Inspiratory and Expiratory Effects of Nasal Breathing

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The purpose of the present study was to assess quantitatively the nasal valve area during normal inspiratory and expiratory maneuvers. The pressure-flow technique of Warren was used to estimate nasal size in 15 adult subjects who had no previous history of nasal surgery or abnormality. Cross-sectional size of the nasal airway was also assessed (1) after insertion of tubing to eliminate alar effects, (2) after administration of 0.1 % Otrivin decongestant to eliminate mucosal effects, and (3) by blocking each nostril individually. Mean areas were 0.63 cm² \pm 0.17 during inspiration and 0.56 cm² \pm 0.14 during expiration. This difference is statistically significant (<0.01) and the effect was maintained under conditions of occlusion of either nostril, insertion of tubing, and administration of Otrivin. These findings suggest that the nasal valve acts as a respiratory brake during expiration possibly to allow adequate time for gas exchange at the alveoli.

Clefts of the lip and palate frequently produce significant nasal deformities, such as deviated septum, vomerine spurs, and atresia of the nostrils, as well as maxillary growth deficits which alter the nasal floor. These abnormalities tend to reduce the size of the nasal airway (Drettner, 1960; Foster, 1962; Aduss and Pruzansky, 1967; Warren et al, 1969).

Warren et al (1969) reported that nasal airway resistance is higher in the cleft population even prior to surgical treatment. This was attributed to the nasal deformities and maxillary growth deficits that so frequently occur. They suggested that high nasal airway resistance has important implications in breathing and speech, since it may lead to mouth breathing and possible dental malocclusion.

Surgical correction of nasal, palatal, and pharyngeal structures often further compromises the nasopharyngeal space. Cosmetic surgery of the cleft nose is one example. The objectives are to restore the symmetry of the alar cartilages, produce an esthetically acceptable nasal tip, and obtain a satisfactory relationship between the lip and nose (Converse et al, 1977). In the unilateral cleft lip, the unaffected nostril often appears to be abnormally large because of developmental hypoplasia or previous surgical intervention of the cleft nostril. The surgeon often reduces the size of the unaffected nostril in order to restore symmetry.

Similarly, secondary restorative procedures for residual palatal incompetence in cleft palate, such as the posterior pharyngeal flap and the prosthetic speech appliance, significantly reduce the nasopharyngeal airspace (Warren et al, 1974). The posterior pharyngeal flap has been shown to have an especially adverse effect on upper airway breathing in children. Indeed, many patients complain of mouthbreathing and snoring after this procedure, and there are reports that the posterior pharyngeal flap sometimes produces sleep apnea, which may result in growth retardation, heart failure, or sudden death (Guilleminault et al, 1976; Kravath et al, 1980).

On the other hand, some surgical procedures may improve the nasal airway. The elimination of spurs, the correction of a deviated septum, and even maxillary osteotomies can reduce nasal airway resistance (Turvey et al, 1984). Orthodontic expansion of the maxillary arch can also have a beneficial effect on nasal respiration (Hershey et al, 1976; Warren, 1980).

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The point to be emphasized is that there is little quantitative documentation of the physiologic status of the nasal airway in individuals with cleft lip or lip and palate other than clinical impressions and some very limited nasal resistance studies (Warren et al, 1969; Drettner, 1960; Warren et al, 1974; Warren, 1980). It is, therefore, not surprising that the literature does not describe the changes that occur in the airway after surgery except for a few anecdotal reports that suggest some problems (Warren et al, 1974; Guilleminault et al, 1976; Kravath et al, 1980). What little is known implies that airway impairment and mouthbreathing are not infrequent, especially in children.

The significance of impaired nasal respiration and mouth breathing in the noncleft population has raised considerable controversy among clinicians. There have been many attempts to establish a causal relationship between dentofacial deformities and nasal airway inadequacy. The most prevalent view has been that mouthbreathing resulting from an inadequate nasal airway is often associated with such deformities as retrognathic mandible, protruding maxillary anterior teeth, high palatal vault, constricted V-shaped maxillary arch, flaccid and short upper lip, flaccid perioral musculature, and a somewhat dull appearance because of a constant open-mouth posture (Morrison, 1931; Strang, 1943; Jennes, 1963: Reid and Donaldson, 1970; Linder-Aronson, 1973, 1979; Quinn, 1978a).

A significant number of clinicians and researchers have questioned the assumption that impaired nasorespiratory function influences growth (Humphrey and Leighton, 1950; Leech, 1958; Watson et al, 1968; Vig et al, 1981). These conflicting views concerning the effects of breathing on facial growth suggest the need for a more quantitative approach to this important question. The controversy stems primarily from our inability in the past to define mouthbreathing in objective terms and evaluate nasal airway impairment quantitatively. Currently, a diagnosis of "mouthbreathing" or nasal obstruction based upon clinical impression alone often determines the therapeutic approach. Clinicians who believe that mouthbreathing is an orthodontically harmful behavior often pursue an aggressive approach to correct this suspected source of abnormal development (Quinn, 1978a, 1978b; Schulhof, 1978; Ricketts, 1968, 1979; Butler, 1960; Ferris et al, 1964; Hyatt and Wilcox, 1961; Speiger and Frank, 1964; Cole, 1976; Rubin, 1968, 1980).

Clearly, an understanding of upper airway respiratory function is of interest to clinicians, especially those involved in treatment of cleft lip and palate patients.

Although much attention has been focused on the nasopharyngeal airspace, turbinates, and septum, the greatest airflow resistance in the normal airway occurs at the nasal valve (Bridger, 1970). The valve is in the region between the upper and lower lateral cartilages and the pyriform aperture just beyond the anterior ends of the inferior turbinates. Cadaveric dissection and intranasal impressions indicate that the crosssectional area of the nasal valve is 0.3 - 0.4 cm² in each nostril (Proctor, 1985). The valve, on the basis of its anatomic and flow-resistive characteristics, has been described in physiologic terms as the main regulator of the nasal airway. The purpose of the present study was to assess the normal nasal airway, and especially the nasal valve area, so that quantitative comparisons with the cleft nasal airway could be made in future studies.

The specific questions asked were:

- 1. Does nasal cross-sectional area differ between inspiration and expiration?
- 2. Does external nares dilation influence nasal cross-sectional size? and
- 3. What are the effects of a nasal decongestant on nasal airway size?

METHODS

The minimum cross-sectional area of the nasal airway was measured in 15 adult subjects, 8 male and 7 female Caucasians, who had no previous history of nasal surgery or abnormality. Each airway was assessed under four different conditions in the following order: (1) the total nasal cross-sectional area was estimated, (2) one nostril was occluded with an appropriately fitted cork and the cross-sectional area estimated for the patent airway; then this procedure was repeated for the other nostril, (3) stiff tygon tubing, approximately 1 cm in length and $\frac{3}{8}$ inch internal diameter (0.71 cm² cross-sectional area), was gently inserted into each nasal vestibule, just deeply enough to dilate the external nares and eliminate alar effects without distorting the nasal valve itself, and (4) each nostril was flooded with 3 drops of 0.1% xylometazoline (Otrivin)¹ nasal solution; then after 15 minutes total crosssectional area was estimated.

The method of measurement involves a modification of the theoretical hydraulic principle and assumes that the smallest cross-sectional area of a structure can be determined if the differential pressure across the structure is mea-

¹ Otrivin 0.1% nasal solution, Geigy Pharmaceuticals, Ardsley, NY.

sured simultaneously with the rate of airflow through it. This method has been used in speech research by Warren and his associates since 1961. Its reliability has been verified in a number of laboratories (Warren and Ryon, 1967; Smith and Weinberg, 1980, 1982).

where k = 0.65, density of air = 0.001 gm/cm³, involves two variables associated with nasal breathing, namely, airway pressure and airflow. Measurement of these two variables simultaneously provides the information necessary for application of the hydraulic principle. The correction factor k was obtained from analog studies that have been reported previously (Warren, 1984). Figure 1 illustrates the technique used.

A nasal cap was selected for each patient that would not distort the nose and would offer only negligible dead space. The pressure drop across the nose was measured by placing one catheter in the mouth and the other in the nasal cap. Each catheter was attached to a pressure transducer. Airflow was measured by a heated pneumotachograph attached to the nasal cap. The subject was asked to breathe in and out of the nose and the resulting pressure and airflow data were sent to a 12-bit analog-to-digital converter to be analyzed by an IBM microcomputer. The data were graphically displayed on a Tektronix 4014 terminal and electronic cursors were used to select corresponding pressure and airflow peaks for the calculation of cross-sectional area. A copy of such data is illustrated in Figure 2.

RESULTS

Figure 3 illustrates mean nasal cross-sectional areas and standard deviations for subjects measured during both inspiration and expiration. Total cross-sectional area was $0.63 \text{ cm}^2 \pm 0.17 \text{ dur-}$ ing inspiration and $0.56 \text{ cm}^2 \pm 0.14$ during expiration. This difference is statistically significant using the paired-t test (p < 0.01).

Figure 4 illustrates mean nasal cross-sectional areas when each nasal passage was measured separately during both expiration and inspiration. Area values for each passage were then added to provide total cross-sectional area. Crosssectional area was never equally distributed between passages. On the average, one passage accounted for 66 percent of the total area, and the other for 34 percent. The area values were 0.69 $cm^2 \pm 0.25$ during inspiration and 0.64 $cm^2 \pm$ 0.22 during expiration. This difference is statistically significant (paired-t test, p < 0.025). Total cross-sectional areas measured during forced respiration through one passage results in area values which are approximately 10 percent greater than when both passages where measured simultaneously.

Figure 5 illustrates mean nasal cross-sectional areas when tygon tubing was inserted into the anterior vestibule. Cross-sectional areas were $0.65 \text{ cm}^2 \pm 0.18$ during inspiration and $0.57 \text{ cm}^2 \pm 0.14$ during expiration. This difference is statistically significant (paired-t test, p < 0.01). Insertion of tygon tubing increased cross-sectional area a nominal 2 percent.



FIGURE 1 Schematic diagram of method used for measurement of nasal cross-sectional areas.



FIGURE 2 Hard copy record of inspiratory and expiratory waveforms. Numerals 1 through 5 indicate the position of the electronic cursors where values were selected for analysis.



FIGURE 3 Mean nasal cross-sectional areas and standard deviations for normal adults.

Figure 6 illustrates mean nasal cross-sectional areas following administration of the nasal decongestant (Otrivin). Cross-sectional areas were $0.77 \text{ cm}^2 \pm 0.18$ during inspiration and $0.70 \text{ cm}^2 \pm 0.14$ during expiration. This difference is statistically significant (paired-t test, p < 0.01). The decongestant increased cross-sectional area about 23 percent over normal values.

Table 1 illustrates the ratio of inspiratory crosssectional areas to expiratory cross-sectional areas for each experimental condition. There are no statistically significant differences among ratios

RIGHT & LEFT NOSTRILS



FIGURE 4 Mean nasal cross-sectional areas given when each passage measured separately is summed to give total area.

(paired-t test, p > 0.29). Therefore, the inspiratory/expiratory effect was consistent under all experimental conditions.

DISCUSSION

The difference in nasal cross-sectional areas between phases of respiration revealed in this study provides another example of the dynamic nature of the respiratory tract. The data suggest that the nasal airway is an active participant in the breathing process rather than a passive conduit of airflow. This finding is not especially surprising since others have observed integrated activity among the resistive segments of the respiratory system (Drettner, 1970, 1979; Blide et al, 1964; Butler, 1960; Ferris et al, 1964; Hvatt and Wilcox, 1961; Speiger and Frank, 1964). Coordinated involvement of the pharynx, glottis and tracheobronchial tree during breathing appears to be of neuromuscular origin (Cole, 1976; Drettner, 1970, 1979). Thus, the changes we observed in the nose between inspiration and expiration apparently follow what had been observed in the larynx and elsewhere (Cole, 1976; Cole et al, 1980; Ingelstedt and Toremalm, 1960; Bartlett et al, 1973; Remmers and Bartlett, 1977; Brain et al, 1977). Gautier et al (1973) and Jackson (1976) provide a plausible explanation for resistive changes in the respiratory cycle. Gautier observed an increase in laryngeal resistance during expiration and suggested that the larynx served as an expiratory brake during breathing. The phasic decrease in glottal opening during expiration increases the duration of expiration and provides adequate time for alveolar gas exchange. Jackson who studied nasocoronary and nasopulmonary reflexes reported that using the larynx as an expiratory brake would be inefficient and suggested that the nose should also act in sequence with the laryngeal response. Dilation of the nasal valve during inspiration and active or passive flattening during expiration could satisfy the requirements of a braking mechanism.

It should be noted that expiratory braking would be useful in speech as well, especially in cleft palate when velar incompetence is present. We have suggested that this phenomenon is the physiologic basis of the nasal grimace (Warren et al, 1985).

The basis of the phasic response of the nasal valve may also explain the paradoxical preference individuals have for nose breathing. Mouthbreathing requires about one-third the effort in terms of work of breathing, yet man is basically a nosebreather. Increased nasal resistance probably contributes to adequate gas exchange.

Our data suggest that the phasic differences in nasal area result from changes in nasal valve patency, although the phenomenon could be the result of effects relating to valve configuration. Insertion of tygon tubes dilated the anterior nares without distorting the nasal valve as was confirmed by subsequent area measurements. More importantly, the phasic difference was not altered, proving that movement of the external nares was not responsible for the effect. Similarly, use of a decongestant also did not alter the phasic response, in spite of the fact that overall nasal airway size increased by 23 percent. The



FIGURE 5 Mean nasal cross-sectional areas when tygon tubing was inserted into the anterior vestibule.



FIGURE 6 Mean nasal cross-sectional areas following administration of the nasal decongestant (Otrivin).

 TABLE 1
 Ratio of Inspiratory Area to Expiratory Area in Normal Adults

Experimental Conditions	MEAN RATIO (Inspiratory Area) (Expiratory Area)
Both passages measured simultaneously	1.11 ± 0.12
Sum of passages measured individually	1.08 ± 0.11
Both passages with tygon tube placement	1.13 ± 0.09
Both passages after Otrivin administration	1.10 ± 0.12

vasoconstriction produced by the decongestant probably opened the airway at the nasal valve by constricting the inferior turbinate which borders on the nasal valve.

Since our technique of nasal airway measurement estimates the smallest cross-sectional size within the airway, it appears that we are measuring the patency of the nasal valve. Our estimates of 0.63 cm² \pm 0.17 during inspiration and 0.56 cm² \pm 0.14 during expiration also are very

close to the measurements made of the valve area in cadavers and by intranasal impressions in vivo (Proctor, 1985; Brain et al, 1977).

Although the nasal valve's primary mechanism of control is unknown, resistance and size of the opening can be altered by the nasal musculature. (Mann et al, 1977; Bridger, 1970; Drettner, 1979). Changes in valve opening significantly affects nasal airway resistance. Normally the valve is responsible for about two-thirds of the resistance within the upper airway (Foster, 1962).

This present study leads to many new questions. We are currently investigating the phasic response in children since nasorespiratory function has been linked by some to dentofacial development. Additionally, individuals with cleft palate demonstrate nasal abnormalities and maxillary deficits which alter the upper airway. Surgical procedures may benefit some individuals and be detrimental to others. Surgeons interested only in a cosmetic result may not always consider the importance of nasal valve function. In fact, we have observed a surprisingly high incidence of mouth breathing in the cleft population and, in many instances, a defective nasal valve appears responsible. These findings will be reported shortly.

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