The effects of enlarged adenoids on developing malocclusion

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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 musculoskeletal and postural anomalies, which may in turn cause dentoskeletal malocclusions (Fig. 3). Hypertrophy of the adenoids, and palate tonnls, are one of the most frequent causes of upper respiratory obstruction (Fig. 4). Philosophies regarding the treatment of adenoid hypertrophy range from dietary considerations to dentofacial orthopadies, change of breathing exercises, and surgical procedures.
from these articles generally fall into three categories:

- That hypertrophied adenoids have a definitive effect resulting in skeletal malocclusion
- That hypertrophied adenoids, coupled with other factors, may aid in the development of skeletal anomalies
- 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 is 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 and 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 two childhood growth peaks. The first growth peak occurs during the change from primary to permanent dentition (between five and 10 years of age) and the second growth peak occurs between 10 and 15 years of age. The study of the early years of life shows that by the age of four, 60 per cent of the craniofacial skeleton has reached its adult size. By the age of 12, 90 per cent 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:

- The cranial base must develop properly
- The naso-maxillary complex must grow down and forward from the cranial base
- The maxilla must develop in a linear and lateral fashion
- A patent airway must develop properly.

The relationship between the naso-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 relationship of the condyle to the glenoid fossae (in the temporal bone) resulting in multiple TMI 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 naso-maxillary
complex is associated with the anterior cranial fossae. The posterior boundary of the maxilla determines the posterior limits of the midface. This structural plan is beneficial to facial and cranial development. The basic structural format of facial growth and development is dependent on, and governed by, the interrelation of multiple functions and morphological aspects. Functional matrices include a phenomenon of bone displacement, the maxillary forward and downward movement equating mandibular growth upward and downward. The displacement and growth phenomenon is responsible for the spatial relationship necessary for functional joint movement resulting in the final result of facial growth. Additionally, morphological adaptations affect dentoosseous development. The integration of the musculoskeletal system affects respiration, masti- cation, deglutition, and speech.

This basic understanding of facial growth and development is relevant as adenoidal tissue enlargement coincides with major facial growth, for example, they occur simultaneously. Facial growth may be restricted by abnormal development of adenoidal tissue resulting in abnormal swallowing and breathing patterns (Fig. 5).

Adenoidal growth and development

Adenoidal 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. This system of tissue includes adenoids and pharyngeal tonsils; lateral pharyngeal tonsils; lateral pharyngeal bands; palate tonsils and lingual tonsils (Fig. 6). 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 ciliated respiratory epithelium which is normally distributed throughout the upper and posterior nasopharyngeal 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 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 two to 12. During adolescence, a decrease in adenoid size is noted as current with the growth of the nasopharynx. Rarely is adenoid tissue present 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. 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. The growth of adenoidal tissue as demonstrated by a bell curve, peaks at near age six and also begins involution at or near this age (Fig. 7). 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. Abnormal adenoidal growth that occurs during childhood, may consume the nasopharnx and extend through the posterior choanae in the nose. This excessive adenoidal growth usually interferes with normal facial growth and can result in abnormal breathing patterns, congestion, snoring, mouth breathing, sleep apnea. Eustachian tube dysfunction/obits media, rhinosis, nasal obstruction, abnormal swellings, swallowing problems, reduced ability to smell and taste, and speech problems. Theoretically, many clinicians believe the blockage should be removed as soon as possible through a surgical procedure called adenoidectomy. However, according to a study conducted by Havas and Longwiner one-third of child study participants, with traditional adenoidectomies, were ineffective with intranasal extensions of the adenoids obstructing the posterior choanae. For this segment of the study population the “powdered-shaver adenoidectomy” was effective in the complete removal of the obstructive adenoid tissue ensuring postural patency.

Upper airway obstruction and mouth breathing

During normal nasal respiration, the nose filters, warms and humidifies the air in preparation for its entry into the body’s lungs and bronchi. This nasal airway also provides a degree of nasal resistance in order to assist the movements of the diaphragm and intercostals muscles by creating a negative intrathoracic pressure. This intrathoracic pressure promotes airflow into the alveoli.

Correct normal resistance is 2–5 cm H2O/L-sec and results in high tracheobronchial airflow which enhances the oxygenation of the most peripheral pulmonary alveoli. In contrast, mouth breathing causes a lower velocity of incoming air and eliminates nasal resistance. Low pulmonary compliance results. According to blood gas studies, mouth breathers have 20 per cent higher partial pressure of carbon dioxide and 20 per cent lower partial pressures of oxygen in the blood, linked to their lower pulmonary compliance and reduced velocity.

Contributing factors in the obstruction of upper airways include: anatomical airway constriction, developmental anomalies, macrognathia, enlarged tonsils and adenoids, nasal polyps and allergic rhinitis. 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.

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 inferior rotation of the mandible, and a change in head posture. These modifications take place in an effort to stabilize the airway. 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. 8). Additionally, during mouth breathing, muscle alterations affect mastication, deglutition and posture since these other muscles are relied upon.

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**Clinical**

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Malocclusion—The issue still in debate

Dental changes associated with nasal airway blockage have been described by CV Tomes in 1872 as adenoid facies. Tomes commented based on his belief that enlarged adenoids were the principle cause of airway obstruction and resulted in noticeable dental changes.17 Tomes reported that children, who were mouth breathers, often exhibited narrow V-shaped dental arches16 (Fig. 9). This narrow jaw is a result of mouth breathers keeping their lips apart and their tongue position low. The imbalance between the tongue and the muscles in the cheek, result in cheek muscles compressing the alveolar process in the presellar region. Simultaneously, the lower jaw positions back (Fig. 10). These observations have been termed the compensatory theory.18

Tomes’s views were supported in the 1950s by numerous leading orthodontists. These supporting clinicians reported airway obstruction as an important etiologic agent in maloclusion. Rubin advocated that in order for these patients to fully be assessed they must be thoroughly evaluated by both a rhinologist and orthodontist.19 Malocclusion is the departure from the normal relationships of the teeth in the same dental arch or teeth in the opposing arch.20

Airway obstruction, coupled with loss of lingual and palatal pressure of the tongue, produces alterations in the maxilla. The position of the tongue also plays an important role in mandibular development. The tongue placed downward can lead to a retrognathic mandible; and an interproximal space can lead to anterior occlusal anomalies.21

Addititionally, maxillary 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. The most commonly found occlusal alterations are cross bite (posterior and/or anterior), open bite, increased overjet, and retroclina-

and Linder-Kroning’s findings were in agreement with the significant correlation between changed mode of breathing and diminished mandibular/palatal plane angle (ML/NA) found in adenotomised children.22

Several authors have taken the position that all lateral incisors 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 etiologic factor in malocclusion. Additionally, Whitaker (1911) found that in a study of 800 children, who underwent adenoidectomy or tonsillectomy only 30 per cent had dental anomalies 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.13 Another orthodontic clinician, Vig, took the position that without documented total nasal obstruction, any surgery or other treatment to improve nasal respiration is empirical and difficult to justify from an orthodontic point of view.17,18

Nasal respiratory evaluation

The relationship of airway obstruction and dentofacial structures/malocclusion is still the subject of investigation and controversy amongst orthodontists. The correlation between functional problems and morphologic characteristics is yet to be solidified. Regardless of varied opinion in this area practitioners should observe each patient carefully.

Suggested protocol

As the patient enters the room, facial and head posture should be noted to see if the lips are closed during respiration. Signs of allergic rhinitis should be noted, as well as histories of frequent colds or sinusitis. Assessment of family history for allergies is important. Sleep history should be evaluated: sleep apnoea, loud snoring, open-mouth posture while asleep.

Patient is asked to seal their lips — difficulty breathing through nose should be noted. One nostril can be occluded and the response noted – same procedure on the other side. (Fig. 11)

The evaluation of nasal airway patency is complicated, especially when the possibility exists that airways may clinically appear inadequate but quite functional physiologically. Lip separating or an open-mouth habit is not an infallible indicator of mouth breathing. Often complete nasal respiration is coupled with dental conditions that cause open-mouth posture.22

Adenoid evaluation

Nasopharyngeal space and the size of adenoids have been evaluated using different methods of assessment:

1. Determination of the roentgenographic adenoid/nasopharyngeal ratio (a lateral cephalometric x ray)23
2. Flexible optic endoscopes (Fig. 12)
3. Acoustic rhinometry24
4. Direct measurements during surgery.

Direct measurements are considered to be the most accurate because space can be assessed in three directions.24 A lateral cephalometric radiograph is an added valuable diagnostic tool for the orthodontist in the evaluation of children with upper airway obstructions16 (Fig. 15).

Treatment of nasal obstruction

Adenoidectomy with or without tonsillectomy is indicated if hypertrophied adenoids (and tonsils) are the cause of upper airway obstruction.25

Powered-Shaver Adenoidectomy — Adenoidectomy coupled with Endoscopic evaluation will assist in achieving adequate removal of adenoids particularly high in the nasopharynx. Use of the powered-shaver technique allows for better clearance of obstructive adenoids.

The end result is more reliable restoration of nasal patency.19 Septal surgery (usually indicated in the child) but may be considered in the presence of a marked nasal septal deflection with impaction. Conservative septal surgery in growing patients will not have an adverse effect in dentofacial growth.19,24,26,27

– Maxillary expansion (RME or SAMx) — an orthodontic procedure that widens the nasal vault14 (Fig. 14).

– Cryosurgery or electrosurgery — this is a viable option for patients with vasomotor rhinitis.17

– Bipolar Radiofrequency Ablation (allergic rhinitis) — performed under local anesthetic

– Inferior turbinectomy

– Adenoidectomy

– Cryopexy

– Inferior turbinate resection

– Inferior turbinoplasty

– Inferior turbinate REM

– Caldwell-Luc

– Rhinoplasty

Conclusion

The effect of adenoids on facial 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 development, cannot ignore the early years of the patient’s growth cycle. By age twelve, 90 per cent of facial growth has already occurred. This is the age when many practitioners begin orthodontic treatment.17 This is the age when 80–90 per cent of cranial midfacial growth is complete, so most formation and/or deformation has occurred.28 To wait until 90 per cent of the abnormality has occurred, before beginning treatment, is not consistent with a preventive philosophy. Interceptive measures must be initiated sooner. Early intervention requires an acceptance of a multidisciplinary approach to total patient health. An integrated approach to patient evaluation, diagnosis and treatment is most effective. Primary care physicians, dentists, allergists, otolaryngologists, and orthodontists must all work together for early prevention and management of young patients with increased nasal airway resistance.

After diagnosis, a comprehensive risk benefit analysis regarding early intervention must be considered. Although hereditary and environmental factors must be considered, the univer-

References

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