Nasal obstruction and facial growth: The strength of evidence for clinical assumptions

Katherine W. L. Vig, BDS, MS, FDS, DOrth
Columbus, Ohio

The orthodontic relevance of nasorespiratory obstruction and its effect on facial growth continues to be debated after almost a century of controversy. The continuing interest in nasal obstruction is fueled by strong convictions, weak evidence, and the prevailing uncertainty of cause and effect relationships that exist. The essence of any debate is to provide opposing evidence from which a majority vote is obtained. Political issues may be appropriately resolved by such means as a majority vote. Scientific issues, however, can only be resolved by data and appropriately structured hypotheses put to the test. One of the problems in debating nasorespiratory obstruction and facial growth is the inability to provide unequivocal answers to such issues as: How much nasal obstruction is clinically significant? At what age is the onset critical and for how long does it have to exist before an effect on facial growth can be expected? To provide unequivocal answers, clinical studies need to be designed to identify and quantify the degree of nasorespiratory obstruction and compare individuals for any clinically relevant differences. The purpose of this article is to review the available evidence. If both data and untested popular beliefs are subjected to the same rigorous criteria, indications for the orthodontic management of patients with nasorespiratory obstruction may gain a more rational approach to treatment recommendations.
(Am J Orthod Dentofacial Orthop 1998;113:603-11)

After more than a century[1] of conjecture and heated argument, the orthodontic relevance of nasal obstruction and its assumed effect on facial growth continues to be debated. To document a relationship between modification of facial growth and mouthbreathing requires defining the term mouthbreathing and the extent to which this may be associated with facial growth. Evidence from animal studies has been extrapolated to explain the human condition but *total* nasal obstruction, as produced by Harvold et al.[2] in monkeys, is extremely rare in human beings. Primates do not have the same nasorespiratory mechanism as human beings and do not readily adapt to mouthbreathing. If human beings have a preference for nasal respiratory mode as infants, this can adapt readily to oronasal respiratory mode in children. The debate, therefore, should focus on whether *partial* nasal obstruction is a risk factor for altered dentofacial growth in children. If treatment interventions that carry a significant risk, questionable benefit, and considerable cost, are to be justified, then good evidence is essential for clinicians to make rational decisions.

As the resolution of any controversy is conditional on the strength of evidence that may be used to refute or accept contentious issues, it behooves clinical scientists to design studies which generate sound data. By designing and establishing a basis for objectively assessing and interpreting results from "good data," progress may eventually be forthcoming and resolve alternative explanations for clinically observed phenomena. Alternative treatments based on rationally derived information, and not merely on accumulated subjective opinions supported by anecdotal case reports, may provide a basis for a more rational approach to the utility and efficacy of alternative treatment strategies. With the possible introduction of health care reforms and containment in the cost of the provision of health care in the United States, important issues in making clinical decisions relate to the risk/benefit/cost ratios.

The purpose of this article is to review some of the available evidence in children, adolescents, and adults that suggest that there may or may not be an association between respiratory mode and facial morphology.

NASAL OBSTRUCTION AND FACIAL MORPHOLOGY

The typical features that are considered characteristic of persons who have difficulty breathing

Originally presented at the CDABO meeting in St. Andrews by the Sea in 1992 as a debate topic on the Counterpoint Session on Nasorespiratory Obstruction.
From the Department of Orthodontics, College of Dentistry, The Ohio State University.
Reprint requests to: Dr. Katherine Vig, Department of Orthodontics, College of Dentistry, 4129 Postle Hall, 305 W 12th Avenue, Columbus, OH 43210

0889-5406/98/$5.00 + 0 **8/1/86381**

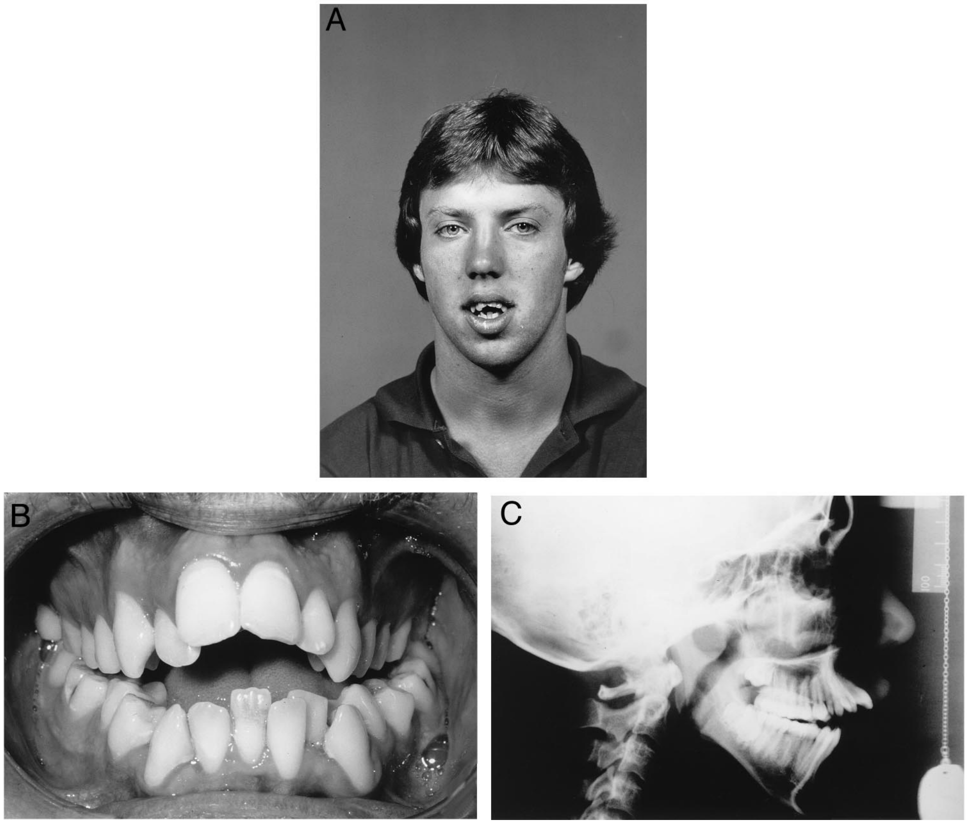

Fig. 1. A, Sixteen-year-old boy with an increase in lower face height, lip apart posture, and narrow alar base. **B**, Anterior view in occlusion. Note anterior open bite, bilateral posterior crossbite associated with transverse maxillary deficiency, and crowding of incisors. **C**, Lateral cephalogram confirms increase in lower facial height, vertical maxillary excess, and skeletal open bite. (Illustrations originally appeared in Int J Adult Orthod Orthog Surg 1989;4:119-28. Reprinted with the permission of Quintessence Publishing Co, Inc.)

through their nose and therefore may be diagnosed as having nasal obstruction, is exemplified by the long-face syndrome[3] (Fig. 1*A*). The pediatrician often refers to this as “adenoidal facies.” The prototype of this condition is considered to include an increase in lower facial height, lip apart posture, narrow alar base, and frequently self reported “mouthbreathing.” Intraorally, the clinician might expect to find a narrow maxillary arch with a high palatal vault and a posterior crossbite with a Class II dental malocclusion (Fig. 1*B* and C). A combination of orthodontics and surgery may be recommended in the non-growing patient, with the outcome goal of this treatment intervention being characterized by balanced vertical facial proportions and the ability for this patient to maintain lip contact at rest (Fig. 2*A*, *B*, and *C*). The issue of whether this patient had nasal impairment before the surgical procedure could have been unequivocally resolved by objective rhinometric tests to evaluate the nasal and oral components of airflow and nasal resistance. This, however, is not a routine procedure.

Some clinicians may feel that if treatment intervention had been considered at an earlier age and “mouthbreathing” evaluated and nasal obstruction diagnosed and corrected, then appropriate orthodontic intervention with maxillary expansion to correct the posterior crossbites might have resolved the vertical facial growth pattern. The concept of early intervention to eliminate the risk factor of altered facial growth and abnormal perioral muscle function and thus improving the long-term stability and facial morphology is attractive. Clinicians seek answers to questions such as:

- How much nasal obstruction is clinically significant?

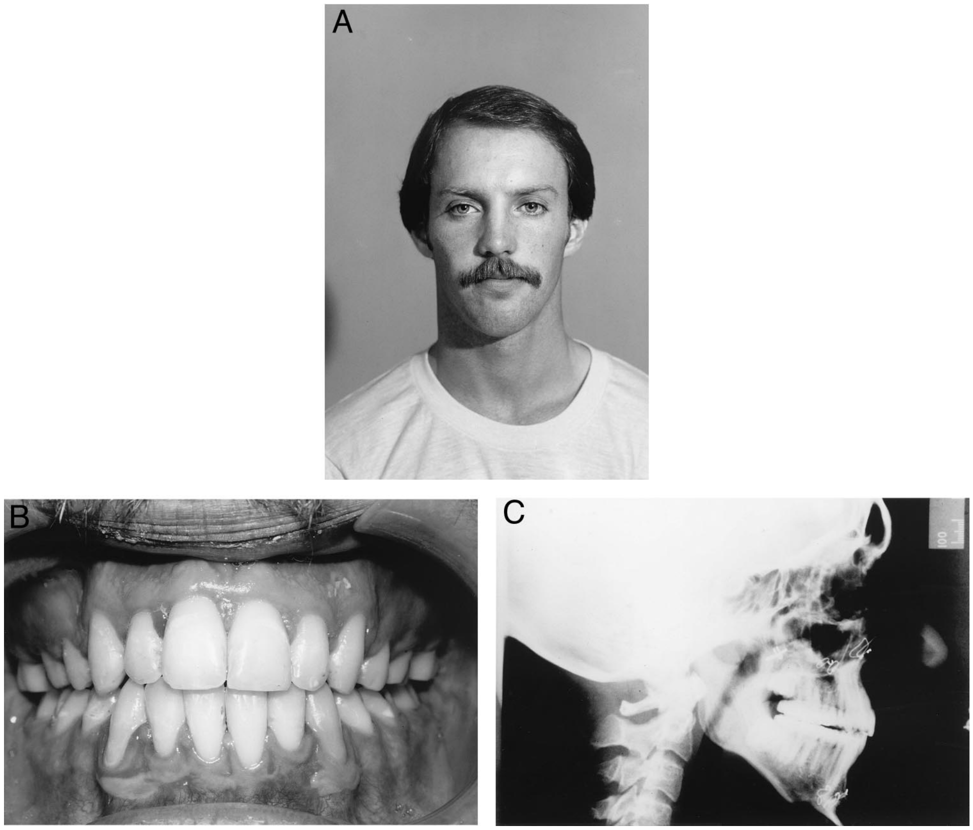

Fig. 2. A, Patient aged 19 years after surgical/orthodontic treatment. Segmental maxillary osteotomy with vertical superior maxillary repositioning and autorotation of the mandible with a genioplasty corrected the skeletal component of the malocclusion. **B**, Anterior view of occlusal relationship after treatment. **C**,Lateral cephalogram 1 year after surgical/orthodontic treatment. (Figs. 2A and B originally appeared in Int J Adult Orthod Orthog Surg 1989;4:119-128. Reprinted with the permission of Quintessence Publishing Co, Inc.)

- At what age is the onset of nasal obstruction critical?
- How long does obstruction of the nasal airway have to exist before a growth effect may be anticipated?
- Is this clinically relevant to Orthodontics?

A review of the literature shows that many of the arguments and propositions presented are based on anecdotal case reports. Well-designed and rigorous prospective randomized clinical trials are not currently available from either dental or medical literature. As a prerequisite for resolving the issue, we need to reliably identify nasorespiratory function and quantify the degree of obstruction. Comparison of persons matched for age and gender with and without nasal obstruction should provide the clinician with information of any clinically relevant differences in facial morphologic characteristics.

METHODS OF MEASURING NASAL OBSTRUCTION

Nasal obstruction is an ill-defined and ambiguous term that is equated with "mouthbreathing" and a lip-apart posture. The misconception that a direct cause and effect relationship exists between incompetent lips and mouthbreathing has continued to be controversial. The establishment of an anterior oral seal for nasal respiration may not be easily achieved in those persons with a lip-apart posture and an increase in lower facial height. An adaptive mechanism for persons who breathe through their nose, but maintain a lip-apart posture, is to obtain a posterior oral seal with the tongue against the soft palate. Some years ago, Ballard and Gwynne-Evans[4] reported their findings that lip incompetence was not necessarily associated with mouthbreathing. Much of the data indicate that it is rare for an person to breathe 100% through the mouth and a

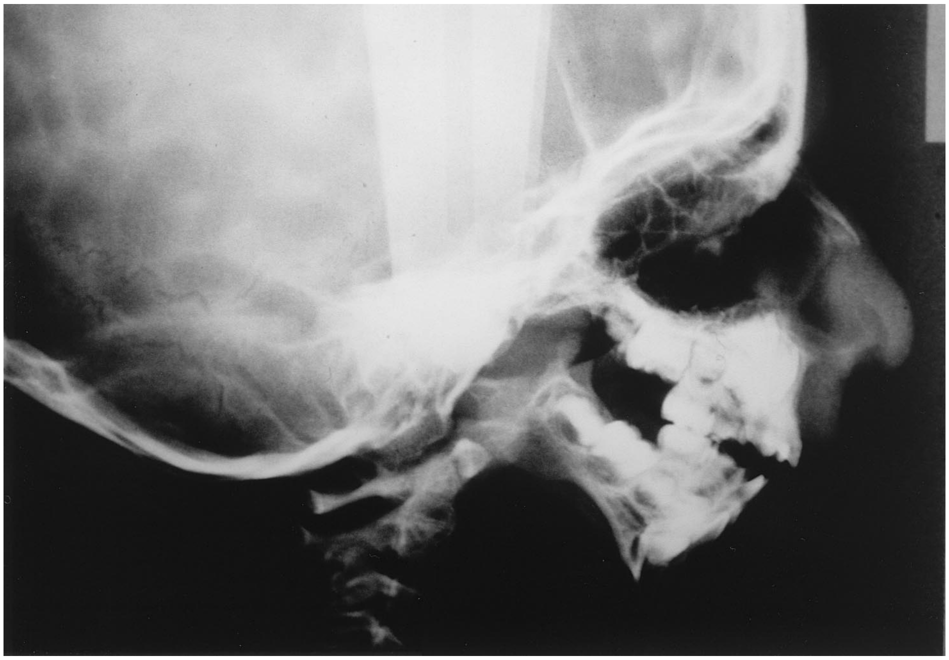

Fig. 3. Lateral cephalogram of child with posterior choanal atresia associated with Treacher Collins syndrome. Note the characteristic openbite, increase in anterior lower face height, retrogenia and associated decreased posterior face height, and sagittal mandibular deficiency. Both nasal and postlingual airways are compromised, and the patient had a tracheotomy.

more common mode of respiration is a combination of simultaneous oral and nasal airflow.[5]

Certain conditions may prevail where total nasal obstruction occurs. Choanal atresia with total bony or soft tissue obstruction of the posterior nares result in a total blockage, preventing nasal airflow. An example is an infant born with this condition, who also had other syndromic features associated with Treacher Collins or mandibulofacial dysostosis (Fig. 3). These neonates may become severely hypoxic at birth, not only from the choanal atresia but also the typical mandibular deficiency, which is a characteristic feature of this syndromic manifestation. In addition, these infants may have a compromised postlingual airway as a result of the mandibular deficiency. The immediate need for a tracheotomy may become necessary in the management of the airway in these neonates. Freng and Kvam[6] examined cephalograms of 51 patients with choanal atresia and found a tendency for sagittal maxillary deficiency with associated mandibular deficiency when stenosis had been present throughout the period of facial growth. Fig. 3 represents the condition in the primary dentition of a child who has a tracheotomy and was diagnosed as having Treacher Collins syndrome as an infant. The characteristic features of the increase in lower facial height and open bite are consistent with those attributed to nasal obstruction but are also characteristic of the syndromic manifestation. With the oral and nasal airways functionless in this child because of the tracheotomy, the facial pattern and open bite could be attributed to the inherited syndromic condition with an additional environment component.

The sequence of events in assuming altered vertical facial growth as a consequence of nasal obstruction in normal biologic variation is:

Nasal obstruction ▶ Lip apart posture ▶ Mouth-breathing ▶ Modification of facial growth

The classic work of Harvold[2] cited earlier in the text was based on total obstruction of the nasal airway in monkeys; this resulted in a cause and effect relationship. However, human studies have indicated that total nasal obstruction is rare, and the most common respiratory mode is a simultaneous oral and nasal airflow.[5] The percentage of nasal versus oral airflow is dependent on a number of variables. Case series[7,8] indicate that children with nasal obstruction experience a downward and backward rotation of the mandible, with subsequent growth producing an increase in the lower facial height.

If there is an association between mouthbreathing and modification in facial growth, there is a need to know which children are at risk. Is this an age dependent condition? How much obstruction of nasal airflow has to occur before an effect on facial growth is observable? Is this a reversible situation and is there a time dependent relationship? These questions depend on the fundamental premise of being able to define nasal obstruction. The nose has an anterior opening designated as the nares, a middle portion that is influenced by the turbinates and their associated vascular mucosa that enlarges and becomes engorged in those persons with allergic rhinitis, and the posterior nares that open into the nasopharynx and may be affected by adenoidal hypertrophy. An important issue in nasal obstruction is therefore to identify where the obstruction occurs. Various methods have been proposed and used in different studies. These include: (1) Cross sectional area, which will be affected by turbulence; (2) Peak nasal flow rate, which was used in studies[7]; (3) Nasal resistance, which may vary over time; and (4) Respiratory mode, which identifies nasal/oral ratio of air flowing through the nose and the mouth.

In comparing these measures of nasal function, how well will one of these measures predict the others? If the evidence from which we make clinical decisions is the result of studies that are not comparable, then one method of treatment cannot necessarily be compared with an alternative. Questions such as: Is nasal obstruction synonymous with increased nasal resistance? Does the term nasal obstruction refer to those persons who are total or 100% nasal breathers? Do aerodynamic estimates provide reliable and valid information, or are imaging methods using radiographic appearances a more reliable measure of nasal obstruction? Is there a good agreement between radiographic findings and rhinomanometric measures?

RADIOGRAPHIC APPEARANCE OF THE ADENOIDS

Cephalometric radiographs and rhinomanometric tests to evaluate nasal obstruction have been available for several decades. The controversy that airway patency and its effect on facial growth have been linked in a causal relationship has a long history of debate and conjecture. The cephalogram is a commonly obtained diagnostic record for orthodontic treatment planning with two-dimensional information of the adenoidal image. Although positive correlations between airflow and airway measurements have been made on cephalometric radiographs, the three-dimensional aspects have been neglected.[9] Various lines and areas[10] have been interpreted by a number of investigators to implicate the enlarged adenoid in a causal relationship with mouthbreathing and the subsequent effect on vertical facial growth (Fig. 4). Linder-Aronson[7] reported in a group of children who had adenoidectomy that they returned to nasal breathing and demonstrated craniofacial growth changes.[7,11,12] These changes in breathing mode and mandibular and maxillary growth were measured 5 years after adenoidectomy[11] as was the incisor position.[12] Conversely, Bushey[13] found no relationship between nasal respiration and linear measurements of the adenoids on lateral skull cephalograms before and after surgical removal of the tonsils and adenoids.

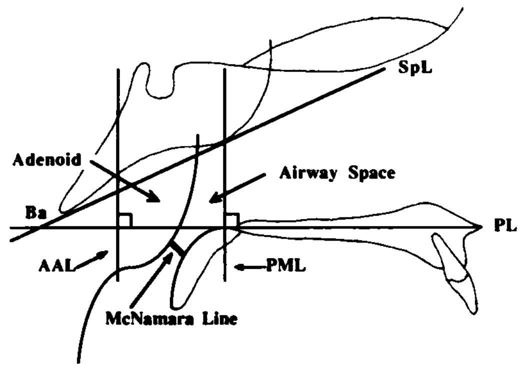

Fig. 4. Diagram of the posterior nasal airway region with adenoidal encroachment in the Shulhof[10] "airway space." The McNamara linear measurement of the minimum distance from soft palate to adenoidal shadow on lateral cephalogram. (*PL,* palatal line; *Ba* = *SpL,* basion to sphenoid tangent; *PML,* perpendicular to *PL* at the posterior nasal spine; *AAL,* perpendicular to *PL* through anterior arch of the atlas.) (Originally appeared in the Am J Orthod Dentofacial Orthop 1991;99: 354-60. Reprinted with permission of Mosby, Inc.)

An illustrative case in Fig. 5 indicates a change in dimension of the adenoids after adenoidectomy. Although the patient demonstrated a predominately oral mode of respiration, this remained unchanged after adenoidectomy. There appeared to be none of the typical modifications in facial growth often attributed to nasal obstruction. There was an initial tendency to increased overbite and reduced lower facial height rather than the typical increase in lower facial height. This illustrates the fallacy of conclu-

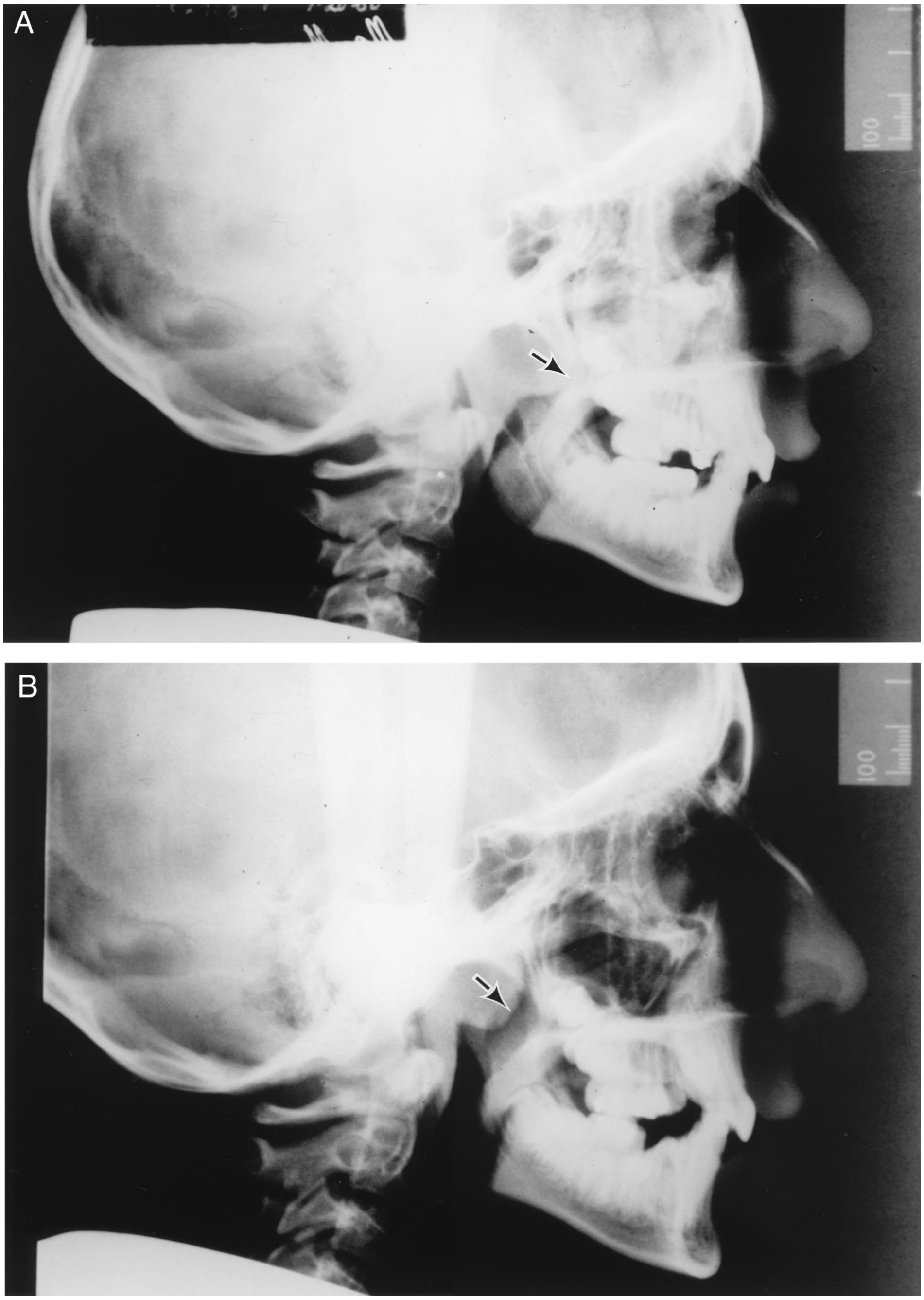

Fig. 5. A, Hypertrophied adenoids in 10-year-old child who was a “mouth breather.” She demonstrated none of the characteristic features of the “adenoidal facies.” **B**, After adenoidectomy the postnasal airway appears increased and patent on the lateral cephalogram. Her predominately oral mode of respiration remained apparently unchanged after surgical reduction of the adenoids.

sions based on a single case report and can only stress the need for evaluating the strength of multifactorial evidence. Qualitative comparisons of evidence constitutes establishment of a hierarchical approach, with an increasing strength from single case report, to a series of cases, followed by case-controlled studies retrospectively to the randomized controlled prospective clinical trial.

RHINOMANOMETRIC STUDIES

Although some clinicians have causally associated nasal obstruction and hypertrophied adenoids with changes in facial growth, another group of patients who frequently have posterior nasal obstruction are those with cleft lip and palate. These patients often have pharyngeal flaps placed to deliberately reduce their nasal airflow during speech. This iatrogenic increase in nasal obstruction reduces hypernasality during speech but at the expense of increasing resistance to nasal airflow. Serial lateral skull radiographs of children who had pharyngeal flaps indicate that there may be an effect on facial growth.[14] However, the relationship between the amount of nasal obstruction that has to be present before an effect on facial growth occurs is not clear. Likewise, the time that must elapse before growth will be effected and the age at which children are most at risk for this intervention has not been identified.

Pharyngoplasty may also adversely affect facial growth by inhibiting sagittal maxillary growth and induce functional adaptations by increasing nasal resistance. But the results of a matched controlled clinical study of pharyngoplasty patients from the Oslo archive[15] indicate that the risk of adversely affecting sagittal or vertical maxillary growth by a pharyngeal flap is inconsequential. To determine the prevalence of mouthbreathing in the cleft lip/palate population, Hairfield et al.[16] evaluated the percentage of nasal breathing in children and adults. They found that approximately two thirds of the subjects had an oral/nasal airflow and one third of the subjects had predominately nasal airway. Adults had the same prevalence of mouthbreathing as children, which supports the contention that surgical repair of the lip/palate compromises nasal respiration.

As cited earlier, quantification of respiration has relied on airway dimensions from cephalometric radiographs and nasal airflow/pressure studies, which were considered valid diagnostic indicators for making clinical decisions for treatment interventions. These have been used as proxys for respiratory mode. The functional aspects of breathing have had various diagnostic methods applied for assessing whether air is flowing through the nose, the mouth, or both. Simple clinical tests to ascertain mouthbreathing include the fogging of a mirror held under the nostrils and cotton wisps that move in the nasal airflow. Too often the determination of respiratory mode is miscalculated by clinicians, especially if self-reported information is relied on from the patient. The directly measured ratio of the oral to nasal airflow provides an objective determination of nasal respiratory function.

FACIAL GROWTH AND RESPIRATION

In an attempt to rationalize vertical dentofacial morphologic characteristics with variations in breathing behavior, Fields et al.[17] used contemporary respirometric techniques to compare respiratory modes of normal and long-faced adolescents. They concluded that the long-faced subjects had a significantly smaller component of nasal airflow, although the tidal volume and minimum nasal cross sectional area were similar. They suggest that significant differences in airway impairment do not have a direct effect on the breathing mode, which may be behaviorally determined rather than being structurally dependent. The form-function interaction that conveniently should explain the causal association between nasal obstruction and facial growth in children appears to be of a multifactorial nature.

FORM/FUNCTION INTERACTIONS

Rapid maxillary expansion (RME) has been recommended for the correction of maxillary transverse deficiency with the additional benefit of increasing nasal airflow. Evidence to support this comes both from patients' self-reported perceptions and from radiographic images. Rhinometric studies after RME indicate that there is no increase in percentage of nasal breathing, nor is there a predictable decrease in nasal resistance. Although a reduction in nasal resistance was frequently measured after expansion, this did not change the respiratory mode of the patient.[18]

The variability associated with nasal resistance and RME may be affected by such factors as the age and gender of the patient at the time of the treatment intervention. Growth modification and nasal obstruction have not taken into account age and gender effects as confounding variables. Determination of nasal respiratory function requires standards that are appropriate for age and gender, similar to the normative standards that exist for incremental changes in craniofacial growth and development in the Bolton standards of dentofacial developmental growth[19] or the Atlas of Craniofacial Growth.[20] Such stratified normative data have not been compiled previously for respiratory mode, nasal resistance, and cross-sectional area on a longitudinal basis for the same series of children over time. This would provide a unique data base for age-related measures to establish if significant age and gender differences

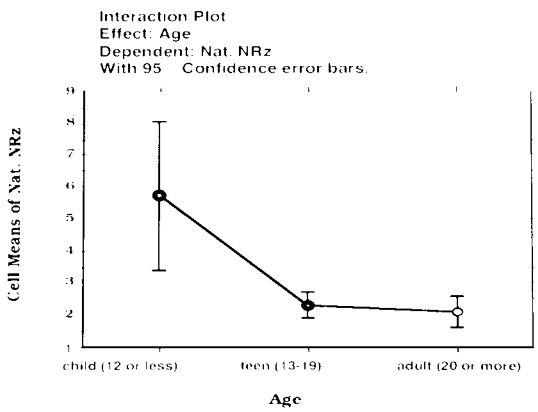

Fig. 6. Effect of age on nasal resistance *(NRz).* Variability around the mean in children reduces with age and levels off during adulthood. (Nat Nrz, natural mode (no nasal decongestant); nasal resistance, measured in cm H_2O/L/sec.

in these variables exist. Results from a cross sectional study[21] indicate significant effects of age and gender on nasorespiratory function in children, adolescents, and adults. Gender considerations revealed significantly greater percentage of nasal respiratory component in women than in men. Thus considerable sexual dimorphism exists at all ages. This was particularly evident with nasal resistance that decreased with age (Fig. 6). Recent data indicate that nasal resistance increases in the geriatric population, returning to the characteristics in childhood. This may contribute to airway impairment in sleep-disordered breathing in the elderly.

ADAPTATION TO ALTERED MORPHOLOGY

Because surgical superior impaction of the maxilla has become an accepted treatment for the correction of vertical maxillary excess,[22] attention has focused on nasal resistance and respiratory mode after orthognathic surgery.[23] These studies indicate that orthognathic surgery, which vertically repositions the maxilla, does predictably reduce nasal resistance, which intuitively should increase the percentage of nasal airflow. This, however, does not apparently occur, and thereby provides another example of why clinicians and researchers should not assume that because one of the parameters of nasal respiration has been affected, that extrapolation to others, such as cross-sectional area, peak nasal flow rate, and respiratory mode will all be similarly affected. The intercorrelations between these various parameters of nasorespiratory function are quite low.[24] The highest correlation, which approaches 0.74, is between respiratory mode (percentage of nasal breathing) and peak nasal flow rate. The other correlations expressed in the relationship between cross-sectional area, nasal resistance, and respiratory mode ranged between 0.4 and 0.6. These measures are, therefore, not interchangable nor are they estimates of the same functional attribute.

The timing of orthognathic surgery for the correction of dentofacial deformities relates to the stability of the outcome after the surgical/orthodontic morphologic changes. Clearly, the nearer the patient is to completion of growth, the less likely it is that the long-term outcome will be affected by continuing growth. However, other factors also play a part in determining the time of surgical intervention; these relate to age, gender, psychosocial impact, and functional considerations.[25]

DISCUSSION

The strength of evidence

For diagnostic tests to be clinically useful, they must consistently be able to differentiate between the presence or absence of a clinical condition or disease. Reduction in the nasal component of respiratory airflow is clearly not in itself a disease, but rather an arbitrary point on a continuum between 100% nasal breathing at one end of the spectrum and zero at the other extreme. There are, therefore, unresolved issues that cloud the issue of the utility of diagnostic tests in this field. The sensitivity and specificity of diagnostic tests for impaired nasal breathing[26] are poor indicators of nasal resistance, peak flow rate, and percentage of nasal airflow. The demonstrated age and gender variations in all of these nasorespiratory parameters require specific values for discrimination between normal and abnormal nasal function to be established. Such values should not merely be based on statistical considerations with percentiles or standard deviations around a mean but should correspond to biologically significant deviations in function, with demonstrated effects on either general health or facial growth.[27] This would provide age and gender "gold standards" for diagnostic identification of treatment needs.

Diagnostic tests

The diagnostic tests of otorhinolaryngologists for assessing impaired nasal respiration are inconsistent.[28] For clinical purposes, a test should measure the severity of the condition and not some inconsistently related covariate. Therefore, if the purpose is to determine a patient's respiratory mode or percentage of nasal airflow, then neither cross-sectional area of the nasal passage, peak nasal airflow, nor nasal resistance are suitable substitutes, because they are all poorly intercorrelated. Each of these parameters may be of interest but is not valid as a proxy for assessing mode of respiration.

Given that the validity of a test measures what it is intended to, it is also important that the test is reliable. Reliability in this context is not confined to reproducibility over time, but more particularly to the accuracy of both positive and negative prediction. To evaluate the "goodness" of a test by experimentally verifying the true-positive rate (sensitivity) and true-negative rate (specificity) of identification of the condition by the test procedure is commonplace in contemporary medical practice. Unless both the presence and absence of the condition are consistently identified, the test is inadequate. Underdiagnosis or overdiagnosis of conditions leads to either deprivation or unnecessary interventions. Clinically, neither of these alternatives are acceptable and render the study of treatment effects invalid in terms of efficacy, utility, or safety.

CONCLUSIONS

Clearly, more objective tests are required, and unambiguous criteria must be established if airway impairment is to be adequately defined and its etiologic significance in relationship to facial growth determined. Only when this issue is resolved will the clinical impact of respiratory function be clarified and the appropriate interventions advocated. If data from studies and untested popular beliefs are subjected to the same rigorous criteria for assessment, the results may eventually influence orthodontic clinical practice and our future rationale for treatment interventions.

Support from NIH/NIDR Grant #DE 06061 on which I have been a Coinvestigator for 10 years is recognized. The data cited in this manuscript has been generated by Dr. Peter Vig and members of his laboratory. I am indebted to Peter Vig for permission to use this data for the debate and subsequent publication. Special thanks to Dr. Timothy Turvey, UNC Department of Oral and Maxillofacial Surgery, with whom I collaborated in the treatment of the patient illustrated in Figs. 1 and 2. My thanks are also extended to the College of Diplomats of the American Board of Orthodontists for inviting me to present this material at their annual meeting in New Brunswick in 1992 as one of the Point/Counterpoint Sessions on Nasorespiratory Obstruction.

REFERENCES

1. Warren DW, Spalding PM. Dentofacial morphology and breathing: a century of controversy. In: Melsen, B, editor. Current controversies in orthodontics. Chicago: Quintessence Pub Co; 1991. p. 45-76.
2. Harvold EP, Chierici G, Vargervik K. Primate experiments on oral sensation and dental malocclusion. Am J Orthod 1973;63:494-508.
3. Schendel SA, Eisenfeld J, Bell WH, Epker BN, Mishelevich DJ. The long-face syndrome: vertical maxillary excess. Am J Orthod 1974;70:398-408.
4. Ballard CF, Gwynne-Evans E. Discussion on the mouthbreather. Proc R Soc Med 1958;50:279-85.
5. Keall CL, Vig PS. An improved technique for the simultaneous measurement of nasal and oral respiration. Am J Orthod 1986;91:207-12.
6. Freng A, Kvam E. Facial sagittal growth following partial, basal resection of the nasal septum: a retrospective study. Eur J Orthod 1979;1:89-96.
7. Linder-Aronson S. Respiratory function in relation to facial morphology and the dentition. Angle Orthod 1979;6:59-71.
8. McNamara JA. Influence of respiratory pattern on craniofacial growth. Angle Orthod 1981;51:269-300.
9. Holmberg H, Linder-Aronson S. Cephalometric radiographs as a means of evaluating the capacity of the nasal and pharyngeal airway. Am J Orthod 1979;76:476-90.
10. Shulhof RJ. Consideration of the airway in Orthodontics. J Clin Orthod 1978;12: 440-4.
11. Woodside DG, Linder-Aronson S, Lundstrom A, McWilliams J. Mandibular and maxillary growth after changed mode of breathing. Am J Orthod 1991;100:1-18.
12. Linder-Aronson S, Woodside DG, Hellsing E, Emerson W. Normalization of incisor position after adenoidectomy. Am J Orthod 1993;103:412-27.
13. Bushey RS. Adenoid obstruction of the nasopharynx. In: McNamara JA, editor. Nasorespiratory function and craniofacial growth. Monograph #9, Craniofacial Growth Series, Ann Arbor: University of Michigan; 1979. p. 199-232.
14. Long RE, McNamara JA. Facial growth following pharyngeal flap surgery: skeletal assessment on serial lateral cephalometric radiographs. Am J Orthod 1985;87:187-96.
15. Semb G, Shaw W. Pharyngeal flap and facial growth. Cleft Palate J 1990;27:217-24.
16. Hairfield WM, Warren DW, Seaton DL. Prevalence of mouthbreathing in cleft lip and palate. Cleft Palate J 1988;25:135-8.
17. Fields HW, Warren DW, Black K, Phillips CL. Relationship between vertical dentofacial morphology and respiration in adolescents. Am J Orthod Dentofacial Orthop 1991;99:147-54.
18. Hartgerink DV, Vig PS, Abbott DW. The effect of rapid maxillary expansion on nasal airway resistance. Am J Orthod Dentofacial Orthop 1987;92:381-9.
19. Broadbent BH Sr, Broadbent BH Jr, Golden WH. Bolton standards of dentofacial developmental growth. St Louis: CV Mosby Co; 1975; p. 140-66.
20. Riolo ML, Moyers RE, McNamara JA, Hunter WS. An atlas of craniofacial growth. Monograph #2 Craniofacial Growth Series. Ann Arbor: University of Michigan; 1979; p. 25-200.
21. Vig PS, Zajac D. Age and gender effects on nasorespiratory function. Cleft Palate Craniofac J 1993;30:279-84.
22. Bell WH, McBride KL. Correction of the long-face syndrome by LeFort 1 osteotomy. Oral Surg Oral Med Oral Pathol 1977;44:493-501.
23. Spalding PM, Vig PS, Lints RR, Vig KWL, Fonseca RJ. The effects of maxillary surgery on nasal respiration. Int J Adult Orthod Orthog Surg 1991;6:189-198.
24. Vig PS, Kowalski CJ. Interrelations between respiratory mode, nasal resistance, flowrate and cross-sectional area. J Dent Res 1991;72:342.
25. Vig KWL, Turvey TA. Surgical correction of vertical maxillary excess during adolescence. Int J Adult Orthod Orthog Surg 1989;4:119-28.
26. Vig PS, Spalding PM, Lints RR. Sensitivity and specificity of diagnostic tests for impaired nasal respiration. Am J Orthod Dentofacial Orthop 1991;99:354-60.
27. Kluemper GT, Vig PS, Vig KWL. Nasorespiratory characteristics and craniofacial morphology. Eur J Orthod 1995;491-5.
28. Drake AF, Keall HJ, Vig PS, Krause CJ. Clinical nasal obstruction and objective respiratory mode determination. Ann Otol Rhinol Laryngol 1988;97:397-402.