Sleep-Disordered Breathing, Orofacial Growth, and Prevention of Obstructive Sleep Apnea

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KEYWORDS

• Upper airway • Abnormal resistance • Sleep states • Orofacial growth • Prevention

KEY POINTS

- Orofacial growth is involved in factors leading to obstructive sleep apnea; untreated abnormal growth leads to upper airway resistance.
- Upper airway resistance due to abnormal orofacial growth leads to increased collapse of upper airway during sleep.
- Recognition and treatment of factors involved in abnormal orofacial growth prevent sleep-disordered breathing.

INTRODUCTION

Often, patients are not diagnosed with sleep-disordered breathing (SDB) or obstructive sleep apnea (OSA) until approximately 40 years of age. This is unfortunate because a diagnosis of SDB at this age is accompanied by various comorbidities, including excessive daytime sleepiness, increased risk of traffic, and industrial accidents and cardiovascular complications. Each year, new epidemiologic studies demonstrate associations between sleep apnea and metabolic dysfunction; psychiatric, neurologic, and ophthalmologic syndromes; and pregnancy complications. Part of the increase in the prevalence of sleep apnea can be attributed to the rising occurrence of obesity in many countries. There is a long known association between the prevalence of OSA and obesity. This phenomenon has been seen in both adults and children.[1]

The presence of OSA in children was shown, however, in 1976,[2] and the observation that OSA is familial and can be noted in both children and adults in the same families was well demonstrated in different parts of the world in the 1990s.[3,4,5] The dissociation of adult and pediatric OSA is thus completely artificial; to the contrary, the underpinnings and development of SDB follow a continuum across age. Acknowledging this longitudinal aspect has led to studies to identify what is behind SDB and what the factors are that lead to abnormal breathing during sleep.

It can be argued that the adult overweight OSA patient is at the end of the line. By this time, clinically relevant comorbidities are present, and detection of OSA at this point is already too late. The absence of education on early identification is responsible for this sad state of medical care. Elements leading to SDB should be detected much earlier; treatment should be attempted at the

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