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COMMENTARY

A century of adenotonsillectomy's failure to fully resolve sleep-disordered breathing: mild malocclusions are maybe not so mild?

Commentary on Cohen-Levy J, Quintal MC, Rompré P, Almeida F, Huynh N. Prevalence of malocclusions and oral dysfunctions in children with persistent sleep-disordered breathing after adenotonsillectomy in the long term. *J Clin Sleep Med*. 2020;16(8): 1357–1368. doi:10.5664/jcsm.8534

Kevin L. Boyd, DDS, MSc

Department of Dentistry and Sleep Medicine, Lurie Children's Hospital, Chicago, Illinois; Private Practice, Chicago, Illinois; The University of Pennsylvania, Museum of Archaeology and Anthropology, Philadelphia, Pennsylvania

The authors of the study by Cohen-Levy et al[1] in this issue of the *Journal of Clinical Sleep Medicine* are to be commended for their well-organized and strongly referenced reporting on the highly prevalent problem of persistent sleep-disordered breathing (SDB) in children following adenotonsillectomy or adenoidectomy solely. Within recently published literature, postsurgical SDB symptom recurrence is described as both a short-term[2,3] and long term[4] problem; and according to the late Christian Guilleminault[5], "it appears that we cannot assume that T&A alone can be relied upon to sufficiently restore normal breathing during sleep. Nasal breathing during wake and sleep is the demonstration of normal respiratory functioning in a child, and persistence of mouth breathing is an indicator for the need for further treatment of sleep-disordered breathing"; there is really nothing new here. Published reports dating from the early 20th century also describe the frequency of recurrent SDB as a common postadenotonsillectomy morbidity.[6–8] Unhealthy body weight, also described as a frequent SDB comorbidity phenotype in the present study, is often additionally associated with other systemic comorbidities (eg, cardiovascular disease, type 2 diabetes, hypertension, etc.) over all age ranges; screening for overweight/obesity in growing children, as determined by body mass index z-score (body mass index percentile) screening assessment, is an especially important inclusion criteria component of the Cohen-Levy study protocol per its known persistence into adolescence and adulthood. Another set of morphological SDB risk indicator traits discussed in the paper with SDB comorbidity and known persistence capability beyond childhood are specific maldevelopments of the interconnected craniofacial and respiratory complexes. Somewhat casually referred to in the present paper as mild malocclusions (eg, constricted transverse jaw dimension [width], deep and narrow palatal vault, retrusive chin and/or midface, steep mandibular plane/vertical direction of craniofacial growth) in nonsyndromic children; these so-called mild malocclusions are also reported elsewhere[9,10] as being frequently associated with SDB comorbidity in childhood and beyond in the absence of appropriately timed and applied orthodontic/dentofacial orthopedic intervention.

Within the discussion section of their paper. the authors describe specific malocclusion traits as sometimes precursors of SDB in preschool age, citing the work of Marino et al,[11] suggesting that various craniofacial abnormalities in preschool children are predisposing factors for SDB, including decreased mandibular and maxillary lengths, skeletal retrusion, and increased lower facial height. However, in the Cohen-Levy study they "...could not establish any direct relationship between abnormal respiratory events during sleep and specific malocclusions, such as cross-bite, deep bite or class II occlusion, whose prevalence in this sample was not significantly different from that in the general population"; this finding was described as "surprising" in light of strong associations depicted in the literature citing the PANIC study,[12] which reported on a fairly large cohort (491) of Finnish children (6–8 years of age) with cross-bite who had higher risk of SDB than those without cross-bite; children with a convex facial profile were deemed at higher risk for SDB than those with a normal facial profile. Furthermore, the Cohen-Levy investigation cites a 2013 meta-analysis[13] as being "in accordance with" their own finding of a noncausal relationship between craniofacial structure and pediatric SDB. I think this could be misleading to readers of this because of the possible implication that any purported relationship between malocclusion phenotypes and SDB behavioral phenotypes must be causal and unidirectional or bidirectional to be clinically relevant. This, I think, may be short-sighted as it ignores the previously cited observations that, quite often, certain malocclusion traits may not be directly causal of SDB, but often at least coexisting/comorbid with SDB behavioral traits. Furthermore, the so-called mild malocclusion phenotypes described and referenced in the Cohen-Levy study can also sometimes be mutually resolved with appropriately timed (ie, early childhood) and applied interventions and with adjunctive approaches such as orofacial myofunctional therapy and surgical revision of restrictive oral tethers (ie, frenectomies),

which the authors also referenced in the present *Journal of Clinical Sleep Medicine* paper.

In summary, I think the paper of Cohen-Levy in this issue of the *Journal of Clinical Sleep Medicine* does not really teach anything new as is evidence by myriad published reports cited here dating from the beginning of the past century; however, this paper will serve as an excellent and well-referenced guide for medical and dental researchers and clinicians who are seeking a better understanding about how suboptimal development of the hard and soft tissues of the intimately connected craniofacial and respiratory complexes might help explain incomplete resolution of SDB, or recurrence of SDB symptoms, after adenotonsillectomy or adenoidectomy solely.

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Address correspondence to: Kevin L. Boyd, DDS, MSc; Dentistry for Children, 1721 N Halsted St, Chicago, IL, 60614; Tel: (312) 988-9855; Email: kbo569@googlemail.com

DISCLOSURE STATEMENT

The author reports no conflicts of interest.