Cardiovascular Disease and Sleep Disordered Breathing: Are Children Vulnerable?


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In recent years, evidence that sleep disordered breathing in children leading to several end organ morbidities has emerged. Of particular importance is the fact that the cardiovascular system may manifest a variety of maladaptive responses to the presence of sleep disordered breathing. The combination of large swings in intrathoracic pressures in the context of obstructed breathing, coupled with recurrent arousals and resultant sleep fragmentation, as well as gas exchange abnormalities, all likely contribute to the development of cardiovascular disease.1 While the exact mechanisms have yet to be elucidated, it is currently thought that alterations in autonomic nervous system tonic and reflexive functions, disruption of endothelial homeostasis, and systemic inflammation may all lead to accelerated atherosclerosis and disruption of vasomotor control.

In the paper by O’Driscoll and colleagues2 in this issue of SLEEP, 50 children with a history of snoring undergoing nocturnal polysomnography were recruited to investigate the effects of sleep-related obstructive events on heart rate (HR) and mean arterial pressure (MAP), 2 of the common surrogate markers of cardiovascular function. Specifically, they assessed these outcomes using finger photoplethysmography, which allows for beat-to-beat noninvasive measurements of HR and MAP throughout the night. Thus, the effects of arousals and/or obstructive respiratory events on HR and MAP could be examined. In addition, fluctuations of MAP and HR were determined by partitioning respiratory events into several phases.

Although photoplethysmography enables noninvasive cardiovascular monitoring with a relatively high temporal resolution, it is not without pitfalls, since data from 9 (18%) of the children were excluded due to motion artifact and possible patient discomfort. Nevertheless, the authors were able to analyze valid data from 30 children with obstructive events, and their findings are intriguing. First and foremost, increases in MAP of 19 mm Hg during non-rapid eye movement (NREM) sleep and 13 mm Hg in rapid eye movement (REM) sleep were found during the time period elapsing from the late phase of an obstructive apnea to the postapneic phase. Similarly, the investigators reported HR increases of 21 beats per minute in NREM sleep and 15 beats per minute in REM sleep for the same segments of the apneic events. Not only are the changes comparable with those previously documented in adults with obstructive sleep apnea (OSA),3,4 but, interestingly, the ∆HR and ∆MAP changes were significantly greater when obstructive events occurred in NREM sleep. Although REM sleep-related obstructive events were associated with more profound oxyhemoglobin desaturations, and therefore it would be expected that vascular changes would be most prominent in REM sleep, this was not the case. The authors speculate that this REM-NREM discrepancy was related to the improved ventilatory responses to hypoxia in NREM sleep,5 such that the larger inspiratory responses in NREM sleep would be responsible for larger oscillations in HR and MAP. Indeed, the degree of oxyhemoglobin desaturation and length of the respiratory event did not significantly predict ∆HR and ∆MAP. These findings would then suggest that autonomic nervous system responses in children are more robust in NREM sleep.

Another important observation in this study is that both subcortical arousals and arousals defined according to the American Sleep Disorders Association criteria were significant predictors of ∆HR and ∆MAP. Since children with OSA are more prone to having subcortical arousals during obstructive respiratory events,6,7 the use of markers of autonomic activation for scoring respiratory events in pediatric polysomnography was previously studied.8 The significant correlation of arousals and ∆HR and ∆MAP changes reported by O’Driscoll et al2 would suggest a potential role for finger photoplethysmography during polysomnography in estimating severity of not only sleep disordered breathing, but also of cardiovascular risk when sleep disordered breathing is present.

The findings of this study may a priori support the contention that cardiovascular disease begins in children, particularly in the context of sleep disordered breathing. However, much additional work needs to be done before this conclusion can be reached. In their attempt to compare the recorded data in children with published data in adults with OSA, the authors only examined events exceeding 10 seconds’ duration (i.e., events meeting adult criteria). The exclusion of events consisting of 2 or more breaths’ duration, as is routinely used in pediatric scoring, makes it difficult to determine whether indeed all children with OSA develop cardiovascular alterations during respiratory events, or whether only children with more severe OSA with longer duration events will be those at risk. In addition, O’Driscoll and colleagues2 elected to analyze events in all chil-
Children, including those with primary snoring (obstructive apnea hypopnea index < 1 event/h of total sleep time), a group that includes a large spectrum of sleep disordered breathing, and may somewhat underscore which disease severity is critical. The increases in MAP in children with OSA as is seen with continuous blood pressure monitoring,9 and the recent findings of relative baroreflex insensitivity secondary to intermittent hypoxia during sleep in both animal models and children,10-12 would further suggest that children with more severe OSA undergo vascular remodeling induced by recurrent obstructive events, and that, therefore, dose-dependent differences in OSA severity should be apparent.

Notwithstanding, the robust increases in HR and MAP in children with obstructive respiratory events during sleep reveal that such changes can occur even in the presence of a priori a normal cardiovascular system. It is therefore possible that autonomic disturbances elicited by sleep disordered breathing during early childhood not only may impose immediate consequences, such as hypertension, but may also adversely impact the cardiovascular system in a sustained fashion into adulthood.

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REFERENCES


