Airway cooling in asthmatic and nonasthmatic subjects during nasal and oral breathing

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It has been suggested that nasal breathing attenuates the airway obstruction that follows physical exertion in asthmatics. In an effort to determine the reason for this protection, we had nine asymptomatic asthmatics and five normal subjects inhale subfreezing air at equal ventilations through either their noses or mouths in a random fashion while we measured the temperature in the retrotracheal esophagus (Trt). Pulmonary mechanics recorded before and after voluntary eucapnic hyperventilation simulating moderately heavy workloads demonstrated a mean fall in forced expiratory volume in one second (as a representative variable) of 28.6% ± 4.8% (SEM) and 7.5% ± 1.9% from control in the oral and nasal challenges, respectively, in the asthmatic subjects ($p < 0.001$). Measurement of Trt during hyperventilation showed a mean fall of 2.7° ± 0.05° C with oral breathing compared with 0.4° ± 0.2° C with nasal breathing in this group ($p < 0.0001$) and a linear relationship between the degree of airway cooling and the severity of subsequent bronchoconstriction ($r = 0.81$). The normal subjects showed similar changes in temperature but did not change their lung function. These data demonstrate that nasal ventilation minimizes airway cooling in both normal and asthmatic individuals through more efficient conditioning of inspired air, and it is through this mechanism that this form of respiration protects against exercise-induced bronchospasm. (J ALLERGY CLIN IMMUNOL 69:354, 1982.)

It has been reported that the bronchospastic response to an exercise challenge can be reduced in asthmatics if they inspire through their noses.[1] This observation has given rise to the concept that there are temperature-sensitive, "irritant-like" receptors in the posterior pharynx, which are essential to the pathogenesis of EIB.[1] Recent studies, however, have failed to find any role for such receptors in either the pharynx[2] or airways,[3, 4] thus opening up questions as to the mechanism by which nasal breathing produces its beneficial effects. Since there is a growing body of evidence that demonstrates that the initial stimulus for the production of airway obstruction is cooling of the intrathoracic airways as a consequence of their participation in the conditioning of inspired air,[5-9] and since the nose is more efficient at warming and humidifying the inspired air than is the mouth,[10-12] we wondered if nasal breathing would reduce respiratory heat loss. If this were so, then it should be possible to record differences in the magnitude of airway cooling between nasal and oral respiration. To assess this possibility, we performed a series of experiments in which we measured Trt and Trc during both forms of breathing and recorded lung mechanics before and after inhalation. Our observations form the basis of this report.

METHODS

Nine atopic persons (six men and three women, mean age 23.4 ± 1.7 yr SEM) with asthma as defined by the American Thoracic Society[13] and five normal individuals (four men and one woman, mean age 29.6 ± 2.3 yr) served as our subjects. All the asthmatics were asymptomatic at the time of investigation, and none was using medication on a regular basis. Therapy was withheld for at least 24 hr before

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Abbreviations used

FEV_1:	Forced expiratory volume in one second
SG_{AW}:	Specific conductance
RV:	Residual volume
Trc:	Temperature in the retrocardiac esophagus
Trt:	Temperature in the retrotracheal esophagus
T_I:	Temperature of the inspired air
$P_{ET_{CO_2}}$:	End tidal carbon dioxide tensions
$\dot{V}_E$:	Minute ventilation
EIB:	Exercise-induced brochospasm

any study day. All the asthmatics and all but one of the normal subjects were nonsmokers. None of our subjects had rhinitis or other pathologic nasal conditions. Informed consent was obtained from each patient.

Using a constant-volume variable-pressure plethysmograph that was serially interfaced to a time-based recorder and a minicomputer, we measured airway resistance and total lung capacity and its subdivisions by the techniques of Dubois et al.[14, 15] Resistance was converted to its reciprocal, conductance, and was expressed as a ratio of conductance to volume, termed SG_{AW}.[16] Four to five measurements of each variable were obtained and the mean was computed. These data were considered acceptable if their coefficients of variation were 5% or less. The subjects next performed maximum forced exhalations in triplicate, using a waterless spirometer. FEV_1 was determined by standard techniques.

Subjects inspired subfreezing air from a compressed source through a heat exchanger that was externally cooled by circulating isopropyl alcohol.[2, 5-9] T_I was continuously recorded in all experiments by a thermocouple situated in the airstream 10 cm upstream from the mouthpiece. The water content of the gas inspired from the exchanger, as measured by a change in weight in glass drying tubes,[2, 5] was less than 0.05 mg H_2O/L of air and for the purposes of this study was considered to be zero.

During hyperventilation, expired gas was directed away from the exchanger into a reservoir balloon that was being constantly evacuated at a known rate. The subjects were coached to respire so as to keep the balloon filled. In so doing, their $\dot{V}_E$ precisely matched the rate of emptying the balloon and could be set to any desired amount.[2, 5, 8] $P_{ET_{CO_2}}$ were continuously monitored at the mouth or nose by means of a Beckman LB-2 analyzer, the output of which was displayed on the oscilloscope of an analogue recorder. A mixing valve at the inspiratory port of the exchanger permitted the addition of sufficient carbon dioxide to keep $P_{ET_{CO_2}}$ at resting eucapnic concentrations. As in other studies, $P_{ET_{CO_2}}$ ranged between 37 and 42 torr.[2, 5, 8] The target $\dot{V}_E$ chosen for each subject matched the maximum amount that each was able to sustain with nose breathing in a preliminary study. Nasal respiration was performed by replacing the mouthpiece on the heat exchanger with a pediatric anesthesia mask that was secured tightly around the subject's nostrils. The inlet diameters of both the mask and mouthpiece were similar.

In seven of the nine asthmatics and five normals, we measured Trc and Trt during both forms of breathing. As in previous studies,[5-9] we defined the anatomic relationships of each subject's esophagus to other mediastinal structures by having him or her swallow a balloon-tipped catheter to locate the site of maximum cardiac artifact and the lowest point in the esophagus at which movement of the trachea produced a pressure artifact. When these distances were known, they were used to select the sites for placement of the thermal probes. After the catheter was removed, two vinyl-sheathed copper constantan thermocouples (outer diameter, 2.0 mm) were inserted into the esophagus, one to the level of the point of maximum cardiac artifact and the other to that of maximum tracheal artifact. No anesthesia was employed. To minimize nasal resistance, one thermocouple was inserted through each nostril. To ensure their constant spatial relationship to each other and to the subjects, the wires were marked and securely taped to the face.

Both thermocouples were matched for response times using water baths of various temperatures, and the entire system was calibrated against a National Bureau of Standards Thermometer. The 90% response time was 1.5 sec and the accuracy was $\pm 0.1°$ C. Simultaneous measurements of Trt and Trc areas were determined prior to, during, and after hyperventilation.

The experiments consisted of having the subjects perform isocapnic hyperventilation of subfreezing air at their predetermined $\dot{V}_E$ via their noses or their mouths in a random fashion for 4 min. Pulmonary mechanics were measured before and approximately 5 min after completion of each challenge. $\dot{V}_E$, T_I, and water content of inspired air were kept constant for both nose and mouth breathing. Both challenges were performed on the same day and were separated by an interval of 1 to 2 hr to allow mechanics to return to baseline. Previous work has demonstrated that this duration of rest between thermal challenges provides adequate time for recovery.[2, 5-9]

Data were analyzed by paired and unpaired t tests and a two-factor analysis of variance.

RESULTS

In the asthmatics, $\dot{V}_E$ averaged 43 ± 1.8 and 44 ± 1.5 L/min (SEM) with nose and mouth breathing, respectively, and T_I equalled $-18° \pm 1.9°$ C nose and $-19° \pm 1.0°$ C mouth. In the normal subjects, the values for these variables were quite similar ($\dot{V}_E = 42.5 \pm 2.5$ L/min nose, 42.6 ± 2.5 L/min mouth; $T_I = -19.8 \pm 2.3°$ C nose, $-19.5 \pm 2.1°$ C mouth). There were no significant differences between the nasal and oral challenges in either group nor were there any significant differences between the asthmatics and normal subjects.

When the asthmatics inhaled subfreezing air through the mouth, the expected changes in pulmonary mechanics were produced (Fig. 1). FEV_1 and SG_{AW} fell 29% and 56%, respectively, from their control values, and RV rose 35%. Although there were no sig-

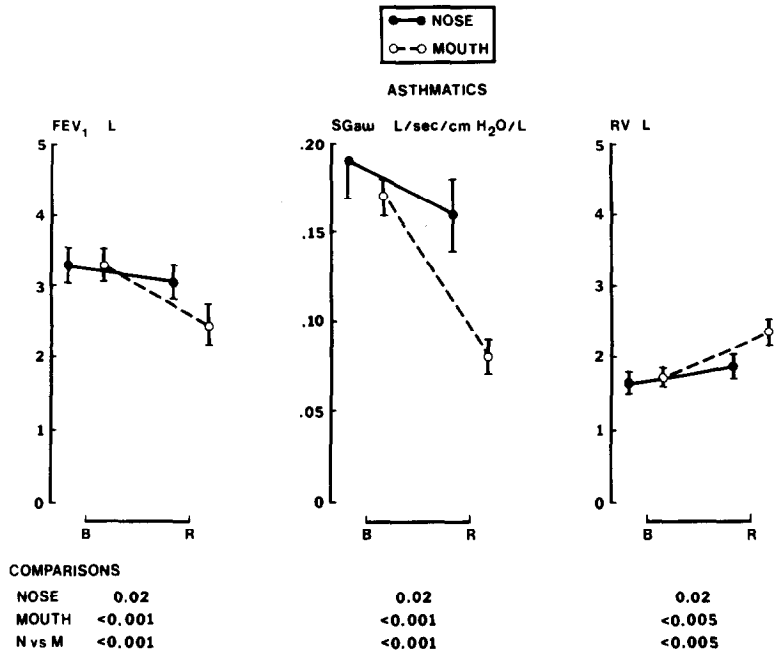

FIG. 1. Alterations in pulmonary mechanics induced by nose and mouth breathing in asthmatic subjects. The data points are mean values and the brackets are one standard error. *B,* Baseline; *R,* response after challenge.

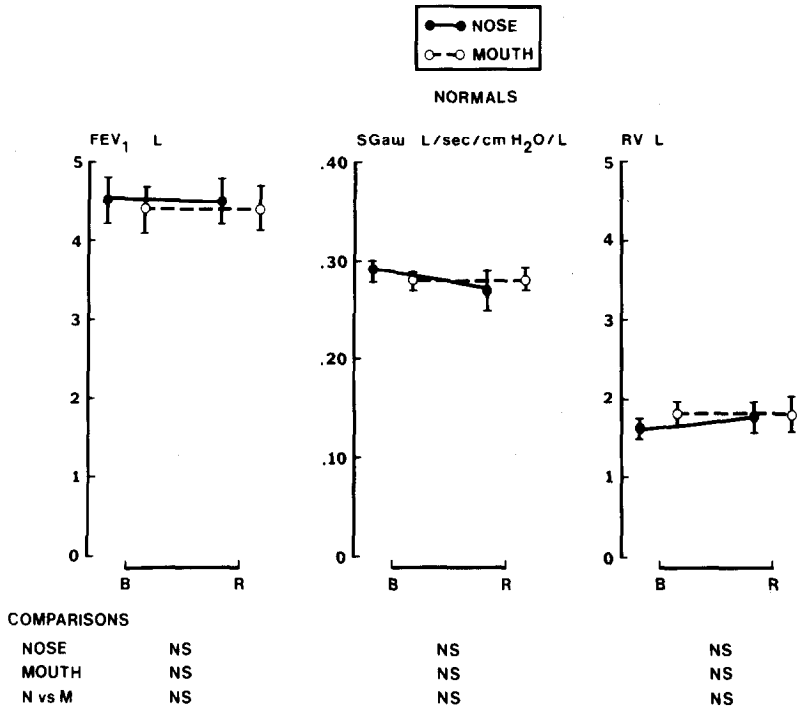

FIG. 2. Alterations in pulmonary mechanics with nose and mouth breathing in normal subjects. The format is identical to that of Fig. 1.

nificant differences in baseline mechanics or in the application of the stimulus, the obstructive response was significantly attenuated with nose breathing. Under these circumstances, FEV_1 and SG_{AW} were reduced only 7.5% and 16%, respectively, whereas RV increased 11%. There were no changes in pulmonary function in the normal subjects with either route of respiration (Fig. 2).

Esophageal temperature measurements for both groups of subjects are shown in Figs. 3 and 4. Trc remained relatively constant during hyperventilation in both groups during both forms of respiration, and

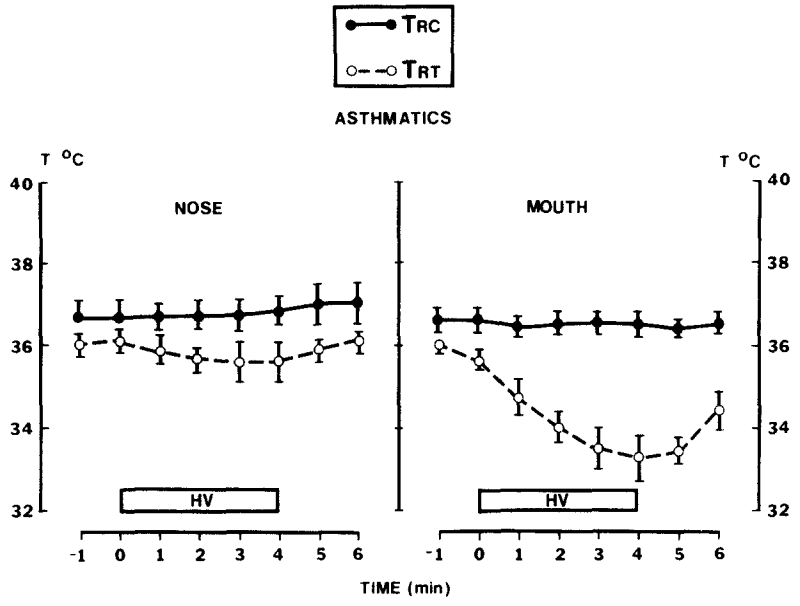

FIG. 3. Changes in Trc and Trt in asthmatic subjects during nose and mouth breathing. The data points are mean values and the brackets represent one standard error. The rectangles on the abscissa denote the time spent hyperventilating *(HV).*

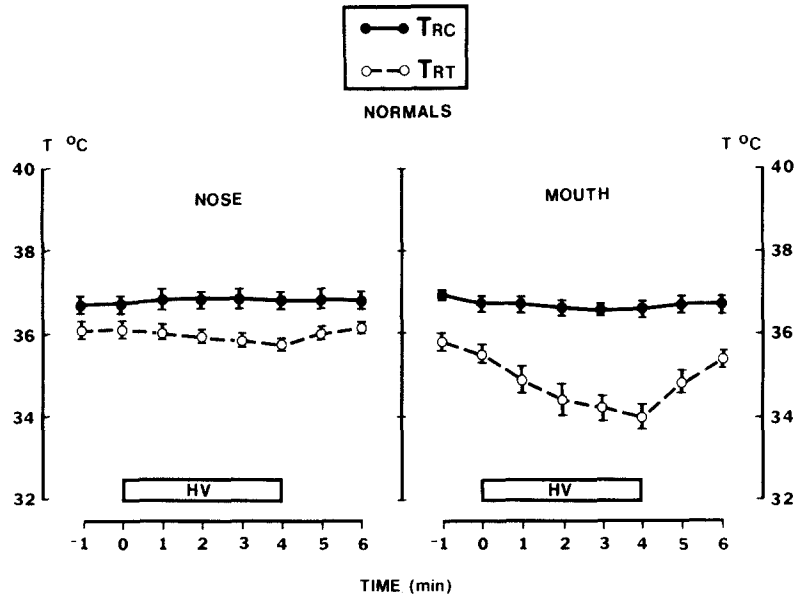

FIG. 4. Changes in Trc and Trt in normal subjects during nose and mouth breathing. The format is identical to that of Fig. 3.

Trt fell more with mouth breathing than with nose breathing. In the asthmatics (Fig. 3), by the fourth minute of hyperpnea, the mean Trt had fallen 0.4° C from its control value with nasal ventilation and 2.7° C when the mouth was used ($p < 0.001$). Similar changes were observed in the normal subjects (Fig. 4) (nose Δ Trt = 0.2° C, mouth Δ Trt = 1.6° C; $p < 0.001$). Although these changes were larger in the asthmatics, they did not reach statistical significance.

Fig. 5 demonstrates that there is a linear relationship between the degree of airway cooling (as measured by a fall in Trt) and the severity of the subsequent bronchoconstriction. It is clear that nose and mouth breathing represent opposite ends of a continuum, with overlap in the middle. At one extreme, when the temperature change is small (as occurs in most subjects with nasal respiration), the obstructive response is small. However, in cases in which the temperature change is great, the response increases as well. At the other end of the spectrum, the mean changes in temperature with mouth breathing are large and therefore produce more severe obstruction.

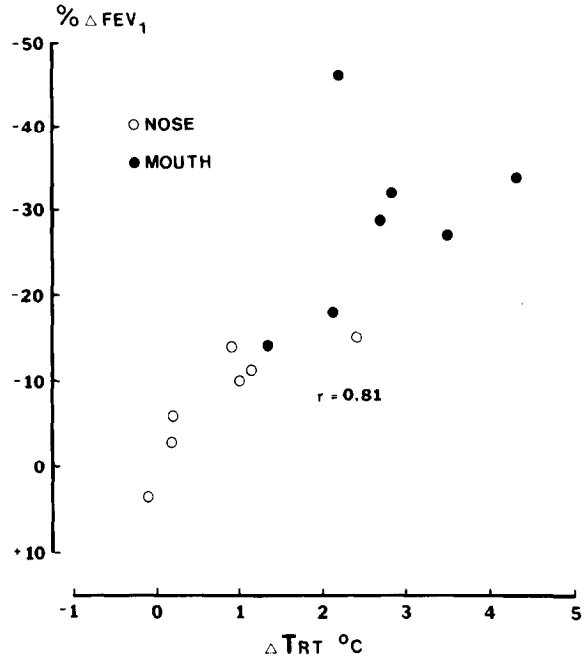

FIG. 5. Relationship between the degree of airway cooling (Δ Trt) and the severity of the obstructive response (%ΔFEV$_1$) with nose and mouth breathing.

DISCUSSION

The results of the present study demonstrate that when asthmatic subjects respire large volumes of air through their noses, their airways are less cooled and they develop less severe bronchial obstruction than with mouth breathing at identical ventilations and inspired air conditions. When these observations are combined with those that document that airway cooling and respiratory heat loss (irrespective of how they are produced) are essential for the production of obstruction[5–9] and that airway and pharyngeal anesthesia have no effect on this response,[2–4] it is an easy matter to explain the protective effect of nasal breathing within the framework of the respiratory heat flux hypothesis of EIB.

Our findings regarding the beneficial consequences of nasal breathing confirm the work of Shturman-Ellstein et al.[1]; however, our observations on airway temperature do not support the ideas put forth by these authors to explain the mechanism of this phenomenon. These investigators had asthmatic children exercise while breathing either through their noses or mouths and found that the former mode of respiration protected against the obstructive consequences of the work. They postulated that the reasons for this behavior were related to the stimulation of irritant-like receptors in either the oropharynx or nasopharynx. No data were offered in support of the existence of such receptors, and, in fact, subsequent investigations have been unable to find any evidence to uphold the concept that thermally or mechanically sensitive neuroreceptors in the posterior pharynx or airways are important in the pathogenesis of EIB.[2–6] Rather, from the current work it can now be appreciated that the reason that nasal breathing minimizes the obstructive response is that the nose is a better heat exchanger than the mouth and as such attenuates the degree of airway cooling.

Based on the data in the literature, our conclusions regarding the efficiency of the nose as a conditioner of inspired air seem quite reasonable. In 1954, Cole[10] showed that nasal breathing heated (and presumably humidified) the inspired air more completely than did oral breathing. When he had normal subjects at rest breathe cold air, he found the temperature of the air at the level of the pharynx to be considerably higher when inspiration took place through the nose than when through the mouth.[10] This temperature difference became much smaller at the level of the trachea. When thermal demands were increased by hyperventilation, the difference in temperature between air inhaled through the nose vs the mouth became greater and extended more deeply into the respiratory tract.

In addition to its effect during inspiration, the nose is also a more efficient reclaimer of heat during expiration. Several investigators have observed the temperature of the expired air at the nasal port to be significantly lower than at the oral port, even though the heat and water content of the expiratory air at the level of the larynx were equal for both nose and mouth breathing.[10, 12] Further, this effect was not dependent on the level of ventilation, at least over moderate ranges.[10, 11]

Our results confirm these earlier findings in that we have shown the superiority of the nose as a heat exchanger and that this superiority becomes more obvious during hyperventilation with subfreezing air. We extend the results of previous work by comparing the type of changes in intrathoracic temperature that can develop with both forms of breathing and by applying these ideas to asthmatic subjects whose airways are far more responsive to cooling than are normal ones.[7–9] Although exact quantitation of the temperatures within the airways was not made in our study, the sharp drop in retrotracheal esophageal temperature shown with oral breathing vs the small change with nasal breathing indicates that the actual differences in the airways are much more dramatic. Thus we have shown a practical and functional application of the difference between nose and mouth breathing.

Does the protective effect of nose breathing on the obstructive response to exercise have any clinical or therapeutic value? The answer seems to be a qualified

yes. Because of the lesser magnitude of respiratory heat loss with nasal breathing, nasal respiration during tasks requiring relatively low levels of ventilation (such as jogging, brisk walking, bicycling, etc.), particularly if performed in cold environments, may prevent totally or significantly attenuate the development of the ensuing bronchospasm. However, it must be remembered that respiration exclusively through the nose can only be performed over a relatively restrictive range of ventilations. Niiminaa et al.[17] examined the point at which individuals switched from nasal to oral breathing and found this to be influenced by the nasal resistance and the subject's perception of his or her work of breathing. In this particular study,[17] the switching point occurred at a mean $\dot{V}_E$ of 35 L/min, whereas in other studies this value has varied from approximately 30 to 60 L/min.[18, 19] Since nasal resistance varies directly with the airflow and indirectly with the temperature of the inspired air,[17, 20, 21] these factors undoubtedly account for at least part of the variability reported in the literature.

In allergic asthmatics the situation is more complex. Since many of these people have chronic rhinitis, their baseline nasal resistance is increased. Thus the nasal work of breathing could be much higher at a given airflow than that in normal subjects, and so the level of ventilation at which the switching point to oral augmentation occurs could be lower. Theoretically, this problem could be overcome with the use of decongestants. Although there is little doubt that this form of therapy can reduce the nasal work of respiration, and so increase airflow, it is also potentially detrimental in that the local vasoconstriction might diminish the heat exchanging properties of a nose and so facilitate airway cooling. It remains to be determined which result will predominate.

In any event, on the basis of the results of this study, we expect that if asthmatics were trained to breathe through their noses they would experience much less severe postexertional obstruction than if the same ventilation occurred through their mouths. Given the ventilatory limits imposed by the nose, however, the work loads performed would be modest.

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