

ORIGINAL PAPER

## Blood pressure and desaturation in children and adolescents with primary hypertension

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### ABSTRACT

**Introduction:** Primary hypertension (PHT) has long ceased to be an adult problem. It commonly affects children and adolescents and is becoming a severe health care problem in many countries. In contrast to secondary hypertension, which occurs mainly in younger children with kidney, cardiovascular, and endocrine disorders, PHT affects older children and adolescents. Potential causes of PHT include being overweight, particularly obese, sleep apnea, and increased sympathetic nervous system (SNS) activity. Vegetative system activity is an essential factor in children's blood pressure (BP). Those with excessive sympathetic system activity have increased heart rate, BP, and other parameters characterizing the positive chronotropic effect. In turn, one of the factors stimulating the SNS is blood desaturation (DES). This study investigates the relationship between ambulatory BP and oxygen DES rates.

**Material and methods:** The degree and number of DES episodes were assessed by finger pulse oximetry during 24-h ambulatory monitoring in 54 boys and girls with PHT. Their results were compared to 52 healthy children without PHT.

**Results:** Several disturbances in blood saturation were found in children with PHT. They had more episodes of DES and profound hypoxia (< 90%), their blood DES was significantly lower, and their DES time was longer. Additionally, higher systolic BP and higher diastolic pressure loads were observed throughout the day and night in children with longer DES times (> 60 s), who also had more DES episodes and lower baseline and average blood saturation.

**Conclusions:** Children with PHT show significant disturbances in blood oxygenation, leading to overactive SNS activity, which may be a crucial element in PHT pathogenesis in children.

### KEY WORDS:

children, primary hypertension, blood desaturation.

### INTRODUCTION

Many factors can cause or contribute to the formation of hypertension in children, of which the most important are kidney diseases, endocrine diseases, aortic coarctation, and vasculitis. These diseases can cause hypertension at any age but are most often described in younger children. Primary hypertension (PHT) occurs most often

in older children and adolescents, and its pathogenesis is very complex. Factors conducive to PHT development include excessive sympathetic system activity, obesity, insulin resistance, and sleep disorders [1]. Therefore, PHT pathogenesis is multifactorial with a complex relationship, where several risk factors can be present simultaneously, further complicating the unambiguous determination of the cause of PHT in each patient.

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In addition, obstructive sleep apnea (OSA) and shallow breathing syndrome (OSAHS) can lead to PHT. Obstructive sleep apnea incidence in adults is relatively high, affecting up to 56% of hypertensive patients [2], and is higher in patients with refractory hypertension. In children, OSA prevalence varies widely at different life stages, from 1.1% at pre-school age to 4% at school age [3]. However, the relationship between OSA and hypertension is not well understood. Blood desaturation (DES) and hypoxemia undoubtedly lead to excessive sympathetic system activation, which changes the circadian rhythm of arterial pressure without a nocturnal physiological decrease in blood pressure (BP).

The instrumental diagnostics of OSA are based on pulse oximetry, home respiratory polygraphy (HRP), and polysomnography (PSG). The American Academy of Pediatrics recommends overnight, attended, in-laboratory PSG as the gold-standard approach for diagnosing obstructive sleep apnea syndrome (OSAS) in children with clinical sleep-disordered breathing manifestations [4].

Pulse oximetry is an ideal screening test at this age due to its ease of execution, low cost, and high diagnostic positive predictive value. Blood oximetry is a valuable tool that can facilitate treatment decisions when PSG is unavailable [5]. Oximetry has been successfully implemented in the publicly funded Canadian healthcare system as an abbreviated and low-cost testing modality for diagnosing OSAS in children [6]. Developing well-validated protocols and devices for continuous oximetry and simultaneous BP monitoring could further improve the value and usefulness of this approach as a diagnostic modality.

Most BP recordings are performed with the cuff-based method introduced by Scipione Riva-Rocci in 1896. However, current ambulatory blood pressure monitoring (ABPM) devices are relatively large and not convenient or practical for daily or long-term use, particularly with children. Many children tolerate repeated cuff inflations and measurements poorly during the night, which can interrupt sleep and cause falsely elevated BP readings [7]. Recently, a new cuffless BP device (SOMNOtouch-non-invasive blood pressure (NIBP); SOMNOmedics GmbH; Randersacker, Germany) uses pulse transit time (PTT) for beat-to-beat BP calculation after a single or double initial cuff-based calibration measurement [8]. We have previously compared BP measured with the cuffless SOMNOtouch-NIBP device with a standard cuff-based device in typical clinical use over 20–24 hours. We found that the pulse wave velocity – BP function created produced a significant correlation between BP obtained from the SOMNOtouch-NIBP device and systolic blood pressure (SBP) measured by an Omron 907 sphygmomanometry device (Langenfeld, Germany) [9]. In addition, many other reports have obtained promising results with 24 h BP assessment using a continuous beat-to-beat measurement technique [10]. This study uses the portable SOMNOtouch-NIBP device for recording different phys-

iological signals, particularly electrocardiogram (ECG), peripheral pulse wave, oxygen saturation, and movement, to obtain a continuous BP value. These physiological signals are intended to guide the diagnosis of cardiovascular disorders. This study assesses the round-the-clock BP profile, blood oxygenation, ECG, and heart rate in a non-invasive manner.

## MATERIAL AND METHODS

### PATIENT COHORT AND DATA COLLECTION

Fifty-four children with PHT were included in the study, with a mean age of  $14.68 \pm 2.58$  (10–18), a mean height of  $168.35 \pm 13.80$  cm, a mean body weight of  $73.94 \pm 22.01$  kg, and a mean body mass index (BMI) of  $25.51 \pm 5.57$  kg/m<sup>2</sup>. Twenty-nine patients were overweight (BMI > 25) or obese (BMI > 30). The control group consisted of healthy children, without tonsillitis.

Hypertension in our children was defined as SBP or diastolic blood pressure (DBP) persistently in the  $\geq 95^{\text{th}}$  percentile for sex, age, and height on at least three separate occasions (2016 European Society of Hypertension Guidelines for the Management of High BP in Children and Adolescents) [11]. For patients aged 16 or older, an absolute cutoff of 140/90 mm Hg was used.

In this study, we used this non-invasive device for simultaneous and continuous monitoring of BP, heart rate, pulse oximetry, physical activity, and sleep time (Figure 1). This device was validated according to the European Society of Hypertension International Protocol Revision 2010 for Validation of BP Measuring Devices in Adults [12], adapted to its distinct characteristics. The systolic blood pressure and DBP calculation is based on a non-linear correlation between PTT (in ms) and BP (in mm Hg). The significant advantage of the cuffless device is that it can monitor many different parameters simultaneously without repeated cuff inflations that many children poorly tolerate [13]. The cuffless device was placed on the left forearm and connected to the photoplethysmography at the left index finger and ECG electrodes. After at least 10 minutes of rest, a manual BP measurement was used as the first calibration measurement for the cuffless device. Individual diaries were used to define awake and asleep BP. Simultaneously, beat-to-beat BP was recorded by the cuffless device. Up to 100,000 beat-to-beat BP recordings per patient were usually collected. The time of the recordings was in the range 20–24 h. The internal movement sensors of the SOMNOtouch device collected the patient's activity and body position, helping to distinguish between physiological and psychogenic BP increases. In addition, it was possible to perform a valid sleep/wake analysis based on the activity profile and body position recording [14].

Pulse oximetry was continuously performed while measuring BP on the middle finger of the left hand. After

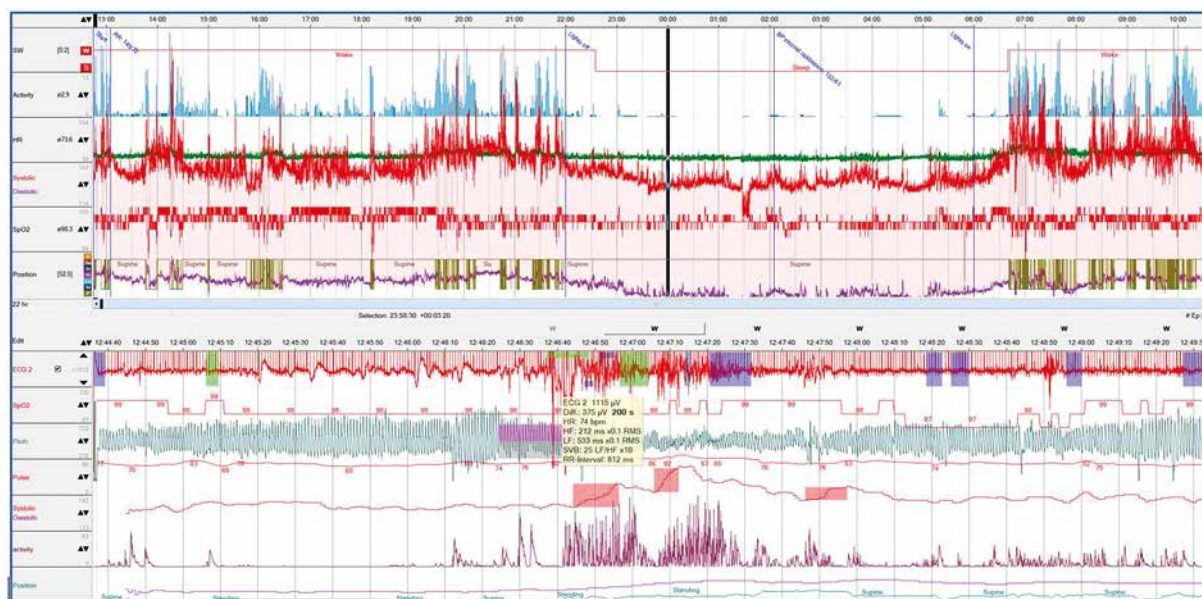


FIGURE 1. A typical recording from an adolescent with primary hypertension using the SOMNOtouch-NIBP device, visualized in the Domino Light v.1.4.0 software

TABLE 1. Anthropometric characteristics of primary hypertension patients and controls

Parameters	PHT	Control	p-value
Age (years)	14.68 ± 2.58	13.36 ± 3.27	0.030
Height [cm]	168.35 ± 13.80	162.04 ± 18.08	0.045
Body weight [kg]	73.94 ± 22.01	64.69 ± 24.61	0.048
BMI [kg/m <sup>2</sup> ]	25.51 ± 5.57	23.69 ± 6.01	0.120

BMI – body mass index, PHT – primary hypertension

recording, the data were sent to SOMNOtouch, where measurements were analyzed using their dedicated software (Domino Light v.1.4.0; SOMNOmedics GmbH). We analyzed the total number of DES, DES index (number/h), minimal saturation (%), average saturation (%), number of DES episodes < 90%, number of DES episodes < 80%, lowest DES duration, medium DES duration, and the total DES time. Oxygen desaturation was defined as a decrease of ≥ 4% in peripheral oxygen saturation (SpO<sub>2</sub>) relative to baseline. This study was approved by the local Ethics Committee, and every participant (≥ 16 years) or their parents/legal guardians gave fully informed consent to participate in the study. The study was performed according to the principles of the Second Declaration of Helsinki.

### STATISTICAL ANALYSIS

Continuous data are presented as mean ± standard deviation and compared using paired t-tests as patients' data distribution was normal. Correlations between different variables were measured using Pearson correlation coefficients which measured the strength of association between two variables. All statistical analyses were performed using the Statistica v.13.0 software. All results with *p* < 0.05 were considered statistically significant.

## RESULTS

### CHARACTERISTICS OF STUDY SUBJECTS

This study included 54 children with PHT aged 14.68 ± 2.58 years and compared them with 52 healthy children aged 13.37 ± 3.27 years. Their anthropometric characteristics are provided in Table 1. Pulse oximetry included all data on blood oximetry status (Figure 2). We used the following parameters to characterize BP: 24 h SBP (SYS 24 h), DBP (DIA 24 h), SBP load (Load SYS 24 h), and DBP load (Load DIA 24 h); day SBP (SYS Day), DBP (DIA Day), SBP load (Load SYS Day), and DBP load (Load DIA Day); night SBP (SYS Night), DBP (DIA Night), SBP load (Load SYS Night), and DBP load (Load DIA Night). Almost all respiratory rate measurements differed significantly between groups (Table 2). Notably, heart rate was higher in children with PHT during the 24 h period (82.43 ± 11.36; *p* = 0.025) and during the day (88.77 ± 10.87; *p* = 0.020) compared to controls (78.12 ± 7.77 and 84.48 ± 7.19, respectively).

The blood saturation and DES results are presented in Table 3. Children with PHT had significantly more DES episodes (7.92 ± 9.29) than control children (4.11 ± 4.20; *p* = 0.010). Moreover, the maximum number of DES episodes in children with PHT was 46, higher than in control children (20). Baseline blood saturation did not differ between children with (98.14 ± 1.18%) and without PHT (98.17 ± 1.32%; *p* = 0.910). The minimum blood saturation value in children with PHT was 76%, lower than in control children (83%). Children with PHT had significantly more hypoxia episodes < 90% (3.71 ± 4.76) than control children (0.78 ± 3.10; *p* = 0.010). The maximal duration of DES < 90% was 10.9 s in children with PHT, longer than in control children (0.8 s).

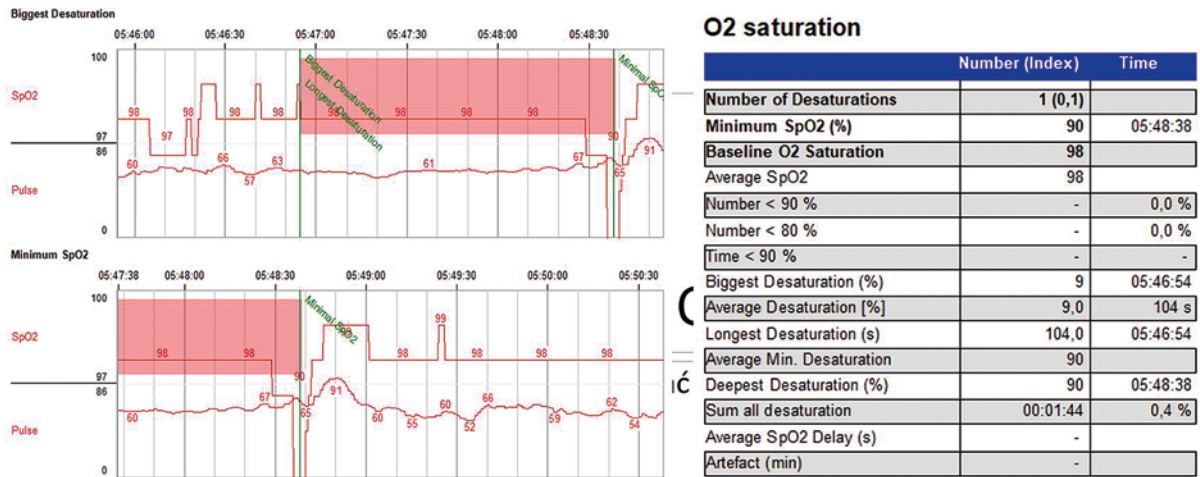


FIGURE 2. A typical blood saturation report for an adolescent with primary hypertension showing their greatest desaturation and lowest SpO2, visualized in the Domino Light v.1.4.0 software

TABLE 2. Respiratory rate measurements of primary hypertension patients and controls

Parameters	PHT	Control	p-value
SYS 24 h [mm Hg]	132.74 ±7.49	116.50 ±7.98	< 0.010
DIA 24 h [mm Hg]	70.71 ±9.77	65.73 ±4.68	< 0.010
Load SYS 24 h (%)	49.92 ±28.45	13.38 ±11.79	< 0.010
Load DIA 24 h (%)	27.80 ±31.05	11.40 ±11.49	< 0.010
SYS day [mm Hg]	133.77 ±13.16	119.64 ±8.05	< 0.010
DIA day [mm Hg]	73.86 ±11.71	69.18 ±5.38	0.020
Load SYS day (%)	48.78 ±29.86	10.30 ±10.12	< 0.010
Load DIA day (%)	24.11 ±27.50	7.92 ±8.39	< 0.010
SYS night [mm Hg]	116.73 ±20.36	109.09 ±8.15	0.020
DIA night [mm Hg]	59.68 ±12.65	57.68 ±5.15	0.330
Load SYS night (%)	49.91 ±33.66	23.07 ±20.82	< 0.010
Load DIA night (%)	28.45 ±26.18	15.79 ±17.87	0.010
Heart rate 24 h	82.43 ±11.36	78.12 ±7.77	0.025
Heart rate day	88.77 ±10.87	84.48 ±7.19	0.020
Heart rate night	67.98 ±11.18	65.84 ±6.58	0.230

DIA – mean diastolic arterial pressure, DIA day – diastolic arterial pressure during day, DIA night – diastolic arterial pressure during night, load DIA 24 h – load of diastolic arterial pressure, load DIA day – load of diastolic arterial pressure during day, load DIA night – load of diastolic arterial pressure during night, load SYS 24 h – load of systolic arterial pressure, load SYS day – load of systolic arterial pressure during day, load SYS night – load of systolic arterial pressure during night, PHT – primary hypertension, SYS 24 h – systolic arterial pressure, SYS day – mean systolic arterial pressure during day, SYS night – systolic arterial pressure during night

TABLE 3. Blood saturation and desaturation of primary hypertension patients and controls

Parameters	PHT	Control	p-value
Number of DES, n	7.92 ±9.29	4.11 ±4.20	0.010
Baseline O <sub>2</sub> saturation (%)	98.14 ±1.18	98.17 ±1.32	0.910
Number < 90%	3.71 ±4.76	0.78 ±3.10	0.010
Time < 90% (s)	0.80 ±2.90	0.25 ±0.31	0.620
Average biggest DES (%)	6.24 ±3.14	6.02 ±2.33	0.730
Average DES (%)	5.27 ±1.39	4.58 ±1.30	0.030
Longest DES (s)	56.34 ±39.92	37.00 ±24.85	0.010
Average min DES (%)	94.17 ±1.38	94.15 ±1.59	0.950

DES – desaturation, PHT – primary hypertension

**TABLE 4.** Comparison of children with primary hypertension and desaturations lasting > 60 or ≤ 60 s

Parameters	DES > 60	DES ≤ 60	p-value
SYS 24 h [mm Hg]	137.92 ±6.61	130.68 ±6.19	0.004
DIA 24 h [mm Hg]	71.25 ±13.44	68.29 ±4.50	0.057
Load SYS 24 h [mm Hg]	53.88 ±31.82	44.07 ±22.33	0.345
Load DIA 24 h [mm Hg]	42.73 ±47.21	14.64 ±10.21	0.024
SYS day [mm Hg]	135.73 ±21.88	134.18 ±6.71	0.785
DIA day [mm Hg]	73.36 ±17.48	71.41 ±5.10	0.666
Load SYS day [mm Hg]	50.86±34.62	42.44 ±22.86	0.444
Load DIA day [mm Hg]	28.49 ±37.49	13.66 ±10.15	0.131
SYS night [mm Hg]	113.82 ±39.23	119.06 ±7.27	0.592
DIA night [mm Hg]	58.18 ±22.88	58.47 ±5.71	0.960
Load SYS night (%)	51.74 ±1.62	47.56 ±22.76	0.759
Load DIA night (%)	47.44 ±34.43	16.35 ±14.76	0.004

DES – desaturation, DIA – mean diastolic arterial pressure, DIA day – diastolic arterial pressure during day, DIA night – diastolic arterial pressure during night, load DIA 24 h – load of diastolic arterial pressure, load DIA day – load of diastolic arterial pressure during day, load DIA night – load of diastolic arterial pressure during night, load SYS 24 h – load of systolic arterial pressure, load SYS day – load of systolic arterial pressure during day, load SYS night – load of systolic arterial pressure during night, SYS 24 h – systolic arterial pressure, SYS day – mean systolic arterial pressure during day, SYS night – systolic arterial pressure during night

**TABLE 5.** Comparison of desaturation (DES) characteristics in the primary hypertension – only with DES longer and shorter than 60 s (DES > 60 and DES ≤ 60) groups

Parameters	DES > 60	DES ≤ 60	p-value
Number of DES, n	9.87 ±9.91	4.52 ±4.86	0.001
Baseline O2 saturation (%)	97.33 ±1.23	98.07 ±1.23	0.020
Average O2 saturation (%)	97.25±1.24	98.05 ±1.29	0.010
Average min DES (%)	91.33 ±3.21	93.20 ±2.69	0.008

DES – desaturation

Blood DES was significantly lower in children with PHT (5.27 ±1.39) than in control children (4.58 ±1.30;  $p = 0.030$ ). Maximal blood DES in children with PHT was 14%, similar to control children (13%). The longest blood DES time in children with PHT (136 s) was longer than in control children (105 s). Mean blood DES time in children with PHT (56.34 ±39.92 s) was significantly longer than in control children (37.00 ±24.85 s;  $p = 0.010$ )

Next, we divided children with PHT into two groups, those with DES lasting > 60 s (DES > 60;  $n = 23$ ) and lasting ≤ 60 s (DES ≤ 60;  $n = 31$ ) (Table 4). The DES > 60 s group had significantly higher SYS 24 h (137.73 ±6.61 mm Hg;  $p = 0.007$ ), Load DIA 24 h (42.73 ±47.21%;  $p = 0.024$ ), and Load DIA Night (47.44 ±34.43%;  $p = 0.004$ ) values than the DES ≤ group (130.35 ±6.19 mm Hg, 14.64 ±10.21%, 16.35 ±14.76%, respectively).

The desaturation > 60 group had lower DES, baseline blood saturation, mean blood saturation, and mean lowest blood DES than the DES ≤ 60 group (Table 5).

A similar analysis was performed with the full cohort, combining children with and without PHT (Table 6). The DES > 60 group had significantly higher DIA 24 h (69.50 ±10.17 mm Hg;  $p = 0.010$ ), Load DIA 24 h (26.66 ±38.13%;  $p = 0.020$ ), and Load DIA Night (28.13

±30.21%;  $p = 0.030$ ) values than the DES ≤ 60 group (66.60 ±4.88 mm Hg, 12.58 ±10.30, and 15.58 ±15.60, respectively). The correlation between the longest DES duration and SYS 24 h in children with PHT is shown in Figure 3.

## DISCUSSION

Many studies on hypertension pathogenesis identify sympathetic system involvement as an important etiological factor. We hypothesized that oxygen DES might be closely associated with BP, particularly on ambulatory measurement, and that it might be enhanced in younger patients by accounting for heart rate as a rough measure of sympathetic activity. This study did not explore sympathetic activation, but it is known from previous studies that permanent or transient hypoxia may intensify this activity. This relationship has been particularly well documented in OSAS. Obstructive sleep apnea is characterized by recurrent upper respiratory tract collapse during sleep with chronic intermittent hypoxemia. Intermittent hypoxemia is recognized as the main potential contributor to PHT pathogenesis during OSA [15, 16]. Many mechanisms can cause OSA in children, including upper airway collapsibil-

**TABLE 6.** Comparison of desaturation (DES) characteristics in the full cohort with DES longer and shorter than 60 s (DES > 60 and DES ≤ 60) groups

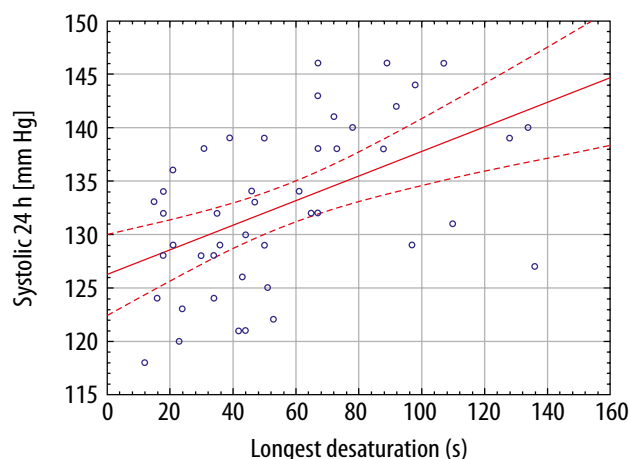
Parameters	DES > 60	DES ≤ 60	p-value
SYS 24 h [mm Hg]	128.59 ±12.14	123.26 ±9.24	0,045
DIA 24 h [mm Hg]	69.50 ±10.17	66.60 ±4.88	0.110
Load SYS 24 h (%)	32.44 ±32.44	27.04 ±23.41	0.440
Load DIA 24 h (%)	26.66 ±38.13	12.58 ±10.30	0.020
SYS day [mm Hg]	129.00 ±17.47	126.29 ±9.46	0.410
DIA day [mm Hg]	72.43 ±12.66	69.98 ±5.70	0.270
Load SYS day (%)	30.76 ±32.91	24.34 ±23.52	0.360
Load DIA day (%)	18.40 ±29.27	10.67 ±9.51	0.100
SYS night [mm Hg]	110.71 ±28.27	113.96 ±8.48	0.470
DIA night [mm Hg]	57.48 ±16.44	58.90 ±7.52	0.620
Load SYS night (%)	34.50 ±37.06	34.07 ±27.71	0.960
Load DIA night (%)	28.13 ±30.21	15.58 ±15.60	0.030

DES – desaturation, DIA – mean diastolic arterial pressure, DIA day – diastolic arterial pressure during day, DIA night – diastolic arterial pressure during night, load DIA 24 h – load of diastolic arterial pressure, load DIA day – load of diastolic arterial pressure during day, load DIA night – load of diastolic arterial pressure during night, load SYS 24 h – load of systolic arterial pressure, load SYS day – load of systolic arterial pressure during day, load SYS night – load of systolic arterial pressure during night, SYS 24 h – systolic arterial pressure, SYS day – mean systolic arterial pressure during day, SYS night – systolic arterial pressure during night

ity, narrower upper airway, and skeletal abnormalities [17]. During development, OSAS prevalence has two peak periods. The first occurs in children aged between 2 and 8 with adenotonsillar hypertrophy. The second occurs with weight gain during adolescence [18]. Only two of our children had symptoms of an abnormal nose air passage caused by hypertrophic tonsils. While most did not show signs of OSA, twenty-nine children were overweight or obese (BMI = 25.51 ±5.57 kg/m<sup>2</sup>). However, we did not find any direct correlation between body weight, blood DES, and BP.

A wide range of symptoms and signs are associated with OSA in children, depending on their age. Snoring or noisy breathing, hypertension, oral breathing, disturbed sleep with frequent changes of position, and nightmares are described predominantly in pre-school age. Excessive sweating, pavor nocturnus, somnambulism, chronic cough, and enuresis often resolve when respiratory sleep disorders are adequately treated. The instrumental diagnostics of the OSA are based on pulse oximetry, HRP, and PSG. Nocturnal pulse oximetry has the characteristics of an ideal screening test at this age, including ease of execution, low cost, and high diagnostic positive predictive value.

While nocturnal PSG remains the gold standard for OSA diagnosis, it is expensive and not always available. Nocturnal oximetry has been proposed as an abbreviated and low-cost testing modality for diagnosing OSAS. A systematic review of 25 original articles found that at least three clusters of DES events and at least three SpO<sub>2</sub> drops < 90% in a nocturnal pulse oximetry recording indicate moderate-to-severe OSAS. Analysis of nocturnal oximetry recordings is relatively easy and can be completed in only a few minutes. Nocturnal pulse oxime-

**FIGURE 3.** Correlation between systolic 24 h and the longest desaturation duration

The fitted line denotes  $\text{systolic 24 h} = 126.23 + 0.11514 \times \text{longest desaturation time}$  ( $r = 0.507$ ;  $p = 3.00 \times 10^{-4}$ )

try is a low-cost and easy-to-use diagnostic modality to identify OSAS among children with sleep-disordered breathing (SDB) symptoms when PSG is unavailable [19]. The second mechanism by which hypoxia can lead to hypertension is the impairment of endothelial-dependent and endothelial-independent vasodilation disorders. Acute exposure to hypoxia reduces endothelium-dependent vascular function in small and large vessels. The decline in microvascular endothelial function was approximately twice as significant as that observed in the large blood vessels, showing the sensitivity of the microvascular endothelium to hypoxia [20, 21].

Our main finding was the confirmation that children with PHT more often have blood DES. We did not analyze the hypertension mechanism, but we observed a higher heart rate in this group. Intermittent hypoxia

during the hours of sleep is likely to increase BP by activating the autonomic nervous system directly. The sympathetic nervous system (SNS) regulates key responses through direct innervation and adrenal activation, including increasing cardiac output by increasing heart rate and myocardial contractility and inhibiting hypoxia-induced systemic vasodilation.

We observed more DES episodes with a maximum number of 46/24 h in our children with PHT. Minimal blood saturation was lower in children with PHT than in the control children. Average DES was also lower in control children. These results confirm our primary hypothesis of the relation between PHT and blood oxygenation. This relationship is usually more pronounced in younger than older individuals, where there is less response to cardiac rhythm activation of the sympathetic system. With age, oxygen needs are lower, increasing tolerance to hypoxia or hypoxemia [22].

Nevertheless, our study should be interpreted in the context of its limitations. First, it is cross-sectional and does not allow for causal inference. Second, it was based on pulse oximetry, and full PSG was not performed. Therefore, we cannot distinguish between obstructive and central sleep apnea. In some patients, oxygen DES may result from central apnea combined with obstructive apnea. Another problem that should be mentioned is that the mean age of the control group was slightly lower than the mean age of the study group.

## CONCLUSIONS

Children with PHT show significant disturbances in blood oxygenation, leading to overactive SNS activity, which may be a crucial element in PHT pathogenesis in children. In our study we found that blood DES was positively associated with outpatient BP during nighttime sleep hours and the daytime. This association can be mediated by heart rate, an approximate measure of sympathetic activity. Our finding implies that correcting hypoxemia with continuous positive airway pressure therapy or other therapeutic methods may be particularly effective in younger patients suffering from hypertension with sleep apnea and a faster heart rhythm.

## DISCLOSURE

The authors declare no conflict of interest.

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