approaches to the management of cardiovascular risk associated with sleep respiratory disorders in pediatric patients. Biomedicines 2024 Feb 9;12(2):411. https://doi.org/10.3390/biomedicines12020411.

[4] Castillo-García M, Solano-Pérez E, Coso C, Romero-Peralta S, García-Borreguero D, Izquierdo JL, et al. Impact of obstructive sleep apnea in cardiovascular risk in the pediatric population: a systematic review. Sleep Med Rev 2023 Oct;71:101818. https://doi.org/10.1016/j.smrv.2023.101818.

[5] Fernandez-Mendoza J, He F, Calhoun SL, Vgontzas AN, Liao D, Bixler EO. Association of pediatric obstructive sleep apnea with elevated blood pressure and orthostatic hypertension in adolescence. JAMA Cardiol 2021 Oct 1;6(10):1144. https://doi.org/10.1001/jamacardio.2021.2003.

[6] Tirosh A, Afek A, Rudich A, Percik R, Gordon B, Ayalon N, et al. Progression of normotensive adolescents to hypertensive adults: a study of 26 980 teenagers. Hypertension 2010 Aug;56(2):203–9. https://doi.org/10.1161/HYPERTENSIONAHA.109.146415.

[7] Campana EMG, Brandão AA, Pozzan R, Magalhães MEC, Fonseca FL, Pizzi OL, et al. Blood pressure in adolescence, adipokines and inflammation in young adults. The Rio de Janeiro Study Arq Bras Cardiol 2013. https://doi.org/10.5935/abc.20130226.

[8] Erlingsdottir A, Indridason OS, Thorvaldsson O, Edvardsson VO. Blood pressure in children and target-organ damage later in life. Pediatr Nephrol 2010 Feb;25(2):323–8. https://doi.org/10.1007/s00467-009-1350-3.

[9] Ai S, Li Z, Wang S, Chen S, Chan JW, Au CT, et al. Blood pressure and childhood obstructive sleep apnea: a systematic review and meta-analysis. Sleep Med Rev 2022 Oct;65:101663. https://doi.org/10.1016/j.smrv.2022.101663.

[10] Chan KC ching, Au CT, Hui LL, Wing YK, Li AM. Childhood OSA is an independent determinant of blood pressure in adulthood: longitudinal follow-up study. Thorax 2020 May;75(5):422–31. https://doi.org/10.1136/thoraxjnl-2019-213692.

[11] Zintzaras E, Kaditis AG. Sleep-disordered breathing and blood pressure in children: a meta-analysis. Arch Pediatr Adolesc Med 2007 Feb 1;161(2):172. https://doi.org/10.1001/archpedi.161.2.172.

[12] Kario K, Hoshide S, Mizuno H, Kabutoya T, Nishizawa M, Yoshida T, et al. Nighttime blood pressure phenotype and cardiovascular prognosis: practitioner-based nationwide JAMP study. Circulation 2020 Nov 10;142(19):1810–20. https://doi.org/10.1161/CIRCULATIONAHA.120.049730.

[13] Amin RS, Carroll JL, Jeffries JL, Grone C, Bean JA, Chini B, et al. Twenty-four–hour ambulatory blood pressure in children with sleep-disordered breathing. Am J Respir Crit Care Med 2004 Apr 15;169(8):950–6. https://doi.org/10.1164/rccm.200309-1305OC.

[14] Kang K, Chiu S, Weng W, Lee P, Hsu W. 24-Hour ambulatory blood pressure variability in children with obstructive sleep apnea. Laryngoscope 2021 Sep;131(9):2126–32. https://doi.org/10.1002/lary.29455.

[15] Crinion SJ, Ryan S, Kleinerova J, Kent BD, Gallagher J, Ledwidge M, et al. Nondipping nocturnal blood pressure predicts sleep apnea in patients with hypertension. J Clin Sleep Med 2019 Jul 15;15(7):957–63. https://doi.org/10.5664/jcsm.7870.

[16] Horne RSC, Yang JSC, Walter LM, Richardson HL, O'Driscoll DM, Foster AM, et al. Nocturnal dipping is preserved in children with sleep disordered breathing regardless of its severity. Pediatr Pulmonol 2013 Nov;48(11):1127–34. https://doi.org/10.1002/ppul.22727.

[17] Nisbet LC, Nixon GM, Yiallourou SR, Biggs SN, Davey MJ, Trinder J, et al. Sleep-disordered breathing does not affect nocturnal dipping, as assessed by pulse transit time, in preschool children: evidence for early intervention to prevent adverse cardiovascular effects? Sleep Med 2014 Apr;15(4):464–71. https://doi.org/10.1016/j.sleep.2013.11.787.

[18] Li AM, Au CT, Sung RYT, Ho C, Ng PC, Fok TF, et al. Ambulatory blood pressure in children with obstructive sleep apnoea: a community based study. Thorax 2008 Sep 1;63(9):803–9. https://doi.org/10.1136/thx.2007.091132.

[19] Leung LCK, Ng DK, Lau MW, Chan CH, Kwok KL, Chow PY, et al. Twenty-four-hour ambulatory BP in snoring children with obstructive sleep apnea syndrome. Chest 2006 Oct;130(4):1009–17. https://doi.org/10.1378/chest.130.4.1009. PubMed PMID: 17035432.

[20] Kang KT, Chiu SN, Weng WC, Lee PL, Hsu WC. Analysis of 24-Hour ambulatory blood pressure monitoring in children with obstructive sleep apnea: a hospital-based study. Medicine (Baltim) 2015 Oct;94(40):e1568. https://doi.org/10.1097/MD.0000000000001568.

[21] Khan MTF, Smith DF, Schuler CL, Witter AM, DiFrancesco MW, Armoni Domany K, et al. Circadian blood pressure dysregulation in children with obstructive sleep apnea. Sleep 2024 Feb 8;47(2). https://doi.org/10.1093/sleep/zsad254. zsad254.

[22] Witter AM, Smith DF, Khan MTF, Nakamura A, Schuler CL, DiFrancesco MW, et al. Relationship of circadian blood pressure dysregulation with left ventricular structure and function in children with obstructive sleep apnea. J Sleep Res 2025 Oct;34(5):e70049. https://doi.org/10.1111/jsr.70049.

[23] Flynn JT, Urbina EM, Brady TM, Baker-Smith C, Daniels SR, Hayman LL, et al. Ambulatory blood pressure monitoring in children and adolescents: 2022 update: a scientific statement from the American heart association. Hypertension 2022 Jul;79(7). https://doi.org/10.1161/HYP.0000000000000215.

[24] Castillo-García M, Solano-Pérez E, Romero-Peralta S, Viejo-Ayuso ME, Silgado-Martínez L, Álvarez-Balado L, et al. Prevalence of high blood pressure in pediatric patients with sleep-disordered breathing, reversibility after treatment: the KIDS TRIAL study protocol. Children 2022 Nov 28;9(12):1849. https://doi.org/10.3390/children9121849.

[25] Berry RB, Brooks R, Gamaldo C, Harding SM, Lloyd RM, Quan SF, et al. AASM scoring manual updates for 2017 (version 2.4). J Clin Sleep Med 2017 May 15;13(5):665–6. https://doi.org/10.5664/jcsm.6576.

[26] Kaditis AG, Alonso Alvarez ML, Boudewyns A, Alexopoulos EI, Ersu R, Joosten K, et al. Obstructive sleep disordered breathing in 2- to 18-year-old children: diagnosis and management. Eur Respir J 2016 Jan;47(1):69–94. https://doi.org/10.1183/13993003.00385-2015.

[27] Lurbe E, Agabiti-Rosei E, Cruickshank JK, Dominiczak A, Erdine S, Hirth A, et al. European society of hypertension guidelines for the management of high blood pressure in children and adolescents. J Hypertens 2016;34(10):1887–920. https://doi.org/10.1097/HJH.0000000000001039. 2016 Oct.

[28] Wühl E, Witte K, Soergel M, Mehls O, Schaefer F. Distribution of 24-h ambulatory blood pressure in children: normalized reference values and role of body dimensions. J Hypertens 2002 Oct;20(10):1995–2007. https://doi.org/10.1097/00004872-200210000-00019.

[29] Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. Sleep Med 2000 Feb;1(1):21–32. https://doi.org/10.1016/S1389-9457(99)00009-X.

[30] De Onis M. Development of a WHO growth reference for school-aged children and adolescents. Bull World Health Organ 2007 Sep 1;85(9):660–7. https://doi.org/10.2471/BLT.07.043497.

[31] Weltgesundheitsorganisation. Length/height-for-age, weight-for-age, weight-for-length. In: Onis M de, editor. weight-for-height and body mass index-for-age; methods and development. Geneva: WHO Press; 2006. p. 312 (WHO child growth standards).

[32] Kwok K li, Ng DK, Chan C hong. Cardiovascular changes in children with snoring and obstructive sleep apnoea. Ann Acad Med Singapore 2008 Aug 15;37(8):715–21. https://doi.org/10.47102/annals-acadmedsg.V37N8p715.

[33] Brooks DM, Kelly A, Sorkin JD, Koren D, Chng SY, Gallagher PR, et al. The relationship between sleep-disordered breathing, blood pressure, and urinary cortisol and catecholamines in children. J Clin Sleep Med 2020 Jun 15;16(6):907–16. https://doi.org/10.5664/jcsm.8360.

[34] Chan KC, Au CT, Yu MW, Wing YK, Li AM. Natural history of REM-OSA in children and its associations with adverse blood pressure outcomes: a longitudinal Follow-Up study. Nat Sci Sleep 2021 Nov;13:1967–84. https://doi.org/10.2147/NSS.S331389.

[35] Mediano O, López-Monzoni S, Castillo-García M, Romero-Peralta S, Viejo-Ayuso ME, Silgado-Martínez L, et al. Cardiovascular risk through hypoxic burden in children with sleep apnea: a secondary analysis of a nonrandomized clinical trial. JAMA Netw Open 2025 Oct 23;8(10):e2538744. https://doi.org/10.1001/jamanetworkopen.2025.38744.

[36] Gulotta G, Iannella G, Vicini C, Polimeni A, Greco A, De Vincentiis M, et al. Risk factors for obstructive sleep apnea syndrome in children: state of the art. Int J Environ Res Publ Health 2019 Sep 4;16(18):3235. https://doi.org/10.3390/ijerph16183235.

[37] Magnusdottir S, Hill EA. Prevalence of obstructive sleep apnea (OSA) among preschool aged children in the general population: a systematic review. Sleep Med Rev 2024 Feb;73:101871. https://doi.org/10.1016/j.smrv.2023.101871.

[38] Spilsbury JC, Storfer-Isser A, Rosen CL, Redline S. Remission and incidence of obstructive sleep apnea from middle childhood to late adolescence. Sleep 2015 Jan;38(1):23–9. https://doi.org/10.5665/sleep.4318.

[39] Baker-Smith CM, Isaiah A, Melendres MC, Mahgerefteh J, Lasso-Pirot A, Mayo S, et al. Sleep-disordered breathing and cardiovascular disease in children and adolescents: a scientific statement from the American heart association. J Am Heart Assoc 2021 Sep 21;10(18):e022427. https://doi.org/10.1161/JAHA.121.022427.

[40] Nisbet LC, Yiallourou SR, Walter LM, Horne RSC. Blood pressure regulation, autonomic control and sleep disordered breathing in children. Sleep Med Rev 2014 Apr;18(2):179–89. https://doi.org/10.1016/j.smrv.2013.04.006.
[41] Gozal D, Hakim F, Kheirandish-Gozal L. Chemoreceptors, baroreceptors, and autonomic deregulation in children with obstructive sleep apnea. Respir Physiol Neurobiol 2013 Jan;185(1):177–85. https://doi.org/10.1016/j.resp.2012.08.019.
[42] Hakim F, Gozal D, Kheirandish-Gozal L. Sympathetic and catecholaminergic alterations in sleep apnea with particular emphasis on children. Front Neurol 2012; 3. https://doi.org/10.3389/fneur.2012.00007.
[43] O'Driscoll DM, Horne RSC, Davey MJ, Hope SA, Anderson V, Trinder J, et al. Increased sympathetic activity in children with obstructive sleep apnea: cardiovascular implications. Sleep Med 2011 May;12(5):483–8. https://doi.org/10.1016/j.sleep.2010.09.015.
[44] Harteveld LM, Nederend I, Ten Harkel ADJ, Schutte NM, De Rooij SR, Vrijkotte TGM, et al. Maturation of the cardiac autonomic nervous system activity in children and adolescents. J Am Heart Assoc 2021 Feb 16;10(4):e017405. https://doi.org/10.1161/JAHA.120.017405.
[45] Baker-Smith CM, Flinn SK, Flynn JT, Kaelber DC, Blowey D, Carroll AE, et al. Diagnosis, evaluation, and management of high blood pressure in children and adolescents. Pediatrics 2018 Sep 1;142(3):e20182096. https://doi.org/10.1542/peds.2018-2096.
[46] Au CT, Chan KC ching, Lee DLY, Leung NMW, Chow SMW, Kwok KL, et al. Effect of surgical intervention for childhood OSA on blood pressure: a randomized controlled study. Sleep Med 2023 Jul;107:9–17. https://doi.org/10.1016/j.sleep.2023.04.006.
[47] Chan KCC, Au CT, Chook P, Lee DLY, Lam HS, Wing YK, et al. Endothelial function in children with OSA and the effects of adenotonsillectomy. Chest 2015 Jan;147 (1):132–9. https://doi.org/10.1378/chest.14-1307.
[48] Chan JYS, Li AM, Au CT, Lo AFC, Ng SK, Abdullah VJ, et al. Cardiac remodelling and dysfunction in children with obstructive sleep apnoea: a community based study. Thorax 2009 Mar 1;64(3):233–9. https://doi.org/10.1136/thx.2007.094904.