

The case for environmental etiology of malocclusion in modern civilizations—Airway morphology and facial growth



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The impact of nasal respiration impairment on craniofacial growth and development remains a topic of interest for orthodontists in their daily encounter with mouth breathing patients. The aims of this article are to critically review the: (1) etiology of nasal obstruction, namely septal deviation, turbinate dysfunction, lymphoid tissue hypertrophy, and soft tissue alteration; (2) diagnostic methods to evaluate nasal obstruction; (3) role of mouth breathing in the development of characteristic malocclusions and associated patterns of facial growth (“adenoid facies”), with a focus on recent research data; (4) indications of medical and surgical treatments with the ongoing debate about removal of lymphoid tissues to avoid facial dysmorphology; (5) diagnosis and treatment of obstructive sleep apnea in growing subjects. Orthodontists play an important role in the early diagnosis of airway impairment. Early clearance of the airways, whether medically or surgically achieved, is gaining more ground between ENT specialists as they became aware of the potential effect on craniofacial development. (Semin Orthod 2016; 22:223–233.) © 2016 Elsevier Inc. All rights reserved.

Introduction

The study of the relationship between malocclusion and environmental factors has been uninterruptedly updated in the orthodontic literature during the last century. The most evaluated aspect has been the potential effect of altered mode of breathing on dentofacial components. Orthodontists have focused on this association mainly because of daily encounters with patients exhibiting complete or partial abnormal respiration. They discovered that aberrations in the nose, the neighboring anatomical entity to the mouth, created a variety of malocclusions and facial dysmorphologies because of the diversity of the adaptation processes.

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In his seminal classification of malocclusion, Edward Angle singled out the relationship between mouth breathing and malocclusion. He described the Class II division 1 malocclusion as “always accompanied and, at least in its early stages, aggravated, if not indeed caused by mouth breathing due to some form of nasal obstruction.”¹ Regarding the Class III malocclusion, he stated that “deformities under this class begin at about the age of the eruption of the first permanent molars, or even much earlier, and are always associated at this age with enlarged tonsils and the habit of protruding the mandible, the latter probably affording relief in breathing.”¹ However, the excessive number of studies that assessed the direct connection between nasal obstruction and facial growth,^{2–14} failed to seal the debate on the orthodontic implications of nasal respiration impairment.^{15–17}

The aim of this article is to explore the various aspects of the association between mouth breathing and dentofacial growth namely, the etiology of mouth breathing, the relationship between malocclusion and mouth breathing, the medical treatment and the optimal timing of lymphoid tissue removal.

Etiology of mouth breathing

The airway tube extends from the nostrils to the lungs. In between, the nose, nasopharynx, and oropharynx are lined up with many important organs and tissues that play an important role in filtering and humidifying the air before it reaches the lungs, and in the immunity of our body. Conversion of nasal to oral breathing can be induced by different factors, whether partial or complete airway obstruction may occur at any of those levels and can develop at any age.

Mouth breathing is classified into two groups: habitual, with adequate nasal potency, and enforced, through nasal resistance or obstruction.¹⁸ The latter may occur in the anterior (maxillary) airway, in the posterior (pharyngeal) airway, or both, since the two sections are not completely independent. The maxillary section has greater resistance in the nasal airway and therefore is more prone to obstruction.

Causes of nasal airway obstruction

Lymphoid tissues hypertrophy

Adenoids hypertrophy constitutes the primary cause of upper airway obstruction, particularly in children, inducing mouth breathing. The adenoids are located at the junction between the nose and the oral cavity, at the roof of the nasopharynx near the Eustachian tube that connects the ear to the oropharynx. In few instances, the hypertrophied adenoids can block the Eustachian tube and limit the drainage from the middle ear into the nasopharynx, which can cause a middle ear effusion.

Interestingly, and unlike other tissue in the body, the adenoids increase in size during childhood to twice of their final adult size with a particular pattern of growth,¹⁹ an observation that Pruzansky²⁰ denied. He suggested, in a cephalometric study, that the lymphoid tissues do not follow a specific growth curve, but respond individually to different environmental factors. Later, in a longitudinal study between ages 3 and 16 years, Linder-Aronson and Leighton²¹ studied adenoids growth behavior on lateral cephalographs, and reported an increase in adenoid size in preschool and primary grade level years, followed by a decrease during preadolescence and early adolescence. These findings support a prevailing practice

by otolaryngologists to delay the removal of the pharyngeal lymphoid tissues until after puberty.

Parallel to the growth of lymphoid tissues, the general growth of the oropharynx complex and face maintain a normal functioning of the nasopharynx.²² Nasopharyngeal obstruction and subsequent change to mouth breathing may be induced if discrepancy in the growth of the lymphoid tissues and the nasopharynx occurs.²²

On the other hand, the tonsils known as the “gate keepers” of the oropharynx, may also lead to airway obstruction if hypertrophied (Fig. 1). In the rare condition when tonsils touch or meet in the midline, they are called “kissing tonsils.” Otolaryngologists classify the tonsillar hypertrophy in a similar grading system to that of adenoid hypertrophy. However, clinical examination for diagnosis is crucial.

Although the adenotonsillar hypertrophy constitute the main cause of airway obstruction in growing individuals, other agents may contribute in increasing nasal resistance in the upper nasal airways such as hard tissues: deviated septum, turbinate irregularities and congenital, traumatic or therapeutic asymmetries of the nasal cavity; and soft tissues: catarrhal and allergic rhinitis, and nasal polyps.

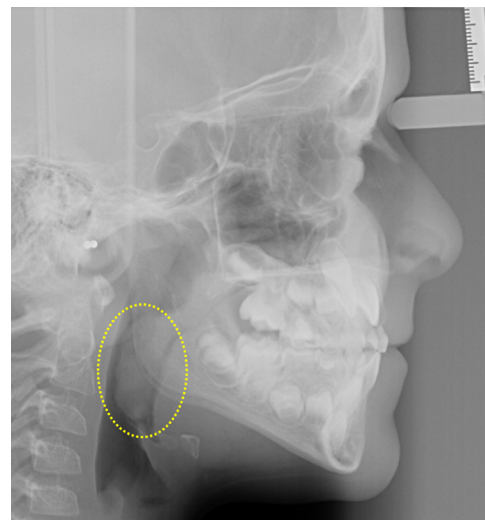


Figure 1. Lateral cephalometric radiograph of a 6-year-old boy. The circle in yellow denotes the hypertrophied tonsils almost blocking totally the pharyngeal airways.

Septal deviation

The nasal septum, which is dividing the nose into two bilateral cavities, both formed by cartilaginous and osseous tissues. It remains one of the most common causes of nasal airway impairment. A septal deviation to one side, caused by genetic or environmental factors such as trauma, is a major cause of airway obstruction. Trauma at birth is shown to induce septal deviation in newborn with different percentage of incidence.^{23–25} The deviation can be diagnosed clinically and cephalometrically (Fig. 2). Upon clinical examination, a bulging mass is seen at the opening of the nostrils unilaterally. Tracking the midsagittal line on the postero-anterior cephalometric radiograph shows a deviation in the radiopaque cartilage mass, usually assuming an S shape.

Turbinates

The lateral walls of the nasal cavity are lined up with three pairs of turbinates: superior, middle, and inferior. The turbinates play a primary role in “filtering,” heating and humidifying the air before it reaches the lungs. They undergo cycles of swelling every 3–7 h, with no change in the total nasal airway resistance. The nasal cycle occurs in nearly 80% of normal people with alternate congestion and decongestion between right and left side.²⁶ The cyclical hypertrophy should not be mistaken for a chronic condition.

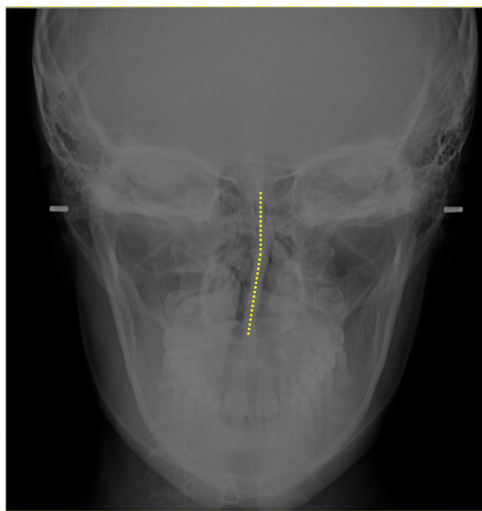


Figure 2. Postero-anterior cephalometric radiograph of an 11-year-old girl. The nasal septum is deviated in its lower half causing a shift to mouth breathing mode.

Similar to adenoids, turbinates’ hypertrophy can be diagnosed on nasal endoscopy, but also on postero-anterior cephalographs. It can disclose a possible extension of the posterior part of the inferior turbinates that would indicate an etiology for mouth breathing, particularly in the absence of septal deviation and/or large adenoids and tonsils. When hypertrophied, inferior turbinates may extend posteriorly and constrict the airways, showing a “foggy” image on lateral cephalograph above the posterior nasal spine (PNS). This “tail” is often overlooked (Fig. 3).²⁷ The pathology may be associated with allergic rhinitis that is treated with either medication (nasal steroids) or surgery (reduction or excision).²⁸

Soft tissues

Nasal obstruction may be caused by an overgrowth or alteration of the soft tissue mucosa lining the nasal cavity, in the presence of a local or general pathology. Catarrhal or allergic rhinitis is a common etiology for chronic mouth breathing. A long period of treatment is usually necessary. When nasal polyps affect nasal respiration, surgical removal is mandatory to recover normal respiration.

Nasal obstruction and mouth breathing: Relationship and diagnosis

Methods to diagnose adenoidal hypertrophy include nasal endoscopy (NE) and radiological imaging.

While nasoendoscopy remains a standard mean to diagnose any nasal airway impairment, cone-beam computerized tomography (CBCT) has gained ground in the orthodontic science. It is progressively used for diagnosis and treatment planning on patients exhibiting complex malocclusions.²⁹

Studies have compared the accuracy of diagnostic methods, i.e., nasal endoscopy and lateral cephalometric radiographs and have been found to be similar, with endoscopy having the advantage of three dimensional evaluation.³⁰ In a recent study comparing CBCT with nasoendoscopy the researchers found that CBCT images were as accurate as NE in evaluating adenoid size especially when used by trained orthodontists.³¹

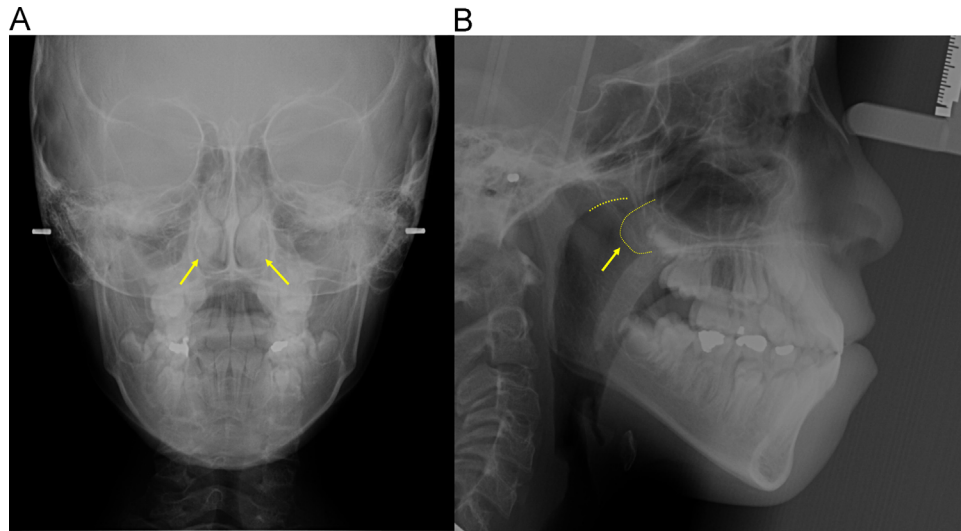


Figure 3. (A) Bilateral obstruction of the airway, in a 9-year, 4-month-old patient, by the hypertrophied inferior turbinates (right and left arrows) shown on a postero-anterior cephalograph. (B) The patient's lateral cephalometric radiograph shows the posterior extension of the inferior turbinate toward the posterior wall of the nasopharynx (arrow). This hypertrophy induced a narrowing of the airways and shift to mouth breathing mode.

On lateral cephalographs, adenoid hypertrophy has been classified into three or four sizes or grades, ranging from small to large, as well as ratios between the size of adenoids and nasopharynx.³² The classification shown in Fig. 4 is based on the percentage of airway obstruction, whereby the following grades are assigned: 1 for less than 50% obstruction, 2 for more than 50% but less than 100%, and 3

when the adenoids totally block air passage (Fig. 4). The subjective method in grading the airway obstruction by adenoids corresponded with the objective measurement of the airway clearance. In a study on 200 growing subjects, Bitar et al.³² found a high correlation between the adenoids grading and the shortest distance between the adenoids and the soft palate ($r = -0.79$).

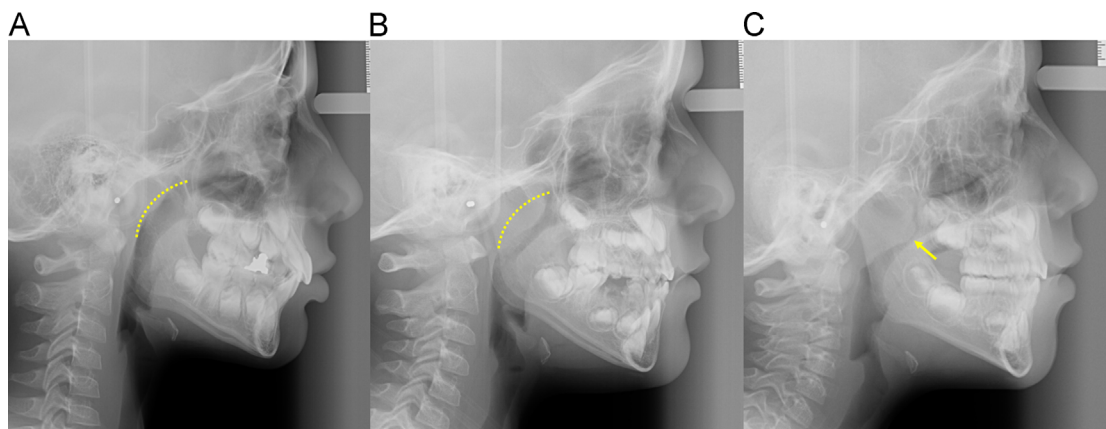


Figure 4. Adenoid hypertrophy: lateral cephalographs of 3 mouth breathers showing different grades of airway obstruction relative to adenoid size. (A) Grade 1 (less than 50% obstruction of airway) in a 8-year, 6-month-old boy (B) Grade 2 (more than 50% obstruction but less than 100% airway obstruction) in a 6-year-old boy. (C) Grade 3 (total obstruction) in a 4-year, 9-month-old boy.

Mouth breathing and facial growth: Relationship and diagnosis

The impact of mouth breathing upon the development of malocclusion appears to be related largely to a low posturing of the tongue (and the subsequent adaptation of other facial muscles) that may influence growth of the jaws, and the occlusion. Mouth breathing has been associated with a narrow upper arch, but apparently no high palatal vault¹¹; posterior crossbite; anterior open bite, usually through excessive eruption of posterior teeth; and a hyperdivergent skeletal pattern.¹¹

In parallel to the above descriptors, children requiring adenoidectomy have been reported to have longer facial height, steeper mandibular plane angle, and a more retrognathic mandible than corresponding controls.²⁻⁷ Similarly, children with enlarged tonsils were found to have more retrognathic and superior-posteriorly inclined mandibles, greater anterior total and lower facial heights, and larger mandibular plane angles.⁸ Moreover, retroclined mandibular incisors, more anteriorly positioned maxillary incisors, decreased overbite, increased overjet, increased incidence of lateral crossbites, shorter mandibular arches, and narrower maxillary dental arches²⁻¹⁴ were related to chronic mouth breathing. Acknowledging the impact of

lymphatic tissues on facial morphology, orthodontists labeled faces with those reported characteristics as “adenoid facies” (apparently at least 100 years ago), but has also been known as “long face syndrome” and “high angle” facial pattern (Fig. 5).³³

Recently, we reported on data collected from the cephalographs of 200 Caucasian children (ages: 1.71–12.62 years, nearly 50% of them <5 years) that were diagnosed by a pediatric otolaryngologist as being chronic mouth breathers, and referred them for cephalometric evaluation of adenoid hypertrophy.³⁴ Facial dysmorphology was observed as early as the second year of life in the youngest patient evaluated (1.71 years). Postero-inferior tilt of the maxilla (average inclination of palatal plane to horizontal: $-7.68^\circ \pm 3.44^\circ$; norm: $0^\circ \pm 2.5^\circ$,³⁵ possibly the initial response to functional alteration, occurred separately or together with one or all of the following modifications, compatible with a hyperdivergent vertical pattern: increased palatal to mandibular plane angle; increased lower face height, steep mandibular plane, mandibular antegonial notching, increased gonial angle, and elongated and thinner symphysis (Fig. 5B). The palatal tilt reached severe levels (8° – 9°) between ages 4 and 5 years. The occlusion ranged from normal with adequate overjet/overbite to malocclusions that

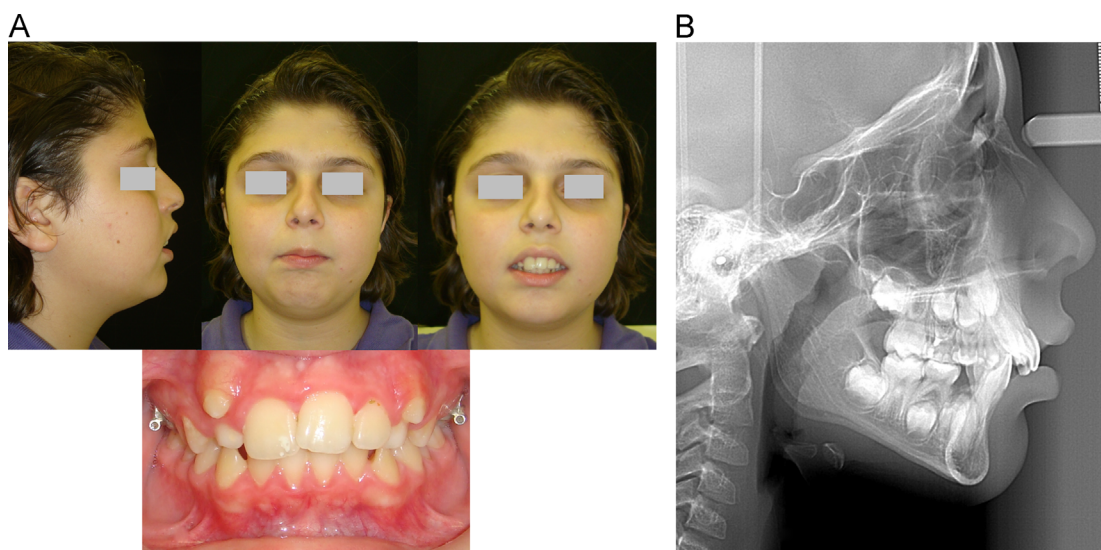


Figure 5. (A) “Long Face” syndrome characteristics in a 9-year, 3-month-old boy mouth breather: lip incompetency, increased lower facial height, narrow width of the nose base and shadows under the eyes. (B) Lateral cephalograph of same patient shows the hyperdivergent vertical pattern: increased palatal to mandibular plane angle (PP/MP = 36°); increased lower face height (LFH/TFH = 57%).

contained one or more of these characteristics: posterior crossbite, increased overjet, Class II molar relationship, open bite, and anterior crossbite.

In addition, when the study group was classified by age into group 1: ≤ 6 years ($n = 124$) and group 2: > 6 years ($n = 76$), airway clearance distance (AD) was more decreased in the younger group and at a statistically significant level (group 1: 3.19 ± 2.32 mm; group 2: 4.78 ± 2.80 mm; $p < 0.05$). Furthermore, we stratified the study group into four subgroups on the basis of palatal to mandibular plane angulation to reflect the facial divergence: group A: $PP-MP \leq 27.5^\circ$, $n=34$; B: $27.5^\circ < PP-MP \leq 32^\circ$, $n = 68$; C: $32^\circ < PP/MP < 36.5^\circ$, $n = 67$; D: $PP-MP \geq 36.5^\circ$, $n = 31$. Statistically significant differences ($p < 0.05$) occurred mainly between the most hyperdivergent group (D) and the hypodivergent (A) and normodivergent (B) groups in the AD distances, albeit the hyperdivergent pattern exhibited the narrowest airways.³⁴

Authors have tackled the issue of differences across age groups. Linder-Aronson et al.² related those potential differences to the effect of normal growth of the nasopharynx leading to increase in airway clearance. However, in our study, both age groups included important characteristics pertinent to hyperdivergence (MP-SN, PP-MP) and to long face syndrome in general, suggesting that this facial pattern on average would last once it was present. Thus, the severity and extent of these morphologic alterations depend on the timing, duration, and rate of oral breathing.

Despite the significant number of studies relating association between mouth breathing and the development of malocclusion, the association is not clear-cut.³⁶⁻³⁸ Recent studies have confirmed the presence of mostly "vertical" alteration of the dentofacial complex rather than a "sagittal" one.³⁹

Medical and surgical treatment

The health-risks associated with bypassing the physiological protective mechanisms of the nasal airway in warming, humidifying and purifying inhaled air, in addition to the associated craniofacial dysmorphoses, often warrant medical and surgical interventions to resolve persistent mouth breathing in children.^{40,41} Treatment will

depend on the underlying etiology of mouth breathing: the most common culprits in children, allergic rhinitis and adenotonsillar hypertrophy, requiring two different approaches.^{40,42-44} The management of allergic rhinitis lies in pharmacological drugs utilized either orally or intranasally. Medicaments include antihistamines, corticosteroids, antileukotrienes, nasal decongestants and intranasal saline douching.⁴⁰ The two drugs most effective in battling nasal obstruction, intranasal corticosteroids and nasal decongestants, raise different safety and tolerability concerns when dealing with children. Stimulatory effects and cardiac-related events generally contraindicate the use of nasal decongestants in children, not to mention the risk of rebound nasal congestion. On the other hand, intranasal corticosteroids raise concerns for possible effects on growth velocity and hypothalamic-pituitary-adrenal axis function.⁴⁵ Although generally regarded as safe in children when used in low doses, especially for compounds with low systematic availability, a small degree of risk cannot be excluded, especially in light of the lack of studies evaluating the final height in children treated with intranasal corticosteroids.

Frequently, allergic rhinitis occurs concomitantly with adenotonsillar hypertrophy in children.⁴⁶ Whether in association with rhinitis or as a separate entity, surgical intervention in the form of adenoidectomy, tonsillectomy or both becomes necessary if resolution of mouth breathing is to be expected. Currently, the indications for adenoidectomy alone versus in conjunction with tonsillectomy for the management of airway obstruction are unclear. Despite the post-operative morbidity associated with adenoidectomy, children are often able to return to normal activity the day after surgical intervention and the risk of post-operative hemorrhage is less than 1%.⁴⁷ However, combined adenoidectomy/tonsillectomy increases the risk of hemorrhage to 3% and may delay the recovery period to 14 days.⁴⁷

While the mean age of onset of allergic rhinitis is 10 years, adenotonsillary hypertrophy is often diagnosed significantly before the age of 5 years, potentially deterring normal craniofacial growth at an earlier, more sensitive period and for a longer number of years. Adenotonsillectomy, and more commonly adenoidectomy, is

therefore among the most common surgeries performed in children.^{46,47} The post-operative assessment of children undergoing such surgeries has highlighted the potential of normalization of breathing towards reversing or stabilizing the craniofacial dysmorphoses associated with mouth breathing. Several authors have described a more anterior direction of symphyseal growth, reversal of the tendency to mandibular rotation, increase in posterior facial height and increased amount of mandibular growth following adenoidectomy or adenotonsillectomy.^{2-4,48-51} Favorable changes in dental arches and dental positions have similarly been reported: increase in maxillary inter-canine width and normalization of upper and lower incisor inclinations.^{6,44,49,51,52} Nevertheless, normalization of growth and craniofacial patterns has been reported to be partial, with the majority of children retaining features of the dolichofacial type of long face syndrome and variations in individual response.^{6,12,53} Similarly, limited research on the potential for myofunctional improvement suggests partial improvement in tongue posture, facial muscle tonicity, mobility of lips/tongue/mandible, deglutition and mastication.^{46,54} When post-treatment changes were followed through time, the incomplete immediate recovery was not found to improve with time and significant disability often remained, particularly in relation to masticatory function and deglutition.⁴⁶ Although it would be prudent to avoid firm conclusions regarding this young area of research, preliminary results on small sample sizes may suggest the need for a multidisciplinary approach involving speech therapy and myofunctional exercises for a more optimal recovery.^{46,54,55}

Clinical implications

The nature and timing of craniofacial growth and the early morphological changes observed with nasal airway obstruction support early surgical intervention to avoid a permanent setting of skeletal dysmorphology that would be difficult to control orthodontically. However, the invasiveness and potential morbidity of an elective surgical intervention in a child necessitate a careful cost-benefit analysis. Several factors favor the delay in surgical intervention: the airway naturally becomes less obstructed with increasing age,

possibly indicating a gradual adjustment or compensation in growth^{32,56}; dental compensation is not uncommon and occlusion often shows no signs of deterioration despite mouth breathing³⁴; research comparing the skeletal effects following adenoidectomy/adenotonsillectomy does not support significant benefits when performed early (<4 years or in the primary dentition) compared to later in childhood (>4 years or mixed dentition).^{57,58}

It is noteworthy that the measures that have been shown to be affected by the timing of surgical intervention are the angular divergence between maxilla and mandible⁵⁸ and anterior lower facial height.⁵⁷ Interestingly, when Bitar et al.³² looked specifically at children with nearly complete adenoid obstruction, features relating to hyperdivergence, increased lower facial height and other long-face syndrome characteristics were present across all ages examined. The data suggest that simply classifying children into mouth breathers and nasal breathers may impede the assessment of possible growth corrections consequent to surgical intervention, and calls for the investigation of the effects of early vs. late treatment while controlling for the severity of obstruction. Further longitudinal research is crucial for the development of prediction equations and evidence based guidelines for when early adenoidectomy and/or tonsillectomy should replace pharmacological treatment and close monitoring of growing children. Such guidelines would be based on the severity and persistence of nasopharyngeal airway obstruction, the presence of early signs of malocclusion and individual risk for the long-face syndrome.³⁴

Obstructive sleep apnea in children

Mostly known as a frequent problem in adults, obstructive sleep apnea syndrome (OSAS) is also common in children and adolescents, and it is considered the severe aspect of the sleep disordered breathing which includes as well primary snoring and upper airway resistance syndrome.

Originating from a different epidemiological background, its diagnosis and therefore its treatment approach can differ from adults. OSAS prevalence varies from 0.69–2.9% in children⁵⁹⁻⁶² and is characterized by prolonged partial upper airway obstruction and/or intermittent complete obstruction that disrupts

normal ventilation during sleep and normal sleep patterns.⁶³ These episodes of obstructive apnea or hypopnea can last for more than 10 s, and are mostly terminated by arousals.

Etiology

Predisposing factors that can lead to partial or total airway narrowing can play a major role in developing OSAS such as hypertrophied tonsils and enlarged adenoid,⁶⁴ allergic rhinitis leading to nasal obstruction because of nasal mucosal edema and mucus secretion.⁶¹

Obesity was related to the increase risk of snoring and in severe cases to OSAS⁶⁵ where it appears to be more prevalent among overweight and obese children, as high as 60%⁶⁶; it is suggested that the lateral pharyngeal walls consisting of muscles, tonsillar tissues, and fat pads, can increase in thickness due to the total volume of fat and therefore limiting the airflow.⁶⁷

Risk factors for OSAS also include medical conditions that involve craniofacial dysmorphism (retrognathia), midface hypoplasia, hypotonia, and syndromes that might affect the tongue position such as Down syndrome.⁶⁸ Smoking in adults was associated with sleep breathing disorders where it leads to obstruction and collapse of the pharyngeal airway by inducing pharyngeal inflammation and mucosal edema, and therefore increasing the risk of snoring.⁶⁹ Similar results were reported with passive parental smoking where the risk factor for snoring in children was increased.⁷⁰

Symptoms

Symptoms of OSAS include snoring accompanied with choking or gasping during sleep resulting in disturbed sleep and recurrent awakenings which lead to daytime fatigue, headaches, dry or sore throat and excessive daytime sleepiness.

Neurobehavioral problems with impaired concentration, daytime hyperactivity, anxiety and depressive symptoms, failure to thrive were also associated with OSAS.^{71,72}

Parents may report loud and noisy breathing with an open mouth accompanied with snoring, and many children will sweat during sleep, especially around their head and neck. Children might accommodate in unusual positions, such

as with their neck hyperextended or propped upon multiple pillows.⁷³

Diagnosis

Considering the clinical history such as rate of growth, snoring, tendency to fall asleep during the day, sleep disturbances may lead to the diagnosis of OSAS in children. A clinical examination that reveals the presence of enlarged tonsils and adenoids can be associated to the previously mentioned signs to confirm the presence of OSAS.^{68,71}

Overnight polysomnography is recognized as the gold standard for diagnosis of OSAS. One of the problems of polysomnography in childhood is that performance and interpretation of the results have not yet been standardized or evaluated for different age groups.

Treatment

Treatment of OSAS in children depends on the etiology and usually requires a multidisciplinary management involving the pediatrician, pediatric or adolescent psychiatrist, ENT specialist, maxillofacial surgeons, orthodontists, speech therapist, and neurosurgeons in some syndromes.

- Adenotonsillectomy is considered first-line treatment if the child has adenoidal vegetations and/or tonsillar hypertrophy.⁷⁴
- Nocturnal masks for continuous positive airway nasal pressure (CPAP devices) may be recommended. Some research indicates that such therapy may be helpful in weight loss.⁷⁵
- For some children, positional (nonsupine) therapy may be indicated if their OSA is worse in certain positions such as supine sleep, shifting their sleeping position to either prone or on their sides may be an important factor in reducing the severity of OSA.⁶⁸
- Treating obesity in children with OSAS and weight loss is also considered an effective treatment option.⁶⁶
- Rapid maxillary expansion (RME): The precise role of maxillary constriction in the pathophysiology of OSA is unclear, but subjects with maxillary constriction have increased nasal resistance resulting in mouth breathing, similar to OSAS patients. The tongue posture was found to result in retroglossal airway narrowing in constricted palate. As RME treats maxillary constriction thereby

increasing the width of the maxilla and possibly reducing any nasal resistance thus modifying the breathing pattern in these patients.^{76–80} This modification involves nasal cavities and, indirectly, the jaw which will be repositioned and this causes the root of the tongue to move forward and it changes the pharyngeal structures.^{81,82}

Conclusion

The impact of mouth breathing upon the development of malocclusion seems to be highly correlated, and it can have dramatic impairment on the facial morphology during growth. The importance of early examination no later than age 5 years of age is highly recommended to detect treatable causal factors such as:

1. *Hard tissue*: deviated septum; turbinate irregularities; congenital traumatic/therapeutic asymmetries of nasal cavity.
2. *Soft tissue*: large adenoids, tonsils, catarrhal and allergic rhinitis, and nasal polyps.

The medical treatment can involve simple intervention, such as steroids to surgical involvement if needed.

Another condition that should also be detected early in children and that is frequently misdiagnosed is the obstructive sleep apnea syndrome (OSAS); it can result from the same causes of mouth breathing and can be aggravated by environmental factors, such as obesity, some specific abnormalities. This syndrome should be addressed as urgent as in some instances it does not only affect the normal facial and total growth of the child, but also can be a life threatening condition.

Despite all the studies and the scientific data that insist on the importance of addressing mouth breathing problems in growing patients, this condition is still widely misdiagnosed, or underestimated within the orthodontic specialty, and even among the medical specialists. More efforts should be invested to promote awareness within the community and the importance of early diagnosis and treatment.

References

1. Angle EH. Treatment of malocclusion of teeth. *Angles System*. Philadelphia: The SS White Dental Manufacturing Co.; 1907.
2. Linder-Aronson S, Woodside DG, Lundstrom A. Mandibular growth direction following adenoidectomy. *Am J Orthod*. 1986;89:273–284.
3. Kerr WJ, McWilliam JS, Linder-Aronson S. Mandibular form and position related to changed mode of breathing—a 5-year longitudinal study. *Angle Orthod*. 1989;59:91–96.
4. Woodside DG, Linder-Aronson S, Lundstrom A, et al. Mandibular and maxillary growth after changed mode of breathing. *Am J Orthod Dentofac Orthop*. 1991;100:1–18.
5. Woodside DG, Linder-Aronson S, Stubbs DO. Relationship between mandibular incisor crowding and nasal mucosal swelling. *Proc Finn Dent Soc*. 1991;87:127–138.
6. Linder-Aronson S, Woodside DG, Helsing E, et al. Normalization of incisor position after adenoidectomy. *Am J Orthod Dentofac Orthop*. 1993;103:412–427.
7. Linder-Aronson S. Adenoids. Their effect on mode of breathing and nasal airflow and their relationship to characteristics of the facial skeleton and the dentition. A biometric, rhino-manometric and cephalometric-radiographic study on children with and without adenoids. *Acta Otolaryngol Suppl Stockh*. 1970;265:1–132.
8. Behlfelt K, Linder-Aronson S, McWilliam J, et al. Dentition in children with enlarged tonsils compared to control children. *Eur J Orthod*. 1989;11:416–429.
9. Behlfelt K, Linder-Aronson S, McWilliam J, et al. Craniofacial morphology in children with and without enlarged tonsils. *Eur J Orthod*. 1990;12:233–243.
10. Behlfelt K, Linder-Aronson S, Neander. Posture of the head, the hyoid bone, and the tongue in children with and without enlarged tonsils. *Eur J Orthod*. 1989;11:416–429.
11. Subtelny JD. Effect of diseases of tonsils and adenoids on dentofacial morphology. *Ann Otol Rhinol Laryngol*. 1975;84:50–54.
12. Oulis CJ, Vadiakas GP, Ekonomides J, et al. The effect of hypertrophic adenoids and tonsils on the development of posterior crossbite and oral habits. *J Clin Pediatr Dent*. 1994;18:197–201.
13. Valera FC, Travitzki LV, Mattar SE, et al. Muscular, functional and orthodontic changes in preschool children with enlarged adenoids and tonsils. *Int J Pediatr Otorhinolaryngol*. 2003;67:761–770.
14. Mattar SE, Anselmo-Lima, Valera FC, et al. Skeletal and occlusal characteristics in mouth-breathing pre-school children. *J Clin Pediatr Dent*. 2004;28:315–318.
15. Warren DW. Effect of airway obstruction upon facial growth. *Otolaryngol Clin North America*. 1990;23:699–712.
16. Tourné LPM. Growth of the pharynx and its physiologic implications. *Am J Dentofacial Orthop*. 1991;99:129–139.
17. Tourné LPM, Scheweiger J. Immediate postural responses to total nasal obstruction. *Am J Dentofacial Orthop*. 1996;110:606–611.
18. Timms DJ. *Rapid Maxillary Expansion*. Chicago: Quintessence; 1981.
19. Scammon RE, Harris JA, Jackson CM, et al. *The Measurement of Man*. Minneapolis: University of Minnesota Press; 1930.
20. Pruzansky S. Roentgencephalometric studies of tonsils and adenoids in normal and pathological states. *Anna Otol Rhinol Laryngol*. 1975;84(suppl 19):55–62.
21. Linder-Aronson S, Leighton BC. A longitudinal study of the development of the posterior nasopharyngeal wall between 3 and 16 years of age. *Eur J Orthod*. 1983;5:47–58.

22. Diamond O. Tonsils and adenoids: why the dilemma? *Am J Orthod* 1980;78:495.
23. Harugop AS, Mudhol RS, Hajare PS, et al. Prevalence of nasal septal deviation in new-borns and its precipitating factors: a cross-sectional study. *Indian J Otolaryngol Head Neck Surg*. 2012;64:248–251.
24. Bhattacharjee A I, Uddin S, Purkaystha P. Deviated nasal septum in the newborn—a 1-year study. *Indian J Otolaryngol Head Neck Surg*. 2005;57:304–308.
25. Kawalski H, Spiewak P. How septum deformations in newborns occur. *Int J Pediatr Otorhinolaryngol*. 1998;44:23–30.
26. Jones N. The nose and paranasal sinuses physiology and anatomy. *Adv Drug Deliv Rev*. 2001;51:5–19.
27. Ghafari J. Biological mechanisms of tooth movement and craniofacial adaptation. In: Davidovitch Z, Mah J, eds. *Society for the Advancement of Orthodontics*. Boston, MA: Harvard; 2004:167–181.
28. Cummings CW, Fredrickson JM, Harker LA, et al. *Otolaryngology Head and Neck Surgery, II*. St. Louis: Mosby; 1998.
29. Haney E, Gansky SA, Lee JS, et al. Comparative analysis of traditional radiographs and cone-beam computed tomography volumetric images in the diagnosis and treatment planning of maxillary impacted canines. *Am J Orthod Dentofacial Orthop*. 2010;137:590–597.
30. Lourenco EA, Lopes Kde C, Pontes A Jr, et al. Comparison between radiological and nasopharyngolaryngoscopic assessment of adenoid tissue volume in mouth breathing children. *Rev Bras Otorrinolaringol (Eng Ed)*. 2005;71(1):23–27.
31. Major MP, Witmans M, El-Hakim H, et al. Agreement between cone-beam computed tomography and nasoendoscopy evaluations of adenoid hypertrophy. *Am J Orthod Dentofacial Orthop*. 2014;146:451–459.
32. Bitar MA, Macari AT, Ghafari JG. Correspondence between subjective and linear measurements of the palatal airway on lateral cephalometric radiographs. *Arch Otolaryngol Head Neck Surg*. 2010;136:43–47.
33. Proffit WR. The etiology of orthodontic problems—respiratory pattern. In: Proffit WR, Fields HW Jr, eds. *Contemporary Orthodontics*. 3rd ed. :137–141.
34. Macari AT, Bitar MA, Ghafari JG. New insights on age-related association between nasopharyngeal airway clearance and facial morphology. *Orthod Craniofac Res*. 2012;15:188–197.
35. Ricketts RM. Perspectives in the clinical application of cephalometrics. The first fifty years. *Angle Orthod*. 1981;51:115–150.
36. Klein JC. Nasal respiratory function and craniofacial growth. *Arch Otolaryngol Head Neck Surg*. 1986;112:843–849.
37. Blum DJ, Neel HB 3rd. Current thinking on tonsillectomy and adenoidectomy. *Compr Ther*. 1983;9:48–56.
38. Vig KWL. Nasal obstruction and facial growth: the strength of evidence for clinical assumptions. *Am J Orthod Dentofac Orthop*. 1998;113:603–611.
39. Agostinho HA, Furtado IÁ, Silva FS, et al. Cephalometric evaluation of children with allergic rhinitis and mouth breathing. *Acta Med Port*. 2015;28:316–321.
40. Scadding G. Optimal management of nasal congestion caused by allergic rhinitis in children. *Pediatr Drugs*. 2008;10:151–162.
41. Jefferson Y. Mouth breathing: adverse effects on facial growth, health, academics, and behavior. *Gen Dent*. 2010;58:18–25.
42. Bresolin D, Shapiro PA, Shapiro GG, et al. Mouth breathing in allergic children: its relationship to dentofacial development. *Am J Orthod*. 1983;83:334–340.
43. Motonaga SM, Berti LC, Anselmo-Lima WT. Mouth breathing: causes and changes of the stomatognathic system. *Braz J Otorhinolaryngol*. 2000;66:373–379.
44. Vieira BBI Sanguino AC, Mattar SE, et al. Influence of adenotonsillectomy on hard palate dimensions. *Int J Pediatr Otorhinolaryngol*. 2012;76:1140–1144.
45. Skoner DP, Rachelefsky GS, Meltzer EO, et al. Detection of growth suppression in children during treatment with intranasal beclomethasone dipropionate. *Pediatrics*. 2000;105:E23.
46. Bueno Dde A, Grechi TH, Trawitzki LV, et al. Muscular and functional changes following adenotonsillectomy in children. *Int J Pediatr Otorhinolaryngol*. 2015;79:537–540.
47. Black AP, Shott SR. Is adenoidectomy alone sufficient for the treatment of airway obstruction in children? *Laryngoscope* 2014;124:6–7.
48. Bahadir O, Caylan R, Bektas D, et al. Effects of adenoidectomy in children with symptoms of adenoidal hypertrophy. *Eur Arch Otorhinolaryngol*. 2006;26:156–159.
49. Zettergren-Wijk L, Forsberg CM, Linder-Aronson S. Changes in dentofacial morphology after adeno/tonsillectomy in young children with obstructive sleep apnoea—a 5-year follow-up study. *Eur J Orthod*. 2006;28:319–326.
50. Mattar SE, Valera FC, Faria G, et al. Changes in facial morphology after adenotonsillectomy in mouth-breathing children. *Int J Paediatr Dent*. 2011;2:389–396.
51. Kallunki J, Marcusson A, Ericsson E. Tonsillectomy versus tonsillectomy—a randomized trial regarding dentofacial morphology and post-operative growth in children with tonsillar hypertrophy. *Eur J Orthod*. 2014;36:471–478.
52. Pereira SR, Bakor SF, Weckx LL. Adenotonsillectomy in facial growing patients: spontaneous dental effects. *Braz J Otorhinolaryngol*. 2011;77:600–604.
53. Hultcrantz E, Larson M, Hellquist R, et al. The influence of tonsillar obstruction and tonsillectomy on facial growth and dental arch morphology. *Int J Pediatr Otorhinolaryngol*. 1991;22:125–134.
54. Valera FC, Trawitzki LV, Anselmo-Lima WT. Myofunctional evaluation after surgery for tonsils hypertrophy and its correlation to breathing pattern: a 2-year-follow up. *Int J Pediatr Otorhinolaryngol*. 2006;70:221–225.
55. Lundeborg I, McAllister A, Graf J, et al. Oral motor dysfunction in children with adenotonsillar hypertrophy—effects of surgery. *Logoped Phoniatr Vocol*. 2009;34:111–116.
56. Cassano P, Gelardi M, Cassano M, et al. Adenoid tissue rhinopharyngeal obstruction grading based on fiberoendoscopic findings: a novel approach to therapeutic management. *Int J Pediatr Otorhinolaryngol*. 2003;67:1303–1309.
57. Arun T, Isik F, Sayinsu K. Vertical growth changes after adenoidectomy. *Angle Orthod*. 2003;73:146–150.
58. Souki BQ, Pimenta GB, Franco LP, et al. Changes in vertical dentofacial morphology after adeno-/tonsillectomy during deciduous and mixed dentitions mouth

- breathing children-1 year follow-up study. *Int J Pediatr Otorhinolaryngol.* 2010;74:626–632.
59. Brunetti L, Rana S, Lospalluti ML, et al. Prevalence of obstructive sleep apnea syndrome in a cohort of 1,207 children of southern Italy. *Chest.* 2001;120:1930–1935.
 60. Anuntaseree W, Rookkapan K, Kuasirikul S, et al. Snoring and obstructive sleep apnea in Thai school-age children: prevalence and predisposing factors. *Pediatr Pulmonol.* 2001;32:222–227.
 61. Ali NJ, Pitson DJ, Stradling JR. Snoring, sleep disturbance, and behaviour in 4-5 year olds. *Arch Dis Child.* 1993;68:360–366.
 62. Gislason T, Benediktsdottir B. Snoring, apneic episodes, and nocturnal hypoxemia among children 6 months to 6 years old. *Chest.* 1995;107:963–966.
 63. American Thoracic Society. Standards and indications for cardio-pulmonary sleep studies in children. *Am Respir Crit Care Med.* 1996;153:866–878.
 64. Hultcrantz E, Lofstrand-Tidestrom B, et al. The epidemiology of sleep related breathing disorder in children. *Int J Pediatr Otorhinolaryngol.* 1995;32:63–66.
 65. Corbo GM, Forastiere F, Agabiti N, et al. Snoring in 9- to 15-year-old children: risk factors and clinical relevance. *Pediatrics.* 2001;108:1149–1154.
 66. Narang I, Mathew JL. Childhood obesity and obstructive sleep apnea. *J Nutr Metab.* 2012;2012:1–8.
 67. Schwab RJ, Gupta KB, Gefter WB, et al. Upper airway soft tissue anatomy in normal and patients with sleep disordered breathing: significance of the lateral pharyngeal walls. *Am J Respir Crit Care Med.* 1995;152:1673–1689.
 68. Tomlinson M. Obstructive sleep apnoea syndrome: diagnosis and management. *Nurs Stand.* 2007;21:49–56.
 69. Bloom JW, Kaltenborn WT, Quan SF. Risk factors in a general population for snoring. Importance of cigarette smoking and obesity. *Chest.* 1988;93:678–683.
 70. Corbo GM, Fuciarelli F, Foresi A, et al. Snoring in children: association with respiratory symptoms and passive smoking. *Br Med J.* 1989;299:1491–1494.
 71. American Academy of Pediatrics. Clinical practice guideline: diagnosis and management of childhood obstructive sleep apnea syndrome. *Pediatrics.* 2002;109:704–712.
 72. Gozal D. Obstructive sleep apnea in children: implications for the developing central nervous system. *Semin Pediatr Neurol.* 2008;15:100–106.
 73. Hoban TF, Chervin RD. Pediatric sleep-related breathing disorders and restless leg syndrome: how children are different. *Neurologist.* 2005;11:325–337.
 74. Erler T, Paditz E. Obstructive sleep apnea syndrome in children: a state-of-the-art review. *Treat Respir Med.* 2004;3:107–122.
 75. Ong CW, O'Driscoll D, Truby H, et al. The reciprocal interaction between obesity and obstructive sleep apnea. *Sleep Med Rev.* 2013;17:123–131.
 76. Haas AJ. Rapid expansion of the maxillary dental arch and nasal cavity by opening the midpalatal suture. *Angle Orthod.* 1961;31:73–86.
 77. Enoki C, Valera FC, Lessa FC, et al. Effect of rapid maxillary expansion on the dimension of the nasal cavity and on nasal air resistance. *Int J Pediatr Otorhinolaryngol.* 2006;70:1225–1230.
 78. Compadretti GC, Tasca I, Bonetti GA. Nasal airway measurements in children treated by rapid maxillary expansion. *Am J Rhinol Allergy.* 2006;20:385–393.
 79. Ramires T, Maia RA, Barone JR. Nasal cavity changes and the respiratory standard after maxillary expansion. *Braz J Otorhinolaryngol.* 2008;74:763–769.
 80. Kilic N, Oktay H. Effects of rapid maxillary expansion on nasal breathing and some naso-respiratory and breathing problems in growing children: a literature review. *Int J Pediatr Otorhinolaryngol.* 2008;72:1595–15601.
 81. Ozbek MM, Memikoglu UT, Altug-Atac AT, et al. Stability of maxillary expansion and tongue posture. *Angle Orthod.* 2009;79:214–220.
 82. Buccheri A, Dilella G, Stella R. Rapid palatal expansion and pharyngeal space. Cephalometric evaluation. *Prog Orthod.* 2004;5:160–171.