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.)

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.

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.

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1073-8746/16/1801-$30.00/0

http://dx.doi.org/10.1053/j.sodo.2016.05.009

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 Pruzansky[20] 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 Leighton[21] 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.

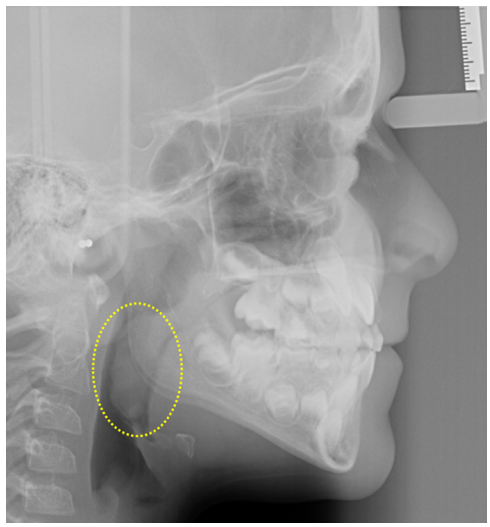

Figure 1. Lateral cephalometric radiograph of a 6-year-old boy. The circle in yellow denotes the hypertrophied tonsils almost blocking totally the pharyngeal airways.