CARDIOVASCULAR INVOLVEMENT IN SLEEP-RELATED BREATHING DISORDERS IN CHILDREN AND ADOLESCENTS

Kardiovaskulárne súvislosti spánkových porúch dýchania u detí a dospievajúcich

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Abstract
Study objectives: The amount of evidence of the role of sleep-related breathing disorders (SBD) in paediatric cardiovascular disease and premature adult cardiovascular disease is rising, proving that not only obstructive sleep apnoea (OSA) but also primary snoring (PS) is associated with higher risk of cardiovascular (CVS) morbidity. The link between SBD and premature CVS morbidity in paediatric patients is still underexamined.
Methods: Our study group consisted of 80 patients examined in the Sleep Laboratory of University Hospital Martin (Slovakia) between 2019 and 2022. The patients underwent a standard all-night polysomnography (PSG).
Results: We determined and graphically demonstrated an increased predisposition to higher BP in patients with OSA and PS compared to healthy controls ($p < 0.001$) but not in patients with OSA compared to PS ($p > 0.05$). In the group of patients with PS, the systolic pressure percentile correlated with duration of snoring during the night ($r = 0.4$; $p < 0.05$). A logistic regression of the whole dataset showed correlation of AHI and average nocturnal heart rate (ANHR, $r = 0.36$; $p < 0.001$) as well as snoring and ANHR ($r = 0.32$; $p < 0.001$). This analysis showed that snoring correlates strongly with total arousal index (TAI, $r = 0.73$, $p < 0.001$) and AHI correlates strongly with TAI ($r = 0.87$, $p < 0.001$).
Conclusion: The presented data confirm the association of SBD with risk factors leading to higher CVS morbidity in children. We proved CVS impairment not only in patients with OSA but also in patients with PS *(Tab. 2, Fig. 2, Ref. 48). Text in* PDF www.lekarsky.herba.sk.
KEY WORDS: obstructive sleep apnoea, primary snoring, hypertension, premature cardiovascular morbidity.
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Abstrakt
Ciele štúdie**:** Dôkazy o poruchách dýchania súvisiacich so spánkom (SBD) v súvislosti s kardiovaskulárnymi ochoreniami detí a o predčasných kardiovaskulárnych ochoreniach dospelých stúpajú, čo dokazuje, že nielen obštrukčné spánkové apnoe (OSA), ale aj primárne chrápanie (PS) je spojené s vyšším rizikom kardiovaskulárnej (CVS) morbidity. Súvislosť medzi SBD a predčasnou morbiditou CVS u pediatrických pacientov je stále nedostatočne preskúmaná.
Metódy: Naša študijná skupina pozostávala z 80 pacientov vyšetrených v Spánkovom laboratóriu Fakultnej nemocnice Martin (Slovensko) v rokoch 2019 až 2022. Pacienti podstúpili štandardnú celonočnú polysomnografiu (PSG).
Výsledky: Zistili sme a graficky sme preukázali zvýšenú predispozíciu k vyššiemu TK u pacientov s OSA a PS v porovnaní so zdravými kontrolami ($p < 0,001$), ale nie u pacientov s OSA v porovnaní s PS ($p > 0,05$). V skupine pacientov s PS percentil systolického tlaku koreloval s dĺžkou chrápania počas noci ($r = 0,4$; $p < 0,05$). Logistická regresia celého súboru údajov ukázala koreláciu AHI a priemernej nočnej srdcovej frekvencie (ANHR, $r = 0,36$; $p < 0,001$), ako aj chrápania a ANHR ($r = 0,32$; $p < 0,001$). Táto analýza ukázala, že chrápanie silne koreluje s celkovým indexom prebudenia (TAI, $r = 0,73$, $p < 0,001$) a AHI silne koreluje s TAI ($r = 0,87$, $p < 0,001$).
Záver: Prezentované údaje potvrdzujú asociáciu SBD s rizikovými faktormi vedúcimi k vyššej morbidite CVS u detí. Poruchu CVS sme dokázali nielen u pacientov s OSA, ale aj u pacientov s PS (tab. 2, obr. 2, lit. 48). *Text v PDF www.lekarsky.herba.sk.*
KĽÚČOVÉ SLOVÁ: obštrukčné spánkové apnoe, primárne chrápanie, hypertenzia, predčasná kardiovaskulárna morbidita.

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Introduction

Sleep-related breathing disorders (SBD) are conditions of abnormal and difficult breathing during sleep, primarily including snoring and sleep apnoea (1). Obstructive sleep apnoea (OSA) in children is sleep-disordered breathing characterised by a combination of repeated episodes of prolonged partial upper airway obstruction (obstructive hypopnea) and/or intermittent complete obstruction (obstructive apnoea) and sleep fragmentation attributable to recurrent arousals from sleep that disturb normal sleep patterns and normal ventilation during sleep; this results in the disruption of normal gas exchange (intermittent hypoxia and hypercapnia) (2–5). These intermittent episodes of partial or

complete cessation of airflow disturb the normal autonomic control of the cardiovascular (CVS) system (6). Further, oxygen desaturations and elevations in carbon dioxide levels cause surges in sympathetic activity and associated cardiovascular changes (7) and repetitive arousals have been linked to increased sympathetic activity (8).

It is well proven that OSA in adults, with disease lasting years, is significantly associated with higher CVS morbidity and mortality. Arterial hypertension, arrhythmias, pulmonary artery hypertension, ischaemic stroke, and coronary artery disease have all been scientifically proven to be associated with OSA in adults (9–11obstructive sleep apnea was a significant predictor of incident coronary heart disease (myocardial infarction, revascularization procedure, or coronary heart disease death). Extrapolating these findings to the younger population should lead us to the conclusion that OSA that develops early in life will harm more as it has more time to do so. The amount of evidence of the role of SBD in paediatric cardiovascular disease (CVD) and premature adult CVD is rising (12, 13), proving that not only OSA but also primary snoring is associated with higher risk of CVS morbidity.

One of the most significant risk factors for OSA in children is obesity. As childhood obesity rates continue to rise in many parts of the world, so does the prevalence of OSA (14). This is particularly concerning given the potential long-term health consequences of OSA, including an increased risk of CVD later in life meaning fewer productive years of life and fewer years of disease-free life. In addition to obesity, other significant risk factors for OSA in children include craniofacial abnormalities, enlarged tonsils or adenoids, and neuromuscular disorders (5, 15).

The prevalence of OSA in paediatric patients is difficult to estimate, as it is often underdiagnosed and underreported. However, it is believed to affect up to 5% of children, with higher rates in certain populations such as those with Down syndrome (5). OSA is also more common in boys than girls and tends to peak between the ages of 2 and 8 years (5, 14).

Primary snoring (PS), also known as simple or non-apnoeic snoring, is regarded as the first stage of sleep-disordered breathing without severe medical consequences for the snorer and co-sleeper (1). Although it is a highly prevalent phenomenon in the general population, our knowledge is limited because of the lack of a consensus on terminology (12). In recent years it has been brought to light due to high prevalence and its possible association with higher CVS morbidity. Snoring signifies a degree of narrowing in the upper airway, and habitual snoring (snoring > 3 nights per week), which affects around 10% of the paediatric population, is the most common symptom associated with OSA (16–18). Subjects with habitual snoring but normal overnight polysomnography (PSG) are often designated as having PS (19).

Several studies have investigated the association between PS and CVD in children. Different studies have found that children with PS have higher blood pressure and a higher prevalence of hypertension compared to children without PS. Another study found that children with PS had higher levels of inflammatory markers, which are associated with increased CVD risk (20). Additionally, studies have found that children with PS have impaired vascular endothelial function compared to children without PS (21–23). While these studies suggest an association between PS and CVD in children, it is important to note that the studies have several limitations. Most of the studies conducted have been cross-sectional in nature, which makes it difficult to establish a causal relationship between PS and CVD. Additionally, the studies have varied in their definitions of PS and CVD, which makes it difficult to compare results across studies.

One of the most significant cardiovascular consequences of OSA is the effect it has on blood pressure. During episodes of OSA, the obstruction of the upper airway leads to a decrease in oxygen levels and an increase in carbon dioxide levels in the blood. This triggers a cascade of physiological responses, including activation of the sympathetic nervous system and the release of stress hormones such as adrenaline and cortisol. These responses lead to situational tachycardia and hypertension, which cause harm throughout fixation of these pathological circuits with increasing levels of angiotensin II, catecholamines, and other vasopressors which affect blood pressure in the awake state as well (25, 26).

Repeated intrathoracic pressure changes during OSA contribute to the higher CVS morbidity (27). Chronic exposure to abnormal sharp fluctuations in blood pressure (BP) during and just after the course of each apnoeic event is thought to lead, eventually, to loss of normal nocturnal ‘dipping’ in average nocturnal BP as compared with daytime BP. Over the course of months to years, this could eventually lead to an increased risk for hypertension (1, 28, 29).

Research on adults has consistently shown that OSA (associated with frequent arousals and hypoxaemia) is associated with an increased risk of hypertension. Over time, this can lead to damage to the arteries and an increased risk of heart attack, stroke, and life-threatening arrhythmias (30). The severity of OSA appears to be directly related to the degree of BP elevation (31). This suggests (and has been scientifically proven) that treating OSA may help to lower BP and reduce the risk of CVD (32). In studies done with children, PS as well as OSA is associated with higher daytime systolic BP (12) and in a 5-year follow-up study patients with PS had worse outcomes in markers of CVS morbidity (carotid intima thickness, flow-mediated dilation) than their sex- and age-matched controls (33).

During OSA episodes, the occurred hypoxaemia and hypercapnia stimulate the sympathetic nervous system, which results in an increase in heart rate. The heart rate may also increase due to the body’s response to the cessation of breathing, which results in an activation

of the fight or flight response. Research has consistently shown that adult and paediatric individuals with OSA have higher resting heart rates than individuals without the condition (34, 35). The degree of heart rate elevation in adults with OSA appears to be related to the severity of the condition and resolves after appropriate treatment (35). The elevated heart rate during OSA episodes as well as during daily activities is an index of disrupted autonomic control and thus autonomic dysfunction (28). A higher resting heart rate has been associated with an increased risk of CVD, including hypertension, coronary artery disease, and stroke. Specifically, it is associated with higher risk of hypertension in paediatric patients regardless of adiposity, ethnicity, or age (36).

Methods

Our study group consisted of 80 patients examined in the Sleep Laboratory of the Clinic for Children and Adolescents of the University Hospital Martin between 2019 and 2022. The patients were referred for examination for suspected sleep-disordered breathing. At the time of examination, they were not diagnosed as hypertensive and had not been prescribed any medication affecting BP and heart rate. Patients with syndromological disorders (Prader Willi syndrome, Down syndrome), known epilepsy, or congenital metabolic diseases were excluded from the study.

The patients were hospitalised at the Clinic for Children and Adolescents, University Hospital Martin, Slovakia, and their BP was measured under standardised conditions by a trained nurse. The measurement was carried out in the morning hours in a sitting position after at least 5 minutes of rest; a total of at least three measurements were carried out with a correctly selected BP handcuff. An average was created from the measurements, which was then converted to a percentile value based on the current height value in centimetres. Anthropometry was done prior to admission to the hospital.

Patients underwent a standard all-night PSG. The PSG recording was evaluated by a trained technician and subsequently validated by a medical somnologist based on criteria established by the American Academy of Sleep Medicine. For the needs of the selected cross-sectional study, these parameters were evaluated – apnoea/hypopnea index (AHI), total snoring time in minutes, total snoring time expressed as a percentage of total sleep time (%TST), average night-time heart rate (ANHR), respiratory arousal index (RAI), leg movement arousal index (LMAI), spontaneous arousal index (SAI), and total arousal index (TAI). The ECG was monitored with 3 leads; the ANHR was recorded.

Based on the results of the performed examinations, the patients were divided into a group of patients with OSA (n = 24, 7 girls), a group of patients with PS (n = 27, 6 girls), and a group of healthy controls (n = 29, 8 girls). The characteristics of our study groups are summarised in Table 1.

To create a compiling dataset for cross-sectional study, the following criteria were set:

- A patient with a sufficiently high-quality PSG recording and an AHI of more than 5/hour of sleep, which corresponds to moderate/severe OSA in paediatrics, was considered a patient with OSA.
- A patient with a sufficiently high-quality PSG recording and an AHI of less than 1/hour of sleep (absence of OSA) and with the presence of snoring during at least 5% of the TST and anamnesis of snoring more than 3 days a week was considered a patient with PS.
- A patient with a sufficiently high-quality PSG with an AHI less than 1 and less than 5% snoring from the TST was considered a healthy control (HC).

Statistical analysis was performed using Microsoft Excel Analysis ToolPak and Systad 11. Descriptive characteristics across groups were compared using single-factor ANOVA with post hoc Tukey–Kramer analysis to establish statistical significance of the differences found. A logistic regression model was used to assess the relation between AHI, snoring (in minutes and %TST), BMI percentile, arousal indices, awake systolic and diastolic BP percentile (adjusted to the height of the patients), and ANHR.

Results

The results of studied parameters expressed as mean value (± SD) are presented in Table 2.

In the group of patients with OSA, 20 patients (83%) had a BMI percentile above 95% and 12 patients (50%) with OSA had confirmed adenotonsillar hypertrophy. In the group of patients with PS, 7 patients (26%)

Table 1. Baseline characteristics of study groups.

	OSA		PS		HC				
	n=24 average	7 girls SD	n=27 average	6 girls SD	n=29 average	8 girls SD	p value		
							OSA vs PS	OSA vs HC	PS vs HC
age (years)	10.8	4.1	10.4	4.1	9.8	4.3	†	†	†
height (cm)	146.6	23.7	141.9	23.5	140.8	24.7	†	†	†
weight	70.9	38.2	49.0	30.0	43.6	31.8	*	*	†
BMI (kg/m2)	30.0	10.4	22.2	7.9	19.5	6.6	*	**	†
BMI percentile	81.8	33.9	65.8	33.8	62.8	33.0	*	**	*

OSA – obstructive sleep apnoea, PS – primary snoring, HC – healthy controls, BMI – body mass index, BMI - body mass index, † = $p > 0.05$; * = $p < 0.05$; ** = $p < 0.001$

Table 2. Studied parameters.

	OSA		PS		HC				
	n=24	7 girls	n=27	6 girls	n=29	8 girls	p value		
	average	SD	average	SD	average	SD	OSA vs PS	OSA vs HC	PS vs HC
SYS percentil	82.0	14.2	84.6	16.3	62.4	32.3	†	*	*
DIA percentil	86.4	21.1	85.4	15.0	68.1	27.1	†	*	*
AHI (1/hour)	39.5	40.7	0.3	0.4	0.2	0.3	*	*	†
snoring (minutes)	165.9	113.2	66.5	51.3	0.6	1.3	**	**	**
snoring (%TST)	41.0	27.9	16.2	14.4	0.1	0.3	**	**	**
ANHR (1/min)	84.6	13.4	76.2	10.6	70.5	10.9	*	**	*
RAI	25.26	36.30	0.12	0.18	0.06	0.14	**	**	†
LMAI	2.30	4.24	2.00	3.67	2.04	3.27	†	†	†
SAI	21.50	13.16	17.54	9.48	12.70	5.81	†	*	*
TAI	49.78	42.79	20.20	8.89	14.79	5.61	**	**	†

OSA – obstructive sleep apnoea, PS – primary snoring, HC – healthy controls, BMI – body mass index, BMI - body mass index, AHI - apnoe--hypopnoe index, %TST - % of total sleep time, ANHR - average night-time heart rate, RAI - respiratory arousal index, LMAI - leg movement arousal index, SAI - spontaneus arousal index, TAI - total arousal index, † = $p > 0.05$; * = $p < 0.05$; ** = $p < 0.001$

had a BMI percentile above 95% and 2 patients (7%) had confirmed adenotonsillar hypertrophy. In the group of HC, 8 patients (27.5%) had a BMI percentile above 95% and none of the patients had confirmed adenotonsillar hypertrophy.

In our cross-sectional study, we were able to determine and graphically demonstrate an increased predisposition to higher BP (both systolic and diastolic) in patients with OSA and PS compared to HC ($p < 0.001$) but not in patients with OSA compared to PS ($p > 0.05$). In particular, a tendency to elevated diastolic pressure was observed. Average values of systolic BP in the monitored groups (82nd BP percentile in OSA vs 84.6th BP percentile in PS vs 62.4th BP percentile in HC) and diastolic BP (86.4th BP percentile in OSA vs 85.4th BP percentile in PS vs 68.1th BP percentile in HC) point to impaired BP regulation not only in patients with OSA but also in patients with PS. In the group of patients with PS, the systolic pressure percentile correlated with duration of snoring during the night ($r = 0.4$; $p < 0.05$). The graphic representation of these data in Figure 1 also displays the loss of physiological dispersion of measured values, with a significant shift towards higher percentiles compared to HC. A whole-dataset correlation of BMI percentile and BP revealed a close relation of BMI and systolic ($r = 0.49$; $p < 0.001$) and diastolic ($r = 0.42$, $p < 0.001$) BP but no correlation with AHI ($r = 0.21$, $p > 0.05$), nor of snoring duration ($p = 0.08$, $p > 0.05$) with BMI percentile. A separate logistic regression of a specific study group did not prove close correlation of AHI and systolic and diastolic BP percentile in either of the studied groups.

We were able to prove that patients with OSA have a higher ANHR than patients with PS ($p < 0.05$) and HC ($p < 0.005$) but patients with PS did not have a significantly higher ANHR compared to HC ($p > 0.05$). A separate logistic regression of patients with PS found correlation of duration of snoring (in minutes) with ANHR ($r = 0.4$; $p < 0.05$). The changes in ANHR are presented in Figure 2.

A logistic regression of the whole dataset from all three study groups showed correlation of AHI and ANHR ($r = 0.36$; $p < 0.001$) as well as snoring (in minutes) and ANHR ($r = 0.32$; $p < 0.001$), and snoring (%TST) and ANHR ($r = 0.31$, $p < 0.001$). This whole-dataset analysis showed that snoring (duration and %TST) correlates strongly with TAI ($r = 0.73$, $p < 0.001$ and $r = 0.67$, $p < 0.001$) and AHI correlates strongly with TAI ($r = 0.87$, $p < 0.001$). ANHR correlated with TAI ($r = 0.42$, $p < 0.001$) and RAI ($r = 0.39$, $p < 0.001$).

Post hoc analysis revealed that sleep fragmentation represented as TAI is highest in patients with OSA (significantly higher than PS and HC; $p < 0.001$). The RAI of the group with diagnosed OSA was significantly higher in comparison to PS and HC (both $p < 0.001$) but did not differ between PS and HC ($p > 0.05$). The SAI was also highest amongst patients with OSA compared to patients with PS and HC ($p < 0.05$) but did not differ between PS and HC ($p > 0.05$).

Discussion

SBD are well-described risk factors for cardiovascular morbidity in adult patients (37). The impact of SBD on the cardiovascular system includes effects on BP, myocardial structure and function, endothelial function, and cardiac autonomic activity (1, 28). These factors alone and especially in combination can lead to significant cardiovascular morbidity. The link between SBD and CVD in children became more apparent over time as we gathered data supporting these events in children and adolescents (28).

Despite the availability of effective treatments, many paediatric patients with OSA continue to experience cardiac morbidity. This highlights the need for increased awareness of the potential cardiovascular consequences of OSA in children, as well as the importance of early

Figure 1. Systolic and diastolic blood pressure regulation amongst studied groups (OSA – obstructive sleep apnoea, PS – primary snoring, HC – healthy controls, SYS – systolic percentile, DIA – diastolic percentile)**.**

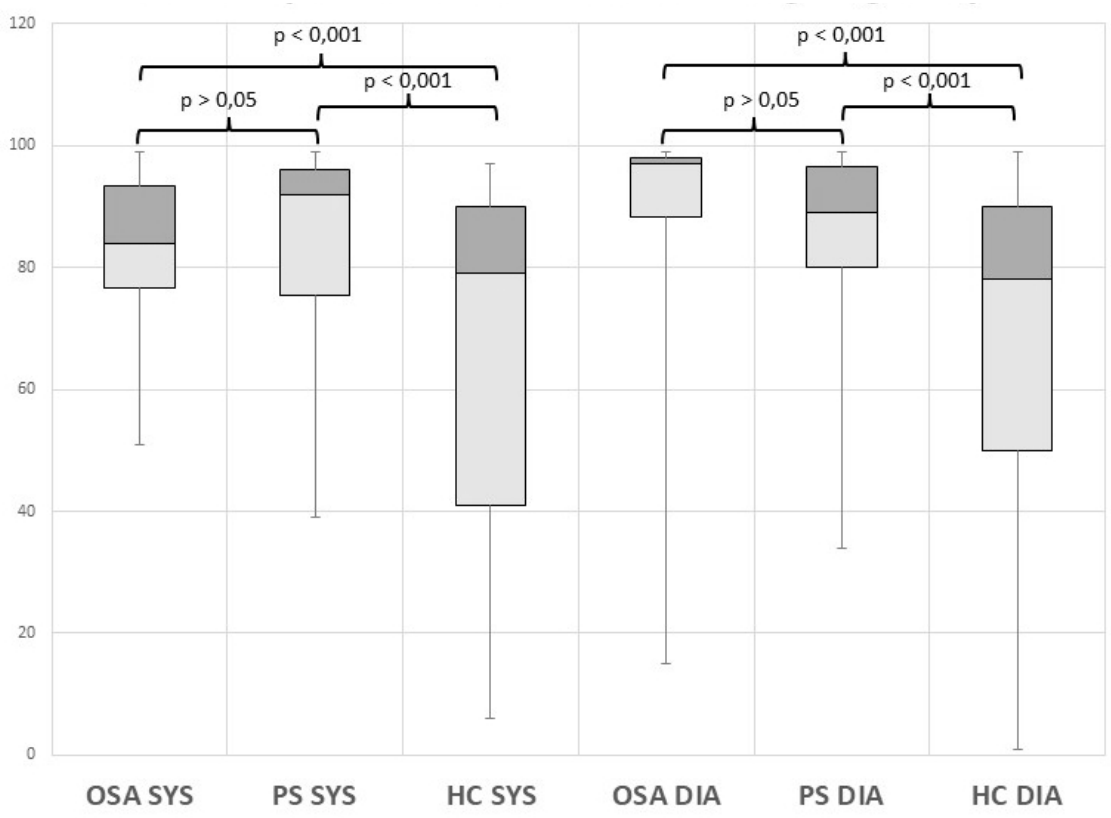
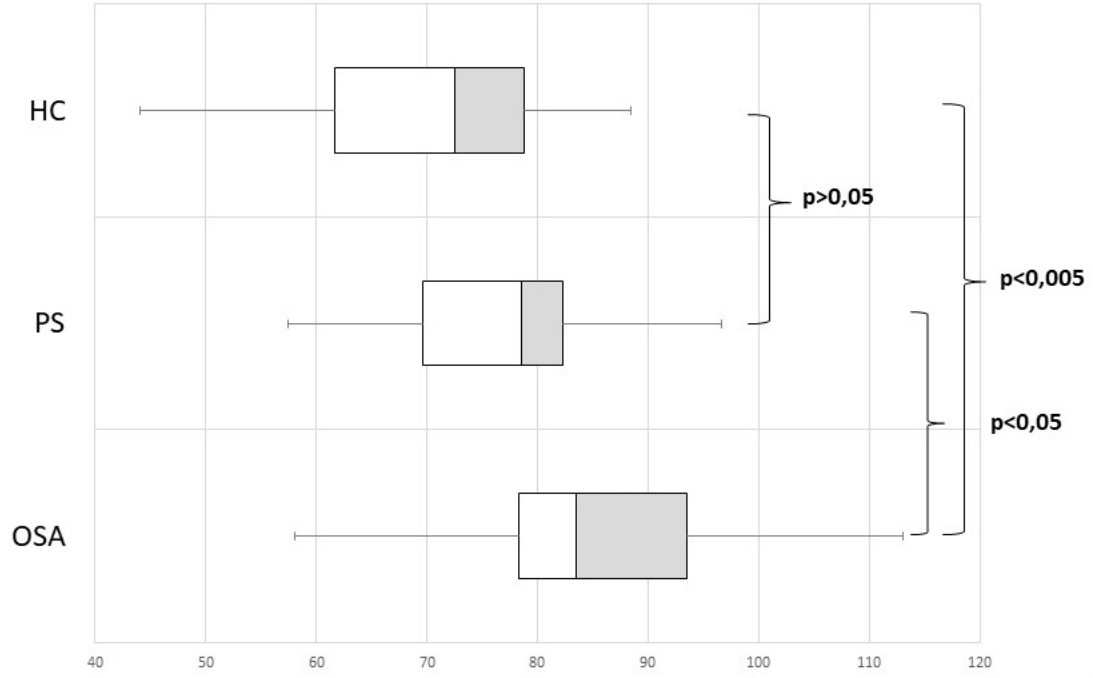


Figure 2. Average night-time heart rate in study groups (OSA – obstructive sleep apnoea, PS – primary snoring, HC – healthy controls)**.**



diagnosis and treatment (38). Ongoing research is needed to better understand the mechanisms by which OSA contributes to cardiac morbidity in paediatric patients, as well as to develop new and more effective treatments.

Our selected group of patients diagnosed with OSA consists of patients only with moderate to severe OSA. Those having mild OSA (AHI > 1–5) were excluded as they might not be exhibiting signs of cardiovascular derangement since previous studies have confirmed that

seriousness of cardiovascular signs correlates with the severity of disease. Thus, these patients were not included for statistical analysis.

Data provided within our dataset confirm the presence of higher BP in the patients (to the moment of PSG, non-hypertensive children) with SBD. Using single-factor ANOVA and post hoc Tukey–Kramer analysis, we confirmed BP dysregulation in both groups with SBD compared to the HC. In the graphic depiction of our findings, it is mentionable that patients with SBD had less physiological dispersion of measured values compared to the HC, which is indicative of dysregulation of BP not only in patients with moderate to severe OSA but also in patients with PS. These data are in line with the observational meta-analysis of Kwok et al. that proved the presence of higher BP in paediatric patients with OSA (39); nevertheless, the data from the time of the study did not take into account BP percentile as a diagnostic criterion for hypertension. This limitation was addressed by Zintzaras (40) in 2007 using defined criteria for meta-analysis which included BP percentile but the heterogeneity of the measurement methodology used in analysis brought conflicting results as the BP was in some cases monitored during PSG in short intervals (15 minutes during the whole night) and in some cases measured as in-office measurement before unattended home PSG.

The data provided from the study of Hinkle et al. (41) are consistent with our findings but the level of correlation of BP and OSA severity was not confirmed in our study. This discrepancy is attributable to the patient selection criteria since Hinkle et al. enlisted only patients with known hypertension, thus they had a significantly different study population. The association of higher BP and higher BMI is an established fact as it is known that obese children have greater odds for hypertension and its consequences (42).

Considering this information, we cannot neglect the influence of BMI percentile on our findings. Of those patients having OSA and a BMI percentile below 95%, none had systolic or diastolic BP above the 95th percentile for their height. Knowing that OSA is closely associated with obesity, it is difficult to establish which of the pathological processes started this vicious circle. Not neglecting obesity as a confounding parameter, there are studies describing the relationship of elevated systolic BP with OSA severity amongst normal-weight children (41) (mainly due to adenotonsillar hypertrophy), which support the idea of BP dysregulation independently being affected by OSA. A possible explanation for the absence of statistical power of correlations of systolic and diastolic pressure percentile with AHI and snoring might be explainable by the method of using the percentile as an adjusted value for BP, where it is not possible to get higher than the 99th BP percentile since every value over the 99th percentile is not expressed as a higher number that could be correlated to higher AHI. Therefore, the need for better methodology for assessment of BP in paediatrics is rising.

In our study group of patients with PS, we found correlation between snoring duration (in minutes) and systolic BP percentile. This finding corresponds with the findings of other authors (12,43). The precise definition of PS is still pending but in line with similar criteria used by Li et al. we confirm that PS is associated with higher BP and in our study also that systolic BP correlates with duration of snoring during sleep, and therefore there is a need to consider this observation when dealing with paediatric patients with hypertension.

The nocturnal decrease in heart rate during sleep is a sign of good function of the autonomic nervous system and the circadian rhythmicity system, which is necessary for adequate regeneration of the organism (44,45). In patients with OSA, there is a marked persistence of higher ANHR during sleep (34). In our study, the mean ANHR in patients with sleep-disordered breathing (SDB) was higher compared to HC and higher in patients with OSA but did not reach the values observed in patients with PS. The sleep fragmentation represented as TAI correlated strongly with ANHR in the whole dataset which might explain the higher ANHR even in the group of PS patients who do not have OSA but have higher ANHR. The SAI (that was higher in PS and OSA groups) corresponding with arousals that occur secondary to the auditory sounds (snoring itself) and are accompanied by elevation in heart rate might explain the higher ANHR in the group of patients with PS who do not have OSA.

The most effective treatment for OSA in terms of BP management is treatment of the OSA itself. In paediatric patients, treatments for OSA involve lifestyle modifications (e.g., weight loss, exercise) and surgical interventions (e.g., removal of enlarged tonsils and adenoids) which may also lead to a reduction in BP, although the evidence is less clear (38). In more severe or resistant cases, treatment with non-invasive ventilation is used. Studies have consistently shown that the use of CPAP machines can lead to a significant reduction in BP in individuals with OSA, even in those who do not have hypertension at baseline (46). Additionally, medications such as antihypertensives may be necessary in some individuals with OSA who have persistent hypertension despite other treatments (47).

The treatment of OSA leads to a lowering of resting heart rate. The most effective treatment for OSA, CPAP therapy, can lead to a reduction in heart rate, particularly in individuals with more severe forms of the condition (35). In addition to CPAP therapy, lifestyle modifications, such as weight loss and exercise, can also lead to a reduction in heart rate in individuals with OSA (48). Medications, such as beta-blockers, may also be used to lower heart rate in individuals with persistent elevations despite other treatments.

Conclusion

According to the data we present, patients with SDB have a higher risk of cardiovascular morbidity. It is necessary to verify the reversibility of these findings in

the treatment of SDB. Patients with OSA and PS should be examined by a cardiologist, undergo an ambulatory BP measurement, and, according to the findings, be treated in cooperation with a paediatric somnologist with the aim of terminating the pathological circuits that have started, which would lead to the development of hypertension, heart failure, arrhythmias, pulmonary hypertension, or ischaemic heart and brain diseases in the future.

Limits of the study

Our study is based upon standardised whole-night PSG which is a valid tool to diagnose SDB in paediatric patients. Patients selected for the study were referred for examination with the suspicion of SDB, thus the selection bias was unavoidable. The study design is cross-sectional and long-term and in-depth observations are needed to elucidate the validity of the findings as well as the study of effectivity of therapeutic interventions. In-office BP measurements were performed by a skilled and empathetic nurse that patients had a chance to get to know before measurement and were performed according to strict criteria. Nevertheless, a single in-office measurement cannot replace ambulatory BP monitoring that can mitigate the risk of whitecoat hypertension. In accordance with our observations, we continue the clinical research and offer the patients the possibility to elucidate current findings with other tools available.*

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