Upper airway obstruction - poor function becomes poor form

Dr. Bradford Edgren explores a condition that can have a profound influence upon the development of the craniofacial process

Educational aims and objectives
The purpose of this article is to delve into the various aspects of upper airway obstruction and its effect on younger patients.

Expected outcomes
Correctly answering the questions on page XX, worth 2 hours of CE, will demonstrate the reader can:
- Define upper airway obstruction.
- Identify craniofacial and dental growth abnormalities that are indicative of the condition.
- Realize why early diagnosis/clinical evaluations are essential to proper treatment.
- Recognize the important role that the orthodontist has in the diagnosis and treatment of upper airway obstruction.

Who could deny the importance of one's appearance in modern society? Individuals with more pleasing and tempting facial attributes are more readily welcomed than those without. While genetics are the initial consideration for all craniofacial development and morphology, malocclusions included, chronic environmental conditions, such as upper airway obstruction (UAO), may be erroneously passed off as genetic predisposition, or more simply, as part of the blueprint of growth.[1] In other words, the inherited genetic makeup of the individual (i.e., genotype) ultimately determines the eventual features and attributes of said individual (i.e., phenotype).

Upper airway obstruction (UAO) includes any abnormal condition of the nose, mouth, throat, or larynx that interferes with normal respiration. UAO is often the result of soft tissue enlargement (i.e., allergies), enlarged adenoids compared to the available airway, tonsilar hypertrophy, and/or inadequate nasal airway development. Research has demonstrated that impaired nasal respiration can have a significant effect upon the normal development of the craniofacial process and dentition/occlusion.[2-6] By the time young, growing patients present themselves for an orthodontic exam, significant alterations in the normal mode of respiration may have already imposed a profound influence upon the development of the craniofacial process.

A child whose chronic upper airway obstruction remains untreated may develop any number of undesirable craniofacial and dental growth abnormalities. These unfavorable growth problems include: constricted nasopharynx and maxillary arch forms, high palatal vaults, larger total and anterior faces, craniocervical extension, forward head posture, more retrognathic mandibles, vertical dysplasias, mandibular prognathism, and facial asymmetries. Additional studies have indicated that UAO may lead to sleep disorders and obstructive sleep apnea syndrome in children.[7-9] Many of these same craniofacial characteristics including forward head posture, craniocervical extension, narrowing of the nasopharynx and oropharynx can be found in adults suffering from obstructed sleep apnea.[10-12] Upper airway obstruction and extended craniocervical posture have also been associated with the signs and symptoms related to temporomandibular disorders.[13]

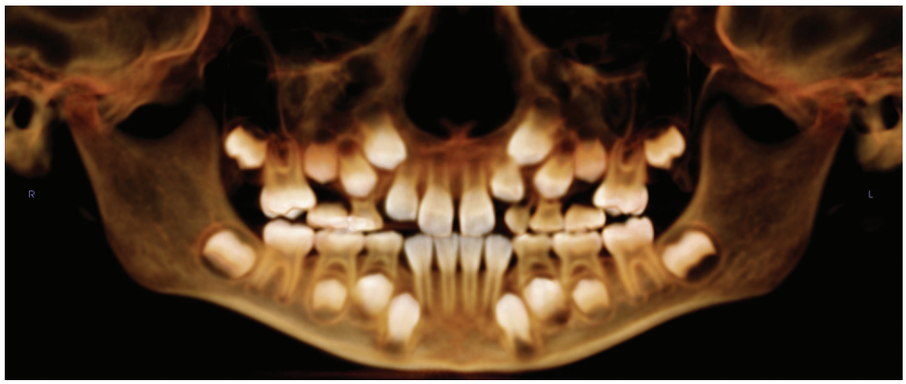

Figure 1: Pretreatment panoramic image demonstrating severe crowding and multiple ectopic and impacted teeth

Early diagnosis is essential to preventing the undesirable effects from upper airway obstruction. Fortunately, the orthodontist is in a unique position to evaluate upper airway obstructions and their affects upon facial and dental development. An orthodontist's initial exam should not be limited to just the evaluation of the presenting malocclusion, but should also include assessments of the mode of breathing and tonsilar enlargement. These preliminary clinical evaluations can then be followed up with adenoid and inferior turbinate enlargement analyses from lateral and frontal cephalograms.[14,15]

"Linder-Aronson and Leighton have shown that the lymphoid tissue on the posterior nasopharyngeal wall is thickest at 5 years of age, and subsequently decreases until 10 years of age."[16] Between 10 and 11 years, there is a slight enlargement, after which the decrease continues.[16] Research

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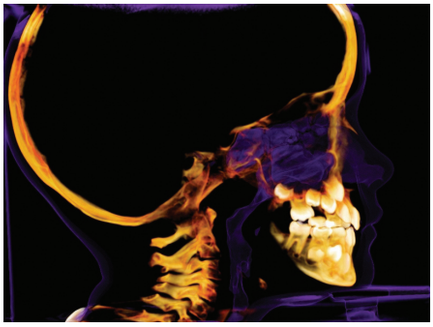

Figure 2: Pretreatment lateral image exhibiting significant adenoid hypertrophy

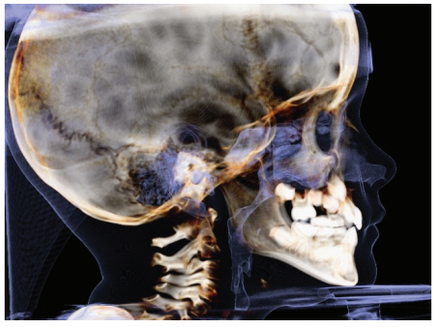

Figure 3: Pretreatment lateral image

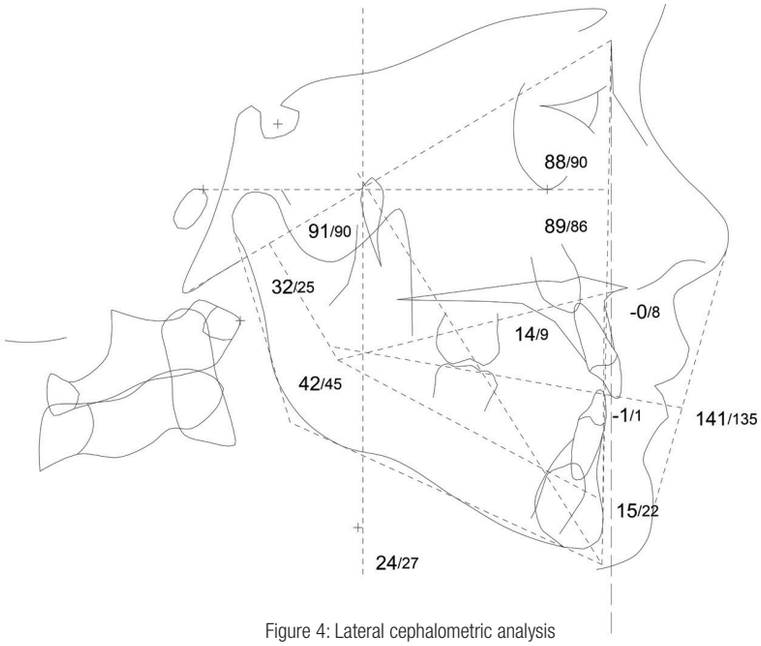

Figure 4: Lateral cephalometric analysis

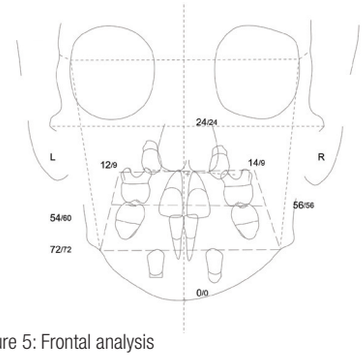

Figure 5: Frontal analysis

Figure 6: Growth Forecast to Maturity without treatment. Note the significant mandibular growth ultimately resulting in an anterior crossbite without orthodontic treatment

during the late 1970s by Linder-Aronson and Henrickson,[17] Handelman and Osborne,[18] and by Schulhof[19] developed objective methods of evaluating airway obstruction due to adenoid enlargement with lateral cephalograms. More recently, studies are evaluating the patency of the airway with cone beam computed tomography. Some of these studies have suggested that CBCT scans are an effective technique when analyzing airway volumes.[20,21] However, other studies still question the dependability and validity of three-dimensional scans in airway evaluation.[22] Regardless of the type of imaging, whether two- or three- dimensional, clinical and radiographic evidence should be used jointly when screening the patency of the airway in the orthodontic patient.

Absolute indications for tonsillectomy and adenoidectomy include, but are not limited to, adenotonsillar hyperplasia with sleep apnea, or abnormal dentofacial growth. Relative indications for adenotonsillectomy are adenotonsillar hyperplasia with upper airway obstruction, dysphagia, and/or halitosis.[23,24] Currently, there has been a significant shift towards upper airway obstruction, away from infection, as a surgical indication for tonsillectomy and/or adenoidectomy.[24]

So, who else is better qualified to evaluate existing abnormal dentofacial growth, or the potential for divergent dentofacial growth, but an orthodontist? Even though upper airway obstruction from adenoid enlargement can resolve spontaneously over time, its consequential negative influence during these periods of rapid facial growth can have remarkable and long-lasting ramifications upon a child's craniofacial growth. Waiting for adenoid enlargement to spontaneously resolve on its own may irreversibly affect a child's craniofacial development during these periods of rapid growth.[20] Shouldn't future, not just current, craniofacial and dental growth be considered, especially if it's aberrant, when evaluating a patient for adenotonsillectomy?

By age 7, a child's craniofacial development has already reached 75% of its total growth. By age 12, 90% of the average child's craniofacial maturation has been realized.[25] So, to wait until age 12, when 90% of a dentofacial deformity has already been established before

Figure 7: Super imposition of lateral cephalometric analysis and growth prediction demonstrating significant mandibular growth compared to maxillary growth

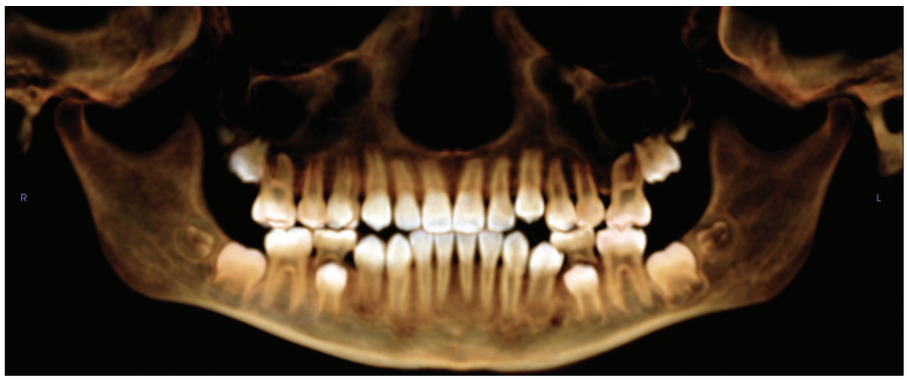

Figure 8: Post Phase I treatment panoramic image at age 8 years 10 months exhibiting resolution of severe crowding

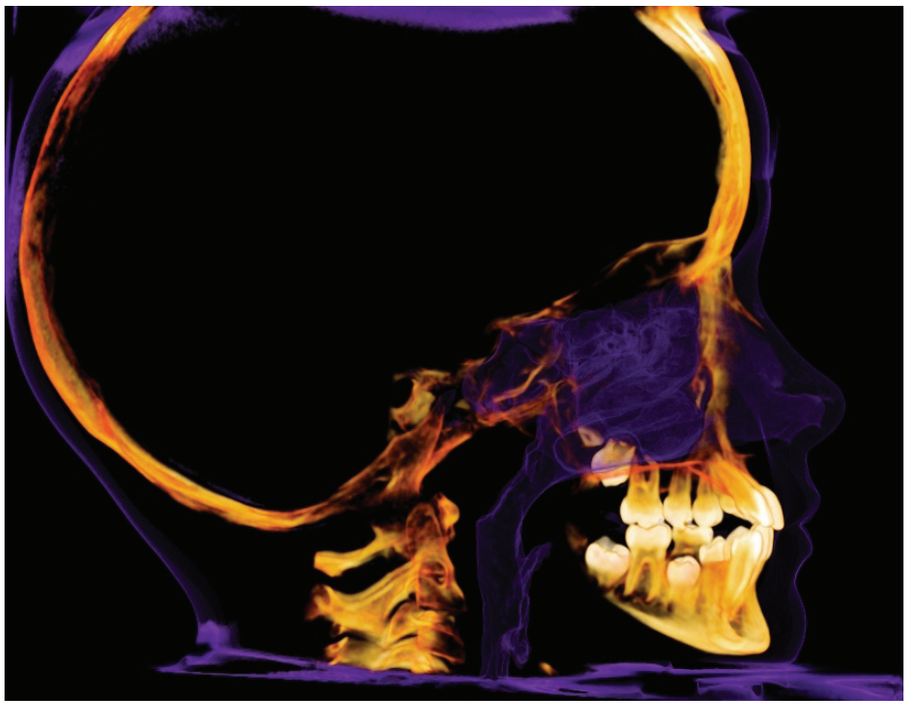

Figure 9: Post Phase I treatment lateral airway image demonstrating improved airway and straightening of the cervical vertebrae

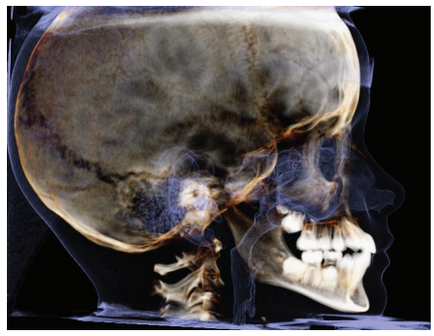

Figure 10: Post Phase I treatment lateral image

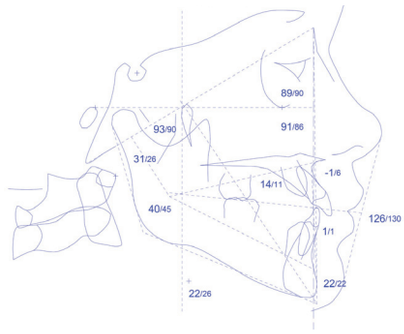

Figure 11: Post Phase I treatment lateral cephalometric analysis

instituting the appropriate treatment, is not consistent with a preventive philosophy.[26] Anterior, posterior, and vertical dentofacial discrepancies are all closely linked to growth. Consequently, interceptive and corrective orthodontic measures, as suggested by the American Association of Orthodontists, should be initiated, at the least, by age 7.[27]

The earlier the reestablishment of normal oropharyngeal function and nasal respiration, the more likely normal dentofacial development will be reinstituted.[16,28,29] Oral respiration may persist for a year or more after the airway has been restored while the original chronic mouth-breathing habit is "unlearned."[27]

This 6-year, 4-month-old female presented in my office with severe crowding, including impacted maxillary and mandibular canines. The ectopic maxillary first permanent molars were erupting into lingual crossbite (Figure 1). Clinically, this young patient had enlarged tonsils and was a mouth breather. The lateral cephalogram demonstrated significant adenoid enlargement (Figures 2 and 3). Cephalometric analysis revealed a skeletal lingual crossbite pattern and a severe skeletal Class III malocclusion due to both the maxilla and mandible (Figures 4 and 5). The growth analysis revealed the potential for excessive mandibular growth (Figures 6 and 7). The patient had a history of dysphagia, odynophagia, and sleep apnea. The patient was referred to an otolaryngologist who subsequently scheduled her for soft tissue ablation of the inferior turbinates and adenotonsillectomy.

The Phase I orthodontic treatment plan included initially banding the upper arch to relieve the severe crowding followed up with rapid maxillary expansion in the future. The lower arch would be banded during Phase I to gain space for the ectopic mandibular canines.

Following adenotonsillectomy and inferior turbinate ablation, her frequent

sore throats, mouth breathing and snoring resolved. The Phase I treatment significantly reduced the dental crowding and resolved the posterior crossbites (Figures 8-13).

The orthodontist is in a distinct position to promote positive airway development by influencing mid-face and maxillary development in those cases where it is deemed deficient. One should note that skeletal lingual crossbite patterns do not always reveal themselves with obvious posterior dental crossbites. It can be challenging to determine the presence of a skeletal lingual crossbite pattern when it appears that there is a normal transverse relationship between the upper and lower jaws without a frontal analysis. Many patients who appear to have a normal transverse skeletal relationship can have a skeletal lingual crossbite pattern,[30] negatively affecting orthodontic treatment outcomes and airway patency. Frontal cephalograms also provide the ability to evaluate the condition of the turbinates. The orthodontic patients we treat are three-dimensional. The routine use of frontal analyses on orthodontic cases adds that third dimension and can only enhance orthodontic diagnosis and airway evaluation; ultimately enhancing treatment outcomes. This case demonstrates how the appropriate orthodontic treatment, treatment timing, and referrals, based upon proper diagnostics, can improve the orthodontic outcome for the patient.

Orthodontists should always evaluate the potential for abnormal growth. When growth is not taken into account, an orthodontic case treated to proper balance at age 12 can become a failed result at maturity due to abnormal craniofacial growth, which can be directly associated with UAO.

Flanary studied the quality of life for children aged 2 through 16 suffering from upper airway obstruction secondary to adenotonsillar hypertrophy (UAO) and obstructive sleep apnea (OSA). The conclusion from this study was that the quality of life for these children does improve after adenotonsillectomy.[31] More recent studies on the improvement of the quality of life following adenotonsillectomy have had similar findings.[32]

The orthodontist is in a unique position to evaluate upper airway obstruction and abnormal craniofacial development. Early diagnosis and appropriate treatment is the key to helping these patients. OP

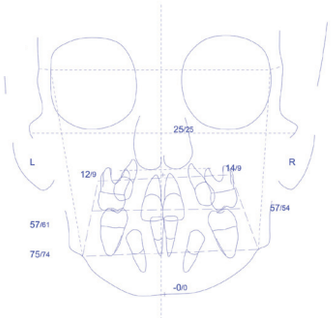

Figure 12: Post Phase I treatment frontal cephalometric analysis demonstrating improved skeletal lingual crossbite pattern

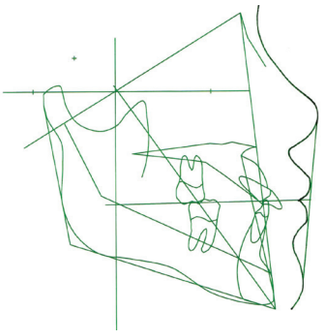

Figure 13: Post Phase I treatment Growth to Maturity illustrating improved growth forecast. Patient still exhibits strong lower jaw growth without additional orthodontic treatment, however, the patient doesn't develop an anterior crosbite

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