Rhythm disturbances in childhood obstructive sleep apnea during apnea-hypopnea episodes

Anant Khositseth, Jittamas Chokechuleekorn, Teeradej Kuptanon, Anchalee Leejakpai
Department of Pediatrics, Faculty of Medicine, Ramathibodi Hospital, Mahidol University, Bangkok, Thailand

ABSTRACT

Background : **Obstructive sleep apnoea (OSA) can result in cardiovascular complications. Nocturnal arrhythmias are reported up to 50% of adult OSA patients. Arrhythmias and heart rate variability in children with OSA have not been well studied.**

Aims : **We sought to study rhythm disturbances in childhood OSA and also to analyze the relationship of heart rate variability to the severity of OSA in children.**

Methods : **In a retrospective cross sectional study, records of children aged < 15 years with history of snoring and suspected OSA, who had undergone polysomnography (PSG) for first time were analyzed. The cardiac rhythm and heart rate variability were studied during PSG.**

Results : **A total of 124 patients diagnosed with OSA were grouped into mild (n = 52), moderate (n = 30), and severe (n = 42) OSA. During PSG, all had sinus arrhythmias and only three patients had premature atrial contractions (PACs). The standard deviation of heart rate (SD-HR) during rapid eye movement (REM) sleep in severe OSA (9.1 ± 2.4) was significantly higher than SD-HR in mild OSA (7.5 ± 1.3, P < 0.0001). The maximum heart rate (max-HR) during REM-sleep in severe OSA (132.1 ± 22.1) was significantly higher than the max-HR in mild OSA (121.3 ± 12.6 bpm, P = 0.016).**

Conclusions : **There was no significant arrhythmia in children with OSA during their sleep. Heart rate variability correlated with the severity of OSA.**

Keywords : Arrhythmia, children, obstructive sleep apnea

INTRODUCTION

Obstructive sleep apnea syndrome (OSAS) in children is the most extreme variant of sleep-disordered breathing (SDB), characterized by intermittent but prolonged episodes of partial obstruction (obstructive hypopnea) and/or complete obstruction (obstructive apnea) of the upper airway, which disrupt normal ventilation and nocturnal sleep physiology.[1] Its prevalence in pediatric populations is approximately 2% (1-4%) with the peak prevalence between 2 and 8 years of age when tonsils and adenoids are largest in relation to the upper airway size.[2-5] OSAS is diagnosed when a child has an apnea–hypopnea index (AHI), number of apnea and hypopnea episodes per hour of sleep > 5, and symptoms of excessive daytime sleepiness based on polysomnography (PSG).[6,7] The association between OSAS and cardiac arrhythmia has been published. Beat-to-beat variation in heart rate is typically seen in SDB.[8] Cardiac arrhythmias are presumed to be common in patients with OSAS, however, the true prevalence and clinical relevance of cardiac arrhythmias remain unknown. The presence and complexity of tachyarrhythmias and bradyarrhythmias may influence morbidity, mortality, and the quality of life for patients with OSAS.[9,10] The most common arrhythmias during sleep include nonsustained ventricular tachycardia (VT), sinus pause/arrest, second degree atrioventricular (AV) conduction block and frequent premature ventricular contractions (PVCs > 2 bpm).[11,12] The prevalence of arrhythmias in the children with OSAS is not well defined.[13,14] In this study, we used standardized data collection, including electrocardiogram (ECG) during an overnight sleep study in our sleep laboratory in pediatric

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Address for correspondence: Dr. Anant Khositseth, Department of Pediatrics, Faculty of Medicine, Ramathibodi Hospital, 270 Rama VI Road, Ratchathewi, Bangkok, Thailand. E-mail: khositseth@gmail.com

population. This study is aimed to find the episode of arrhythmias and the association of arrhythmia and severity of the OSAS during nocturnal sleep in children with OSAS.

MATERIALS AND METHODS

We retrospectively reviewed records of children with history of snoring who underwent overnight PSG to confirm the diagnosis and grading the severity of OSA. Overnight PSG was performed according to the American Thoracic Society standards[1] (Alice 3; Respironics; Murrysville, PA; or Seista; Compumedics; Abbotsford, Australia). No sedation or sleep deprivation was used. The following parameters were recorded during the study: (1) Electroencephalogram (EEG); (2) right and left electrooculogram; (3) submental and tibial electromyogram; (4) ECG; (5) oronasal airflow thermistor or cannula; (6) oxygen saturation monitoring (Health dyne Oximeter model 930; Respironics); (7) chest and abdominal wall motion by computer-assisted respiratory inductance plethysmograph; and (8) snoring microphone. Sleep stages were determined according to the Rechtschaffen and Kales criteria.[15] Arousals were defined as recommended by the American Academy of Sleep Medicine (AASM).[16] Obstructive apnea was defined as the cessation of airflow despite breathing effort for more than two respiratory cycles. Obstructive hypopnea was defined as the decrease of airflow by > 50%, but < 80% of baseline associated with desaturation of ≥4% or arousal despite breathing effort. The OSA was diagnosed according to AASM's diagnostic criteria for OSA including respiratory events as OSA and hypopnea.[16] The ratio of the numbers of obstructive apnea to hypopnea in 1 hour defined as AHI. In children, an apnea index > 1 was statistically abnormal, and the upper limit of AHI was reported to be 1.5.[16] In this study, AHI > 1.5 was used to define OSA. The severity of OSA was graded to be mild (AHI > 1.5 and <5), moderate (AHI 5-10), and severe (AHI > 10).

Arrhythmias defined as premature atrial contraction (PAC), PVC, AV block including second and third degree AV block, atrial flutter (AFl), atrial fibrillation (AF), supraventricular tachycardia (SVT), VT, and ventricular fibrillation (VF) were reviewed in single lead ECG.

Standard deviation of heart rate (SD-HR), minimal heart rate (min-HR), and maximum heart rate (max-HR) in each stage of sleep were obtained.

Statistical analysis

Student's *t* test was used for continuous variables to compare between two groups and analysis of variance (ANOVA) test to compare among three groups. *P* value of < 0.05 was considered statistically significant.

RESULTS

A total of 213 cases with history of suspected OSA underwent PSG in the 3-year study period, in which 89 cases were excluded. These were due to CPAP or BipAP (positive airway pressure machines) during the study ($n = 32$), broken recordings ($n = 20$), repeat study ($n = 15$), and miscellaneous causes ($n = 22$) like central hypoventilation in 4, airway/pulmonary abnormalities in 5, congenital heart disease in 2, Prader–Willi syndrome in 4, etc., A total of 124 cases (mean age 6.7 ± 3.4 years, 26% females) were diagnosed as OSA and enrolled in this study. We graded severity of OSA into three groups: Mild OSA ($n = 52$), moderate OSA ($n = 30$), and severe OSA ($n = 42$). Mean age, sex, body weight, and height were not significantly different in each group [Table 1]. However, the body mass index (BMI) in severe OSA was significantly higher than BMI in mild OSA. The severity of obesity based on ideal weight for height in each group was not significantly different.

Lead II ECG tracing during PSG in each stage of sleep demonstrated sinus arrhythmias, defined as sinus rhythm with varying R–R intervals, in all cases. Only three cases (2.4%) had occasional PAC. There were no episodes of other arrhythmias including PVC, AV block, AFl, AF, SVT, VT, and VF. Most cases, 100 of 124 cases (80.7%) had episode of sinus bradycardia (heart rate was less than normal value for age).[17]

SD-HR, Min-HR, and Max-HR in each stage of sleep including rapid eye movement (REM) and nonrapid eye movement (NREM) in each group were compared as shown in Table 2. In NREM sleep, the min-HR, the max-HR, and the SD-HR in mild, moderate, and severe OSA groups were not significantly different [Table 2]. In REM sleep, the min-HR in REM sleep was not significant different in each group, however, the SD-HR and max-HR were significantly higher in severe OSA than in mild OSA (9.1 ± 2.4 compared with 7.5 ± 1.3, $P < 0.0001$, and 132.1 ± 22.1 compared with

Table 1: Demographics data in each group according to severity of obstructive sleep apnea

Variables	Severity of obstructive sleep apnea			*P* value
	Mild (*N*=52)	Moderate (*N*=30)	Severe (*N*=42)	
Age (years)	6.9±3.4	6.3±2.7	6.9±4.0	NS
Body weight (kg)	26.7±16.7	27.6±17.6	34.8±23.9	NS
Height (cm)	117.9±18.9	116.4±16.9	121.3±24.5	NS
BMI (kg/m^2)	17.7±4.9	18.8±6.0	21.1±7.3	0.03
Sex (Male: Female)	2.1:1	2.3:1	5:1	NS
No obesity (%)	59.6	60.0	40.5	NS
Overweight (%)	17.3	10.0	16.7	NS
Mild obesity (%)	11.5	6.7	14.3	NS
Moderate obesity (%)	5.8	13.3	14.3	NS
Severe obesity (%)	3.8	6.7	9.5	NS
Morbid obesity (%)	1.9	3.3	4.8	NS

BMI: Body mass index, NS: Not significant

Table 2: Variables of heart rate in each group according to severity of obstructive sleep apnea

Variables of heart rate (beats/min)	Severity of obstructive sleep apnea			*P* value
	Mild (*N*=52)	Moderate (*N*=30)	Severe (*N*=42)	
SD: HR-NREM	7.4±1.1	7.7±1.4	7.8±1.5	NS
SD: HR-REM	7.5±1.3*	8.4±1.6	9.1±2.4*	<0.0001
Min HR-NREM	41.1±18.4	42.2±21.2	38.8±21.8	NS
Min HR-REM	57.8±8.4	56.2±11.8	61.2±9.3	NS
Max HR-NREM	95.9±28.1	100.4±36.1	92.4±42.9	NS
Max HR-REM	121.3±12.6**	125.8±13.4	132.1±22.1**	0.016

HR: Heart rate, Max: Maximum, min: Minimum, NREM: Nonrapid eye movement, REM: Rapid eye movement, SD: Standard deviation

121.3 ± 12.6, $P = 0.016$, respectively).

Subgroup analysis in each group including mild OSA with or without obesity, moderate OSA with or without obesity, and severe OSA with or without obesity demonstrated that the SD-HR, min-HR, and max-HR in NREM and REM sleeps were not significantly different, except min-HR in REM sleep in severe OSA with obesity (57.3 ± 7.7 was significantly less than min-HR in REM sleep in severe OSA without obesity (66.2 ± 9.0), $P = 0.004$.

DISCUSSION

All the cases in the present study were diagnosed by overnight PSG, which is the gold standard for the diagnosis of OSAS. Using new criteria,[16] we graded severity of OSA into mild, moderate, and severe OSA.

Arrhythmias including AF, nonsustained VT, sinus pause/arrest, second-degree AV block, and PVC have been described in adults with OSA.[11] Nocturnal arrhythmias were reported in up to 50% of adults with OSA.[18] However, the incidence and prevalence of arrhythmias in SDB is still poorly defined, because of small number of cases, lack of controls, and other confounding variables in the reported studies.[14]

Several mechanisms have been postulated for cardiac arrhythmias in OSAS including hypoxia, and sympathetic activation, but the exact mechanisms remain speculative. Proposed mechanisms might start with repetitive pharyngeal collapse during sleep resulted in oxygen desaturation, followed by persistent inspiratory efforts against an obstructed airway, and termination by arousal from sleep in combination with varieties of autonomic, hemodynamic, humoral, and neuroendocrine responses.[19,20]

This study demonstrated that the incidence of arrhythmias in children with OSA was quite low, and we found only PACs, which are benign arrhythmias. Although sinus arrhythmia was found in all patients but this finding should not be accounted for arrhythmia since it is a normal variation found commonly in a healthy child. Sinus bradycardia was common, however, there was no clinical significance. Even in normal subjects, rhythm changes could be found during sleep including sinus pause (>2 s), sinus bradycardia (<40 bpm), first degree AV block, and Wenckebach second degree AV block.[21] We speculated that the lower incidence of arrhythmia during sleep in childhood OSA was due to two reasons, first, the underlying mechanisms of OSA, which are much different between children and adults. Secondly, most elderly adults have predisposing factors for arrhythmias by their own risks including coronary artery disease, obesity, and myocardial dysfunction.

Normally, during the non-REM sleep, the HR decreases 5-10% (55-60 bpm) when compared with the HR during the wakeful period.[22] During the REM sleep, the HR increases with the max-HR accompanying increase in the autonomic arousal mechanism associated with the activation of neuronal systems around pontine systems.[23] REM is a unique sleep stage from a physiological point of view because the heart rate and breathing rate are similar to wakeful period. During REM sleep it is common for arrhythmias from autonomic nervous system instability. Our data suggested that in severe OSA there might be more deviation of the max-HR during REM sleep. This could be indirect evidence of more heart rate variability in severe OSA.

CONCLUSIONS

There was no significant arrhythmia in children with OSA during their sleep. Heart rate variability represented by standard deviation of heart rate was significantly higher in severe OSA when compared with mild OSA.

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