The effects of enlarged adenoids on a developing malocclusion

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Introduction
The aims of this article are:
1) to highlight the skills and tools that assist the clinician in identifying upper airway obstruction;
2) to improve the diagnosis of adenoid hypertrophy; and
3) to improve the classification and treatment of associated malocclusions.

The methodology used in this literature analysis consists of a thorough review of narrowly tailored research and journal articles. The paradigm explored in each article involves upper airway obstruction, adenoid hypertrophy and malocclusion. The results and conclusions stemming from these articles generally fall into three categories:

1) That hypertrophied adenoids have a definite effect resulting in skeletal malocclusion.
2) That hypertrophied adenoids, coupled with other factors, may aid in the development of skeletal anomalies.
3) That adenoid hypertrophy has no effect on airway obstruction and malocclusion.

The research in this area is expansive, but largely inconsistent. Thus, the cause and effect relationship of adenoid hypertrophy and malocclusion must be carefully examined on a case by case basis. Regardless of the various researchers’ conclusions, one theory remains common — that airway obstruction caused by adenoid hypertrophy and malocclusion are related. The degree of that relationship and what it affects are still under debate. This paper attempts only to highlight the positive existence of this relationship and its possible effects regarding dentofacial growth and development.

Basic Facial Growth & Development
Developments in the understanding of human craniofacial growth have stemmed from histological and embryological studies, radiographic cephalometry, correlation of growth and facial anomalies analysis of surgical interventions, animal research and other science fields. Despite these studies, we are still waiting for a definite consensus regarding the controlling mechanism of craniofacial tissue.

Postnatal facial growth is influenced by genetic and environmental factors. Most facial growth and development occurs during the two childhood growth peaks. The first growth peak occurs during the change from primary to permanent dentition (between 5 and 10 years of age), and the second growth peak occurs between ten and fifteen years of age.

The study of the early years of life shows that by the age of four, 60 percent of the craniofacial skeleton has reached its adult size. By the age of twelve, 80 percent of facial growth has already occurred. By age seven the majority of the growth and development of the maxilla is complete, and by age nine the majority of the growth and development of the mandible is complete.

Proper facial growth is affected either positively or negatively, early in life, by the sequential occurrences of four major factors:
1) The cranial base must develop properly.
2) The naso-maxillary complex must grow down and forward from the cranial base.
3) The maxilla must develop in a linear and lateral fashion.
4) A patent airway must develop properly.

The relationship between the naso-maxillary complex and the cranial base is significant for aesthetic reasons and proper function. The naso-maxillary complex determines the relationship of the condyle to the glenoid fossae (in the temporal bone) resulting in multiple TMJ problems. An improper airway will affect the global individual growth.

The simultaneous growth of these factors is not nearly as significant as how these factors interrelate during facial growth and development. For example, the basic design of the face is established by a series of interrelated factorial developments.

Adenoidal Growth & Development
When adenoids are fully developed, the adenoidal mass increases, and the adenoid becomes more prominent, widening the pharynx. Enlarged adenoids are associated with other problems such as enlarged tonsils, posterior nasopharynx walls, and by age nine the majority of craniofacial growth has already occurred. This study of the early years of life shows that growth and development of the craniofacial complex (Fig. 1). Important motions such as chewing and swallowing depend largely on normal craniofacial development. Any restriction to the upper airway passage can cause nasal obstruction, possibly resulting in various dentofacial and skeletal alterations.

Upper respiratory obstruction often leads to mouth breathing (Fig. 2). Habitual mouth breathing may result in muscular and postural anomalies that may in turn cause dentofacial malocclusions. Hypertrophy of the adenoids, and palate tonsils, are one of the most frequent causes of upper respiratory obstruction (Fig. 3). Philosophers regarding the treatment of adenoid hypertrophy range from dietetic control and environmental modifications to dentofacial orthopedics, change of breathing exercises and surgical procedures.

Lymphoid tissue is normally present as part of the Waldeyer’s tonsillar ring in the form of a nasopharyngeal tonsil (Linder-Aronson 1970). The Waldeyer’s ring is the system of lymphoid tissue that surrounds the pharynx.

Bacteria may play a role in adenoid hyperplasia. Specifically, different pathogens, such as Haemophilus influenzae and Staphylococcus aureus, have been associated with lymphoid tissue hyperplasia. The adenoid lymphoid structures are lined with respiratory and non-respiratory epithelium that is normally distributed throughout the upper and posterior nasopharynx walls. During the presence of disease, the distribution of the ductritic cells (antigen presenting cells) is altered. The result is that there is an increase in dendritic cells in the crypts and extracellular areas, and a decrease in surface epithelium dendritic cells.

Lymphoid tissue is normally not apparent in the early infant stage of life. Marked symptoms of adenoid development are most common in the childhood age range of 2-12. During adolescence a decrease in adenoid size is noted as current with the growth of the nasopharynx. Rarely, adenoid tissue present

Abstract
This article reviews upper airway obstruction caused by hypertrophied adenoids and the possibilities of a subsequent malocclusion. Early diagnosis and treatment of pathological conditions that can lead to the obstruction of the upper airways is essential to anticipate and prevent alterations in dental arches, facial bones and muscle function. Correct nasal breathing facilitates normal growth and development of the craniofacial complex (Fig. 1). Important motions such as chewing and swallowing depend largely on normal craniofacial development. Any restriction to the upper airway passage can cause nasal obstruction, possibly resulting in various dentofacial and skeletal alterations.

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in adults, and when it is noted it is usually in an atrophic condition. The cause of the involution of the Waldeyer's ring is still under investigation.4,5 The imbalance in the relationship between the enlargement of the nasopharynx/nasopharyngeal airway and the concomitant growth of adenoid tissue can result in reduced patent nasopharyngeal airway and increased nasopharyngeal obstruction.6,7

The growth of adenoidal tissue as demonstrated by a bell curve peaks at or near age six and also begins involution at or near this age as well (Fig.6). Facial growth is coupled with adenoidal growth. As the cranial base forms the roof of the nasopharynx, a close examination of the growth and development of the craniofacial complex becomes significant for evaluation of the size and configuration of the nasopharyngeal airway. Any abnormal development regarding this craniofacial complex may affect the nasopharyngeal airway. Adenoidal adenoidal growth occurs during childhood may consume the nasopharynx complex may affect the nasopharyngeal airway. Any abnormal development regarding this craniofacial complex may affect the nasopharyngeal airway. Adenoidal growth is associated with adenoidal hyper trophy of the tonsils and adenoids, nasopharyngeal polyps and allergic rhinitis.5,6 However, for purposes of this paper the focus shall be on enlarged adenoids as the major contributing factor. There are numerous studies that link adenoid hypertrophy with nasopharyngeal airway obstruction to the development of skeletal and dental abnormalities.6

Airway obstruction, resulting from nasal cavity or pharynx blockage, leads to mouth breathing, which results in postural modifications such as open lips, lowered tongue position, anterior and posterior rotation of the mandible, and a change in head posture. These modifications take place in an effort to stabilize the airways. As previously discussed, facial structures are modified by postural alterations in soft tissue that produce changes in the equilibrium of pressure exerted on teeth and the facial bones (Fig.7). Additionally, during mouth breathing, muscle alterations affect mastraction, deglutition and phonation because other muscles are relied upon for support.6

Theoretically, many clinicians believe the blockage of their lower pulmonary compliance and reduced ventilation were the principle cause of airway obstruction and resulted in noticeable dentofacial changes.7

Contributing factors in the obstruction of upper airways include: anatomical airway constriction, developmental anomalies, macroglossia, enlarged tonsils and adenoids, nasopharyngeal polyplas and allergic rhinitis.8,9 However, for purposes of this paper the focus shall be on enlarged adenoids as the major contributing factor. There are numerous studies that link adenoid hypertrophy with nasopharyngeal airway obstruction to the development of skeletal and dental abnormalities.6

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Malocclusion: The Issue Still in Debate

There is a cause and effect relationship between adenoids, nasal obstruction and malocclusion? Dentofacial changes associated with nasal airway blockage have been described by C.Y. Tones in 1872 as adenoid facies. Tones coined this term based on his belief that enlarged adenoids were the principle cause of airway obstruction and resulted in noticeable dentofacial changes.7

Tones reported that children who were mouth breathers often exhibited narrow V-shaped dental arches (Fig. 8). This narrow jaw is a result of mouth breathers keeping their lips apart and their tongue position low. The imbalance between the tongue pressure and the muscles in the cheek results in cheek muscles compressing the alveolar process in the premolar region. Simultaneously, the lower jaw postures back. These simultaneous actions have been termed the compressor theory9 (Fig.9).

Tones’ views were supported in the 1950s by numerous leading orthodontists. These supporting clinicians reported airway obstruction as an important aetiological factor in malocclusion. Rubin advocated that in order for patients to fully be assessed they must be thoroughly evaluated by both a rhinologist and an orthodontist.3 Malocclusion is the departure from the normal relation of the teeth in the same dental arch or to teeth in the opposing arch.4

Airway obstruction coupled with loss of lingual and palatal pressure of the tongue produces alterations in the maxilla. The positioning of the tongue also plays an important role in mandibular development. The tongue displaced downward or forward can lead to a retrognathic mandible, and an interposed tongue can lead to anterior occlusal anomalies.6

Additionally, maxillary and mandibular changes can be viewed in the transverse direction, producing a narrow face and palate often linked with cross bite; in the anteroposterior direction, producing maxillary retrusion; and in the vertical direction, causing an increase in palatal inclination as related to the cranial base and excessive increases of the lower anterior face height.10

The most commonly found occlusal alterations are cross bite (posterior and/or anterior), open bite, increased overjet and retroclination of the maxillary and mandibular incisors.8,11 Maloney and Linder-Aronson’s findings were in agreement with the significant correlation between changed mode of breathing and diminished mandibular/palatal plane angle (ML/NA) found in adenotonsillarized children.12

Several authors have taken the position that adenoids are not consistently found to be associated with adenoids, mouth breathing, nor a particular type of malocclusion, and that there is no cause and effect relationship between adenoids, nasal obstruction/mouth breathing and malocclusion.

Proponents of this position believe that the V-shaped palate was inherited and not acquired through mouth breathing. Hartsook (1946) on a review of literature related to mouth breathing concluded that mouth breathing is not a primary etiological factor in malocclusion. Additionally, Whittaker (1911) found that in a study of 800 children who underw ent adenotonsillectomy or tonsillectomy, only 50% had dental abnormalities that needed orthodontic intervention.

There is some suggestion that adenoids and hypertrophic tonsils are a consequence of a thyroid hormone deficiency. This hormone deficiency acts as a catalyst for activating the organism’s defense mechanisms, which include hypertrophy of lymphoid tissue.11 Another orthodontic clinician, Vig, took the position that without documented tonsillectomy or adenoidectomy or other treatment to improve nasal respiration is empirically and difficult to justify from an orthodontic point of view.12

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5) Patient is asked to seal the lips, difficulty breathing through nose should be noted. One nos- tril can be occluded and the re- sponse noted to the same proce- dure on the other side. (Fig. 10)

The evaluation of nasal air- way patency is complicated, es- pecially when the possibility ex- ists that airways may clinically appear inadequate but be quite functional physiologically. Lip separation or an open-mouth habit is not an infallible indicator of mouth breating. Often com- plete nasal respiration is coupled with dental conditions that cause open-mouth posture.5)

Adenoid Evaluation
Nasopharyngeal space and the size of adenoids have been evaluated using different meth- ods.

1) Determination of the roent- genographic nasal index (a lateral cephalometric x-ray),
2) Flexible optic endoscopes (Fig. 11),
3) Acoustic rhinometry, and
4) Direct measurements during surgery.

Direct measurements are considered to be the most accu- rate because space can be as- sessed in three directions.2,12 A lat- eral cephalometric radiograph is an added valuable diagnostic tool for the orthodontist in the evalua- tion of children with upper air- way obstructions.14 (Fig. 12).

Treatment of Nasal Obstruction
1) Adenoidectomy with or with- out tonsillectomy is indicated if hypertrophied adenoids (and tonsils) are the cause of upper airway obstruction.3
2) Powered-Shaver Ade- noidectomy—Adenoidectomy coupled with Endoscopic Visua- lization will assist in achiev- ing adequate removal of ade- noids particularly high in the nasopharynx. Use of the pow- ered-shaver technique allows for better clearance of obstruc- tive adenoids. The end result is more reliable restoration of nasal patency.10
3) Septal surgery (rarely indi- cated in the child), but may be considered in the presence of a marked nasal septal deflection with impaction. Conservative septal surgery in growing pa- tients will not have an adverse effect in dentofacial growth.3,10,20
4) Maxillary expansion (RME or SAME)—an orthodontic pro- cedure that widens the nasal vault.17 (Fig. 15).
5) Cryosurgery or electro- surgery—this is a viable option for patients with vasovagal rhinitis.5
6) Bipolar Radiofrequency Abla- tion (allergic rhinitis)—per- formed under local anesthesia.
7) Use of nasal sprays.

Conclusion
The effect of adenoids on fa- cial expression, malocclusion and mode of breathing has been a topic of debate and investigation by practitioners in the field for the last one hundred years. A review of the literature exposes several theories.

A healthcare provider with a practice philosophy based on prevention of malocclusion de- velopment cannot ignore the early years of the patient’s growth cycle. By age twelve, 90 percent of craniofacial growth is complete, so treatment has occurred. This is the age when many practitioners begin ortho- dontic treatment.1 But this is the age when 85-90 percent of craniofacial growth has already oc- curred. This is the age when many practitioners begin ortho- dontic treatment.2

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Reference

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References