

Sleep and Cardiovascular System in Children

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KEYWORDS

• Sleep • Children • Cardiovascular • Congenital

KEY POINTS

- Cardiovascular (CV) physiology and pathophysiology differ in children compared with adults.
- Obstructive sleep apnea (OSA) and central sleep apnea (CSA) occur in children due to diverse and multifactorial causes.
- Congenital heart disease (CHD), craniofacial anomalies, clinical syndromes, and the related sleep and CV morbidity are unique to the pediatric age group.

INTRODUCTION

Current subspecialty pediatric practice provides comprehensive medical care for a wide range of ages, from premature infants to children, and often includes adults with complex medical and surgical issues that warrant multidisciplinary care. Normal physiologic variations involving different body systems occur during in sleep and these vary with age, stage of sleep, and underlying health conditions. The practice of pediatric sleep medicine is exciting due to the broad changes in cardiopulmonary physiology seen with normal growth and maturation and the plethora of congenital or acquired medical and surgical pathologies affecting this population. Sleep-related breathing disorders in children occur along a spectrum of severity, ranging from mild snoring to severe obstructive apnea, central apnea, and nocturnal hypoventilation. There is limited information available on the bidirectional effect of sleep-disordered breathing (SDB) causing CV morbidity and vice versa, especially in predisposed populations. This article is a concise review of the CV physiology and pathophysiology in children during SDB contributing to CV morbidity, congenital and acquired CV pathology resulting in SDB, and the relationship between

SDB and CV morbidity in different clinical syndromes and systemic diseases in the expanded pediatric population.

PHYSIOLOGY AND PATHOPHYSIOLOGY

Cardiovascular Physiology of Sleep in Children

With normal maturation of the neonate into childhood, changes in CV function during sleep reflect maturation of the autonomic nervous system (ANS), with higher parasympathetic control observed with increasing age.¹ An important indicator of ANS activity is beat-to-beat heart rate variability (HRV), and in healthy infants and children, age and sleep stage influence short-term HRV.²

Heart rate (HR) and blood pressure (BP) are lower during non-rapid eye movement (NREM) sleep than during wakefulness throughout maturation from the neonatal period to childhood. During transition from quiet wake to quiet sleep stage, HR decreases by 4 beats per minute to 8 beats per minute and BP decreases by approximately 14 mm Hg³ along with a reduction in cardiac output. During tonic rapid eye movement (REM) state, however, an 8% drop in HR along with a simultaneous drop in BP (up to 25 mm Hg) and a

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significant reduction in cardiac output (up to 9% drop compared with QW) has been observed.^{4,5} HRV is also higher in REM sleep than in NREM sleep, especially during phasic REM sleep. Cutaneous vasoconstriction is noted briefly during REM sleep, along with regional vasoconstriction to specific organ systems.

Pathophysiology

A diminished HR variability with respiration during specific sleep stages has been described in infants with underlying medical or surgical disease, in infants at risk for sudden infant death syndrome (SIDS) or those who later succumbed to SIDS, and in those with hypoventilation syndromes.⁶ Autonomic modulation shifts toward parasympathetic activity from wake to NREM and reverses to less parasympathetic modulation during REM sleep in normal children. With moderate SDB, however, autonomic modulation is impaired. This leads to higher arrhythmia vulnerability, especially during REM sleep,⁷ although the frequency of nocturnal arrhythmias is less common in children compared with adults with SDB.⁸ Children with more disrupted sleep (increased activity, wake after sleep onset, and long wake episodes) had lower respiratory sinus arrhythmia at baseline and reactivity, suggesting increased ANS arousal, which interferes with sleep.⁹

Sleep duration inversely predicts cardiometabolic risk in obese adolescents, even when controlling for physical activity, anthropometry, and adiposity. Long sleep duration has been significantly associated with lower ambulatory systolic and diastolic BP.^{10,11} In a cross-sectional study involving more than 300 healthy prepubertal children, however, sleep regularity was more prominently associated with both metabolic regulation (insulin resistance) and inflammation (high-sensitivity C-reactive protein) than sleep duration.¹² The CV pathophysiology of OSA and CSA is discussed.

Although cardiac variations during sleep in newborns, infants, and children are complex in a medically susceptible child, these variations are of clinical significance, especially during phasic excitatory stages of sleep.

SLEEP-DISORDERED BREATHING AND CARDIOVASCULAR DISEASE IN CHILDREN

Obstructive Sleep Apnea

OSA is secondary to intermittent upper airway obstruction (UAO) during sleep, with increased work of breathing, worsened negative intrathoracic pressures, frequent arousals, sleep disruption, and disturbances in blood gas exchange. Long-standing UAO results in CV complications,

such as endothelial dysfunction, chronic systemic inflammation via oxidative stress, and increased sympathetic tone resulting in BP abnormalities and CV structural modifications.¹³ In healthy individuals, sympathetic output increases in REM sleep and during arousals,¹⁴ with stimulation of chemoreceptors by hypoxia and hypercapnia also increasing sympathetic activity. In patients with OSA, abnormal gas exchanges and arousals augment the sympathetic tone even more along with surges in certain catecholamines,^{15,16} which result in vasoconstriction and further elevations in BP.

Few data are available regarding CV morbidity in children with OSA syndrome. In children, exaggerated sympathetic responses and increased HR fluctuations were observed in those with OSA compared with controls^{17,18} and treatment of OSA has shown reduction in both sympathetic tone and plasma and urine levels of catecholamines.^{19,20}

In small cohort studies of children with snoring and OSA, investigators have reported increased diastolic BP and higher awake BP and systolic BP during REM sleep.^{21,22} Pediatric studies with 24-hour ambulatory BP monitoring showed increased BP variability and decreased nocturnal BP dipping.^{23,24} In a meta-analysis of pediatric studies, even though not statistically significant, it was observed that during wakefulness, moderate to severe OSA syndrome was associated with an 87% and 121% higher risk for increased systolic and diastolic BP, compared with mild or no OSA, and during sleep, the random effects odds ratio for elevated SBP was 1.2 (95% CI, 0.29–5.02) and fixed effects OR for elevated diastolic BP was 2.23 (95% CI, 0.61–8.16).²⁵ In summary, the trend is that higher systemic BP (especially the diastolic BP) is observed with greater severity of OSA in children.

Intermittent nocturnal hypoxemia increases oxidative stress and production of leukocyte adherence molecules, which may result in endothelial dysfunction and vascular injury. Chronic hypoxemia, increased sympathetic tone, and exaggerated negative intrathoracic pressures during inspiration against an obstructed airway are important OSA-related consequences affecting preload and afterload on the cardiac ventricles.¹³ Chronic mechanical strain to the cardiac ventricles, with reductions in the right ventricular (RV) and left ventricular (LV) stroke volumes due to episodic surges in afterload after fluctuations in the pulmonary and systemic arterial pressures are seen with obstructive events.^{26,27} As discussed previously, OSA is associated with LV dysfunction along with RV changes. Amin and colleagues²⁸ reported that children with higher

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