Upper airway obstruction in the pediatric dental patient

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This article presents clinical observations as well as a review of the literature regarding upper airway obstruction in the pediatric dental patient. Dentists will derive enhanced skills to identify the various types of upper airway obstruction and provide appropriate recommendations for intervention or referral to a physician to minimize detrimental effects on dental development.

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Anatomical airway constriction, developmental anomalies, macroglossia, enlarged tonsils and adenoids, nasal polyps, and allergic rhinitis all may contribute to upper airway obstruction. Developmental, environmental, and hereditary factors are influential in skeletal growth of the face. Modified facial growth results from unbalanced muscle activity, with the application of negative pressure to the pharyngeal airway. The height of the face below the anterior nasal spine may increase along with the angle of the mandible, resulting in a longer and narrower face; this appearance may be referred to as *long-face syndrome*.[1] Facial appearance may reflect adenoid facies, as indicated by obstruction with narrow cheeks, pinched nostrils, shadows beneath the eyes, open mouth posture, and a dull expression (Fig. 1).[2]

A pediatric patient's tongue is disproportionately large. In combination with a retrognathic mandible and higher floor of the mouth, a size/space discrepancy may result, leading to a habitual anterior tongue position that may be associated with a dental open bite (Fig. 2). The structural effects of macroglossia are especially apparent in children with Down syndrome. Narrow arches and a high vaulted palate cause the oral cavity to diminish in size, resulting in anterior positioning of the tongue.[3]

When tongue posture is normal, the tongue rests on the hard palate. As the tongue is displaced downward, the support for the lingual surfaces of the maxillary molars is lost, as is the counterbalance for the buccinator muscles. Flaccid lips decrease the labial support of the maxillary anterior teeth. These muscle/soft tissue imbalances and postural relationships may result in a posterior cross bite, an anterior open bite, and protrusion of the maxillary incisors (Fig. 3). The lips separate in response to the interruption of air flow and the tongue moves forward and downward as activity of the genioglossus and mylohyoid muscles increases. It appears that this anterior repositioning of the tongue occurs in an effort to open the oropharynx for improved respiration.[4]

In the author's experience, a habitual anterior tongue position often results from efforts to open the pharyngeal airway, particularly when the palatine (or faucial) tonsils are grossly enlarged (Fig. 4); tongue-thrust swallowing and an anterior open bite also may be present. Although they often occur simultaneously, a cause-and-effect relationship between tongue-thrust swallowing and anterior open bite has not been established.

An anterior open bite may result from numerous entities, including parafunctional sucking habits and skeletal growth. To determine if aberrant skeletal growth has created the open bite, a cephalometric radiograph is required. An overextended crib may be used to reposition the tongue and train it to a more appropriate position in the oral cavity if the open bite is determined to be dental (Fig. 5). If the anterior positioning of the tongue is deemed to be necessary for adequate pharyngeal air flow, the size and influence of the palatine tonsils should be evaluated prior to use of this appliance or any other intervention.

Hypertrophied palatine tonsils have not been associated with skeletal growth changes, although they can cause significant airway blockage during restorative procedures. This is of particular importance during in-office sedation: protective responses are assumed to be intact during conscious sedation and one would expect a child to adjust his or her head if airway patency is compromised. The deeply sedated child with enlarged tonsillar tissue is at risk for obstruction if his or her chin is rolled forward and touches the chest, as the protective reflexes may not be intact. When performing restorative procedures in the mandible, the dentist may place the child's head in this position inadvertantly.[5] For mild difficulty with nasal breathing, a nasal dilator may be placed to enhance air exchange during use of the rubber dam (Fig. 6).[6]

The pharyngeal tonsils, also referred to as the *adenoids*, are located just posterior to the nasal cavities in the nasopharyngeal area. Rapid, disproportionate growth of the adenoids may obstruct the flow of air. The adenoid tissue may contact the soft palate (either directly or approximately), blocking air flow from the nasal passages to the pharynx. A lateral cephalometric radiograph may be utilized to determine the size of the adenoid tissue relative to the size of the nasopharyngeal dimension. The hypertrophy also may be caused by allergy, infection, or enlargement of the nasal turbinates. In addition to the possibility of altering facial growth, enlarged adenoids may create problems with speech, swallowing, or auditory tube function.[7] The size of the adenoids decreases with age through a process called *involution*. Involution seems to reverse at puberty; after puberty, the adenoids continue to shrink.[8]

Nasal polyps result from chronically inflamed nasal mucosa and originate from the maxillary and ethmoid sinus ostia, near the maxillary turbinates. Clinically, they may appear as pedunculated, shiny, gray, grape-like masses, filling the space between the nasal turbinates and septum. Their gray color can be used to

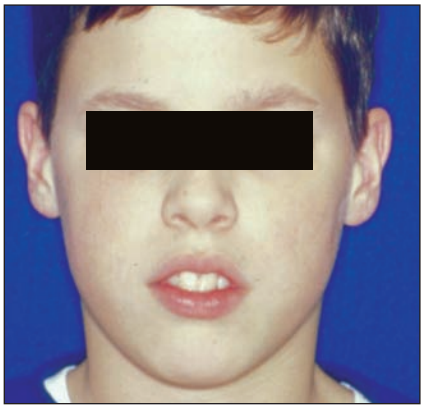

Fig. 1. A patient with adenoid facies.

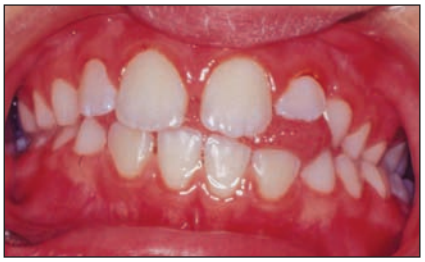

Fig. 2. Habitual anterior tongue with open bite.

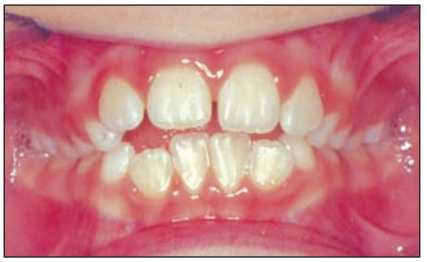

Fig. 3. Constriction of the maxillary arch due to improper tongue position.

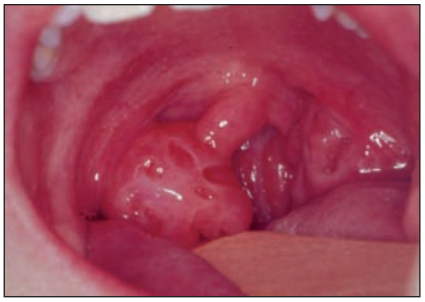

Fig. 4. A case of enlarged palatine tonsils.

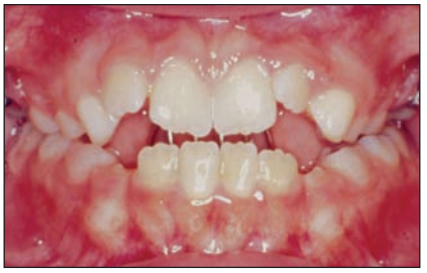

Fig. 5. An overextended crib used to treat an anterior open bite.

distinguish them from the turbinates, which are well-vascularized and pink in color. The nasal airway also may be smaller due to the normal growth pattern of children between 9 and 13 years of age, a period when the nasal airway size decreases.[9]

Relief measures for airway obstruction

Until natural involution of the adenoidal tissues occurs, pharmacological intervention and environmental controls may offer adequate relief for obstruction caused by allergies. Dust often is responsible for allergies in children. Reduction of fomites can begin in a child's bedroom. Cotton blankets that can be laundered and non- or hypo-allergic pillows can be used in place of down comforters and pillows; some mattress and pillow covers specifically designed for allergen reduction also are available. Allergists often recommend removing carpet, draperies, and stuffed animals. Dusting with a damp cloth rather than a dry one is helpful. Doctors also may prescribe allergy shots/immunotherapy, antihistamines, leukotriene inhibitors, or steroids. Nasal beclomethasone has been successful in reducing adenoidal hypertrophy in children aged 5–11.[10] According to a 2003 study, performing surgical intervention (involving an adenoidectomy) on children 1.5–4.0 years of age has little effect on vertical growth patterns of the craniofacial complex.[11] However, malocclusion may improve as the airway improves.[12]

Additional information regarding the possibility of upper airway obstruction can be obtained by questioning the child and his or her parents regarding mouthbreathing, snoring, incidence of infection, and sleep patterns. Mouthbreathing may occur to compensate for obstruction of the nasal airways. Before air enters the lungs, it is cleansed, warmed, and humidified by the nasal passages. Pollen, bacteria, and foreign particles are not screened when this important function is bypassed and the possibility of infection increases as a result. In addition, dry air carries less oxygen than moist air, diminishing the amount of oxygen carried to the lungs.

Nasal air flow may be evaluated by closing one nostril manually, placing a mouth mirror just beneath the opposite nostril, and breathing; the mirror should fog with expiration if air flow is normal. Air flow alternates between nostrils normally but simultaneous cessation of air flow should not occur. Before any validity is assigned to this test, the presence of temporary congestion due to allergy or infection should be determined.

Both skeletal and dental relationships may be affected by mouthbreathing. In a 1983 study by Bresolin et al, chronic allergic mouthbreathers had significantly greater maxillary and total anterior facial height and overjet; increased gonial angles, higher palatal vaults, and posterior crossbite also were noted.[13]

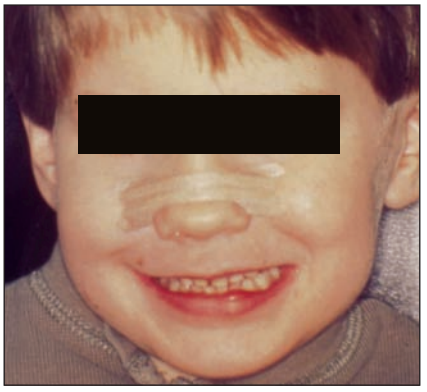

Fig. 6. A patient with a nasal dilator strip.

Sleep apnea

In 1999, Lofstrand-Tidestrom et al examined 4-year-old children with a positive history of snoring every night.[14] These children were found to be mouthbreathers, with night-time breathing disturbances, high-vaulted palates, shorter mandibular dental arches, and maxillary arch constriction.[14] Of the children with obstructive sleep apnea (OSA), 75–100% of them snore, which is considered to be the hallmark among children.[15] Delayed growth and development, palatal constriction, mandibular retrusion, and hyponasal voice may be observed in patients with sleep apnea. Parents may report hyperactivity, noisy breathing/

snoring, poor concentration skills, multiple upper airway infections, otitis media, nightmares/night terrors, headaches, and rhinitis.

Based on the pathophysiology and clinical appearance, sleep apnea is classified into three types. *Central apnea* originates in the brain. Respiratory effort is absent due to lack of an excitatory impulse from the brainstem to the respiratory muscles. An episode lasting longer than 20 seconds is considered to be pathologic. *Obstructive apnea* is experienced more often by older children. It is caused by nasal polyps, enlarged adenoids or tonsils, macroglossia, subglottic stenosis, or laryngomalacia. *Mixed apnea* is a combination of both central and obstructive apnea. OSA may result in hyperextension of the neck while sleeping, diaphoresis, enuresis, and even death.[16]

A mnemonic tool that may be helpful when gathering a patient history is the word *BEARS*, an acronym for *B*edtime problems, *E*xcessive daytime sleepiness, *A*wakenings at night, *R*egularity and duration of sleep, and *S*noring. Many parents also make a sleep audio or videotape.

Polysomnography (PSG), which measures a number of parameters, provides the most reliable results but it must be performed by a trained technician. The main measurement is the frequency of the respiratory events per hour that relate to apnea and hypopnea. Modalities that may be used at home include pulse oximetry, a chest wall motion detector, a nasal air flow meter, and a heart rate monitor. Epidemiological studies report that up to 2% of all children have sleep apnea syndrome. These studies have defined three categories of morbidity: neurobehavioral, cardiovascular, and somatic growth.[15] The dentist may identify failure to thrive due to increased metabolic expenditure, resulting from efforts to breathe during sleep. Due to the serious consequences of obstruction in a sedated child, any child at risk for obstructive sleep disorder who requires sedation for treatment should be treated in a hospital setting.

A 2002 study reported that when rapid maxillary expansion was performed either with an acrylic bonded appliance or with surgical assistance, the width of the nasal floor and lateral movement of the outer walls of the nasal cavity both increased, resulting in nasal resistance decreasing; subjects who used an appliance for rapid expansion showed the same dimensional changes as older subjects who were assisted surgically.[17] Improving the dimensions of the nasomaxillary structures and the nasopharyngeal airway with rapid maxillary expansion also has reduced or eliminated nocturnal enuresis.[18,19] Functional appliances designed to reposition the mandible forward may offer a benefit by increasing the oropharyngeal airway.[20,21]

It must be remembered that adaptive behavior and morphologic changes vary considerably within individuals and that the cause-and-effect relationship for each of these options may affect each child differently. Because adaptive postural changes established much earlier for the purpose of resolving an airway deficiency may be responsible for morphologic facial changes, a reliable evaluation of the airway must be made before any intervention can take place. Measurements of nasal patency may be considered reliable for patients with a remarkable history of ear, nose, throat, or lung disease, provided the measurements are obtained during an asymptomatic time.[22] When upper airway obstruction appears to be present, consultation with primary care provider and/or referral to a specialist is recommended.

Summary

Without consistent findings, questions persist regarding how the various entities of upper airway obstruction contribute to malocclusion and the growth of the craniofacial complex. Continued research with nasal-oral examinations, rhinomanometry, and cephalometric studies during growth are necessary to gain more information regarding upper airway obstruction and its effect on malocclusion and facial growth. Facial growth and the developing occlusion should be monitored to identify the possible influential aspects of a child's airway. As the ability to diagnose and intervene advances, dentists may be able to improve the quality of life significantly for many children.

Author information

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