Effect of alae nasi activation on maximal nasal inspiratory airflow in humans

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Gold, Avram R., Philip L. Smith, and Alan R. Schwartz. Effect of alae nasi activation on maximal nasal inspiratory airflow in humans. J. Appl. Physiol. 84(6): 2115–2122, 1998.—The upper airway is a complicated structure that is usually widely patent during inspiration. However, on inspiration during certain physiological and pathophysiological states, the nares, pharynx, and larynx may collapse. Collapse at these locations occurs when the transmural pressure (Ptm) at a flow-limiting site (FLS) falls below a critical level (Ptm’). On airway collapse, inspiratory airflow is limited to a maximal velocity (VImax) determined by (−Ptm’)/Rus, where Rus is the resistance upstream to the FLS. The airflow dynamics of the upper airway are affected by the activity of its associated muscles. In this study, we examine the modulation of VImax by muscle activity in the nasal airway under conditions of inspiratory airflow limitation. Each of six subjects performed sniffs through one patent nostril (pretreated with an alpha agonist) while flaring the nostril at varying levels of dilator muscle (ala nasi) EMG activity (EMGan). For each sniff, we located the nasal FLS with an airway catheter and determined VImax, Ptm’, and Rus. Activation of the alae nasi from the lowest to the highest values of EMGan increased VImax from 422 ± 156 to 753 ± 291 ml/s (P < 0.01) and decreased Ptm’ from −3.6 ± 3.0 to −6.0 ± 4.7 cmH2O (P < 0.05). Activation of the alae nasi had no consistent effect on Rus. VImax was positively correlated with EMGan, and Ptm’ was negatively correlated with EMGan in all subjects. Our findings demonstrate that alae nasi activation increases VImax through the nasal airway by decreasing airflow collapsibility.

Starling resistor; airway collapsibility; upper airway

THE UPPER AIRWAY is a complicated structure exhibiting a range of behaviors during breathing. Normally, the upper airway is widely patent throughout inspiration. During certain physiological and pathophysiological conditions, however, the upper airway collapses during inspiration. Collapse of the upper airway may completely or partially obstruct inspiratory airflow (V1). Obstructive sleep apnea represents complete V1 obstruction at the nasopharynx, oropharynx, or hypopharynx (19, 23, 25, 36). Snoring (2, 3, 14, 15, 24), snoring (4, 12), and stridor (6, 11, 20) represent partial V1 obstruction at the nares, pharynx, larynx, and extrathoracic trachea. Whenever partial V1 obstruction occurs, collapse of a flow-limiting site (FLS) limits airflow to a maximal level (VImax) (13, 14). Under these circumstances, V1 does not exceed VImax as inspiratory effort increases progressively. Thus, although the upper airway FLS may vary in location, the phenomena of collapse and V1 limitation are similar throughout the upper airway.

The airflow dynamics of the upper airway are affected markedly by the activity of its associated muscles. It is now recognized that upper airway muscles act in at least two ways. First, their contraction may affect airflow caliber by dilating or constricting the airway (16, 17, 21, 31, 37, 38, 40, 41). Second, their contraction may reduce collapsibility by stiffening the airway (16, 17, 21, 34). Hypothetically, either action may affect the level of VImax during upper airway collapse and airflow limitation. However, it is unclear how upper airway muscle activity actually modulates VImax (1, 27).

Methods have been developed to determine the mechanism for alterations in VImax (14, 34). It has been established that the level of maximal flow through biological tubes is determined by two physiological parameters. The first is the transmural pressure (Ptm) at which the FLS collapses [the critical Ptm (Ptm’)], which is an index of airway collapsibility. The second is the airway resistance upstream to the FLS (Rus), which reflects the caliber of the upstream airway segment. Thus, study of airflow dynamics during V1 limitation can help us to determine whether changes in VImax are related to changes in airway caliber or collapsibility.

In this study, we examined the modulation of VImax by muscle activity under conditions of V1 limitation. The study of maximal V1 dynamics requires the simultaneous measurement of muscle activity at the onset of V1 limitation, VImax, and the Ptm’ of the FLS. Such a study is most easily performed in the nasal airway because it is easy to instrument, demonstrates V1 limitation, and contains the dilator naris portion of the nasalis muscle (the alae nasi) that is under voluntary control. In a recent study, we used a nasal catheter to locate the FLS of the nasal airway and to characterize nasal V1 dynamics under conditions of flow limitation (14). To examine how upper airway muscles influence VImax, we used the catheter method to study the effect of varying alae nasi activation on VImax and its determinants (Ptm’ and Rus).

METHODS

Subjects. For this study, we selected only subjects who were able to voluntarily flare their nasal alae. Our six subjects (five men and one woman) were all Caucasian health care workers. One subject had a history of allergic rhinitis, but none of the subjects had a disorder of the lower respiratory tract. Each subject was free of upper respiratory tract symptoms on the day of study. The methods used were approved by the Research and Development Committee of the VA Medical Center-Northport, and informed consent was obtained from the subjects.
Experimental apparatus. Our methods for studying maximal nasal airflow dynamics with the use of a nasal catheter have been previously presented in detail (14). Briefly, a nasal mask was connected in series with a pneumotachograph (model 3813, Hans Rudolph, Kansas City, MO) measuring \( V_i \) (Fig. 1). Mask pressure (Pmask) was measured with a pressure transducer (model 231D, Spectramed, Oxnard CA) from a pressure port in the mask. Nasopharyngeal pressure (Pnp), a pressure downstream to the collapsible segment of the nasal airway, was measured by using anterior rhinometry through the left nostril.

To measure the pressure at the nasal FLS (PFLS) and to detect a change in its location, we identified the nasal FLS as the site downstream to which nasal airflow pressure continued to decrease beyond \( V_{i,\text{max}} \) (14). To measure PFLS, we placed the lateral port of a pressure catheter (1.9 mm OD) immediately downstream to the FLS (Fig. 1) (14). We placed the lateral port of a second pressure catheter 2 mm distal to the first (Fig. 1) to detect a shift of the FLS with alae nasi activation. If the Ptm’ of the FLS at the nasal opening decreased sufficiently with alae nasi activation, then another more collapsible site might become the FLS (Fig. 2). The surface electromyogram (EMG) activity of the alae nasi was monitored by using two electrodes fixed to the left ala and a ground fixed to the forehead. To obtain the moving average EMG (EMGan), the EMG signal was band-pass filtered (10–1,000 Hz), amplified, full-wave rectified, and passed through a low-pass moving-average filter with a time constant of 200 ms (model MA821, CWE, Ardmore, PA).

Each of the six subjects performed a series of flow-limited inspiratory efforts (sniffs) through the right nostril. The subjects were seated and began their sniffs at functional residual capacity. For each sniff, we recorded the raw EMG signal, EMGan, \( V_i \), Pmask, PFLS, and nasopharyngeal pressure (Pnp) simultaneously on a polygraph recorder (model 78, Grass Instruments, Quincy, MA). A microcomputer was used to digitize all the physical signals with an acquisition frequency of 500 Hz and to store them for subsequent analysis (Easyest LX, Keithley ASYST, Taunton, MA).

Because the determination of the Ptm’ of the nasal FLS depends on the accurate measurement of rapidly changing signals, the pressure and flow signals obtained during several sniffs were analyzed by fast Fourier transform. We determined that >99% of the signal power was <5 Hz. The frequency-response system characteristics of each catheter-transducer-amplifier combination were also examined, and the amplitude of each was >95% at 10 Hz.

Experimental protocol. Each of our subjects performed a series of sniffs while varying the level of alae nasi activity by voluntarily flaring the nasal ala. Subjects controlled the level of alar flaring by viewing the EMGan on the polygraph tracing (Fig. 3). We randomized the order of EMG activity levels and asked subjects to replicate each level of activation for three consecutive sniffs. Each subject was allowed to coordinate alae nasi activation with inspiratory effort comfortably. Reproducibility of the maximal nasal \( V_i \) dynamics was facilitated by spraying subjects’ nostrils with a long-acting nasal decongestant (0.05% oxymetazoline hydrochloride).

Data analysis. For each sniff, we determined the three parameters characterizing maximal nasal \( V_i \): \( V_{i,\text{max}} \), Ptm’, and Rus. To accomplish this, we identified the moment \( V_{i,\text{max}} \) was attained and obtained the corresponding values of EMGan (EMGan*), PFLS (PFLS*), and Pmask (Pmask*; Fig. 4). We then fitted the values into the following equations

\[
P_{\text{tm'}} = P_{\text{FLS'}} - P_{\text{mask'}}
\]

\[
\text{Rus} = -\frac{P_{\text{tm'}}}{V_{i,\text{max}}}\]

As in a previous investigation of alae nasi activity (9), EMGan activity was normalized by dividing the EMGan*, in arbitrary units, by the peak value obtained during maximal voluntary flaring of the nasal alae. Using this method, we expressed the EMGan* as a percentage of each subject’s maximum.

To examine the relationship of alae nasi activity to maximal \( V_i \) dynamics, we plotted and regressed each subject’s values of \( V_{i,\text{max}}, \text{Ptm'}, \) and Rus on the corresponding values of EMGan* by using a least squares linear regression. We quantified the strength of the relationships with the correlation coefficients. For each subject, we also calculated the mean values for \( V_{i,\text{max}}, \text{Ptm'}, \) and Rus at the lowest and highest values of EMGan* observed (5 or 6 values for each mean). We compared high and low values for the group of subjects by using a paired t-test.

RESULTS

All six subjects demonstrated a FLS at the nasal opening. Voluntary alar flaring produced values of EMGan* that ranged from 1.3 ± 1.6 (range, 0–3.8%) to 72.3 ± 11.0% (range, 55.2–81.6%) of maximal EMG activity. Although all subjects were able to match their maximal EMG activity throughout the study, they were unable to synchronize \( V_{i,\text{max}} \) with values of EMGan* above 82% of maximal activity. Alae nasi activation did not cause downstream migration of the FLS in any subject.
The effects of alae nasi activity on $V'_{\text{Imax}}$, $P_{\text{tm}}$, and $R_{\text{us}}$ are illustrated in Fig. 5 and Table 1. Alae nasi activity significantly increased $V'_{\text{Imax}}$ and decreased $P_{\text{tm}}$ (Table 1). All subjects demonstrated a significant correlation between EMG$_{\text{an}}$ and both $V'_{\text{Imax}}$ and $P_{\text{tm}}$ (Fig. 5).

Alae nasi activity did not consistently affect $R_{\text{us}}$ (Table 1). Figure 5 demonstrates a negative correlation...
between EMGan and Rus for two subjects (subjects 3 and 6), a positive correlation for two subjects (subjects 1 and 2), and no correlation for two subjects (subjects 4 and 5).

**DISCUSSION**

In this study, we examined how an upper airway muscle affects $V_{\text{Imax}}$. We accomplished this by studying the effect of varying alae nasi activity on nasal $V_{\text{Imax}}$. Our methods allowed us to attribute any increase in $V_{\text{Imax}}$ to a decrease in either Ptm or Rus. Patients sniffed through one nostril while flaring the ala with varying levels of effort. Airflow dynamics were studied with a nasal catheter at the onset of $V_{\text{Imax}}$. In all subjects, high levels of alae nasi activity were associated with an increased $V_{\text{Imax}}$ and decreased nasal Ptm. We also found that alae nasi activity increased $V_{\text{Imax}}$ in one of two ways (Eq. 2). First, decreases in the Ptm of the nasal FLS in each subject caused an increase in $V_{\text{Imax}}$. Second, decreases in Rus in some subjects also increased $V_{\text{Imax}}$.

Our method for assessing the response of flow dynamics to alae nasi activity depends on establishing the precise location of the nasal FLS. We accomplished this by locating the point in the airway at which the pressure in the nasal catheter continued to decrease beyond $V_{\text{Imax}}$. This methodology required a high level of precision. As demonstrated in Fig. 4A (PFLS), even a decrease of 0.2 cmH2O in catheter pressure lasting 50 ms after $V_{\text{Imax}}$ is enough to establish a downstream location for the nasal catheter. Because we used a computer to record both the catheter pressure and the $V$ every 2 ms, our methods had the necessary precision to identify the nasal FLS. In previous use of this methodology (14) and in the present study, we have demonstrated a FLS at the nasal opening in 10 of 11 unique subjects (1 subject participated in both studies). One subject had a FLS located at a constriction at the

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**Fig. 4. Polygraph recording of 2 sniffs performed at different levels of alae nasi activity. A: alae nasi activity is 0% of maximum that subject can develop. $V_{\text{Imax}}$ (vertical line) is 530 ml/s. Critical transmural pressure (Ptm) of FLS ($-0.5$ cmH2O) is calculated by subtracting critical Pmask from critical PFLS (PFLS’ - Pmask’). B: subject has voluntarily flared the nostrils, and EMGan activity is 89% of maximum at $V_{\text{Imax}}$. $V_{\text{Imax}}$ has increased to 1,020 ml/s. Ptm of FLS has decreased to $-2.7$ cmH2O. In this example, alae nasi activation has increased $V_{\text{Imax}}$ and decreased nasal Ptm. Pnp’ = Pnp at $V_{\text{Imax}}$.**
junction of the greater alar and lateral cartilages (the limen nasi). None of our subjects had a FLS at the nasal valve. In contrast to our experience, previous investigators (2, 3) have suggested that the nasal FLS is usually deeper within the nasal airway at the nasal valve. We believe, however, that the nasal FLS is usually located at the nasal opening and that the methods previously employed (2, 3) lacked the precision to accurately localize the nasal FLS. Therefore, because of the precision of the catheter method, we have been able to establish the exact location of the FLS, and have determined it to be at the nasal opening for most of our subjects.

The catheter method allowed us to determine the effect of alae nasi activity on airflow dynamics in the nose. We recognize, however, that our methodology may have influenced our measurements of nasal function. First, we placed two catheters in the nasal opening; this may have narrowed the lumen and increased Rus. The combined area of the catheters was 0.06 cm², which represented no more than 6% of the average area of the nasal opening (>1.0 cm²; 18), suggesting that the effect of these catheters on Rus was minimal. Second, we recognize that the lower margin of the nasal mask funneled air into the nasal opening (Fig. 1). This may have produced marked pressure losses caused by convect-


Table 1. Effect of alae nasi EMG activity on $V_{\text{Imax}}$, Ptm', and Rus

<table>
<thead>
<tr>
<th></th>
<th>Lowest Activity</th>
<th>Highest Activity</th>
<th>Difference</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_{\text{Imax}}$, m/s</td>
<td>422 ± 156</td>
<td>753 ± 291</td>
<td>331 ± 151</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Ptm', cmH$_2$O</td>
<td>−3.6 ± 3.0</td>
<td>−6.0 ± 4.7</td>
<td>−2.4 ± 2.0</td>
<td>&lt;0.05</td>
</tr>
<tr>
<td>Rus, cmH$_2$O·l$^{-1}$·s</td>
<td>9.5 ± 7.2</td>
<td>8.8 ± 6.4</td>
<td>−0.7 ± 3.4</td>
<td>NS</td>
</tr>
</tbody>
</table>

Values are means ± SD. EMG, electromyogram; $V_{\text{Imax}}$, maximal inspiratory airflow; Ptm, transmural pressure; Rus, resistance upstream from flow-limiting site; NS, not significant.

Previous investigators have differed on the mechanism by which alae nasi activity increases nasal airflow (1, 27). Strohl and associates (38) have demonstrated that, in humans breathing at submaximal flows, maximal alar flaring decreases nasal resistance by 29% compared with inspiration without alar flaring. This finding suggests that the major effect of the alae nasi is to dilate the nasal airway and decrease resistance under non-flow-limited conditions (at submaximal levels of airflow). Others (5, 8, 15) believe that the chief effect of the alae nasi is to stiffen the airway (decrease collapsibility), thereby preventing flow-limited conditions from developing.

Our findings suggest that the alae nasi may have increased airflow in both of these ways. First, the alae nasi decreases resistance by dilating the nasal airflow (38), an effect that occurs consistently at submaximal flows and variably at $V_{\text{Imax}}$. Second, our findings demonstrate that alae nasi activation decreased the Ptm' of the nasal FLS. When the Ptm' falls, airway pressure must become more negative for the FLS to collapse and limit flow, and greater levels of nasal V'I can be accommodated (Eq. 2) as inspiratory effort rises with exertion. Once nasal V'I limitation occurs, however, further increases in inspiratory effort no longer generate any increase in nasal airflow, which can be supplemented only by mouth breathing (26). Thus, nasal V'I can increase substantially when the alae nasi activate, because they prevent the development of flow limitation with increasing ventilatory effort and because they decrease nasal resistance over the entire range of submaximal airflow.

In this study, we found the nasal valve to be located downstream to the nasal FLS. Its downstream location should determine its role in modulating airflow dynamics as follows. The nasal valve is the narrowest site within the nasal airway and would be expected to contribute significantly to the resistance of the downstream segment of the airway (15). As the downstream resistance rises (e.g., during congestion of the inferior turbinate), greater inspiratory effort is required to reach $V_{\text{Imax}}$. The level of nasal $V_{\text{Imax}}$, however, should not be affected by changes in downstream resistance. Nasal $V_{\text{Imax}}$ can be affected only by the collapsibility of the FLS and by the nasal Rus rather than the downstream resistance. Thus, increasing the resistance at the nasal valve should decrease submaximal flows at any level of inspiratory effort without affecting the nasal $V_{\text{Imax}}$.

A model of the nasal airway in which changing the resistance at the nasal valve does not affect $V_{\text{Imax}}$ appears to conflict with previously reported observations. Several studies (22, 28, 29) have demonstrated an increased peak nasal $V_{\text{Imax}}$ after decongestion or surgical widening of the downstream nasal airway segment. Because the studies did not monitor the alae nasi, however, we do not know the contribution of changes in alae nasi activity to their findings. Decreasing nasal resistance (increasing submaximal airflow) could increase alae nasi activity and increase nasal $V_{\text{Imax}}$. An increase in $V_{\text{Imax}}$ delivered to the upper or...
lower airway has been demonstrated to increase respiratory drive in humans (7, 10, 30). Such a mechanism can be elucidated if alae nasi activity is examined at various levels of downstream resistance.

Our findings in the nasal airway may also apply to the phenomena of collapse and flow limitation in the pharynx and the larynx (4, 6, 11, 12, 19, 20, 25, 35, 36). Regardless of the site in the upper airway, the principles of flow through a collapsible tube (the Starling resistor model) have accounted for airflow dynamics under flow-limited conditions. The Starling resistor is a passive model, with a FLS, an upstream segment, and a downstream segment with fixed mechanical properties. The upper airway, however, has a complex musculature with activity that changes dynamically with respiration. It is well recognized that the state of neuromuscular activity can dramatically alter the flow dynamics in one such site are influenced by voluntary activation of a single muscle group. Our methods have allowed us to determine the effect of this muscle group on V′\text{Imax} and to discern differences in its activity.

Moreover, like the function of the alae nasi in the nasal airway, the pharyngeal dilator muscle activity increases V′\text{Imax} by decreasing the collapsibility of the pharyngeal airway (32, 33). Thus, under conditions of flow limitation, dilator muscles from different FLS in the upper airway increase airflow by decreasing airflow collapsibility regardless of their effect on Rs. This mechanism of action can only be examined after carefully locating the FLS and characterizing its function and that of the upstream segment.

This research was supported by National Institutes of Health Grants RR-05736, HL-37379, and HL-50381.

Received 28 July 1997; accepted in final form 17 February 1998.

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