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 upper 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 embryologic 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 occur during the 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 nasso-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 nasso-maxillary complex and the cranial base is significant for aesthetic reasons and proper facial bone, muscle and soft tissue support. To allow proper downward and forward rotation of the mandible, the maxilla must be adequately developed in width, for acceptance of the mandible. Any limitation on mandibular rotation may affect the ability of the soft tissue to fill the glenoid fossae (in the temporal bone) result in multiple TMD 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. The nasso-maxillary complex is associated with the anterior cranial fossae. The posterior boundary of the maxilla determines the posterior limits of the midface. This structural plane is significant to facial and cranial development.

This system of tissue includes adenoids and pharyngeal tonsils, lateral pharyngeal tonsils, lateral pharyngeal bands, palatine tonsils and lingual tonsils (Fig. 5). Tonsils and adenoids have disparate embryonic origins and cytology even though they are both part of Waldeyer's ring. Bacteria may play a role in adenoid hyperplasia. Specifically, different pathogens, such as Haemophilus influenza and Staphylococcus aureus, have been associated with lymphoid tissue hyperplasia. The adenoid lymphoid structures are lined with a respiratory type epithelium that is normally distributed throughout the upper and posterior nasopharynx walls. During the presence of disease, the distribution of the dendritic cells (antigen presenting cells) is altered. The result is that there is an increase in dendritic cells in the crypts and extracryptal areas, and a decrease in surface epithelial 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.

This system of tissue includes adenoids and pharyngeal tonsils, lateral pharyngeal tonsils, lateral pharyngeal bands, palatine tonsils and lingual tonsils. 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 motor functions such as chewing and swallowing depend largely on normal craniofacial development. Any restriction to the upper airway passages 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 palatine tonsils, are one of the most frequent causes of upper respiratory obstruction (Fig. 5). Philosophers regarding the treatment of adenoid hypertrophy range from dietary control and environmental modifications to dentofacial orthopedics, change of breathing exercises and surgical procedures.

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 motor functions such as chewing and swallowing depend largely on normal craniofacial development. Any restriction to the upper airway passages 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 palatine tonsils, are one of the most frequent causes of upper respiratory obstruction (Fig. 5). Philosophers regarding the treatment of adenoid hypertrophy range from dietary control and environmental modifications to dentofacial orthopedics, change of breathing exercises and surgical procedures.