A technique to measure the ability of the human nose to warm and humidify air

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Rouadi, Philip, Fuad M. Baroody, David Abbott, Edward Naureckas, Julian Solway, and Robert M. Naclerio. A technique to measure the ability of the human nose to warm and humidify inhaled air. J. Appl. Physiol. 87(1): 400-406, 1999.—To assess the ability of the nose to warm and humidify inhaled air, we developed a nasopharyngeal probe and measured the temperature and humidity of air exiting the nasal cavity. We delivered cold, dry air (19–1°C, <10% relative humidity) or hot, humid air (37°C, >90% relative humidity) to the nose via a nasal mask at flow rates of 5, 10, and 20 l/min. We used a water gradient across the nose (water content in nasopharynx minus water content of delivered air) to assess nasal function. We studied the characteristics of nasal air conditioning in 22 asymptomatic, seasonally allergic subjects (out of their allergy season) and 11 nonallergic normal subjects. Inhalation of hot, humid air at increasingly higher flow rates had little effect on both the relative humidity and the temperature of air in the nasopharynx. In both groups, increasing the flow of cold, dry air lowered both the temperature and the water content of the inspired air measured in the nasopharynx, although the relative humidity remained at 100%. Water gradient values obtained during cold dry air challenges on separate days showed reproducibility in both allergic and nonallergic subjects. After exposure to cold, dry air, the water gradient was significantly lower in allergic than in nonallergic subjects (1,430 ± 45 vs. 1,718 ± 141 mg; \( P = 0.02 \)), suggesting an impairment in their ability to warm and humidify inhaled air.

THE NOSE FUNCTIONS TO condition the temperature and humidity of inspired air (6, 7, 13, 14, 19). How the nasal glands, blood vessels, nerves, epithelium, and other resident cells function in concert in health and disease to achieve this goal remains largely unexplored. One end result of nasal conditioning is to prepare air for gas exchange by the alveoli, which occurs at 37°C and 100% relative humidity (RH).

The air-conditioning capacity of the nose protects the lower airways by warming and humidifying the air from ambient conditions that range from temperatures from −42 to 48°C and from 0 to 100% RH (17). Nasal conditioning occurs at a resting ventilation of ∼5 l/min to sustained flow rates of 20 to 30 l/min before nasal breathing is supplemented with oral breathing (9). The importance of nasal conditioning is exemplified by the work of Shurman-Ellstein et al. (20). Inhalation of the same volume of dry air through the mouth, in contrast to the oronasal route, caused a greater reduction in forced expiratory volume in 1 s in asthmatic patients (20).

At present, the nasal air conditioning process is poorly understood. We undertook the present study to evaluate the influence of inspired air conditions and inspiratory flow rates on nasal air conditioning and used this approach to test the hypothesis that nasal air conditioning is similar in asymptomatic allergic subjects out of their allergy season and normal, nonallergic subjects. Investigations into nasal conditioning of patients with allergic rhinitis are important because of the high prevalence of allergic rhinitis in the US population and its association with lower airway disease (10).

The air-conditioning capacity of the nose can be investigated under different atmospheric conditions. Exposure of the nose to hot, humid air (HHA; 37°C and >90% RH) or cold, dry air (CDA; 0°C and <10% RH) allows assessment of nasal function under two extreme conditions. Similarly temperature and RH measurements can be performed at basal or increased nasal airflow, thereby testing the conditioning capacity of the nose at rest and during increased flow rates, as achieved during moderate exercise. The conditioning property can be investigated in healthy noses as well as in those affected with inflammation, such as allergic rhinitis. The interaction of nasal conditioning and allergic inflammation, with its resultant mucosal hyperreactivity, has not been investigated in detail.

Evaluation of the conditioning capacity of the nose has been hampered by the difficulty of sampling air as it exits the nose. Therefore, we developed a probe to measure the temperature and RH of air exiting the nasal cavity. The probe, equipped with temperature and humidity sensors, is inserted through one nostril into the nasopharynx and continuously samples the airstream immediately after it exits the other nostril. By measuring airstream temperature and RH as it enters the nose and as it exits into the nasopharynx, one can assess the nasal air-conditioning capacity at controlled air temperatures, RHs, and flow rates.

MATERIALS AND METHODS

Nasal probe construction. Nasal probes were constructed from a 14-Fr suction catheter (4-mm outside diameter × 15-cm length; Kendall Healthcare Products, Mansfield, MA). Temperature and RH sensors were inserted into the catheter through circular holes 1 cm from the rounded probe tip. The holes were fashioned to accommodate the sensors and to allow circulation of air around them.

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The temperature sensor was a 10-kΩ glass-bead thermistor (Thermometrics, Edison, NJ), which measures air temperature from −20 to 60°C with an accuracy of ±1°C. The glass bead was suspended in the lumen at the tip of the catheter between two opposing holes. This mounting arrangement permits a rapid response time such that the thermistor settles within 1°C of the final temperature within 3 s after being subjected to a 50°C abrupt change in inflowing air temperature. The RH sensor, a solid-state, thin-film semiconductor device, measures the RH of air over a range of 0–100% with an accuracy of ±2% (Thuder Scientific, Albuquerque, NM). The sensor is constructed of noble metals and a semiconductor molecular diffusion barrier. This structure is highly porous, allowing water molecules easily to penetrate the sensor. As the molecular density varies, so does the conductivity of the device. The variation in conductivity is directly related to the RH. The sensor settles to within ±2% in <5 s after a 50% step change in ambient RH.

The low-level output signals from the temperature and RH sensors were amplified, digitized, and recorded with Atlantis for Windows software (Lakeshore Technologies, Chicago, IL). Readings from the sensors were continuously and simultaneously collected and plotted on the computer screen.

Probe insertion. All patients were seated in a chair during the entire study. Using a nasal speculum and a light source, we inspected each nostril for significant septal deviation or inferior-turbinate hypertrophy, which might hinder probe insertion. The more-patent nostril was chosen as the conduit for the probe and was sprayed with two puffs (0.2 ml) of 0.05% oxymetazoline hydrochloride (Nostrilla, Ciba Self-Medication, Woodbridge, NJ), followed by two puffs (0.2 ml) of 4% lidocaine (Roxane Laboratories, Columbus, OH). Ten minutes later, the probe was slowly inserted through the nose into the nasopharynx. To prevent the sensors from contacting nasal secretions during insertion, the probe tip was covered with thin nylon sheeting that was withdrawn after the probe was positioned in the nasopharynx. The rationale for positioning the probe in the nasopharynx is that it samples the end result of nasal warming and humidification. Nasal endoscopy with a flexible nasopharyngoscope was then performed in the same nostril in which the probe was inserted, for confirmation of its position and status of nasal condition. Because the effects of lidocaine, oxymetazoline, and probe insertion on the conditioning capacity of the nostril are currently unknown, we sealed the “probe” nostril with a wax plug. Thus the probe sensors detected inspired-air conditioning by the unmanipulated nostril.

“Sniff” test. To ensure that the probe sensors retained an adequate response time when positioned in the nasopharynx, the subject forcefully inhaled room air through the nose with the mouth closed. This maneuver created an airflow transiently exceeding 100 l/min. A rapid change in both temperature and RH readings during the sniffs reflected adequate response times for the studies presented here.

Exposure conditions. Preconditioned air was delivered through a nasal continuous positive airway pressure mask (Respirronics, Murrysville, PA) firmly applied to the face. A spacer was also placed to avoid pressure from the mask over the nasal bridge. A second probe, similar to the one in the nasopharynx, was positioned within the mask for measuring the temperature and RH of inhaled air before its entry into the nose. For CDA challenges, dry air from compressed air tanks was chilled by passage at prechosen flow rates through copper tubing submerged in refrigerated ethanol (FTS Systems, Stone Bridge, NY). CDA was directed into the nasal mask through flexible rubber tubing; air flowing into the mask then entered the patent nostril and exited through the mouth. The precise temperature delivered to the nose depended on the flow rate and averaged −19°C at 5 l/min to 10,5°C at 10 l/min and 0.8°C at 20 l/min. For delivery of HHA challenges, air from the compressed air tank was passed through a bubble humidifier immersed in a hot water bath. In this way, air at 37°C and >90% RH was delivered to the nasal mask.

The patients were instructed to breathe through their mouth while air was blown continuously and unidirectionally through the nose at flow rates of 5, 10, and 20 l/min. This experimental design permitted the development of steady-state conditions that were easily measurable by the probe and circumvented the potential problems associated with exhalation. The range of flow rates from 5 to 20 l/min spans flows at rest to values at which most individuals switch from nasal to oronasal breathing (9). It should be noted also that air blown unidirectionally prevents air exiting from the lung at 37°C and 100% RH from condensing on the probe and interfering with the sampling of air in the nasopharynx. The duration of air sampling in the mask and nasopharynx was 22 min, with data collected only during the last 15 min of each challenge used for analysis. The initial 7-min period of each challenge was disregarded because it reflects the time necessary for the temperature in the nasopharynx to reach a steady state.

Data handling. During CDA and HHA challenges, temperature and RH values were collected simultaneously by the sensors located in the mask and in the nasopharynx. Water content of air (WC) is a function of its temperature and RH, and was computed (2) from temperature and RH as

$$WC = \left[ 2.32 + \left( \frac{0.59 \cdot T}{1 - 0.011 \cdot T} \right) + 0.00063 \cdot T^3 \right] \cdot RH/100 - D$$

where WC is in milligrams per kilogram; T and RH are the temperature and relative humidity values of air at 760 mmHg, respectively; and D is the density of air (1.2 g/l at 20°C). Total water lost by the nasal mucosa [water gradient (WG)] during a 15-min challenge was calculated from the difference in water contents of inspired and nasopharyngeal air, the flow rate, and the challenge duration (15 min).

We also calculated a “total WG” as the sum of nasal WGs during each of three flow rates tested (5, 10, and 20 l/min). This measurement approximates the area under the curve. It is an easy way to compare differences between groups and increases appreciation of variability between subjects and changes of individual subjects’ responses to interventions.

Subjects. Eleven healthy, nonallergic subjects were recruited for this study on the basis of the absence of a history of chronic or frequently recurrent nasal symptoms and negative skin-prick testing to seasonal and perennial allergens. Twenty-two seasonally allergic subjects with positive skin prick testing were also studied outside their allergy season (Table 1). They were asymptomatic and taking no medications for their nasal symptoms within the 1 mo before the study. During their allergy season, 19 subjects used over-the-counter antihistamines and/or decongestants, 2 used prescription nonsedating antihistamines, and 1 used intranasal steroids. In studies not using all subjects, the age and gender differences of individual subjects’ responses to interventions.

Table 1. Patient characteristics

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<th>Allergic Subjects</th>
<th>Normal Subjects</th>
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<tr>
<td>n</td>
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<td>11</td>
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<tr>
<td>Age (mean), yr</td>
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<td>Males/group</td>
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were similar to the group as a whole. Exclusion criteria
included a history of perennial allergic or nonallergic rhinitis
and the presence of nasal symptoms suggestive of a common
cold at the time of study. All subjects underwent nasal
endoscopy before and after probe placement. This demon-
strated no mucosal or structural abnormalities. The study
was approved by the Institutional Review Board at the
University of Chicago, and all subjects gave informed, written
consent before participation.

Study design. On presentation to the laboratory, the sub-
jects rested for 15 min before the initiation of the experiment
so that their noses adjusted to the room temperature and RH
in the laboratory, which were usually 22°C and
10% RH. We
prospectively assigned allergic and nonallergic subjects ran-
donically to breathe HHA and CDA on two separate visits. We
also tested the reproducibility of the response to CDA chal-
lenge in both groups by repeating the CDA challenge on
separate days two times in allergic subjects and three times
in nonallergic subjects. All visits were separated by at least
48 h to avoid potential effects of one challenge on another.

Statistical analysis. Statistical analysis was performed
with parametric statistics. Multiple, repeated measurements
were compared by ANOVA, and post hoc analysis was per-
formed by using Fisher’s test of least significant difference.
Comparison of results within the same group of subjects
undergoing different exposures was done by paired t-test.
Nonpaired comparisons (e.g., allergic vs. nonallergic subjects)
were performed with the nonpaired t-test. Two-tailed P
values ≤ 0.05 were considered to indicate significance. The
data are presented as means ± SE.

RESULTS

Sniff test. To ensure that the temperature and RH
sensor continued to function after placement in the
nasopharynx, subjects were asked to breathe room air
and perform the sniff test periodically during the
experiment. The data from a representative patient are
shown in Fig. 1. Room air at 22°C, 30% RH was warmed
up in the nasopharynx to 32°C when the subject was
quietly breathing. With forceful inhalation, the tempera-
ture dropped briefly to 22°C and was then restored back
to 32°C. The RH tracing behaved in a similar manner.
At rest, the RH in the nasopharynx was 100%. With
forceful inhalation, there was a brief drop in the RH to
between 10 and 20%, followed by its restoration to
100%. This maneuver shows the adequacy of time
responses for our studies. Sniffs performed during and
after CDA or HHA exposures showed similar results.

Exposure to HHA. Eleven nonallergic and 8 allergic
subjects were subjected to HHA (37°C; >90% RH) at
flow rates of 5, 10, and 20 l/min, each lasting 22 min.
The mean nasopharyngeal temperatures at 5, 10, and
20 l/min were 35.7 ± 0.24, 36.0 ± 0.3, and 35.9 ± 0.4°C,
respectively (Fig. 2). There was no statistically signifi-
cant difference in nasopharyngeal temperatures at the
three flow rates (P = 0.56) and no difference between
allergic and nonallergic subjects (P > 0.05).

Exposure to CDA. Eleven nonallergic and 8 allergic
subjects were exposed to CDA at flow rates of 5, 10, and
20 l/min. The tracing from one individual is shown in
Fig. 3. The temperature in the mask dropped from 31 to
26°C and then plateaued at ~23°C. The nasopharyn-
geal temperature changed from 36 to 32°C. Increasing
the flow to 10 l/min lowered the mask temperature to
20°C, and the nasopharyngeal temperature decreased
to 25°C. At a flow of 20 l/min, the mask temperature
decreased to ~5°C and the nasopharyngeal temperature
to 20°C. RH in the mask was consistently at 0%
and that in the nasopharynx at 100% with all three flows in all subjects.

The nasopharyngeal temperatures at 5, 10, and 20 l/min for all subjects during exposure to CDA were 33.4 ± 0.7, 30.5 ± 1.1, and 25.9 ± 1.4°C, respectively (Fig. 2). The nasopharyngeal temperature fell significantly with increasing flow rates (P < 0.0001). Post hoc analysis of the nasopharyngeal temperatures obtained at different flow rates showed a statistically significant difference between the temperatures obtained at 5 l/min compared with that obtained at both 10 and 20 l/min (P < 0.05), as well as a significant difference between temperatures obtained at 10 compared with 20 l/min (P < 0.05). Furthermore, nasopharyngeal temperature during CDA was consistently lower than those during HHA at each flow rate (P < 0.01; Fig. 2).

The sum of nasopharyngeal temperatures recorded at the three different flow rates during a single experiment was calculated for all subjects after HHA and CDA exposure. The average sum of nasopharyngeal temperatures after HHA exposure was 107.7 ± 0.8°C, whereas that after CDA exposure was 89.8 ± 2.7°C. There was a statistically significant drop in the sum of nasopharyngeal temperatures after CDA exposure compared with HHA exposure (P = 0.0001).

WG. The WG across the nose was calculated for all three flow rates in subjects (n = 19) after HHA and CDA exposure. After HHA inhalation, the WG at 5, 10, and 20 l/min was 23.3 ± 12.5, −52.2 ± 31.8, and −159.2 ± 66.7 mg, respectively (Fig. 4). ANOVA and post hoc analysis showed a significant difference between WG at 5 and 20 l/min (P < 0.05), as well as between 10 and 20 l/min (P < 0.05) flow rates. After CDA challenge, the WG at 5, 10, and 20 l/min. was 297.6 ± 12.6, 509.1 ± 32.0, and 794.1 ± 62.1 mg, respectively (Fig. 4). Again, ANOVA and post hoc analysis showed that the WGs at 10 and 20 l/min were significantly higher than that at 5 l/min (P < 0.05) and that the WG at 20 l/min was significantly higher than that obtained at 10 l/min (P < 0.05).

WG values were higher during CDA than during HHA at all three flow rates (P < 0.01). The mean total WG after HHA exposure was −188.1 ± 109.6 mg, suggesting that water condenses on the surface of the nasal mucosa during this process. After CDA challenge, the mean total WG at all three flow rates was 1,600.7 ± 96.2 mg, which represents the average amount of water evaporated from the nasal mucosa to warm and humidify the inspired air. There was a statistically significant difference in total WG values obtained during HHA visits and CDA visits in all subjects (P = 0.0001).

Reproducibility. The reproducibility of the nasal response to CDA challenge was studied in eight nonallergic subjects on three separate visits (Table 2). During all three visits, there were flow-dependent significant increases in the WG across the nose. After CDA challenge, the mean total WG values for the three visits were not statistically different (P = 0.56) (Fig. 5). The coefficient of variation (%) (SD/mean × 100%) of total

Fig. 3. Representative data from 1 nonallergic subject after exposure to CDA. The y-axis reflects temperature and relative humidity, and x-axis is time. Subject was exposed to 3 increasing flows of CDA (5, 10, and 20 l/min) for a total duration of 22 min each. RH(NP), relative humidity in the nasopharynx; RH(MK), relative humidity of inhaled air as measured in mask applied to patient’s nose; T(NP), nasopharyngeal temperature; T(MK), temperature of inhaled air in mask.

Fig. 4. Water gradient across the nasal cavity after exposure to either HHA or CDA at 3 flow rates (5, 10, and 20 l/min). Values are means ± SE of 19 subjects. *P < 0.05 vs. 5 l/min, †P < 0.05 vs. 10 l/min, **P < 0.01 vs. CDA values at same flow rates.
The reproducibility of CDA response in nonallergic subjects was studied in two CDA visits (Table 3). On each visit, there were significant differences in the WG across the nose, and the mean total WG was similar on each visit (P = 0.8145; Fig. 5). The coefficient of variation of total WG in these patients ranged between 12 and 22.3% and averaged 8.8%.

Allergic vs. nonallergic subjects. The response to CDA inhalation was compared between 11 nonallergic subjects and 22 allergic subjects. Allergic subjects had significantly lower nasopharyngeal temperatures than did nonallergic subjects at 5 l/min (31.7 ± 0.5°C vs. 34.6 ± 0.9°C, respectively; P = 0.0004) and 10 l/min (28.2 ± 0.5°C vs. 32.2 ± 1.5°C, respectively; P = 0.003). When allergic were compared with nonallergic subjects, there was a significant difference in WG values obtained at 5 and 10 l/min as well as in the total WG (Fig. 6).

**DISCUSSION**

Our capacity to measure the ability of asymptomatic allergic and nonallergic individuals to warm and humidify air in the nasopharynx is novel. Our demonstration of a difference in the ability of these groups to perform this function is exciting. Unfortunately, we have no knowledge of how inflammation or hyperreactivity affects nasal air conditioning. On the basis of a theoretical model, Hanna and Scherer (12) predict that the blood temperature distribution along the airway wall and the total cross-sectional area and perimeter of the nasal cavity are the two most important parameters of the human air conditioning response. Allergic inflammation would be predicted to affect both parameters. Because the allergic individuals were asymptomatic and outside their allergy season, we do not know the state of inflammation in their nasal mucosa nor the blood flow and shape of the nasal cavities at the time of testing. Endoscopic evaluation of the nasal cavity showed no evidence of inflammation in the allergic subjects or differences compared with the nonallergic, normal subjects.

We developed a means to quantify the ability of the nose to warm and humidify air. The technique involves the use of one nostril as a conduit for a catheter containing sensors for continuous sampling of air exiting the opposite nostril. The measurements obtained in the nasopharynx do not determine where the changes occurred during the passage of air through the nasal cavity, but rather they yield the end result of nasal conditioning. The exact location for conditioning of air within the nose may vary in individuals or disease states and may influence nasal function, but it is the air exiting from the nose that would be expected to influence the lower airway. Another reason for sampling the nasopharynx is the ability to place the probe in the same location reproducibly.

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The air delivered to the nose for conditioning was controlled. We studied two relatively extreme condi-
tions: HHA (37°C, >90% RH humidity) and CDA (0–5°C, <10% RH). Airflows approximating the range of nasal flows during rest and mild exercise were chosen (9). Unidirectional flow was chosen instead of bidirectional flow as occurs with breathing. This procedure was selected to provide steady-state conditions that might not be reached because of rapid changes that might occur as a result of heat sinks within the nose, the measuring devices, and the air-delivery system. Additionally, the unidirectional flow prevented the mixing of exhaled air from the lungs and reduced the likelihood of HHA from the lungs condensing on the sensors and giving inappropriate assessment of nasal function. This latter phenomenon was of particular concern for the RH sensor.

Although we expected the air to be fully saturated, we needed to ensure that the humidity sensor was functioning. To this end, we developed the sniff test. By forcible inhaling of air through a partially occluded nose, velocities of air estimated to exceed 100 l/min can be obtained for a few seconds. To our advantage, these flow rates exceeded the nasal mucosa’s ability to saturate air fully at reduced temperatures, and therefore a drop in RH and temperature was detected by the nasopharyngeal probe, documenting its functionality.

When warm, moist air was breathed (air similar to that in the alveoli), the nose absorbed a little water. Normally, the temperature of the anterior nasal mucosa is 32°C (7). This surface temperature is slightly below that of the inspired air (37°C), and water would be expected to condense on the nasal mucosal surface (13, 14).

Breathing of CDA led to wide individual variability in the temperature recorded in the nasopharynx. Nevertheless, the RH remained at 100%, although the absolute humidity or water content varies with temperature. Furthermore, we demonstrated the reproducibility of these observations by performing repeated measurements on the same individual on different days. Our measurements proved to be fairly reproducible for in vivo human studies.

Similar measurements have been made previously by only a few investigators and were performed only in small numbers of healthy subjects (1, 6, 7, 13, 14, 19). Some studies employed discontinuous sampling techniques, whereas others used invasive techniques, such as cricothyroid membrane puncture in which 5 of 16 subjects fainted as a result of the procedure (13). Our preliminary data measuring temperature and RH after exposure to CDA are consistent with previously published results.

For a full understanding of the ability of the nose to condition air, we must both observe the organ as a whole and examine its individual components. These structural components include a pseudostratified, columnar epithelium with a continuous basement membrane (18); a subepithelial capillary system (5), a distensible, cavernous, vascular network; tubuloalveolar, seromucous glands and goblet cells (21); parasympathetic and sympathetic innervation (16); immunohistochemical evidence of a nonadrenergic, noncholinergic nervous system (3, 4); and the presence of submucosal inflammatory cells. Focusing solely on the individual components of the nose or an isolated element of allergic inflammation may distort the importance of their contribution to overall function. Similarly, focusing solely on the whole response may miss compensatory shifts in the reactivity of the individual components. For example, air may be conditioned to the same extent during an allergic response but only because of a compensatory response in the glands and blood vessels. Our challenge will be to determine the reason for the difference between asymptomatic allergic and nonallergic subjects.

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REFERENCES