

Editor's Note:

Human beings are obligate nasal breathers. Should the nasal respiratory mechanism malfunction, the human organism is capable of employing a compensatory oral respiratory mechanism. This month, Allen J. Moses reviews obstructive breathing conditions, both nasal and oral, which pose important diagnostic criteria upon interceptive and corrective treatment judgments for the dentist.

Moses describes an appliance termed a "passivator" which supports the mandible in an anterior and slightly open bite yet neither allows nor causes undesirable orthodontic tooth movements. This appliance is reported to support the patient's mandible during sleep, prevent collapse of the pharyngeal musculature, allow greater volume of space for tongue activity and prevent incorrect swallowing and snoring.

Moses feels that dentists must share a common interest with physicians of the nose and throat while developing an expertise in measurement, diagnosis and treatment of neuromuscular disorders which may affect the stomatognathic system. By understanding the role of breathing problems in oral, facial and dental development, dentists can play an equally important role in preventing many of these problems.

Moses completed his BS degree in 1964 at the University of Illinois and in 1968 received his DDS degree from the University of Illinois College of Dentistry. He has a special interest in temporomandibular joint problems and their effects on the rest of the body. He has taught at the U of I College of Dentistry and lectured at the National College of Chiropractic Medicine. He has published in various journals including the CDS Review. Moses is a Fellow in the International College of Craniomandibular Orthopedics. He currently maintains a private practice of general dentistry in Arlington Heights, Illinois.

In a previous article, "Thumb Sucking or Thumb Propping?" (CDS Review, December 1987), the differentiation was made that thumb suckers are nose breathers and thumb proppers are mouth breathers. In proppers, the thumb is an orthopedic appliance to keep the lips apart, mouth open and facilitate mouth breathing in these nasally obstructed children.

The deleterious consequences of propping may be narrow V-shaped upper arch, low tongue position, abnormal swallow, distal jaw position and forward head

posture. It is no coincidence that these signs characterize many TMJ patients. Understanding the distinction between sucking and propping and timely referral presents an ideal interceptive situation. The purpose here is to elaborate on the subject of breathing and its effect on the types of neuromuscular problems dentists commonly treat and what dentists can do to affect their patients' breathing.

Nasal function/dysfunction

The nose is an incredible organ. In addition to its olfactory function, it is the carburetor of the body. The nose provides for proper humidification, filtration and warming of inspired air.

The nasal pulmonary reflex controls the volume of inspired air. Additionally the nose has a role in immunity, voice resonance and certain hormonal responses. Human beings are normally nasal breathers.¹

There are numerous pathological conditions and abnormalities which can obstruct nasal breathing the role of the backup breathing organ, but the air is not properly filtered, humidified and warmed. This results in constriction of bronchi and depressed elasticity of the lungs. With less tidal volume there follows decreased oxygenation and increased partial pressure of carbon dioxide in the blood in mouth breathers.²

Some of the effects of impaired nasal respiration reported in the literature are: Abnormal facial development, headaches, malocclusion, poor sleep, sore throats, excessive daytime sleepiness, dry mouth, frequent infections, earaches and sinus problems.³

At this point, one might think of the breathing possibilities as nose and/or mouth, but one other possibility exists - no breathing. The condition known as apnea is very serious. The causes and consequences will be discussed.

Animal experiments

Egil Harvold did some interesting experiments on rhesus monkeys to examine the consequences of mouth breathing. The monkey, like humans and most animals, is a nose breather. Harvold asked the question, "What changes would occur if the primate were forced to utilize 100 percent oral respiration?" To achieve this, he obstructed the nasal passages with silastic button plugs permanently sewn in place. No nasal inspiration was possible and only a small amount of nasal expiration could occur.⁴

The experimental animals were kept in this condition for three years. The jaws gradually lowered in rest positions and the tongue position also lowered to establish an oral airway. The shape of the tongue changed and became long and

narrow. The total volume of the tongue, however, did not change. The palate collapsed laterally and the canines went into crossbite. The size of the mandible remained the same, but its habitual position relative to the skull changed.

Harvold clearly shows that oral respiration in monkeys alters neuromuscular activity and causes changes in bone resulting in malocclusion.⁵

In another experiment, Harvold reduced the oral space by displacing the tongue downward against the mandible. He did this by sewing objects in the posterior palatal vault of the monkeys which simulated the effect of tonsils. These objects did not interfere with the occlusion or mastication.

In time, changes in the shape and position of the tongue were noted as the tongue tried to avoid the sensory interference. Indentations formed in the back of the tongue which were not sores. Sensory input altered the shape of the tongue and the following skeletal adaptation occurred: Change in mandibular positions and shape, extrusion of teeth, open bites, increased face height, opening of the gonial angle and steep mandibular plane angle.⁶

Harvold further points out that there are two types of movement involved – rhythmic, such as respiration and tonic, which involves changes in posture. Changes in rhythmic movement cause few skeletal variations but tonic changes cause significant alterations in skeletal morphology.⁷ The muscles with which the dental clinician is primarily concerned, are those involved in tonic changes, such as the diaphragm, pterygoids, masseters and temporalis.

Harvold's controlled experiments confirm and clarify many of our clinical observations. Sten Linder-Aronsen's study of human children establishes convincing statistical correlation that conditions which can obstruct nasal air flow and cause oral respiration may cause deviations in craniofacial growth, particularly increased face height.⁸

Human breathing problems

Several of the more common causes of nasal obstructive breathing in human beings are: Allergy, small external nares, enlarged tonsils and adenoids, vasomotor rhinitis, deviated septum, trauma, enlarged turbinates, congenital abnormalities and nasal polyps.⁹

As a referring doctor, one has to decide between allergist, ENT, pediatrician or sleep lab. Referrals should always be for an evaluation by the specialist of choice, accompanied by a note of one's findings. Thomas Weimert, professor of Otorhinolaryngology at the University of Michigan, found in a five-year study of 1,360 patients referred to him by orthodontists for suspicion of airway compromise that 28 percent had no evidence of significant obstruction.¹⁰

Dentists are important as both referrers and treating doctors. As treating doctors, early is best. At age 2-3, when one sees the finger habit, the allergic shiners, the large tonsils, the mouth breathing, etc., that is the time to begin. At age 4, 60 percent of the facial growth is complete.¹¹ At age 7, 70 percent of facial growth is complete. At age 12, 90 percent of craniofacial growth is complete.

Both structural and functional normalizations must be achieved to get ideal growth patterns. This is not always possible if the growth potential is lost.

Children do not begin to complain of nasal obstructive problems until approximately age 16.¹² It seems children have no frame of reference from which to complain as this is the only way they know how to live. At about age 16, they start to realize that this kind of condition is not normal.

Allergy is by far the most common and important cause of nasal obstruction. The clinical signs of allergy which one may note are nasal salute, nasal crease (turned up nose), allergic shiners, upper lip incompetence, marginal eyelid eczema, nonfebrile pharyngitis (from mouth breathing), lymphoid nodules in the throat and blue-gray color to nasal mucosa.¹³

Allergy must be screened out first. If one parent or sibling is allergic, the child has a 40 percent chance of being allergic. If two parents are allergic, the child has a 70 percent chance of being allergic.¹⁴ Allergy is an example of a normal protective mechanism gone awry – an exaggerated response to proteins and large polypeptide molecules which ordinarily do not warrant such an extreme response. The conventional treatment of allergies includes antihistamines, desensitization, avoidance and prevention.

Probably the most important thing a concerned dentist can do to help prevent allergy is to counsel and educate mothers on the importance of breast-feeding.¹⁶ If mothers must bottle-feed, the dental profession can begin the education process by recommending use of soy formulas which are not as allergenic.

Human infants do not produce immunoglobulins of their own for the first three months of life. Transplacental transfer from mother and breast milk are the two sources. Children not breast fed do not get this immunity and, as such, are more susceptible to childhood illnesses. According to a six-year study by Glaser, bottle fed children have four times as many respiratory conditions as breast fed children.¹⁷

Measurement and diagnosis

Dentists have some very important tools to aid in the diagnosis of nasal breathing problems. The top two photographs (page at right) are of panoramic radiographs. The patient in Figure 1 has a patent nasal airway and the one in Figure 2 has an obstructed nasal area. The bottom two photos, Figure 3 and Figure 4, are

cephalometric radiographs in which the presence of enlarged adenoids can be noted in the nasopharynx. (Radiographs courtesy of Dr. Dan Schwarb, Mt. Clemens, Michigan)

It is enlightening and gratifying to deal with ENTs in today's technological environment. Dentistry prides itself on the refinement of diagnostic skills by the newer electronic means, such as the electromyograph and computerized mandibular scanning. Otorhinolaryngologists have equally sophisticated measuring tools available to them, such as rhinomanometry and polysomnography to determine the validity of their treatment and provide documentation.

Rhinomanometry is a reproducible means of measuring nasal airway resistance. The University of North Carolina has developed SNORT, an acronym which stands for Simultaneous Nasal Oral Respirometric Technique. Using a custom fitted face mask with separate valves attached to the nose and mouth and attached to a flow meter, air pressure transducer, recorder and computer, it can give the nasal versus the oral inspiration, expiration and their ratios.¹⁸

Jerold Principato has established age-related norms of nasal resistance using an anterior rhinomanometric technique. Normal nasal resistance for an adult is 2.5 to 3.5 cm of water per liter per second. At readings above 7.0 at rest, an individual must go to mouth breathing to get adequate air. Between 4.0 and 7.0, orthodontic intervention is the treatment of choice. Above 7.0, surgery is usually considered.¹⁹

After allergy, the second most common cause of nasal obstruction is adenoid hypertrophy.²⁰ Swollen adenoids block the distal part of the nasal airway, thus decreasing nasal patency by increasing the resistance to airflow. Mouth breathing is the result, often accompanied by a lower tongue position, extended head position, lower mandibular posture and narrowed upper arch. This used to be called adenoid facies but the more popular term today is long face syndrome (LFS).

Accompanying hypertrophied adenoids often are enlarged lateral pharyngeal tonsils. These tissue enlargements situated in the airway also alter air flow resistance. Enlarged turbinates is another common cause of nasal airflow obstruction. Chronic rhinitis is the result of thickening of the tissues lining the inferior turbinates, particularly at the posterior end. The increased mucus production causes increased pharyngeal symptoms and the need to constantly clear the throat, better known as postnasal drip.²¹ Children who have hypertrophied adenoids, tonsils and inferior turbinates develop long face syndrome 30 percent of the time. In contrast, children with normal respiratory airways develop long face syndrome 2 percent of the time.²²

Harry Richter, professor of otorhinolaryngology at University of Michigan, feels that the safety of nasal surgery has been well established, and that a clear nasal airway is a prerequisite for developing normal facial form. He writes, that in Current Therapy in Otolaryngology and Head and Neck Surgery, the authors reviewing indications for nasal surgery only allude to a relationship between adenoid hypertrophy and dentofacial development, but they do not yet feel that it is a proper indication for surgery. Quality of life considerations allow for surgical treatment of these patients on a selected basis.²³

It is particularly the quality of life criteria that is making the ENTs and dentists increasingly aware of the existence and importance of sleep apnea.

Apnea literally means want of breath. It can be of central nervous system origin or obstructive. Obstructive sleep apnea is a collapse of the upper airway causing total obstruction of breathing. Episodes usually over 10 seconds long with over 30 per night and over seven per hour cause chronic oxygen deprivation resulting in symptoms such as morning headaches, intellectual deterioration, irritability, impotence, hypertension, cerebral edema and cardiac arrhythmias.²⁴

Snoring is obstructive sleep breathing. Not all snorers have apnea, but 90 percent of apnea patients snore. Human beings are the only animals to sleep in a ventral positions. The sound of snoring originates in the collapsible part of the airway from the epiglottis to the carinum. This area has no rigid support – only the muscles keep it open. During sleep, human get some muscle flaccidity and in combination with nasal obstructions, such as hypertrophied adenoids and tonsils, the pharyngeal space can be occluded. The combination of long soft palate and uvula can also occlude the airway by collapsing when the subject lies in the ventral position.²⁵

Polysomnography can help diagnose and document this condition so that it can be appropriately treated. This is noninvasive testing done while the patient is asleep. It is usually done in a sleep lab of a hospital. Patients are measured for nasal air flow, arterial flow, heart, lungs, blood oxygen saturation, air flow turbulence and brain wave patterns during a 5-6 hour sleeping period.

Treatment

Depending on the objective findings, proper steps can be taken ranging from uvulopalatopharyngeoplasty (UPPP) to tonsillectomy and adenoidectomy to turbinectomy or medication such are antihistamine sprays, proper sleep posture or dental orthopedic appliances.

Dentists are an integral part of the health care team. As referrers, one has to know when and whom to refer. Allergies generally must be treated first, before consideration of any surgery. The greatest number of surgical failures are in

allergic patients.²⁶ The greatest number of orthodontic relapses are in patients who have not had their breathing problems successfully treated.²⁷

What can a dentist do to treat? Normalization of face form following adenoidectomy in a child can take five years. Donald Timms, in a study of 26 patients treated with Rapid Maxillary Expansion, reported a 37 percent mean drop in nasal resistance with 7 mm of expansion. In another larger study of children with a previous history respiratory disease treated with Rapid Maxillary Expansion, 82 percent reported improvement in number of upper respiratory tract infections and 60 percent reported improvement of allergic rhinitis.²⁸

Functional orthopedic appliances can be designed which support patients vertically during sleep and prevent collapse of pharyngeal musculature. It may be that dentists can do as much to clear the airway for the snorer/apnea patient by fabricating the appropriate appliance, as the physician can do with surgery.

Any appliance which is placed in the mouth has an effect on the volume of the tongue box. This is a term coined by Dr. Henri Petit. The tongue box has the pharynx as the posterior wall. The lateral and anterior walls are the teeth. The palate is the superior wall and the inferior wall is the floor of the mouth. Petit emphasizes that normal oral function cannot be achieved with inadequate tongue space. It is important that intraoral appliances do not reduce the volume of space for the tongue or they will set up the deleterious neuromuscular effect of pushing the tongue back into the nasopharynx and adversely affect nose breathing.²⁹

If an obstructive nasal breathing problem exists, dentists must direct themselves toward constructing an appliance which does not compromise tongue space. Appliances which increase tongue space may, in certain cases, be helpful in alleviating nasal obstructive breathing problems. The mandibular orthotic which corrects for deficient vertical dimension and brings the jaw anteriorly may also have a favorable effect on the tongue box.

This may account for why maxillary orthotics, which compromise the swallow and decrease tongue space by their palatal bulk, sometimes do not achieve success when mandibular appliances do.

Activity levels are at their highest or close to it bilaterally for temporalis, masseter, medial pterygoid and superior head of the lateral pterygoid. In an edge to edge bite there are much lower EMG levels in these muscles and almost a cut-off of activity in temporalis.³⁰

By supporting the patients both vertically and anteroposteriorly during sleep and allowing adequate room for the tongue, it has the ability of preventing collapse of pharyngeal musculature. And because it supports the teeth in the exact relationship that they are to each other at minimal EMG activity, it allows no undesirable orthodontic movement to occur.

Passivator

- Prevents "orthodontic" movement
- Does not interfere with correct swallow
- Prevents incorrect swallow
- Allows adequate tongue space
- Helps prevent collapse of pharyngeal musculature
- May improve nose breathing
- May improve snoring

References

1. Proetz A. Physiology of the Nose, Ed. 2, St. Louis, Annals Pub. Co., pp 119-169, 153.
2. Rubin RM. Mode of respiration and facial growth. Am J Orthod, 1980, 78:504-510.
3. Timms DJ. Lecture. RME for medical reasons. Upper Airway Compromise Dentofacial Development Symposium III, Virginia Beach, VA. 1986.
4. Harvold EP, Chierici EG, Vergervik K. Experiments on the development of dental malocclusions. Am J Orthodont. 1972. 61:38-4.
5. Harvold EP. Lecture. Experiments on bone remodeling. Upper Airway Compromise Dentofacial Development Symposium III, Virginia Beach, VA. 1986.
6. Harvold EP, Vergervik K, Chierici G. Primate experiments on oral sensation and dental malocclusion. Am J Orthodont. 1973. 63:494-508.
7. Vargevik H, Harvold EP. Experiments on interaction between orofacial function and morphology. Monograph of Upper Airway Compromise Dentofacial Development Symposium III, Virginia Beach, VA. 1986.
8. Linder-Aronsen, Sten Lecture. Vertical dysplasia update. Amer. Assoc. Orthodontists Annual Meeting, 1985.
9. Rubin RM. Lecture. Allergy, adenoids and airway: early recognition for optimal facial growth. Amer. Assoc. Orthod. Annual Meeting. 1982.
10. Weimert T. Airway obstruction in orthodontic practice. Jour. Clin. Orthod. 1986. 20:96-104.
11. Nelson WE, Vaughan VC and McKay RJ. Textbook of Pediatrics. WB Saunders, pp. 23-42, 1969.

12. Principato J. Lecture. Nasal airway assessment and management in the orthodontic patient. Amer. Assoc. Orthodontists Annual Meeting, 1982.
13. Rubin, Lecture. Controversies regarding etiology of the Long Face Syndrome. Upper Airway Compromise Detrofacial Development Symposium III, Virginia Beach, VA. 1986.
14. Ibid.
16. Rubin, Lecture. Controversies regarding etiology of the Long Face Syndrome. Upper Airway Compromise Detrofacial Development Symposium III, Virginia Beach, VA. 1986.
17. Glaser J. The prophylaxis of allergic disease in infancy. Pediatrics. 1962. 29:835-841.
18. Vig P. Lecture. Respiration: a critique of conventional wisdom and an approach towards a rational orthodontic perspective. Amer. Assc Orthodontists Annual Meeting, 1982.
19. Principato J. Lecture. Nasal airway assessment and management in the orthodontic patient. Amer. Assoc. Orthodontists Annual Meeting, 1982.
20. Ibid
21. Ibid.
22. Shapiro GC and Shapiro PA. Nasal airway obstruction and facial development. Clin. Rev. Allergy, 1984. 2:225-235.
23. Gates GA, Current therapy in otolaryngology: Head and Neck Surgery. C.V. Mosby, 1982-1983.
24. Fairbanks DNF, Lecture. Snoring and obstructive sleep apnea and children. Upper Airway Compromise Detrofacial Development Symposium III, Virginia Beach, VA. 1986.
26. Rubin. Lecture. Controversies regarding etiology of the Long Face Syndrome. Upper Airway Compromise Detrofacial Development Symposium III, Virginia Beach, VA. 1986.
25. Ibid.
27. Principato J. Lecture. Nasal airway assessment and management in the orthodontic patient. Amer. Assoc. Orthodontists Annual Meeting, 1982.
28. Timmons DJ, Rapid Maxillary Expansion. Chicago Quintessence Pub Co., pp. 41-45, 1981.
29. Petit, Henri Monograph, Upper airway problems and pre-orthodontic
30. Miller A, Lecture. Muscle Recruitment and the Chewing System. American Academy of Craniomandibular Disorders Annual Meeting, March 1987.