



EDITORIAL

Establishment of nasal breathing should be the ultimate goal to secure adequate craniofacial and airway development in children[☆]

O estabelecimento da respiração nasal deve ser o objetivo final para garantir desenvolvimento craniofacial e respiratório adequados em crianças

Carlos Torre^a, Christian Guilleminault^{b,*}

^a University of Miami, Division of Sleep and Otorhinolaryngology, Coral Gables, United States

^b Stanford University, Sleep Medicine Division, Stanford, United States

As shown for many years and studied again by Chambi-Rocha et al.¹ in this issue of the Journal, chronic mouth breathing during active craniofacial development of a child may result in anatomical changes that directly affect the airway.² These changes may result in greater airway instability and collapsibility that potentially lead to other problems later in life, such as sleep-disordered breathing.³ Prior investigations of children with mouth breathing have shown a correlation with abnormal orofacial growth.⁴ There is also a continuous interaction between nasal breathing and appropriate sucking, swallowing, and mastication to optimize orofacial growth.⁵ This is especially important in children, in whom the naso-maxillary complex continuously grows from infancy, throughout the pre-pubertal period, and until the

completion of puberty. In fact, maximal orofacial growth takes place during the first two years of life; by age 6 years, nearly 60% of the adult face has developed. Therefore, establishing adequate nasal breathing early in life is essential to maximize the growth of the skeletal complex and the upper airway.^{6,7}

The continuous interaction between the nasomaxillary complex and the mandible during nasal breathing is also important to guide the growth of the entire facial-skeletal complex in a forward and horizontal orientation. This interaction lessens the angulation of the occlusal plane, which shortens the airway length, creates intraoral space to accommodate the tongue, leads to a shorter soft palate, and potentially improves the function of airway dilator muscles to help maintain the airway open.^{8,9} Therefore, it is reasonable to assume that, in order to maximize the potential of adequate craniofacial and airway development, the ultimate goal should be the establishment of continuous nasal breathing. This, in conjunction with other oral functions such as sucking, swallowing, and mastication, are critical functions that will continuously stimulate the intermaxillary cartilage from birth until 13–15 years of age. While active, this synchondrosis will allow facial growth through an osteo-chondral ossification mechanism.⁷

DOI of original article:

<http://dx.doi.org/10.1016/j.jpmed.2017.05.007>

[☆] Please cite this article as: Torre C, Guilleminault C. Establishment of nasal breathing should be the ultimate goal to secure adequate craniofacial and airway development in children. J Pediatr (Rio J). 2017. <http://dx.doi.org/10.1016/j.jpmed.2017.08.002>

* Corresponding author.

E-mail: cguil@stanford.edu (C. Guilleminault).

<http://dx.doi.org/10.1016/j.jpmed.2017.08.002>

0021-7557/© 2017 Published by Elsevier Editora Ltda. on behalf of Sociedade Brasileira de Pediatria. This is an open access article under the CC BY-NC-ND license (<http://creativecommons.org/licenses/by-nc-nd/4.0/>).

Fitzpatrick et al. were the first to describe how continuous mouth breathing leads to a significant increase in upper-airway resistance.³ Then, in the 1980s, a number of groundbreaking experiments shed some light into these observations, when a group of newborn Rhesus monkeys had their nasal passages blocked during the first six months of life.¹⁰ At the end of this period, the monkeys were found to have narrowing of dental arches, decreased maxillary arch length, anterior cross-bite, maxillary overjet, and increased anterior facial height. EMG recordings of different orofacial and neck muscles also revealed an abrupt induction of rhythmic discharge patterns, very different from the nearly continuous low amplitude and desynchronized discharges most normal subjects have at rest.^{11,12} Interestingly, at the end of the six-month period, the Rhesus monkeys were allowed to breathe normally through their noses, which led to normalization of muscle discharge and restoration of adequate orofacial growth.

In humans, restoration of normal nasal breathing continues to be a challenge even after correction of anatomical problems contributing to nasal resistance. Several theories attempt to explain why it is so challenging to re-train a person to become a nasal breather after years of mouth breathing. When there is a "nasal disuse", there is a loss of proprioception and a functional "de-afferentation" that prevent return to normal nasal air-exchange, even after the anatomical factors contributing to nasal obstruction are corrected.¹³ Chronic mouth breathing also results in "under-ventilation" of the nose, which may lead to accumulation of inflammatory cells in the nasal mucosa that cause nasal resistance.¹⁴ Finally, the same anatomic disturbances in orofacial growth that result from chronic oral breathing, in particular the narrowing of the dental arches, may limit the intranasal space and may also cause deviation of the nasal septum secondary to its compression by the high arched palate in a cephalo-caudal orientation.¹⁵

Comparing humans to the Rhesus monkeys may help us understand why there is so little room for error in our species and why it is important to address abnormal nasal breathing and poor oral functions early in life to maximize the growth potential of the craniofacial skeleton. The development of speech in *Homo sapiens*, as well as the switch to bipedalism, led to the elongation of the airway with the development of a poorly supported 2–4 mm oropharynx that lacks an epiglottis lock against the palate.¹⁶ In addition, to facilitate speech, there was an anterior migration of the foramen magnum and a regression of the maxillo-mandibular complex in order to establish the 1:1 ratio of the supra vocal cord tract necessary for adequate speech production.¹⁷ The regression of the skeletal complex came at the expense of teeth. Compared to humans, which have 32 teeth, other monkey species such as the chimpanzee can have up to 44 teeth. The result of this compromised skeletal complex was a pushback of the tongue, which led it to become an obstructive element by becoming part of the upper airway.¹⁸ In monkeys and most other species, the tongue is confined to the oral cavity and does not block the airway.

Nasal breathing during sleep is essential to stimulate adequate ventilation, activate reflexes that help maintain the tonicity of the muscles that stabilize the upper air-

way, and to avoid the airway instability that results from mouth breathing.¹⁹ Addressing mouth breathing during sleep is essential, considering that, at birth, the child spends nearly 80% of the time asleep, and even at 6 years of age they continue to have a prolonged sleeping time where they can spend up to 25% of their day sleeping. Studies monitoring nasal and mouth breathing during sleep have shown that normal individuals spend 96% of their sleep time breathing through their nose.²⁰ This observation was confirmed by other studies showing that normal children between the ages of 4 and 6 years of age oscillate between 0 to 10% of the time breathing through their mouth during sleep, with a mean of 4% of the time.

Considering all this, it is therefore essential to address any problems such as chronic mouth breathing that contributes to poor skeletal and airway development in children. Under these circumstances, patients may not have enough room to accommodate the tongue or other structures, such as the palatine and lingual tonsils, that may become obstructive elements during sleep. Limited airway space from poor skeletal development may also prevent patients from maintaining adequate airway patency as they progress through the natural stages of sleep and their muscles relax. The combination of these elements may ultimately result in airflow limitation during sleep that leads to frequent arousals and drops in the blood-oxygen saturation levels, which defines what is known as obstructive sleep apnea.

Conflicts of interest

The authors declare no conflicts of interest.

References

1. Chambi-Rocha A, Cabrera-Domínguez ME, Domínguez-Reyes A. Breathing mode influence on craniofacial development and head posture. *J Pediatr (Rio J)*. 2018;94:x–y. Q2
2. Mcnamara J. Influence of respiratory pattern on craniofacial growth. *Angle Orthod*. 1981;51:269–300.
3. Fitzpatrick M, McLean H, Urton A, Tan A, O'Donnell E. Effect of oral or nasal breathing route on upper airway resistance during sleep. *Eur Respir J*. 2003;22:827–32.
4. Linder-Aronson S. Dimensions of face and palate in nose breathers and habitual mouth breathers. *Odontol Rev*. 1969;14:187–200.
5. Vargervik K, Harvold E. Experiments on the interaction between orofacial function and morphology. *Ear Nose Throat J*. 1987;66:201–8.
6. Guilleminault C, Partinen M, Praud JP, Quera-Salva MA, Powell N, Riley R. Morphometric facial changes and obstructive sleep apnea in adolescents. *J Pediatr*. 1989;114:997–9.
7. Souki BQ, Lopes PB, Pereira TB, Franco LP, Becker HM, Oliveira DD. Mouth breathing children and cephalometric pattern: does the stage of dental development matter? *Int J Pediatr Otorhinolaryngol*. 2012;76:837–41.
8. Liu SY, Huon LK, Powell NB, Riley R, Cho HG, Torre C, et al. Lateral pharyngeal wall tension after maxillomandibular advancement for obstructive sleep apnea is a marker for surgical success: observations from drug-induced sleep endoscopy. *J Oral Maxillofac Surg*. 2015;73:1575–82.
9. Camacho M, Liu S, Certal V, Capasso R, Powell NB, Riley R. Large maxillomandibular advancements for obstructive sleep apnea:

- 169 an operative technique evolved over 30 years. *J Craniomaxillo-*
170 *fac Surg.* 2015;43:1113–8.
- 171 10. Harvold E, Tomer B, Vargervik K, Chierici G. Primate
172 experiments on oral respiration. *Am J Orthod.* 1981;79:
173 359–72.
- 174 11. Vargervik K, Miller A, Chierici G, Harvold E, Tomer B.
175 Morphologic response to changes in neuromuscular patterns
176 experimentally induced by altered modes of respiration. *Am*
177 *J Orthod.* 1984;85:115–24.
- 178 12. Miller AJ, Vargervik K, Chierici G. Experimentally induced neu-
179 romuscular changes during and after nasal airway obstruction.
180 *Am J Orthod.* 1984;85:385–92.
- 181 13. Lee S, Guilleminault C, Chiu H, Sullivan S. Mouth breath-
182 ing, “nasal disuse,” and pediatric sleep-disordered breathing.
183 *Sleep Breath.* 2015;19:1257–64.
- 184 14. Gelardi M, Carbonara G, Maffezzoni E, Marvisi M, Quaranta N,
185 Ferri R. Regular CPAP utilization reduces nasal inflammation
186 assessed by nasal cytology in obstructive sleep apnea syndrome.
187 *Sleep Med.* 2012;13:859–63.
15. Akbay E, Cokkeser Y, Yilmaz O, Cevik C. The relationship
188 between posterior septum deviation and depth of maxil-
189 lopalatal arch. *Auris Nasus Larynx.* 2013;40:286–90.
- 190 16. Dedhia RC, Rosen CA, Soose RJ. What is the role of
191 the larynx in adult obstructive sleep apnea? *Laryngoscope.*
192 2014;124:1029–34.
- 193 17. Mendes A, Alves-Pereira M, Castelo Branco NA. Voice acoustic
194 patterns of patients diagnosed with vibroacoustic disease. *Rev*
195 *Port Pneumol.* 2006;12:375–82.
- 196 18. Behlfelt K, Linder-Aronson S, Neander P. Posture of the head,
197 the hyoid bone, and the tongue in children with and without
198 enlarged tonsils. *Eur J Orthod.* 1990;12:458–67.
- 199 19. Michels D, de S, Rodrigues A, da MS, Nakanishi MM, Sampaio AL,
200 et al. Nasal involvement in obstructive sleep apnea syndrome.
201 *Int J Otolaryngol.* 2014;2014:717419.
- 202 20. Fitzpatrick MF, Driver HS, Chatha N, Voduc N, Girard AM. Par-
203 titioning of inhaled ventilation between the nasal and oral
204 routes during sleep in normal subjects. *J Appl Physiol* (1985).
205 2003;94:883–90.
- 206

UNCORRECTED PROOF