Relation of Nasal Air Flow to Nasal Cavity Dimensions

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Objective: To investigate the relationship between nasal cavity dimensions and airflow based on measures of acoustic rhinometry (AR) and peak nasal inspiratory flow (PNIF) in a very large sample of mixed rhinologic and nonrhinologic patients.


Setting: Secondary referral ambulatory center and hospital.

Patients: The study population comprised 2523 consecutive adult patients, mainly white, referred to the Department of Otolaryngology–Head and Neck Surgery, Sørlandet Hospital, Kristiansand, Norway, for evaluation of sleep-related disorders (eg, snoring, sleep apnea) or chronic nasal complaints.

Intervention: The subjects underwent AR and PNIF at baseline and after decongestion of the nasal mucosa with xylometazoline hydrochloride. Questionnaires and height and weight measurements were obtained prior to the nasal recordings.

Main Outcome Measure: Associations between measures of AR (volume and area) and PNIF.

Results: Nearly linear relationships were found between PNIF in 4 categories and nasal cavity volumes and minimal cross-sectional areas (analysis of variance, \( P < .001 \); post hoc analysis, \( P < .01 \)). Adjusted associations between 5 of 6 AR measures and PNIF both at baseline and after decongestion were significant (\( P < .001 \) in 9 cases and \( P = .03 \) in 1 case).

Conclusions: Our study indicates statistically significant associations between nasal cavity dimensions and PNIF. The most important structural determinant for PNIF is the minimal cross-sectional area of the nasal cavity.


FUNCTIONING OF THE HUMAN nose is greatly dependent on airflow dynamics. Individual variation in nasal cavity geometry is thought to affect flow rate and flow pattern and hence nasal function. Despite considerable research in this field, disagreement remains about the nature of airflow. Recent advances in medical imaging using computational fluid dynamics enables numerical simulation of airflow patterns in the nasal cavity, a valuable tool for quantifying flow factors in the human nose.1,2 However, the methods are highly specialized and mainly used for research purposes. Furthermore, some challenges remain, eg, the nasal cavities are treated as rigid, static structures2 and analysis is typically based on relatively few models, making the results less applicable to different noses.3

For clinical purposes acoustic rhinometry (AR) and peak nasal inspiratory flow (PNIF) are often used in the evaluation of nasal geometry and airflow, mainly because of their simplicity and noninvasive nature. Surprisingly, a recent larger trial has failed to show any significant associations between the measures these 2 methods generate.4 Knowledge about the in vivo relations between nasal cavity dimensions and airflow is limited, and the claim that dimensions of the nasal cavity affects nasal airflow needs further elucidation.

Our study aimed to investigate the relationship between nasal geometry and airflow using AR and PNIF in a very large sample of mixed rhinologic and nonrhinologic patients.

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CME available online at www.jamaarchivescme.com and questions on page 537
A clinical survey was conducted on 2523 consecutive adult patients, mainly white, referred to the Department of Otolaryngology–Head and Neck Surgery, Sorlandet Hospital, Kristiansand, Norway, for evaluation of sleep-related disorders (eg, snoring, sleep apnea) or chronic nasal complaints between 2001 and 2007. All referred patients were included, along with patients with prior nasal surgery and those taking nasal medications. The subjects underwent AR and PNIF measurements were all obtained on the same day, prior to the nasal recordings.

ACOUSTIC RHINOMETRY

Acoustic rhinometry measures nasal airway cross-sectional area as a function of longitudinal distance along the nasal passage-way following the path of an acoustic pulse. The method is appropriate for anatomic assessment of the nasal airway. An impulse acoustic rhinometer (RhinoMetrics SRE2100 [Rhinoscan version 2.5, build 3.2.3.0]; RhinoMetrics, Lyngby, Denmark) was handled by 3 trained operators throughout our study. Recordings were performed in accordance with published protocols. Three curves from both nasal cavities were averaged to get a mean curve for each side. To account for variations between nostrils due to the nasal cycle, mean values from the left and right side were calculated. The following measures were recorded: minimum cross-sectional area (MCA) in centimeters squared between 0 and 3.0 cm (MCA1), between 3.1 and 5.2 cm (MCA2), and between 0 and 5.2 cm (MCA3) behind the nostril; and nasal cavity volume (NCV) in centimeters cubed between 0 and 3.0 cm (NCV1), between 3.1 and 5.2 cm (NCV2), and between 0 and 5.2 cm (NCV3) behind the nostril. After the initial recordings at baseline, the nasal mucosa was decongested with topical xylometazoline hydrochloride (Otrivin 1 mg/mL; Novartis, Berne, Switzerland), 1 dose given in each nasal cavity, applied in a standardized manner using a hand pump. Ten minutes after administration, allowing the decongestant to take effect, recordings (AR and PNIF) were repeated. Recordings from the posterior nasal cavity were not obtained because they are not considered reliable owing to loss of acoustic energy and consequent underestimation distal to constrictions.

PEAK NASAL INSPIRATORY FLOW

Peak nasal inspiratory flow is a physiological measure indicating the peak nasal airflow achieved during forced inspiration. The method is suggested to be reliable and reproducible and in concordance with other objective tests. In our study, a portable peak flow meter (In-check DIAL; Alliance Tech Medical Inc, Granbury, Texas) was used. Patients were carefully instructed in a standardized technique using the same nasal flow meter equipped with face masks. Three satisfactory maximal inspirations were obtained with the patient in an upright position. The mean value was calculated for subsequent analysis. Maximum flow registration was set to 120 L/min. Peak flows exceeding 120 L/min were recorded as 120 L/min. Recordings were repeated after topical administration of xylometazoline as described in the previous subsection. Calculations were based on both continuous and categorized data; PNIF was divided into the following 4 groups: (1) 0 to 59 L/min, (2) 60 to 89 L/min, (3) 90 to 119 L/min, and (4) greater than 119 L/min.

POSSIBLE CONFOUNDERS

On enrollment, each subject completed a short questionnaire including questions about age, sex, smoking habits, and presence of allergy and asthma. Height and weight were measured at the time of enrollment, using the same measuring device for each subject. Body mass index was calculated as weight in kilograms divided by height in meters squared.

STATISTICAL ANALYSIS

Data were described with mean and standard deviation or median with range when not normally distributed. Unadjusted associations between PNIF categories and AR were calculated using analysis of variance (ANOVA). To uncover adjusted relations between the 2 objective measures, several linear regression models were fitted (only subjects with PNIF values between 0 and 119 L/min were included in these analyses). In addition, both measures were standardized (their mean was subtracted and the values were divided by standard deviation) so that the results could be interpreted as percentage changes. Because of a very large sample size, the model fit was very good. P < .05 was considered statistically significant. When using ANOVA, Bonferroni correction was applied to correct for multiple testing. All analyses were performed with SPSS version 13 (SPSS Inc, Chicago, Illinois).

Sample characteristics are presented in Table 1. Nasal recordings are listed in Table 2. The mean MCA3 at baseline (0.43 cm²) was slightly below the mean values from normative data. The mean PNIF (83 L/min) was not directly comparable with normative data because values above 120 L/min were recorded as 120 L/min, thereby lowering the mean. After decongestion, the mean MCA1 increased by 7%, the mean MCA2 increased by 39%, and the mean MCA3 increased by 9%. The mean NCV1 increased by 4%, the mean NCV2 increased by 51%, and the mean NCV3 increased by 33%. Similarly, the mean PNIF increased by 14% after decongestion. In most subjects, the MCA for the whole nasal airway was located within 3 cm from the na-ris. However, in 17% (baseline) and 12% (after deconges-tion) of the cases, the MCA was located more posteriorly. This was most likely owing to hypertrophy of the anterior part of the inferior turbinate.

Overall, the unadjusted associations between PNIF in 4 categories and AR were always statistically significant both at baseline and after decongestion (ANOVA, P < .001). When performing pairwise comparisons, 14 of 18 possible tests were
significant for baseline values and 15 of 18 tests for values after decongestion (data not shown). The differences at baseline between PNIF categories and MCA1, MCA2, MCA3, and NCV3 are depicted in Figure 1 and Figure 2.

The adjusted associations between AR and PNIF were all in the expected positive direction. A positive estimate of β level means that, for example, a reduction in MCA3, indicating narrower nasal cavities, was correlated with a reduction in PNIF, indicating lower nasal flow. Of 12 AR measures tested, 10 were strongly associated with PNIF. The association between PNIF and MCA1 (β = 38.71; 95% confidence interval [CI], 29.95-47.47), MCA2 (β = 12.11; 95% CI, 7.90-16.32), and MCA3 (β = 53.88; 95% CI, 43.48-64.28) at baseline indicated that a change in MCA1, MCA2, and MCA3 of 1 cm² corresponded with a change in PNIF of 38.71, 12.11, and 53.88 L/min, respectively. Similarly the association between PNIF and NCV2 (β = 4.20; 95% CI, 3.16-5.24) and NCV3 (β = 3.48; 95% CI, 2.54-4.42) at baseline indicated that a change in NCV2 and NCV3 of 1 cm³ corresponded with a change in PNIF of 4.20 and 3.48 L/min, respectively. The association between PNIF and NCV1 did not reach significance. Adjusted associations between PNIF and AR after decongestion of the nasal mucosa showed similar results. The association between PNIF and MCA1 (β = 38.54; 95% CI, 29.30-47.79), MCA2 (β = 3.62; 95% CI, 0.44-6.79), and MCA3 (β = 39.79; 95% CI, 28.86-50.72) indicated that a change in MCA1, MCA2, and MCA3 of 1 cm² corresponded with a change in PNIF of 38.54, 3.62, and 39.79 L/min, respectively. Similarly the association between PNIF and NCV2 (β = 3.16; 95% CI, 2.04-4.29) and NCV3 (β = 2.29; 95% CI, 1.46-3.12) after decongestion indicated that a change in NCV2 and NCV3 of 1 cm³ corresponded to a change in PNIF of 3.16 and 2.29 L/min. The association between PNIF and NCV1 did not reach significance.

To better quantify the strength of the relations among the AR and PNIF measures, we applied linear regression to standardized values, which enabled us to express the correlations in percentage changes. The results are listed in Table 3. As modeled in the previous paragraph, a 10% change in MCA1 at baseline and after decongestion resulted in a 22% and 20% change in PNIF, respectively. A 10% change in MCA2 at baseline and after decongestion resulted in a 14% and 5% change in PNIF, respectively. The strongest associations were found between PNIF and MCA3 at baseline and after decongestion, where a 10% change resulted in a 25% and 22% change in PNIF, respectively. For nasal cavity volumes the relations were slightly weaker. A 10% change in NCV2 at baseline and after decongestion caused a 19% and 15% change in PNIF, respectively. Finally, a 10% change in NCV3 at baseline and after decongestion caused an 18% and 13% change in PNIF, respectively. No significant associations between NCV1 and PNIF were found.

Crude differences between subjects with chronic nasal complaints and subjects with sleep-related complaints were assessed. There were only minor differences in structural nasal measures between these subgroups; subjects with chronic nasal complaints exhibited a slightly lower mean MCA3 compared with subjects with sleep-related complaints. Accordingly, mean PNIF was somewhat lower in
After Decongestion

Lam et al\(^4\) included 290 subjects evaluated for obstructive sleep apnea, with PNIF recordings for 156 subjects. Our article by Lam et al\(^4\) were all in the expected direction in-between AR and PNIF. Still, the correlations presented in the article from Lam et al\(^4\) were crude associations, while our results suggest that both area and volume are important determinants for PNIF.

Our findings are in line with the assumed physiological principles at work; an increase in the size of the nasal airway should allow a higher airflow, as predicted by Poiseuille's law. This is of course a gross simplification because the human nose does not apply directly to the equation. However, area remains an important determinant for nasal air flow, as reflected by our results. Based on the unadjusted associations between PNIF in 4 categories and both volume and MCA of the nasal cavity, the relationship appears to be nearly linear.

The MCA3, representing the physiological nasal valve, seems to be the most important AR measure with regard to nasal flow (PNIF). Both PNIF and MCA3 are significantly associated with subjective nasal obstruction, as we have previously shown.\(^10\) The apparent association between these measures strengthens the internal validity and underlines their importance in clinical decision making. Thus, the combined use of AR and PNIF increases diagnostic power, especially when the tests are mutually confirmatory. Furthermore, our results emphasize

### Table 3. Centered Values of Acoustic Rhinometry and Peak Nasal Inspiratory Flow at Baseline and After Decongestion\(^a\)

<table>
<thead>
<tr>
<th>Variable</th>
<th>At Baseline</th>
<th>P Value</th>
<th>After Decongestion</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>MCA1</td>
<td>0.22* (0.17 to 0.27)</td>
<td>&lt;.001</td>
<td>0.20* (0.15 to 0.25)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>MCA2</td>
<td>0.14* (0.09 to 0.19)</td>
<td>&lt;.001</td>
<td>0.09 (0.07 to 0.10)</td>
<td>.03</td>
</tr>
<tr>
<td>MCA3</td>
<td>0.25* (0.21 to 0.30)</td>
<td>&lt;.001</td>
<td>0.22 (0.17 to 0.26)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>NCV1</td>
<td>0.02 (−0.04 to 0.07)</td>
<td>.55</td>
<td>0.04 (−0.05 to 0.05)</td>
<td>.87</td>
</tr>
<tr>
<td>NCV2</td>
<td>0.19* (0.15 to 0.24)</td>
<td>&lt;.001</td>
<td>0.15 (0.10 to 0.19)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>NCV3</td>
<td>0.18* (0.13 to 0.23)</td>
<td>&lt;.001</td>
<td>0.13 (0.09 to 0.18)</td>
<td>&lt;.001</td>
</tr>
</tbody>
</table>

Abbreviations: MCA1, minimal cross-sectional area between 0 and 3 cm behind the nostril (centimeters squared); MCA2, MCA between 3.1 and 5.2 cm behind the nostril (centimeters squared); MCA3, MCA between 0 and 5.2 cm behind the nostril (centimeters squared); NCV1, nasal cavity volume between 0 and 3.0 cm behind the nostril (cubic centimeter); NCV2, NCV between 3.1 and 5.2 cm behind the nostril (cubic centimeter); NCV3, NCV between 0 and 5.2 cm behind the nostril (cubic centimeter).

\(^a\)Significant variables were adjusted for \(^*\)age and sex; \(\text{none}\); \(\text{d}^\text{age, allergy, asthma; and age and asthma.}\)
the importance of evaluating and treating the narrowest segments of the nasal cavities in patients with flow limitation caused by structural nasal obstruction. Thus, nasal airway management with enhancement of flow properties seemingly depends on adequate correction of nasal valve dysfunction.

One might argue that the correlations between AR and PNIF measures should be even higher, given the aforementioned relations between area and flow. However, the relationship between the structure of the nasal passage and the functional movement of air through it is complex. Dynamic changes in nasal resistance are not fully reflected in AR, which is a static measure. In addition, PNIF may be affected by several factors such as collapse of the soft tissues at the entrance to the naris (the “Venturi” effect), inspiratory effort and compliance, and downstream resistance in the small intrapulmonary bronchioles.

In addition, it is plausible that different subgroups within the sample (i.e., those with sleep-related complaints and nasal complaints), exhibiting slight physiological differences in nasal function and structure, could potentially display different associations than those seen in the sample as a whole. However, the associations between PNIF and AR measures within these subgroups remained statistically significant and in agreement with results from the main analysis. This indicates that the relation between area and flow is universal and highly related to nasal anatomy. Despite statistically significant associations between several measures, the correlation coefficients remained relatively low ($r^2$ between 0.15 and 0.27). Therefore, the structural and functional measures did not fully support each other. However, correlations as a way of expressing an association between a pair of variables can be very misleading, especially when data have a wide range of values, which inflates the size of correlation. In addition, correlations are just crude measures of an association and cannot account for possible confounders. Our findings, however, emphasize that there are strong associations between PNIF and several AR measures.

The study was based on a selected sample that is likely to differ from the general population in terms of nasal anatomy and physiological features. This is partly reflected by an overrepresentation of smokers and a preponderance of male sex due to a large number of male subjects with sleep apnea and snoring in the sample. Respiratory comorbidity could potentially affect the PNIF measurements by limiting the inspiratory effort. Since asthma and allergy were self-reported, limitations are applicable to interpretation and extrapolation of the results. However, the prevalence of self-reported asthma and allergy in our study agrees well with reported prevalences. Another limitation is the lack of distinctions between asthma and chronic obstructive pulmonary disease.

The adjusted analyses were based on subjects with PNIF values between 0 and 119 L/min. One might argue that the exclusion of subjects with higher PNIF values could lead to bias. However, PNIF values above 120 L/min are of only minor clinical interest, since such high peak flows generally excludes nasal obstruction. Furthermore, given the size of our sample and the heterogeneity of our recordings, the range of PNIF is satisfactory to evaluate relations to AR.

Measurements of AR and PNIF represent different aspects of the nasal cavities, namely, structure and flow. Both techniques are highly variable and, for PNIF, highly effort dependent. Their reliability depends on optimal cooperation from the subject, correct instructions from the investigator, and standardized techniques. Close attention was paid to these elements. Because PNIF is based on forced inspiration, it is a relatively crude measure of nasal airflow and does not characterize resting nasal airflow. In addition, the measure provides only a global characterization (overall flow). However, the method has proven to be reliable for assessment of nasal patency and is a valuable tool for clinical purposes.

Our results are based on a very large sample using modern equipment and trained personnel and adjusting for confounders of importance. Therefore, we regard our conclusions as strong. We found statistically significant associations between nasal cavity dimensions and airflow, based on AR and PNIF. Furthermore, our results verify that AR and PNIF correlate well, which strengthens the internal validity. We emphasize the combined use of PNIF and AR as objective diagnostic tools for evaluation of the nasal airway, as well as the importance of identifying and treating the narrowest segments of the nasal cavities for enhancement of flow in patients with structural nasal obstruction.

In conclusion, our study indicates statistically significant associations between nasal cavity dimensions and PNIF. The most important structural determinant for PNIF is the minimal cross-sectional area of the nasal cavity.

Submitted for Publication: June 4, 2008; final revision received September 2, 2008; accepted October 14, 2008.

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Author Contributions: Dr Kjærgaard had full access to all the data in the study and takes responsibility for the integrity of the data and the accuracy of the data analysis. Study concept and design: Kjærgaard and Steinsvåg. Acquisition of data: Kjærgaard and Steinsvåg. Analysis and interpretation of data: Kjærgaard, Cvancarova, and Steinsvåg. Drafting of the manuscript: Kjærgaard, Cvancarova, and Steinsvåg. Critical revision of the manuscript for important intellectual content: Kjærgaard and Steinsvåg. Statistical analysis: Kjærgaard and Cvancarova. Obtained funding: Steinsvåg. Administrative, technical, and material support: Steinsvåg. Study supervision: Steinsvåg.

Financial Disclosure: None reported.

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