

Nasal resistance and flow resistive work of nasal breathing during exercise: effects of a nasal dilator strip

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Gehring, J. M., S. R. Garlick, J. R. Wheatley, and T. C. Amis. Nasal resistance and flow resistive work of nasal breathing during exercise: effects of a nasal dilator strip. *J Appl Physiol* 89: 1114–1122, 2000.—Using posterior rhinomanometry, we measured nasal airflow resistance (R_n) and flow-resistive work of nasal breathing (WONB), with an external nasal dilator strip (ENDS) and without (control), in 15 healthy adults (6 men, 9 women) during exclusive nasal breathing and graded (50–230 W) exercise on a cycle ergometer. ENDS decreased resting inspiratory and/or expiratory R_n (at 0.4 l/s) by $>0.5 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$ in 11 subjects (“responders”). Inspired ventilation (\dot{V}_I) increased with external work rate, but tended to be greater with ENDS. Inspiratory and expiratory R_n (at 0.4 l/s) decreased as \dot{V}_I increased but, in responders, tended to remain lower with ENDS. Inspiratory (but not expiratory) R_n at peak nasal airflow (\dot{V}_n) increased as \dot{V}_I increased but, again, was lower with ENDS. At a \dot{V}_I of ~ 35 l/min, ENDS decreased flow limitation and hysteresis of the inspiratory transnasal pressure-flow curve. In responders, ENDS reduced inspiratory WONB per breath and inspiratory nasal power values during exercise. We conclude that ENDS stiffens the lateral nasal vestibule walls and, in responders, may reduce the energy required for nasal ventilation during exercise.

work of breathing

RECENTLY, AN ADHESIVE EXTERNAL nasal dilator strip (ENDS) device (Breathe Right, CNS, Chanhassen, MN) has become available as a purely mechanical means of lowering nasal airflow resistance (R_n). ENDS is advocated for the relief of nasal obstruction associated with nasal congestion or deviated septum (14, 21), reduction of snoring, and improving sleep quality (15, 22). The device has also been widely adopted by athletes for use in sporting competition in an attempt to promote nasal route breathing during exercise (20).

Some recent studies have examined the effect of ENDS on R_n in normal subjects at rest, but these studies have produced conflicting results, with ENDS significantly reducing R_n in some (14) but having no effect in others (11, 23). There is even less information concerning the influence of ENDS on nasal airflow dynamics during stimulated breathing. Vermoen and co-workers (23) have recently shown that, in normal

subjects, ENDS increases the forced inspiratory volume in 1 s (FIV_1). Our laboratory has also shown that ENDS decreases inspiratory R_n (at 1.0 l/s) during voluntary hyperpnea (10). Limited studies that examined the effect of ENDS on gas exchange and performance characteristics during exercise in athletes have been performed (20), but there are no studies addressing the effect of ENDS on nasal airflow dynamics during exercise. Thus, despite the increasing use of this device in sports, its effects on R_n under exercise conditions remain unknown.

A potential energetic advantage associated with reduced R_n while breathing nasally during exercise is a reduction in the work of breathing. However, the effect of ENDS on the energetics of nasal breathing at either rest or higher ventilatory levels has not been studied. Consequently, the aim of the present study was to examine, in healthy subjects, the effects of ENDS on nasal airway pressure-flow relationships and the flow-resistive work of nasal breathing (WONB) during exercise. To study the potential range of effects of ENDS on nasal airflow dynamics, we confined our observations to nasal-only breathing. During exercise, most subjects switch from nasal-only breathing to oronasal breathing (24) at a minute ventilation of ~ 35 l/min (12). Consequently, we also paid particular attention to the effects of ENDS on nasal airflow dynamics at this level of inspired ventilation (\dot{V}_I).

METHODS

Subjects

Fifteen healthy, adult, Caucasian subjects (six men and nine women; age: 27.0 ± 1.6 yr, mean \pm SE) volunteered to participate in the study. Subjects had no known medical problems, and, in particular, they had no current symptoms of nasal disease, snoring, or allergic rhinitis. All subjects gave written, informed consent, and the protocol was approved by the Western Sydney Area Health Service Ethics Committee.

Nasal Dilator Strips

The ENDS used in the present study is a commercial product (Breathe Right, 3M, Sydney, Australia), available in

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two different sizes for adults: small/medium and medium/large. Subjects in the present investigation were studied while using an ENDS device of the size appropriate for them.

Application of Nasal Strips

Each subject's nasal dorsum was cleaned with an alcohol pad before the ENDS was positioned. All ENDS were placed in accordance with the manufacturer's directions, which specify that the device should be positioned midway over the nose, with the tape-covered springs extending down the external lateral nasal walls along the nasal crease and the tabs at each end of the nasal strip should be adhered to the flare of the nostril.

Nasal Airway Flow Dynamics

Nasal airway pressure-flow relationships were assessed using a modification of standard posterior rhinomanometry (25). Subjects breathed exclusively via a nasal continuous positive airway pressure mask (Sullivan, ResMed, Sydney, Australia) while nasal airflow (\dot{V}_n) was measured using a heated pneumotachograph (Fleisch no. 2, Gould, Bithoven, Netherlands) coupled to a differential pressure transducer (± 10 cmH₂O, Celesco Transducer Products, IDM Instruments, Dandenong, Victoria, Australia). Care was taken to avoid any pressure being exerted on the external nasal walls by the mask. The nasal mask, once fitted to each subject, was tested in situ for air leaks. If leaks were detected, the mask was repositioned until a seal was obtained. An occluded mouthpiece (SensorMedics, Middle Park, Victoria, Australia) was placed in the subject's mouth and connected to a differential pressure transducer (± 100 cmH₂O, Celesco Transducer Products IDM Instruments). The other port of the transducer was connected to the nasal mask. With the occluded mouthpiece in place, there was no oral route airflow, and the pressure inside the mouthpiece reflected oropharyngeal pressure. Thus the output of the pressure transducer reflected transnasal pressure (P_{tn}). The \dot{V}_n and P_{tn} signals were digitized (400 Hz; MacLab 16s, ADInstruments, Castle Hill, New South Wales, Australia) and stored on a Macintosh computer for later analysis. The pressure and airflow signals were in phase to 12 Hz. Tidal volume (V_T) was determined by on-line integration of \dot{V}_n .

Exercise Protocol

Subjects were studied at rest and also during progressive graded exercise on a cycle ergometer (E022E, Siemens-Elerna). Each subject performed, in random order, two runs (30–150 min apart), one with and one without (control) an ENDS device. Baseline data were collected over a 2-min period before exercise commenced at a pedal rate of 60 rpm (held constant throughout the entire exercise run) and with an imposed external work rate of 50 W. The external work rate was then increased in 30-W increments every 2 min until subjects were unable to maintain the task.

Data Analysis

Nasal airflow dynamics. Inspiratory and expiratory R_n values were calculated as P_{tn}/ \dot{V}_n from values measured directly from transnasal pressure-flow curves obtained from three to five consecutive steady-state breaths occurring during the last 30 s of each work rate. To provide a comparison between resting and exercise values, measurements of inspiratory and expiratory R_n were made at 0.4 l/s, the highest common resting \dot{V}_n . However, \dot{V}_n was >0.4 l/s over the majority of each breath during exercise, and, consequently,

we also measured R_n at peak \dot{V}_n . The \dot{V}_I was calculated from the inspired V_T and breathing frequency data for each of the steady-state breaths analyzed.

During exercise, there was counterclockwise hysteresis of the inspiratory transnasal pressure-flow relationship. Inspiratory P_{tn} values were taken from the ascending (i.e., early in inspiration) limb of the inspiratory transnasal pressure-flow relationship. In addition, the magnitude of the hysteresis of the inspiratory transnasal pressure-flow relationship was estimated (at a \dot{V}_I of ~ 35 l/min) as the difference between the transnasal pressures measured from the ascending and descending limbs of the inspiratory transnasal pressure-flow plots at an inspiratory \dot{V}_n of 1.0 l/s (i.e., hysteresis at 1.0 l/s).

Flow-resistive WONB. Individual breath P_{tn}-V_T plots were constructed for 3–5 steady-state breaths that occurred during the last 30 s of each work rate. The flow-resistive WONB was calculated using planimetry (KP-27 Compensating Polar Planimeter, Koizumi, Japan) to measure the area enclosed by the P_{tn}-V_T plot for each breath. Separate analyses were conducted for inspiration and expiration and summed to obtain the total WONB/breath. The calculated WONB/breath (in cmH₂O \times liters) was then converted into J/breath using a standard conversion factor (1 cmH₂O \times liters = 0.09806 J). In addition, nasal power (NP, W) was calculated from the WONB/breath and the breathing frequency.

Statistical analysis. Where applicable, individual breath data were averaged to obtain individual subject values. Individual subject values were then pooled, and group mean (\pm SE) results were calculated. Primarily because of varying levels of fitness, there was considerable variation in the maximum external work rate and maximum \dot{V}_I levels achieved by individual subjects during control. Consequently, we have confined our comparison between ENDS and control values to external work rates ≤ 230 W or to \dot{V}_I levels < 55 l/min. In addition, because fewer subjects reached the higher external work rates and higher levels of \dot{V}_I , our multiple comparison analyses within a single condition are confined to external work rates ≤ 140 watts or \dot{V}_I levels < 55 l/min. For analysis, we pooled data post hoc according to the level of \dot{V}_I . We aimed to obtain data at \dot{V}_I levels of $\sim 10, 20, 30, 40,$ and 50 l/min for analysis; however, the actual achieved values (bins) were $11.6 \pm 0.3, 20.3 \pm 0.3, 29.4 \pm 0.4, 39.3 \pm 0.3,$ and 50.5 ± 0.4 l/min.

Results obtained with ENDS and during control were compared using a Wilcoxon signed-rank test for paired single comparisons, whereas multiple comparisons were made using Friedman's ANOVA with appropriate adjustments, where necessary, for unbalanced data. When the ANOVA demonstrated a significant effect, the Wilcoxon signed-rank test with Bonferroni correction was used to test individual comparisons. The relative effect of ENDS at rest and during exercise was examined using linear regression analysis. $P < 0.05$ was considered significant, except when Bonferroni corrections were employed, in which case $P < 0.005$ was considered significant.

RESULTS

R_n at 0.4 l/s at Rest

Inspiratory R_n (at 0.4 l/s) decreased significantly from 3.04 ± 0.71 cmH₂O \cdot l⁻¹ \cdot s during control to 1.52 ± 0.23 cmH₂O \cdot l⁻¹ \cdot s with ENDS, and expiratory R_n also decreased significantly, from 2.98 ± 0.52 to 1.56 ± 0.15 cmH₂O \cdot l⁻¹ \cdot s during control and with ENDS, respectively ($P < 0.009$). However, there was considerable between-subject variability in the re-

sponse to ENDS, such that R_n (at 0.4 l/s) decreased by $>0.5 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$ with ENDS in eight subjects during both inspiration and expiration, two subjects during inspiration only, and one subject during expiration only (Fig. 1). These subjects were classified as inspiratory and/or expiratory responders, as appropriate.

\dot{V}_I

\dot{V}_I increased with progressive graded exercise during both control and with ENDS ($P < 0.0001$, over 0–140 W; Fig. 2). However, \dot{V}_I tended to be slightly greater

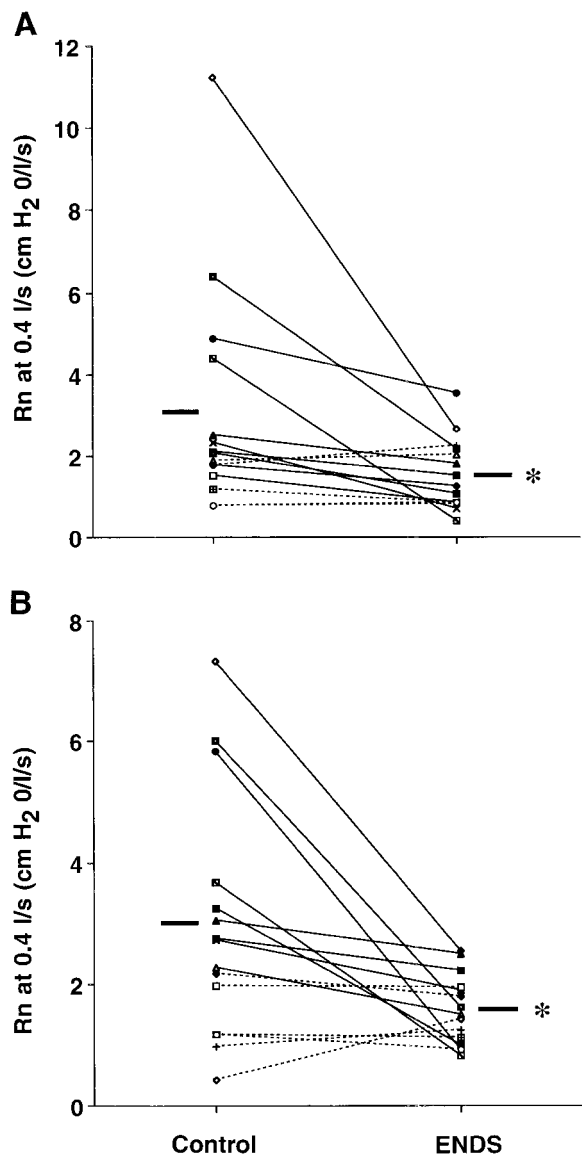


Fig. 1. Inspiratory (A) and expiratory (B) nasal airflow resistance (R_n) at 0.4 l/s with an external nasal dilator strip (ENDS) and without (control) in 15 subjects at rest. ENDS lowered nasal resistance by $>0.5 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$ in 8 subjects during inspiration and expiration, 2 subjects during inspiration only, and 1 subject during expiration only. These subjects were classified as responders (solid lines), whereas the remaining subjects were classified as nonresponders (dotted lines). Group mean R_n (horizontal bars) was significantly lower with ENDS. Different symbols represent individual subjects. * $P < 0.05$ compared with control.

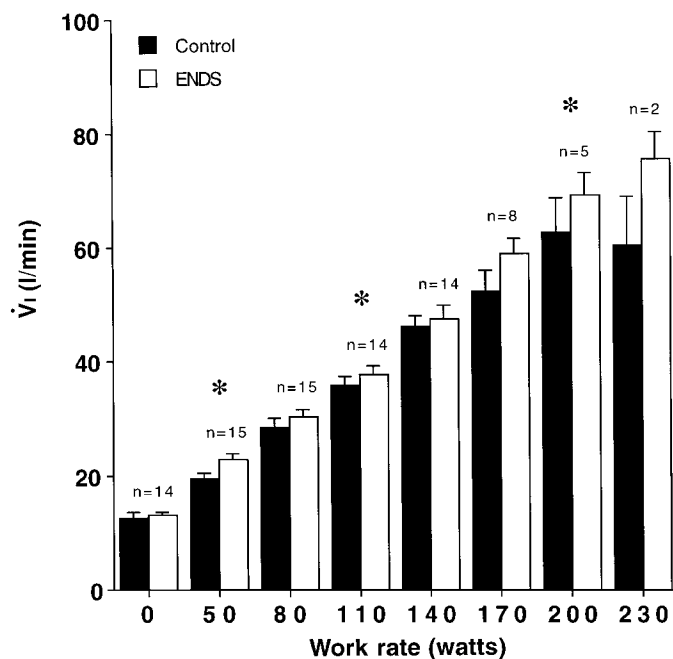


Fig. 2. Ventilation (\dot{V}_I) and external work rate with ENDS and during control. In general, ENDS tended to increase \dot{V}_I at all external work rates when compared with control. Data are means \pm SE; n , no. of subjects. * $P < 0.05$ compared with control.

with ENDS at all external work rates, with significant increases achieved for the whole group at 50, 110, and 200 W ($n = 5$ –15, all $P < 0.05$ compared with control; Fig. 2).

R_n at 0.4 l/s During Exercise

For the whole group, during both control and ENDS, inspiratory and expiratory R_n at 0.4 l/s decreased as \dot{V}_I increased, reaching a plateau at 30–40 l/min ($n = 10$ –15, $P = 0.0001$). However, there was a tendency for R_n at 0.4 l/s to be lower with ENDS when compared with control. When the data were analyzed separately for the responder and “nonresponder” subgroups, it was found that the effect of ENDS was predominantly due to the responders (Fig. 3). In the nonresponder subgroup, there was no significant effect of ENDS at any level of \dot{V}_I ($n = 2$ –6, all $P > 0.08$).

R_n at Peak \dot{V}_n During Exercise

With progressive graded exercise (up to 230 W), the peak inspiratory \dot{V}_n , at which inspiratory R_n at peak \dot{V}_n was calculated, increased significantly from 0.72 ± 0.05 l/s at rest to a maximum of 2.28 ± 0.15 l/s during control and from 0.79 ± 0.05 l/s at rest to a maximum of 2.81 ± 0.20 l/s during ENDS (both groups, $n = 15$, $P = 0.0001$). Expiratory peak \dot{V}_n also increased with exercise from 0.58 ± 0.05 l/s at rest to a maximum of 2.70 ± 0.16 l/s during control and from 0.66 ± 0.05 l/s at rest to a maximum of 3.01 ± 0.23 l/s during ENDS (both groups, $n = 15$, $P = 0.0001$).

For the whole group, inspiratory (but not expiratory) R_n at peak \dot{V}_n increased with increasing \dot{V}_I during control ($n = 10$ –15, $P = 0.017$) but remained relatively

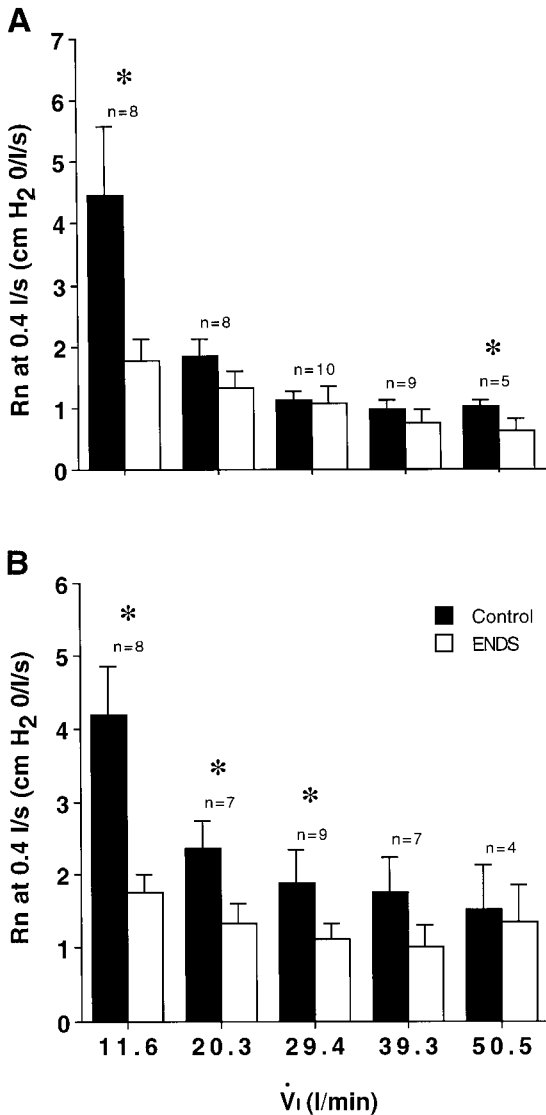


Fig. 3. Effect of exercise on inspiratory (A) and expiratory (B) Rn at 0.4 l/s, with ENDS and during control, in relation to mean \dot{V}_I (bins, see text) in responder subjects. Data are means + SE; n, no. of subjects. * $P < 0.05$ compared with control.

constant over all levels of \dot{V}_I with ENDS ($n = 10-15$, $P = 0.41$). When the data were analyzed separately for the responder and nonresponder subgroups, however, it was found that the effect of ENDS on both inspiratory and expiratory Rn at peak \dot{V}_n was again primarily due to the responders (Fig. 4). In the nonresponder subgroup, there was no significant effect of ENDS at any level of \dot{V}_I ($n = 2-6$, all $P > 0.18$).

The relative magnitude of the effect of ENDS on inspiratory Rn at peak \dot{V}_n at a \dot{V}_I of 29.4 ± 0.4 l/min correlated significantly ($r = 0.82$, $P = 0.0002$) with the relative magnitude of the effect of ENDS on resting inspiratory Rn at 0.4 l/s (Fig. 5).

Hysteresis of the Inspiratory Transnasal Pressure-Flow Curve

Comparison of values for the transnasal pressure differences between the ascending and descending limbs

of the inspiratory transnasal pressure-flow curve (i.e., hysteresis) at a \dot{V}_I of 35.2 ± 0.7 l/min for control and 35.1 ± 0.8 l/min with ENDS ($P = 0.91$) showed that hysteresis at 1.0 l/s decreased with ENDS in 10 subjects by 0.14–12.86 cmH₂O, but was unchanged or increased slightly in the remaining subjects.

Transnasal pressure-flow relationships obtained in one subject during exercise at an external work rate of 140 W during control and with ENDS are shown in Fig. 6. ENDS greatly reduced hysteresis of the inspiratory transnasal pressure-flow curve, as well as resulting in a reduction in the tilt of the curve, which indicates a fall in the overall Rn. In addition, ENDS tended to greatly reduce inspiratory airflow limitation, a phenomenon that occurred in many of the subjects during exercise at the higher external work rates. For the whole group, hysteresis at 1.0 l/s decreased significantly with ENDS when compared with control (from 2.77 ± 1.18 to 0.46 ± 0.16 cmH₂O, $n = 15$, $P < 0.002$).

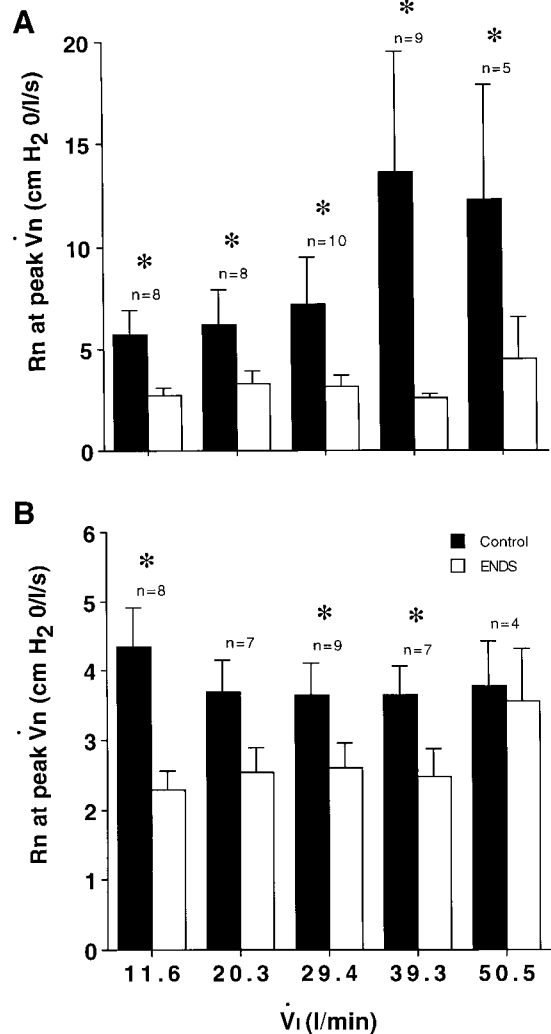


Fig. 4. Effect of exercise on inspiratory (A) and expiratory (B) Rn at peak nasal airflow (\dot{V}_n), with and without ENDS, in relation to mean \dot{V}_I (bins) in responder subjects. Data are means + SE; n, no. of subjects. * $P < 0.05$ compared with control.

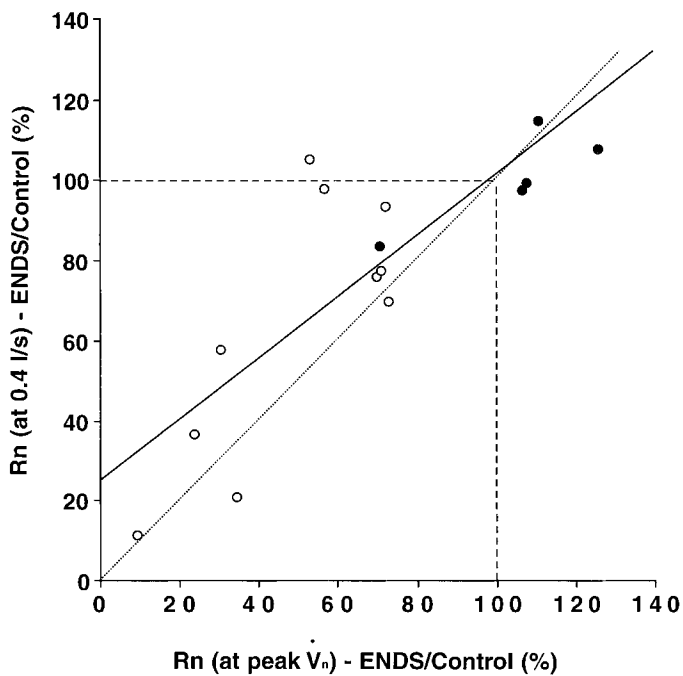


Fig. 5. Relationship between the relative effect of ENDS on inspiratory R_n at peak \dot{V}_n during exercise ($\dot{V}_I = 29.4 \pm 0.4$ l/min) and the relative effect of ENDS on inspiratory R_n at 0.4 l/s at rest. Data are shown for responders ($n = 10$, \circ) and nonresponders ($n = 5$, \bullet). Solid line, linear regression line ($R = 0.82$, $P = 0.0002$); dotted line, identity line; dashed lines, no effect of ENDS.

When the data were analyzed separately for the responder and nonresponder subgroups, it was found that the decrease in hysteresis at 1.0 l/s with ENDS was primarily due to the responder subjects, in which hysteresis at 1.0 l/s decreased from 3.95 ± 1.67 cmH₂O during control to 0.58 ± 0.24 cmH₂O with ENDS ($n = 10$, $P < 0.007$). A feature of the nonresponder subgroup

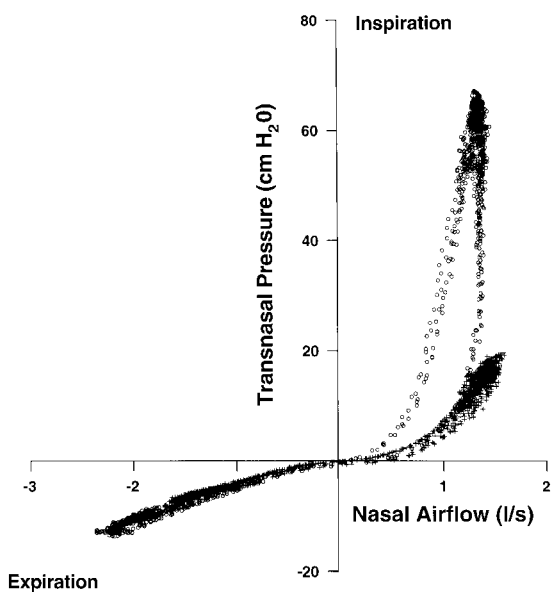


Fig. 6. Transnasal pressure-flow relationship with ENDS (+) and during control (o) in one responder subject at an external work rate of 140 W. See text for discussion.

was the relative absence of hysteresis during control. Furthermore, ENDS had no significant effect on hysteresis at 1.0 l/s in the nonresponder subgroup (0.41 ± 0.17 cmH₂O during control vs. 0.23 ± 0.09 cmH₂O with ENDS, $n = 5$, $P = 0.14$).

WONB During Exercise

For the whole group, during both control and ENDS, inspiratory and expiratory WONB increased as \dot{V}_I increased ($n = 9-14$, $P = 0.0001$). However, when compared with control, WONB tended to be lower with ENDS. When the data were analyzed separately for the responder and nonresponder subgroups, it was found that the effect of ENDS was predominantly due to the responders (Fig. 7). In the nonresponder subgroup, there was no significant effect of ENDS on WONB at any level of \dot{V}_I ($n = 3-6$, $P > 0.20$).

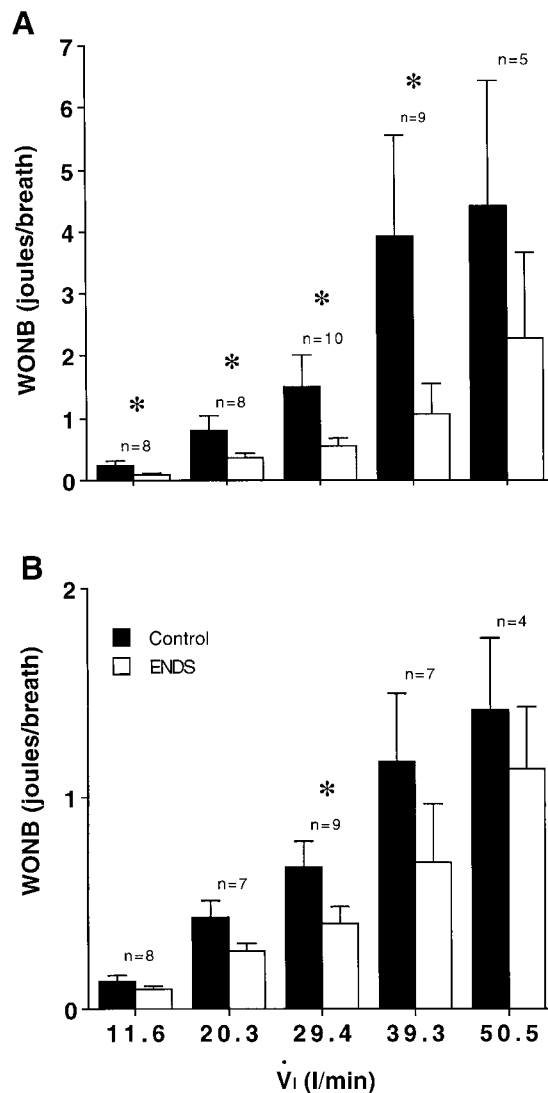


Fig. 7. The effect of exercise on inspiratory (A) and expiratory (B) work of nasal breathing (WONB) per breath with and without ENDS in relation to mean \dot{V}_I (bins) in responder subjects. Data are means + SE; n , no. of subjects. * $P < 0.05$ compared with control.

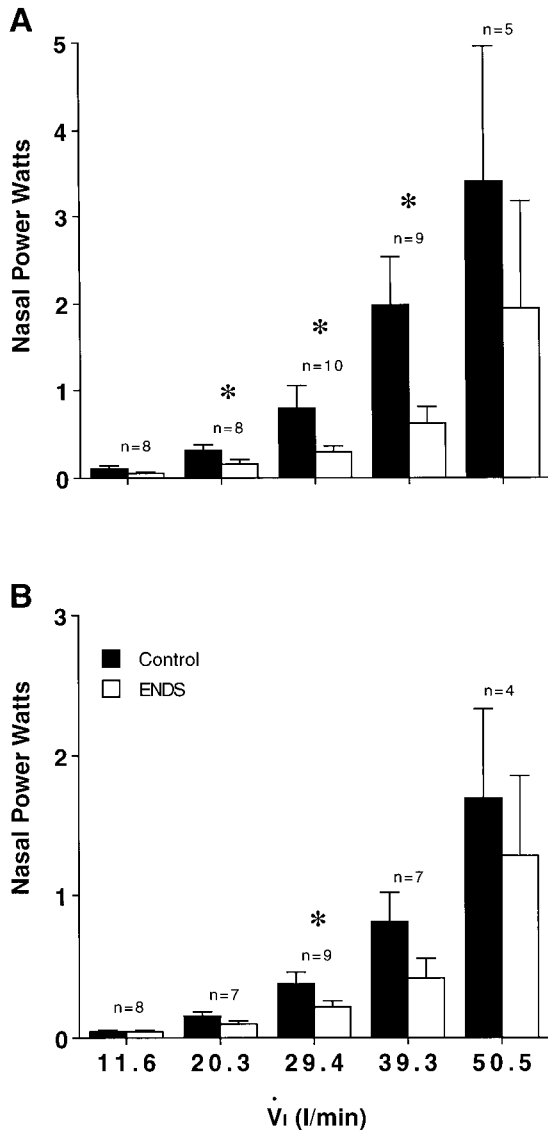


Fig. 8. The effect of exercise on inspiratory (A) and expiratory (B) nasal power with and without ENDS in relation to mean \dot{V}_I (bins) in responder subjects. Data are means + SE; n, no. of subjects. * $P < 0.05$ compared with control.

NP During Exercise

For the whole group, during both control and ENDS, inspiratory and expiratory NP increased as \dot{V}_I increased ($n = 9-14$, $P = 0.0001$). However, NP tended to be lower with ENDS than without. This effect of ENDS was again predominantly due to the responders (Fig. 8). In the nonresponder subgroup there was no significant effect of ENDS on NP at any level of \dot{V}_I ($n = 3-6$, $P > 0.06$).

DISCUSSION

The principal findings of this study were that 1) ENDS decreased resting inspiratory and/or expiratory R_n at 0.4 l/s by $>0.5 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$ in 11 (responders) of the 15 healthy subjects, 2) during progressive graded exercise with nasal-only breathing, \dot{V}_I tended to be

greater with ENDS than during control, 3) inspiratory and expiratory R_n at 0.4 l/s decreased as \dot{V}_I increased during exercise (however, responder inspiratory and expiratory R_n at 0.4 l/s tended to be lower with ENDS than without), 4) only inspiratory R_n at peak \dot{V}_n increased as \dot{V}_I increased during exercise (again responder inspiratory and expiratory R_n at peak \dot{V}_n was lower with ENDS than without), 5) the relative magnitude of the effect of ENDS on inspiratory R_n at peak \dot{V}_n during exercise was significantly correlated with the effect of ENDS on inspiratory R_n at 0.4 l/s at rest, 6) at a \dot{V}_I of ~ 35 l/min, hysteresis at 1.0 l/s of the inspiratory limb of the transnasal pressure-flow curve was less with ENDS in 10 of 15 subjects, and 7) during progressive graded exercise, ENDS significantly reduced the inspiratory flow-resistive WONB and NP values in responders.

The ENDS device is thought to lower R_n via dilation of the vestibule and nasal valve region of the nasal airway (7, 14). However, the effectiveness of the device in lowering R_n in normal healthy subjects is controversial. Some studies have reported that ENDS reduces R_n in normal subjects by an average of 23% during relaxed tidal breathing (9, 14), whereas other studies have shown no significant change in R_n with ENDS (11, 23). Our laboratory previously demonstrated a large range in the magnitude of the response to ENDS in normal healthy subjects at rest, with some subjects failing to respond or, even, increasing R_n with ENDS (10). In the present study, we again demonstrated considerable variability in the effectiveness of ENDS in lowering resting R_n and also showed that individuals may "respond" to ENDS during one or both phases of respiration. In our laboratory's previous study (10), there was a tendency for subjects with higher values of resting R_n to respond to ENDS. This finding was maintained in the present study, which included some subjects who also participated in that previous study (see Fig. 1).

The mechanisms that determine which subjects will respond to ENDS are not known. As discussed, resting R_n may have an influence. However, this influence will depend on the definition of what constitutes a 'response' to ENDS. In the present study, this has been defined as an ENDS-induced decrease in resting inspiratory and/or expiratory R_n at 0.4 l/s of $>0.5 \text{ cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$. Consequently, subjects with a low resting R_n are inherently less likely to have the capacity for R_n to be lowered with ENDS by an amount sufficient to meet this definition. Recently, Amis et al. (1) demonstrated that there is considerable between-subject variability in lateral nasal vestibule wall compliance and suggested that this may explain, at least in part, the occurrence of responders and nonresponders.

During exercise, R_n is known to fall (4, 5, 13, 25), most likely because of sympathetic vasoconstriction in the nasal mucosa (13). This effect has been shown to persist for up to 30 min after exercise (13, 18). Consequently, in the present study, we used a rest period of 30-150 min between the exercise runs. When we evaluated R_n at a constant flow rate (0.4 l/s), the fall in R_n

with exercise was demonstrated under both control and ENDS conditions, reaching a plateau at a \dot{V}_I of ~ 30 – 40 l/min. However, in responder subjects, ENDS tended to decrease both inspiratory and expiratory R_n at 0.4 l/s at all levels of \dot{V}_I .

Because the transnasal pressure-flow relationship is curvilinear (25), measures of R_n are often expressed at a particular airflow rate for comparison purposes. We chose to express R_n at 0.4 l/s, as this represented the highest common airflow at rest in our subjects. However, during exercise, nasal inspiratory and expiratory airflow rates were considerably >0.4 l/s for most of each breath (see Fig. 6). Consequently, R_n at 0.4 l/s, while allowing a direct comparison between resting and exercise values, does not reflect the effective R_n present over most of the breath under exercise conditions. Therefore, we also analyzed R_n at peak \dot{V}_n and, in contrast to the findings for R_n at 0.4 l/s, inspiratory R_n at peak \dot{V}_n increased during exercise under control conditions. Thus, despite exercise-induced nasal mucosal vasoconstriction, R_n at peak \dot{V}_n increases with progressive graded exercise. Most likely, this occurred because of the progressive increase in peak \dot{V}_n that accompanied the exercise task. Again, because of the curvilinear nature of the transnasal pressure-flow relationship, the increase in peak \dot{V}_n resulted in a progressive increase in R_n at peak \dot{V}_n . However, in responders, ENDS lowered inspiratory and expiratory R_n at peak \dot{V}_n at almost all levels of \dot{V}_I when compared with control values. Furthermore, for the whole group (responders and nonresponders), the effect of ENDS during exercise was correlated with its effect at rest (see Fig. 5).

A feature of the findings in the present study was the effect of ENDS on the hysteresis of the inspiratory limb of the transnasal pressure-flow curve. Recently, Shi and co-workers (17) related inspiratory transnasal pressure-flow hysteresis to late inspiratory collapse of the lateral nasal vestibule walls, which was, in turn, associated with a reduction in alae nasi muscle activity towards the end of inspiration. This hysteresis is an important source of pressure loss during inspiration and represents work done by the respiratory muscles that does not lead to increased inspiratory airflow. In the present study, this reduction in hysteresis at 1.0 l/s was, again, most predominant in the responder subgroup (7 of 10 subjects demonstrating a fall in inspiratory transnasal pressure-flow hysteresis at 1.0 l/s were responders). Indeed, a feature of the nonresponder subgroup was the relative lack of inspiratory transnasal pressure-flow hysteresis at 1.0 l/s under control conditions. If such hysteresis is related to late inspiratory lateral nasal vestibule wall collapse, these findings suggest that ENDS nonresponders either maintain alae nasi activity longer during inspiration than ENDS responders, or have intrinsically stiffer lateral nasal vestibule walls. Alternatively, these subjects may have a lower resting R_n , which falls further during exercise, thus making the transnasal pressures to which the nasal walls are exposed lower than in the responders. In any case, it would appear that ENDS

acts to stiffen the lateral nasal vestibule walls, thus defending against late inspiratory nasal vestibule wall collapse. This effect was also demonstrated by the reduction of inspiratory airflow limitation that occurred at the higher levels of \dot{V}_I during control in most of the subjects (see Fig. 6).

A reduction in hysteresis-related pressure losses during inspiration, together with a reduction in R_n , might be expected to result in a decrease in WONB. Indeed, ENDS resulted in a significant reduction in the inspiratory WONB per breath and in NP at almost all levels of \dot{V}_I in responders but not in nonresponders. WONB has received little attention in the literature. In the present study, with exclusive nasal route breathing, we measured oropharyngeal pressure and airflow at the nostrils. Consequently, our values for WONB represent the component of the work of breathing that is related to overcoming airflow resistance in the nose (i.e., the flow-resistive WONB). Using the same approach, Cole and co-workers (2) measured total WONB at rest in normal subjects at ~ 0.20 J/l. In the present study, total WONB at rest averaged 0.31 J/l. However, if the two subjects with the highest resting R_n were excluded, the value was 0.22 J/l, the same as that reported by Cole and co-workers. At rest, inspiratory WONB is also known to be more than 50% greater than that measured during expiration (2); this, too, was reflected in the present study. The major effect of ENDS on WONB was on the inspiratory component of total WONB.

In the present study, WONB was calculated on a per breath basis. Whereas this approach gives specific insights into the mechanics of individual breaths, it is NP (i.e., WONB per unit time) that is likely to provide a parameter more relevant to the energetics of nasal breathing during exercise. NP was previously measured in a study performed by Schultz and Horvath (16) and linked to the switching point from exclusively nasal to oronasal breathing during exercise in normal subjects. According to those authors, WONB, average NP, average transnasal pressure during inspiration, and average transnasal power during expiration, are the most reliable predictors of the onset of oral augmentation of nasal breathing during exercise. Because ENDS reduces WONB and NP during exercise, it would seem likely (although not tested in the present study) that ENDS will prolong the period of nasal breathing during exercise, such that switching to oronasal breathing may occur at a higher \dot{V}_I than under control conditions. Switching to the oral breathing route during exercise reduces nasal airflow. Consequently, the reductions in R_n , WONB/breath, and NP achieved at \dot{V}_I above ~ 35 l/min during exercise in the present study are unlikely to be achieved when oral route breathing is permitted. However, significant reductions in these parameters were achieved at \dot{V}_I below ~ 35 l/min, indicating that, in responders, ENDS is likely to be effective in reducing WONB for the period of an exercise task in which breathing remains exclusively nasal.

During the exclusive nasal breathing in the present study, \dot{V}_I tended to be greater with ENDS than during control at all external work rates. Thus it would appear that unloading of the nasal airway with ENDS results in a slight increase in \dot{V}_I for the same level of exercise stimuli. Furthermore, this increased \dot{V}_I is achieved with less WONB. Whether this finding translates into an energetic advantage during exercise appears to warrant further study. One previous study examined the influence of ENDS on oxygenation and exercise performance in athletes and failed to demonstrate any significant effect on maximum oxygen consumption and maximum power output (20). However, the concept of responders was not addressed in that study. In addition, it should also be emphasized that our findings refer to nasal-only breathing and, therefore, only apply to exercise up to the switching point from nasal-only to oronasal breathing.

Integration of findings from the present study with previous concepts concerning the control of R_n during exercise allows for further insights into the mechanisms by which R_n may be minimized during exercise. The total R_n for each nasal passage may be modelled as two variable resistors in series, whereas the combination of the two nasal passages behaves as two variable resistors in parallel. At rest, about two-thirds of the R_n for each nasal passage occurs in the region of the ostium internum and the remaining one-third in the cartilaginous nasal vestibule (8). During exercise, inspiratory R_n falls because of a combination of sympathetic nervous system-mediated mucosal vasoconstriction (13), which primarily alters the component of total R_n related to the ostium internum, and alae nasi muscle recruitment, which lowers nasal vestibule resistance (3, 17, 25). However, as \dot{V}_I increases, late inspiratory waning of alae nasi muscle activity allows nasal vestibule airflow resistance to increase in association with inspiratory collapse of the lateral nasal vestibule walls (17) and the development of a flow limiting segment in the nasal airway (see Fig. 6). The importance of this mechanism is reflected in the development of inspiratory transnasal pressure-flow hysteresis (17).

The usefulness of ENDS in minimizing inspiratory R_n during exercise may then be twofold. First, the recoil force exerted by ENDS may act to dilate the vestibule region. This may be the effect that predominates at low levels of inspiratory nasal airflow. During exercise, as nasal mucosal vasoconstriction occurs, the impact of ENDS on total inspiratory R_n at low nasal airflow becomes minimal (see Fig. 3). However, at higher nasal airflows, inspiratory nasal vestibule wall collapse represents an increasing component of total R_n . The major effect of ENDS is to stiffen the lateral nasal vestibule walls, thus preventing nasal vestibule narrowing and the development of inspiratory nasal airflow limitation (see Fig. 6). Therefore, ENDS prevents the increase in inspiratory R_n at peak inspiratory nasal airflow that occurs as \dot{V}_I increases (see Fig. 4). Because the recruitment of alae nasi activity during exercise appears to be regulated by mechanisms that

track R_n (6), it would be of interest to assess the interaction between ENDS and alae nasi recruitment. One effect of ENDS may be to "derecruit" alae nasi during exercise, although Sullivan et al. (19) were only able to slightly reduce alae nasi recruitment during exercise using internal nasal splinting.

It is somewhat less clear as to the mechanisms responsible for the lowering of expiratory R_n with ENDS, especially at high \dot{V}_I , when positive intraluminal pressures might be expected to distend and, thereby, stiffen the lateral nasal vestibule walls. Amis et al. (1) previously showed that ENDS exerts a horizontal outward force on the lateral nasal vestibule walls of ~ 22 gm and that this force remains relatively constant over a wide range of nasal widths. Consequently, this force remains constant (both in terms of its magnitude and its direction) within an individual throughout both inspiration and expiration. During expiration, nasal valve luminal cross-sectional area is determined by the dynamic equilibrium reached between lateral nasal vestibule wall inward recoil and the outward force exerted by positive intraluminal pressures. When the ENDS device is in place, an additional outward force is generated, and a new dynamic equilibrium point will be reached, at which nasal wall inward recoil force equals the sum of the outward forces exerted by the positive intraluminal pressure plus the ENDS recoil force. This additional inward nasal wall recoil force can only be generated by further distending the nasal wall, thereby increasing the nasal valve luminal cross-sectional area and lowering expiratory R_n . The key point is that the ENDS recoil force is constant, present throughout all of expiration and always in an outward direction, irrespective of nasal valve luminal diameter and, therefore, lateral nasal vestibule wall position.

In summary, we have shown that normal, healthy subjects who respond to ENDS by decreasing inspiratory and/or expiratory R_n at rest also have a lower R_n with ENDS during exercise. In these responder subjects, during exclusive nasal breathing, ENDS decreases R_n and stabilizes the lateral nasal vestibule walls, thus decreasing the inspiratory pressure losses associated with hysteresis of the inspiratory limb of the transnasal pressure-flow relationship. These effects of ENDS result in a significant decrease in the WONB, particularly during inspiration, and a tendency for \dot{V}_I to increase at any given external work rate. We speculate that this is likely to translate into an energetic advantage of using ENDS during exercise that involves nasal route breathing.

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