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Pediatric sleep-disordered breathing: Role of the dentist

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Objective: In this review article, the role of the dentist in the evaluation and treatment of snoring and Obstructive Sleep Apnea Syndrome (OSAS) in children is described. Snoring and OSAS in children is receiving increased awareness, with reported rates approximating 10% of children who regularly snore, and up to 4% who suffer from OSAS. OSAS in children may have serious developmental and behavioral consequences. Apnea Hypopnea Index (AHI) is the main outcome of the polysomnography test, but its diagnostic values differ from children to adults, as do treatment approaches. **Data Sources and Study Selection:** A comprehensive literature search of

publications from 1973 to 2017 in the PubMed Direct databases was performed to collect information about snoring and OSAS in children. The search was limited to peer-reviewed articles written in English with a few exceptions in other languages. **Conclusion:** Dentists play a significant role in early detection of OSAS, helping in reducing and preventing its serious consequences. A multidisciplinary treatment team, which manages and treats OSAS, should include the dentist in addition to the sleep specialist and the ENT physician. (*Quintessence Int* 201#;##:1–7; doi: ##.###/j.qi.a#####)

Key words: adenoidectomy, obstructive sleep apnea, pediatric, sleep-disordered breathing, snoring, tonsillectomy

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Pediatric sleep-related disorders are common and may occur in up to 30% of children.^{1,2} Inadequate or disturbed sleep can have a negative impact on the physical as well as on the mental health of the child.^{3–8} Sleep-related breathing disorders (SRBD) include a wide spectrum of disorders with a severity ranging from primary snoring to obstructive sleep apnea syndrome (OSAS). Snoring and OSAS in children is receiving increased awareness, with reported rates approximating 10% of children who regularly snore,^{9,10} and up to 4% who suffer from OSAS.¹¹ There is debate whether primary snoring predicts future OSAS^{12,13} or not.^{14–16}

OSAS is characterized by frequent repetitive events of full (apnea) or partial (hypopnea) collapse and block-

age of the upper airway for at least 10 seconds during sleep, leading to a drop in oxygen saturation followed by a micro arousal.^{17,18} General underlying mechanisms involve sleep-induced hypotonus that causes a drop in intrapharyngeal pressure, and narrowing of the air column during inspiration.¹⁹ Adenotonsillar hypertrophy is the most widely recognized contributor to the pathophysiology of pediatric OSA. A combination of anatomical and craniofacial factors (eg, retrognathia and micrognathia, macroglossia or retro-positioning of the tongue), lymphoid tissue, upper airway inflammation, neuromuscular factors, and obesity are interactively involved.^{20,21}

Consequently, a ventilator effort against an obstructed (partial or complete) airway is associated with recurrent oxyhemoglobin desaturation and arousals from sleep.²² This, as mentioned above, results in recurring episodes of obstruction. The extent of the OSAS is measured by polysomnography test (PSG). PSG is a laboratory test performed during sleep; its main outcome is the Apnea Hypopnea Index (AHI), which represents the sleep apnea severity and reflects the average number of apnea and hypopnea events per hour of sleep. The severity of OSAS in adults is categorized by the American Sleep Disorder Association,²³ according to the patient's AHI as following:

- mild OSAS, 5 to 15 AHI
- moderate OSAS, 15 to 30 AHI
- severe OSAS, more than 30 AHI.

However, for children, the thresholds are lower:

- mild OSAS, 1 to 5 AHI
- moderate OSAS, 5 to 10 AHI
- severe OSAS, more than 10 AHI.

PSGs have several drawbacks, especially price, time, and long waiting lists, and it is not universally available. Nocturnal pulse oximetry is a valuable, immediate, low-priced screening option for SRBD in children. Dentists may use nocturnal pulse oximetry for this purpose.^{24,25} However; pulse oximetry is inferior to PSG and is not sufficient for a final diagnosis of OSAS.²⁶ Additionally, video observations and documentations are very useful

tools for recording children during sleep and observing sleep apnea.²⁷ Another tool that may help in identifying of SRBD in adults, and in children as well, are built-in scaled questionnaires, such as the Epworth Sleepiness Scale (ESS) in adults,²⁸ or pediatric sleep questionnaires, which have been found to be useful and beneficial in identifying SRBD in children or in monitoring improvement after treatment.^{29,30}

In adults, OSAS has many consequences, such as cardiovascular morbidity,³¹ stroke,³² and daily tiredness. Unfortunately, OSAS studies in children are rather limited, and the etiology, clinical presentation, and complications, as well as the mechanism and management of this disorder, are less well established.

CLINICAL PRESENTATION OF OSAS AND ITS CONSEQUENCES IN CHILDREN

Most studies deal with toddlers, preschool-, and school-aged children, and relate to the major cause of sleep apnea in children as mainly derived from the adenotonsillar size.³³ The nasopharyngeal airway is the narrowest at 4.5 years of age, mostly due to adenoidal structures affecting upper airway patency. The adenoids reach their greatest size at 7 to 12 years.³⁴ In addition, age 12 is considered to be an age at which 90% of face development is completed.³⁵ Therefore, while no age limits were determined for childhood in the definition of SRBD, 12 years of age is referred to as an acceptable childhood SRBD cut off point, determined due to tonsils and adenoids size, which have a major role in children's SRBD.^{33,34}

The presentation of SRBD in children differs from adults. Among children, even a significant SRBD may manifest only by brief obstructing events, without a clinically significant drop in blood oxygen levels.^{36,37} Children may also compensate by having frequent brief awakenings to reestablish the airway, resulting in fragmented sleep.³⁸

Despite the above findings, children's breathing disorders during sleep are related to the following short- and long-term physical and mental impairments:

- Nocturnal disorders: snoring, non-relaxed sleep, difficulty in breathing through the nose, hissing, and bedwetting.³⁹
- Neuropsychologic disorders: difficulty concentrating, attention deficit disorder, hyperactivity that may be associated with apathy, difficulties at school, daily fatigue, and sometimes shyness.^{39,40} Wong et al⁴¹ found increased risk of addiction to alcohol and drugs in adults who were diagnosed in childhood with sleep disorders.
- Metabolic and cardiovascular disorders: pulmonary hypertension,⁴² insulin resistance, and increase in inflammatory mediators has been reported in child populations.⁴³⁻⁴⁸ However, these disorders may be connected to frequently accompanied obesity.⁴⁹
- Growth and facial development variations: nearly 50% of growth hormone secretion occurs during the third and fourth non-REM sleep stages. Growth hormone decrease due to disturbed sleep⁵⁰ may lead to failure to thrive.⁵¹ Additionally, over the years, children may possibly develop abnormal facial characteristics, such as elongated face, small chin, overcrowded teeth, high and narrow palatal arch, and crossbite (Fig 1).⁵²⁻⁵⁴

The main question is whether these facial features have appeared because of the syndrome, especially in relation to mouth breathing, or are associated with the syndrome but not as cause and effect connection. A landmark study using primates demonstrates a relationship between dentofacial growth modification and the establishment of mouth breathing.⁵⁵ Also, much newer studies support this concept of cause and effect.⁵⁴⁻⁵⁹ However, according to other studies the association between mouth breathing and chronologic change of facial features was not determined.⁶⁰⁻⁶²

POSSIBLE CAUSES OF OSAS IN CHILDREN

Enlarged tonsils and adenoids^{63,64} and chronic nasal obstruction⁶⁵ are the most frequent reasons for OSAS in children. Other causes include congenital syndromes

with specific skeletal characteristics such as Pierre Robin syndrome⁶⁶ or Down's syndrome.⁶⁷ Cleft palate⁶⁸ and obesity⁶⁹ can also cause OSAS in children.

TREATMENT MODALITIES OF OSAS IN ADULTS

In adults the most common and most efficacious line of treatment modality is the CPAP (continuous positive airway pressure) device, which consists of a mask connected to an air pump that keeps the upper airway open during sleep.⁷⁰ The second line treatment is a removable mandibular repositioning dental appliance aimed to protrude the mandible along with the base of the tongue during sleep, thus increasing the pharyngeal space.^{71,72} These interventions are not usually suitable for use in children, with a few exceptions discussed below. When surgery is indicated for adult snoring and OSAS in order to widen the upper airway, uvulopalatopharyngoplasty (UPPP) is utilized (designed to remove soft tissues such as tonsils, part of the uvula, and part of the soft palate),⁷³ with maxillomandibular expansion,⁷⁴ and maxillomandibular advancement surgeries.⁷⁵ These are highly invasive, less common, and mostly recommended only for adults.

Behavioral lifestyle-related modifications, such as weight loss,^{76,77} are very important in adults and may be beneficial in children.

TREATMENT MODALITIES OF OSAS IN CHILDREN

In addition to behavioral lifestyle-related modifications, tonsillectomy and adenoidectomy are considered treatments of choice in children. These were shown to improve both snoring and AHI scores, and improve quality of life and attention deficit disorders.⁷⁸⁻⁸⁰ Correction of chronic nasal airway obstruction is also a useful mode of therapy in children.^{69,81,82} Accepted treatments for adults such as CPAP devices may cause facial deformities such as midface retrusion, and are therefore not recommended in children, particularly before the growing processes ends.⁸³⁻⁸⁵ Mandibular repositioning



Figs 1a to 1e Elongated face, concave profile, and tooth crowding in a child with Pierre Robin syndrome and OSAS.



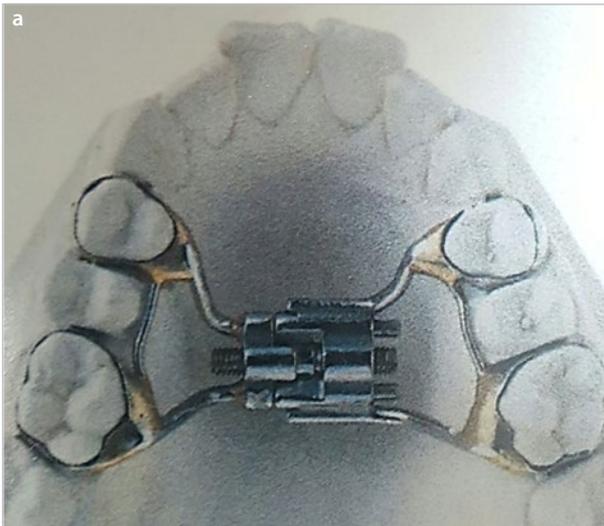
devices⁸⁶ are rarely used in children. However, in children with skeletal class II due to a retruded mandible, similar devices are used to enhance osteogenic growth of the mandible in order to achieve a more permanent protrusive location.⁸⁷ This treatment is proven to be an effective method to reduce the AHI,⁸⁵ but it should be kept in mind that skeletal and orthodontic side effects can be irreversible. In addition, less common nonsurgical procedures include rapid expansion of the maxilla, performed with the aid of a rapid palatal expander or quad helix (Fig 2). This procedure may enhance proper development of the jaws and bite and is effective in the reduction of upper airway resistance to airflow, which subsequently may reduce AHI.^{88,89}

THE ROLE OF THE DENTIST

The American Academy of Pediatrics recommends that physicians routinely ask parents about snoring to

screen for OSAS and milder forms of SRBD.⁹⁰ Early recognition and prevention of OSAS in children is important in order to prevent, or at least minimize, further complications such as neurocognitive, psychologic, and facial permanent deformities in adult life.⁹¹⁻⁹³ The role of the dentist in the area of SRBD is essential in diagnosing and referring patients for further evaluation in a sleep laboratory and by an ear, nose, and throat (ENT) physician. During routine dental examination, the dentist should pay attention to the following signs and symptoms: poor ability to concentrate, poor school performance, failure to thrive, mouth-breathing, nasal speech, recurrent airway infections. The use of scaled pediatric sleep questionnaires aimed especially to the parents may be recommended.^{29,30} In addition, the dentist should pay attention to various craniofacial and oral abnormalities such as elongated narrow face, small chin, tooth crowding, high-arched palate, adenotonsillar hypertrophy, and obesity.^{21,34}

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Figs 2a and 2b Rapid maxillary expansion devices (courtesy technician Yaniv Ben Atar, institution, city).

Special consideration should be given to children with congenital syndromes accompanied by cranio-mandibular abnormalities. Additionally, the dentist is a treatment provider when dental devices are used. An orthodontist should also be involved when rapid maxillary expansion is considered.

CONCLUSION

Dentists play a significant role in the early detection of OSAS, helping to reduce and prevent its serious consequences (Fig 3). A multidisciplinary treatment team that manages and treats OSAS should include the dentist in addition to the sleep specialist and the ENT physician.

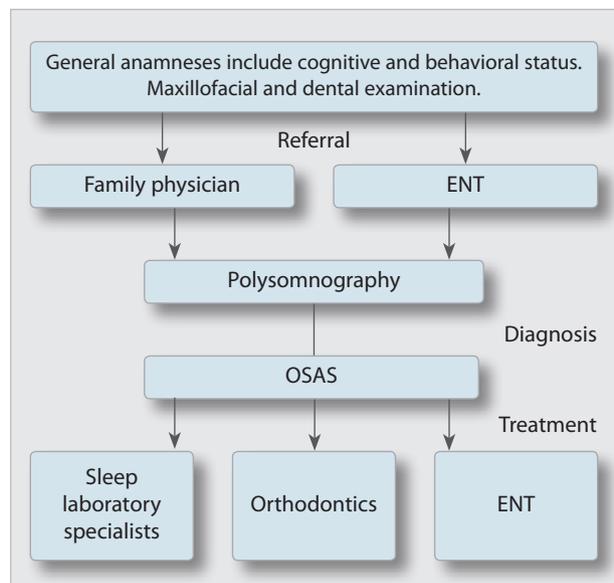
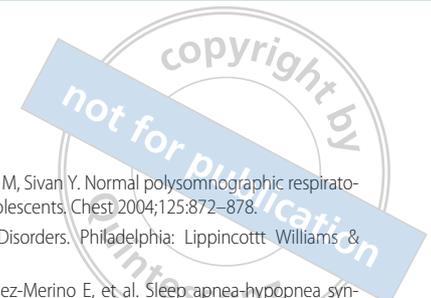


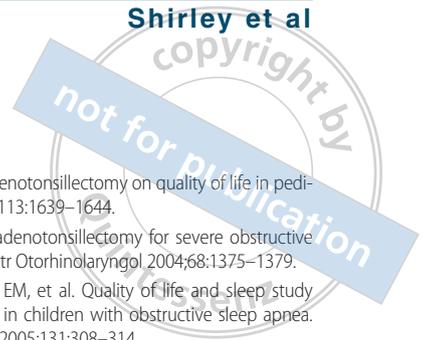
Fig 3 The role of the dentist in referral, diagnosis, and treatment.

REFERENCES

- Anders TF, Halpern LF, Hua J. Sleeping through the night: a developmental perspective. *Pediatrics* 1992;90:554–560.
- Beltramini AU, Hertzog ME. Sleep and bedtime behavior in preschool-aged children. *Pediatrics* 1983;71:153–158.
- Beebe DW. Neurobehavioral morbidity associated with disordered breathing during sleep in children: a comprehensive review. *Sleep* 2006;29:1115–1134.
- Chervin RD, Archbold KH. Hyperactivity and polysomnographic findings in children evaluated for sleep-disordered breathing. *Sleep* 2001;24:313–320.
- O'Brien LM, Mervis CB, Holbrook CR, et al. Neurobehavioral correlates of sleep-disordered breathing in children. *J Sleep Res* 2004;13:165–172.
- Stein MA, Mendelsohn J, Obermeyer WH, et al. Sleep and behavior problems in school-aged children. *Pediatrics* 2001;107:E60.
- Verhulst SL, Rومان R, Van Gaal L, et al. Is sleep-disordered breathing an additional risk factor for the metabolic syndrome in obese children and adolescents? *Int J Obes Lond* 2009;33:8–13.
- Vlahandonis A, Nixon GM, Davey MJ, Walter LM, Horne RS. A four year follow-up of sleep and respiratory measures in elementary school-aged children with sleep disordered breathing. *Sleep Med* 2013;14:440–448.
- Ali NJ, Pitson DJ, Stradling JR. Snoring, sleep disturbance, and behaviour in 4–5 year olds. *Arch Dis Child* 1993;68:360–366.
- Teculescu DB, Caillier I, Perrin P, et al. Snoring in French preschool children. *Pediatr Pulmonol* 1992;13:239–244.
- Lumeng JC, Chervin RD. Epidemiology of pediatric obstructive sleep apnea. *Proc Am Thorac Soc* 2008;5:242–252.



12. Nieminen P, Tolonen U, Löppönen H. Snoring and obstructive sleep apnea in children: a 6-month follow-up study. *Arch Otolaryngol Head Neck Surg* 2000;126:481–486.
13. Topol HI, Brooks LJ. Follow-up of primary snoring in children. *J Pediatr* 2001;138:291–293.
14. Bonuck KA, Chervin RD, Cole TJ, et al. Prevalence and persistence of sleep disordered breathing symptoms in young children: a 6-year population-based cohort study. *Sleep* 2011;34:875–884.
15. Li AM, Au CT, Ng SK, et al. Natural history and predictors for progression of mild childhood obstructive sleep apnoea. *Thorax* 2010;65:27–31.
16. Li AM, Zhu Y, Au CT, et al. Natural history of primary snoring in school-aged children: a 4-year follow-up study. *Chest* 2013;143:729–735.
17. Azagra-Calero E, Espinar-Escalona E, Barrera-Mora JM, et al. Obstructive sleep apnea syndrome (OSAS). Review of the literature. *Med Oral Patol Oral Cir Bucal* 2012;17:925–929.
18. Huang L, Quinn SJ, Ellis PD, Williams JE. Biomechanics of snoring. *Endeavour* 1995;19:96–100.
19. Quinn SJ, Daly N, Ellis PD. Observation of the mechanism of snoring using sleep nasendoscopy. *Clin Otolaryngol Allied Sci* 1995;20:360–364.
20. Arens R, Muzumdar H. Childhood obesity and obstructive sleep apnea syndrome. *J Appl Physiol* 2010;108:436–444.
21. Kheirandish-Gozal L, Gozal D (eds). *Sleep disordered breathing in children*, 1 edn. New York: Springer Science, 2012.
22. Horner RL. Pathophysiology of obstructive sleep apnea. *J Cardiopulm Rehabil Prev* 2008;28:289–298.
23. No authors listed. Sleep-related breathing disorders in adults: recommendations for syndrome definition and measurement techniques in clinical research. The Report of an American Academy of Sleep Medicine Task Force. *Sleep* 1999;22:667–689.
24. Brouillette RT, Morielli A, Leimanis A, et al. Nocturnal pulse oximetry as an abbreviated testing modality for pediatric obstructive sleep apnea. *Pediatrics* 2000;105:405–412.
25. Redline S, Tishler PV, Schluchter M, et al. Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. *Am J Respir Crit Care Med* 1999;159:1527–1532.
26. Kirk VG, Bohn SG, Flemons WW, Remmers JE. Comparison of home oximetry monitoring with laboratory polysomnography in children. *Chest* 2003;124:1702–1708.
27. Sivan YA, Schonfeld KT. Screening obstructive sleep apnoea syndrome by home videotape recording in children. *Eur Respir J* 1996;9:2127–2131.
28. Johns MW. A new method for measuring daytime sleepiness: the Epworth sleepiness scale. *Sleep* 1991;14:540–550.
29. Chervin RD, Hedger K, Dillon JE, Pituch KJ. Pediatric sleep questionnaire (PSQ): validity and reliability of scales for sleep-disordered breathing, snoring, sleepiness, and behavioral problems. *Sleep Med* 2000;1:21–32.
30. Chervin RD, Weatherly RA, Garetz SL, et al. Pediatric sleep questionnaire: prediction of sleep apnea and outcomes. *Arch Otolaryngol Head Neck Surg* 2007;133:216–222.
31. Marin JM, Carrizo SJ, Vicente E, Agusti AG. Long-term cardiovascular outcomes in men with obstructive sleep apnoea-hypopnoea with or without treatment with continuous positive airway pressure: an observational study. *Lancet* 2005;365(9464):1046–1053.
32. Gibson, GJ. Sleep disordered breathing and the outcome of stroke. *Thorax*, 2004;59:361–363.
33. Tagaya M, Nakata S, Yasuma F, et al. Relationship between adenoid size and severity of obstructive sleep apnea in preschool children. *Int J Pediatr Otorhinolaryngol* 2012;76:1827–1830.
34. Arens R, Marcus CL. Pathophysiology of upper airway obstruction: a developmental perspective. *Sleep* 2004;27:997–1019.
35. Proffit W. *Contemporary orthodontics*, 4th edn. St Louis: Mosby-Elsevier, 2006.
36. Marcus CL, Omlin KJ, Basinski DJ, et al. Normal polysomnographic values for children and adolescents. *Am Rev Respir Dis* 1992;146:1235–1239.
37. Uliel S, Tauman R, Greenfeld M, Sivan Y. Normal polysomnographic respiratory values in children and adolescents. *Chest* 2004;125:872–878.
38. Carney PR. *Clinical Sleep Disorders*. Philadelphia: Lippincott Williams & Wilkins, 2005.
39. Lombart M, Chiner E, Gomez-Merino E, et al. Sleep apnea-hypopnea syndrome in a pediatric population: differences between children with tonsillar hypertrophy and those with concomitant disease. *Arch Bronconeumol* 2007;43:655–661.
40. Beebe DW, Ris MD, Kramer ME, et al. The association between sleep disordered breathing, academic grades, and cognitive and behavioral functioning among overweight subjects during middle to late childhood. *Sleep* 2010;33:1447–1456.
41. Wong MM, Brower KJ, Fitzgerald HE, Zucker RA. Sleep problems in early childhood and early onset of alcohol and other drug use in adolescence. *Alcohol Clin Exp Res* 2004;28:578–587.
42. Chan J, Edman JC, Koltai PJ. Obstructive sleep apnea in children. *Am Fam Physician* 2004;69:1147–1154.
43. Bhattacharjee R, Kim J, Alotaibi WH, et al. Endothelial dysfunction in children without hypertension: potential contributions of obesity and obstructive sleep apnea. *Chest* 2012;141:682–691.
44. Canapari, CA, Hoppin, AG, Kinane TB, et al. Relationship between sleep apnea, fat distribution, and insulin resistance in obese children. *J Clin Sleep Med* 2011;7:268–273.
45. De la Eva RC, Baur LA, Donaghue KC, Waters KA. Metabolic correlates with obstructive sleep apnea in obese subjects. *J Pediatr* 2002;140:654–659.
46. Ingram DG, Singh AV, Ehsan Z, Birnbaum BF. Obstructive sleep apnea and pulmonary hypertension in children [epub ahead of print 11 Jan 2017]. *Paediatr Respir Rev* doi: 10.1016/j.prrv.2017.01.001.
47. Lee, LA, Li HY, Lin YS, et al. Severity of childhood obstructive sleep apnea and hypertension improved after adenotonsillectomy. *Otolaryngol Head Neck Surg* 2015;152:553–560.
48. Tsaousoglou M, Bixler EO, Calhoun S, et al. Sleep-disordered breathing in obese children is associated with prevalent excessive daytime sleepiness, inflammation, and metabolic abnormalities. *J Clin Endocrinol Metab* 2010;95:143–150.
49. Tauman R, O'Brien LM, Ivanenko A, Gozal D. Obesity rather than severity of sleep-disordered breathing as the major determinant of insulin resistance and altered lipidemia in snoring children. *Pediatrics* 2005;116:66–73.
50. Mehta A, Hindmarsh PC. The use of somatotropin (recombinant growth hormone) in children of short stature. *Paediatr Drugs* 2002;4:37–47.
51. Nieminen P, Lopponen T, Tolonen U, et al. Growth and biochemical markers of growth in children with snoring and obstructive sleep apnea. *Pediatrics* 2002;109:e55.
52. Marcus CL, Lopponen T, Tolonen U, et al. Diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2012;130:576–584.
53. Nunes WR, Di Francesco RC. Variation of patterns of malocclusion by site of pharyngeal obstruction in children. *Arch Otolaryngol Head Neck Surg* 2010;136:1116–1120.
54. Pirila-Parkkinen K, Pirttiniemi P, Nieminen P, Tolonen U, Peltari U, Löppönen H. Dental arch morphology in children with sleep-disordered breathing. *Eur J Orthod* 2009;31:160–167.
55. Harvold EP, Vargervik K, Chierici G. Primate experiments on oral sensation and dental malocclusions. *Am J Orthod* 1973;63:494–508.
56. Cuccia AM, Lotti M, Caradonna D. Oral breathing and head posture. *Angle Orthod* 2008;78:77–82.
57. Flores-Mir C, Korayem M, Heo G, et al. Craniofacial morphological characteristics in children with obstructive sleep apnea syndrome: a systematic review and meta-analysis. *J Am Dent Assoc* 2013;144:269–277.
58. Peltomaki T. The effect of mode of breathing on craniofacial growth: revisited. *Eur J Orthod* 2007;29:426–429.
59. Solow B, Sandham A. Cranio-cervical posture: a factor in the development and function of the dentofacial structures. *Eur J Orthod* 2002;24:447–456.



60. Acar M, Turkcan I, Ozdas T, et al. Obstructive sleep apnoea syndrome does not negatively affect oral and dental health. *J Laryngol Otol* 2015;129:68–72.

61. Katyal V, Pamula Y, Martin AJ, Daynes CN, Kennedy JD, Sampson WJ. Craniofacial and upper airway morphology in pediatric sleep-disordered breathing: Systematic review and meta-analysis. *Am J Orthod Dentofacial Orthop* 2013;143:20–30.

62. Kim KB. How has our interest in the airway changed over 100 years? *Am J Orthod Dentofacial Orthop* 2015;148:740–747.

63. Muzumdar H, Arens R. Diagnostic issues in pediatric obstructive sleep apnea. *Proc Am Thorac Soc* 2008;5:263–273.

64. Tsubomatsu C, Shintani T, Abe A, et al. Diagnosis and treatment of obstructive sleep apnea syndrome in children. *Adv Otorhinolaryngol* 2016;77:105–111.

65. Nixon, GM, Brouillette RT. Obstructive sleep apnea in children: do intranasal corticosteroids help? *Am J Respir Med* 2002;1:159–166.

66. van Lieshout MJ, Joosten KF, Hoeve HL, et al. Unravelling Robin sequence: considerations of diagnosis and treatment. *Laryngoscope* 2014;124:E203–E209.

67. Marcus CL, Keens TG, Bautista DB, et al. Obstructive sleep apnea in children with Down syndrome. *Pediatrics* 1991;88:132–139.

68. Muntz H, Wilson M, Park A, et al. Sleep disordered breathing and obstructive sleep apnea in the cleft population. *Laryngoscope* 2008;118:348–353.

69. Urschitz MS, et al. Risk factors and natural history of habitual snoring. *Chest* 2004;126:790–800.

70. Sullivan CE, Issa FG, Berthon-Jones M, Eves L. Reversal of obstructive sleep apnoea by continuous positive airway pressure applied through the nares. *Lancet* 1981;1(8225):862–865.

71. Haviv Y, Bachar G, Aframian DJ, Almozni G, Michaeli E, Benoliel R. A 2-year mean follow-up of oral appliance therapy for severe obstructive sleep apnea: a cohort study. *Oral Dis* 2015;21:386–392.

72. Haviv Y, Benoliel R, Bachar G, Michaeli E. On the edge between medicine and dentistry: review of the dentist's role in the diagnosis and treatment of snoring and sleep apnea. *Quintessence Int* 2014;45:345–353.

73. Khan A, Ramar K, Maddirala S, Friedman O, Pallanch JF, Olson EJ. Uvulopalatopharyngoplasty in the management of obstructive sleep apnea: the mayo clinic experience. *Mayo Clin Proc* 2009;84:795–800.

74. Guilleminault C, Li KK. Maxillomandibular expansion for the treatment of sleep-disordered breathing: preliminary result. *Laryngoscope* 2004;114:893–896.

75. Holty JE, Guilleminault C. Maxillomandibular advancement for the treatment of obstructive sleep apnea: a systematic review and meta-analysis. *Sleep Med Rev* 2010;14:287–297.

76. Peppard PE, Young T, Palta M, Dempsey J, Skatrud J. Longitudinal study of moderate weight change and sleep-disordered breathing. *JAMA* 2000;284:3015–3021.

77. Strobel RJ, Rosen RC. Obesity and weight loss in obstructive sleep apnea: a critical review. *Sleep* 1996;19:104–115.

78. Flanary VA. Long-term effect of adenotonsillectomy on quality of life in pediatric patients. *Laryngoscope* 2003;113:1639–1644.

79. Mitchell RB, Kelly J. Outcome of adenotonsillectomy for severe obstructive sleep apnea in children. *Int J Pediatr Otorhinolaryngol* 2004;68:1375–1379.

80. Stewart MG, Glaze DG, Friedman EM, et al. Quality of life and sleep study findings after adenotonsillectomy in children with obstructive sleep apnea. *Arch Otolaryngol Head Neck Surg* 2005;131:308–314.

81. Mansfield LE, Diaz G, Posey CR, Flores-Neder J. Sleep disordered breathing and daytime quality of life in children with allergic rhinitis during treatment with intranasal budesonide. *Ann Allergy Asthma Immunol* 2004;92:240–244.

82. Brouillette RT, Lavergne J, Leimanis A, et al. Differences in pulse oximetry technology can affect detection of sleep-disordered breathing in children. *Anesth Analg* 2002;94(1 Suppl):S47–S53.

83. Fauroux B, Lavis JF, Nicot F, et al. Facial side effects during noninvasive positive pressure ventilation in children. *Intensive Care Med* 2005;31:965–969.

84. Roberts SD, Kapadia H, Greenlee G, Chen ML. Midfacial and dental changes associated with nasal positive airway pressure in children with obstructive sleep apnea and craniofacial conditions. *J Clin Sleep Med* 2016;12:469–475.

85. Villa MP, Pagani J, Ambrosio R, et al. Mid-face hypoplasia after long-term nasal ventilation. *Am J Respir Crit Care Med* 2002;166:1142–1143.

86. Ramar K, Dort LC, Katz SG, et al. Clinical practice guideline for the treatment of obstructive sleep apnea and snoring with oral appliance therapy: an update for 2015. *J Clin Sleep Med* 2015;11:773–827.

87. Ilizarov GA. The principles of the Ilizarov method. *Bull Hosp Jt Dis Orthop Inst* 1988;48:1–11.

88. Villa MP, Malagola C, Pagani J, et al. Rapid maxillary expansion in children with obstructive sleep apnea syndrome: 12-month follow-up. *Sleep Med* 2007;8:128–134.

89. Villa MP, Rizzoli A, Miano S, Malagola C. Efficacy of rapid maxillary expansion in children with obstructive sleep apnea syndrome: 36 months of follow-up. *Sleep Breath* 2011;15:179–184.

90. Section on Pediatric Pulmonology, Subcommittee on Obstructive Sleep Apnea Syndrome American Academy of Pediatrics. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics* 2002;109:704–712.

91. Konstantinopoulou S, Tapia IE. Neurocognitive and behavioural outcomes following intervention for obstructive sleep apnoea syndrome in children. *Paediatr Respir Rev* 2016;20:51–54.

92. Padmanabhan V, Kavitha PR, Hegde AM. Sleep disordered breathing in children: a review and the role of a pediatric dentist. *J Clin Pediatr Dent* 2010;35:15–21.

93. Tan HL, Alonso Alvarez ML, Tsaoussoglou M, et al. When and why to treat the child who snores? *Pediatr Pulmonol* 2017;52:399–412.