EDITORIAL

Natural History and Management of Pediatric Obstructive Sleep Apnea—Emerging Concepts

Commentary on Spilsbury et al. Remission and incidence of obstructive sleep apnea from middle childhood to late adolescence. SLEEP 2015;38:23–29.

Hari Bandla, MD[1]; Lynn A. D'Andrea, MD[2]

[1]University of Chicago, Chicago, IL; [2]Medical College of Wisconsin, Milwaukee, WI

Pediatric obstructive sleep apnea (OSA) is a common medical disorder associated with neurocognitive and cardiovascular dysfunction. Two core concepts of pathophysiology that predispose patients to OSA include an anatomically narrow upper airway and/or alterations of neuromuscular tone. Obstructive sleep apnea occurs in all ages, ranging from premature infants to the elderly. Thus, it is anticipated that pediatric OSA is a disorder of continuum. However, a multitude of changes occur in children during the various growth periods and these changes include increase in upper airway size, lymphoid hyperplasia followed by regression, and alterations in body mass index with central adiposity distribution. Furthermore, pubertal alterations in hormones may lead to dynamic changes in the collapsibility of the upper airway. In summary, these changes are likely associated with alterations in the natural history of pediatric OSA during critical periods of growth and development.

In this issue of *SLEEP*, Spilsbury and colleagues report longitudinal changes in the incidence, remission, and prediction of OSA at two critical stages of growth and development in children: middle childhood and late adolescence.[1] The authors found that obstructive sleep apnea (OSA) identified in a community-based cohort of children rarely persisted from middle childhood through late adolescence. Rather 91% of middle childhood cases remitted, the majority of them spontaneously. Most adolescents with OSA were incident cases (i.e., new cases). This extends findings reported by the Childhood Adenotonsillectomy Trial (CHAT) which estimated that approximately one-half of 5- to 9-year-old children with OSA randomized to watchful waiting had remission of OSA based on polysomnography (PSG) findings after 7 months.[2]

Risk factors for OSA in middle childhood included prematurity, being African American, and lower socioeconomic standards. Snoring was also a risk factor, but children who snored during childhood were not the same children with snoring and OSA during adolescence. Risk factors for OSA in adolescence included male gender, prior history of adenoidectomy or tonsillectomy, and obesity. The authors speculate that history of a prior adenoidectomy or tonsillectomy is a possible marker for abnormal upper airway anatomy. They also note that adolescents with obesity and OSA were also obese as children, although at that time they did not have OSA.

According to the Centers for Disease Control, obesity has more than doubled in children and quadrupled in adolescents in the past 30 years.[3] In the United States, the percentage of children aged 6–11 years of age who were obese increased from 7% in 1980 to nearly 18% in 2012. The percentage of adolescents aged 12–19 years of age who were obese increased from 5% to nearly 21% over the same time period. In 2012, more than one-third of children and adolescents were overweight or obese. Childhood obesity is associated with short-term or immediate effects, but perhaps more importantly it is associated with long-term adverse health effects. As noted by Gottlieb in a commentary in this journal 10 years ago, as the epidemic of obesity extends to every younger ages, the obesity-related sleep-disordered breathing (SDB) typical in adults will become increasingly prevalent in childhood.[4] A recent study estimated that the prevalence of OSA among obese children and adolescents was as high as 60%.[5] OSA may be more severe in obese children and adolescents compared to age-matched, non-obese children and adolescents.[6]

There are several mechanisms that interact to increase the risk of OSA among obese children and adolescents. First, similar to non-obese children, obese children and adolescents can have upper airway obstruction secondary to adenotonsillar hypertrophy. Unfortunately, studies have shown persistence of OSA in up to one-half of the obese children with OSA undergoing adenotonsillectomy.[7] This is significantly higher than the rates in non-obese children with OSA who underwent surgery.[8,9] Another study demonstrated rapid body mass index gain is an independent risk factor for recurrence of OSA one year after adenotonsillectomy.[10] Additional anatomic risk factors can include long soft palate, enlarged fat pads, enlarged tongue, and increased neck circumference. Moreover, functional factors such as altered neuromuscular tone in the upper airway can be associated with OSA in obese children and adolescents secondary to greater upper airway collapsibility. Measurements of airway mechanics have shown that in obese children with OSA, there is a positive critical closing pressure of the pharynx and less genioglossus muscle activity, causing the airway to collapse during sleep even with mild negative inspiratory pressure.[11] Finally, mechanical factors that are associated with SDB in obese children and adolescents include central obesity with an excess mechanical load on the chest, leading to decreased chest wall compliance with decreased lung volumes and increased risk of hypoventilation or gas-exchange abnormalities.

Taken together, these findings suggest that screening for pediatric OSA should be tailored to specific age ranges. In

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Address correspondence to: Hari Bandla, MD, FAASM, University of Chicago, Division of Sleep Medicine, 5841 S Maryland Avenue, C 104E, MC 2117, Chicago, IL 60637; Tel: (773) 702-5216; Email: hbandla@uchicago.edu

particular, overweight and obese adolescents should be considered at increased risk for OSA. We agree with Spilsbury and colleagues[1] that their findings underscore the need to prevent obesity in childhood, which may reduce the likelihood of OSA in adolescents. Also children who have had adenotonsillectomy are at increased risk for adolescent OSA because they likely have additional risk factors for OSA.

The major limitation of all studies in this area is the lack of standardization of what constitutes a clinically significant OSA index. In the report by Spilsbury, an obstructive apnea-hypopnea index (OAHI) > 5 was used to diagnosis children and adolescents with OSA. Similar to other studies, the incidence of OSA increased if the OAHI cutoff was lowered to > 1.[12] Polysomnography is still considered the "gold standard" for the diagnosis of OSA, but lack of standardization of the diagnostic criteria puts one at risk to miss children with clinically significant disease or to treat children when it was perhaps not clearly indicated.

In summary, because OSA in middle childhood usually remitted by adolescence, and most adolescent cases were therefore incident cases, criteria other than concern alone over OSA persistence or incidence should be used when making treatment decisions for pediatric OSA. Given the possibility of remission of OSA in a significant proportion of children by late childhood, treatment of all children diagnosed with OSA, in the absence of current symptomatology might not be necessary. The observations from the study by Spilsbury[1] in conjunction with the CHAT study[2] has given momentum to the concept of conservative management of pediatric OSA in a select group children with continuous monitoring, thus avoiding a surgical procedure and related complications. The greater importance of middle childhood obesity compared to snoring in predicting adolescent OSA provides support for screening, preventing, and treating obesity in childhood. Additional longitudinal studies in more diverse populations might further clarify these concepts.

DISCLOSURE STATEMENT

The authors have indicated no financial conflicts of interest.

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