Upper airway muscle activity and upper airway resistance in young adults during sleep

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Henke, Kathe G. Upper airway muscle activity and upper airway resistance in young adults during sleep. J. Appl. Physiol. 84(2): 486-491, 1998.—To determine the relationship between upper airway muscle activity and upper airway resistance in nonsnorers and snoring young adults, 17 subjects were studied during sleep. Genioglossus and alae nasi electromyogram activity were recorded. Inspiratory and expiratory supraglottic resistance (Rinsp and Rexp, respectively) were measured at peak flow, and the coefficients of resistance (Kinsp and Kexp, respectively) were calculated. Data were recorded during control, with continuous positive airway pressure (CPAP), and on the breath immediately after termination of CPAP. Rinsp during control averaged 7 ± 1 and 10 ± 2 cmH₂O·l⁻¹·s⁻¹ and Kinsp averaged 26 ± 5 and 80 ± 27 cmH₂O·l⁻¹·s⁻² in the nonsnorers and snorers, respectively (P = not significant). On the breath immediately after CPAP, Kinsp did not increase over control in snorers (80 ± 27 for control vs. 46 ± 6 cmH₂O·l⁻¹·s⁻² for the breath after CPAP) or nonsnorers (26 ± 5 vs. 29 ± 6 cmH₂O·l⁻¹·s⁻²). These findings held true for Rinsp. Kexp did not increase in either group on the breath immediately after termination of CPAP. Therefore, 1) increases in upper airway resistance do not occur, despite reductions in electromyogram activity in young snorers and nonsnorers, and 2) increases in Rexp and expiratory flow limitation are not observed in young snorers.

respiratory muscles; obstructive sleep apnea; snoring

Reduced upper airway muscle activity with sleep onset is thought to be an important mechanism in the development of obstructive apnea and snoring (15). However, studies have shown marked upper airway muscle activity still present during sleep in nonsnorers and in patients with obstructive sleep apnea (OSA) (19, 20, 26, 27). Removing or reducing the phasic muscle activity via nasal continuous positive airway pressure (CPAP), then abruptly reducing mask pressure to atmospheric, results in airway closure in OSA (13) and airway narrowing in snorers (7). This suggests a significant role for phasic inspiratory upper airway muscles in helping maintain airway patency. In contrast, it has recently been demonstrated that alcohol or flurazepam ingestion, which has been shown to differentially suppress upper airway muscle activity, did not cause an increase in inspiratory supraglottic resistance in young, non-snorers (5). This suggests that phasic upper airway muscle activity may not be essential in maintaining inspiratory airway caliber or patency in these individuals. Also, little is known about the role of phasic upper airway muscle activity in maintaining airway caliber during expiration. It was the purpose of this study to determine the relationship between upper airway muscle activity and airway resistance, both inspiratory and expiratory, during sleep in young, nonapneic snorers and nonsnorers.

Methods

Seventeen normal-weight, young adults (10 men, 7 women) <30 yr of age were studied. Eleven subjects denied snoring, and six subjects reported nightly snoring. Subjects reported to the laboratory ~2–3 h before bedtime for setup. Electroencephalogram (C3/A2, O2/A1), electrooculogram, and genioglossus electromyogram (EMGgg) were used for sleep staging. Rib cage and abdominal effort were monitored using inductance plethysmography (Respirtrace), which was calibrated by the isovolume technique (9). The rib cage and abdominal signals were summed and represented tidal volume. This volume signal was calibrated by comparison with the integrated flow signal on a breath-by-breath basis.

For the EMGgg monitoring, a pair of fine wire electrodes were inserted perorally into the anterior tongue muscle. The electrode insertion site was anesthetized by use of cotton pledgets soaked in 2% lidocaine. Alae nasi electrical activity (EMGan) was recorded bilaterally with surface electrodes placed over the nasal folds. Subjects performed voluntary maneuvers to elicit maximum activation of the muscles being studied. Maximum activation of the genioglossus was obtained while subjects protruded the tongue against a device designed to measure tongue strength (Iowa Oral Performance Instrument, Breakthrough) (16), which was attached to a pressure transducer (Statham). This maneuver has previously been shown to elicit maximum EMGgg (23). Electromyograms (EMGs) were rectified and integrated using a time constant of 100 ms. Peak EMG activity was measured as the peak height, from electrical zero, and expressed as a percentage of voluntary maximum. Tonic EMG activity was measured as the minimum EMG activity during expiration.

All subjects wore a nasal mask and breathed exclusively through the nose throughout the study. Silicone impression material was applied around the edges of the mask to prevent leaks around the face. Mouth leak was detected by changes in the baseline of the flow signal when mask pressure (Pmask) was changed. If mouth leak was detected, the mouth was taped shut. The mask was attached to a CPAP machine, which was used to generate a low-pressure (<0.5 cmH₂O) bias flow. Flow was measured at the mask with a low-dead-space pneumotachograph (Hans Rudolph, Inc.) attached to a symmetrical differential pressure transducer (Validyne). Pmask was measured using a transducer (Statham). Saline-filled catheters were used to measure hypopharyngeal and nasopharyngeal pressures. To ensure that any effect of the topical anesthesia was gone before data collection, these catheters were placed first so that a minimum of 30 min passed before lights out. One nasal passage was anesthetized with 2% lidocaine, and the first catheter was passed through the nares so that the tip was past the oropharynx but did not touch the glottis. To place the nasal catheter, a cotton pledget was inserted into the nares until it touched the posterior wall of the nasopharynx, then removed, and the catheter was inserted to the same depth. The catheters were secured at the tip of the nose, then attached to pressurized saline-filled intravenous bags and to saline-filled pressure transducers.
upper airway resistance and muscle activity

(Statham). The pressure transducers were kept at a fixed point relative to the catheter tip. Supraglottic pressure (Psg) was calculated as Pmask minus hypopharyngeal pressure and transnasal pressure as Pmask minus nasal pressure. Zero pressure was determined at zero flow. Flow was calibrated with a rotameter, and pressures were calibrated against a known pressure. Before the study, pressure and flow were tested and shown to be in phase up to 10 Hz.

Resistance (inspiratory \(R_{\text{insp}}\) and expiratory \(R_{\text{exp}}\)) was also characterized by the flow-pressure relationship before the point of flow limitation. The following equation was used to describe this curve:

\[
P/V = K\dot{V}
\]

where \(P\) is pressure, \(\dot{V}\) is flow, and the coefficient \(K\) was calculated separately for inspiration \((K_{\text{insp}}})\) and expiration \((K_{\text{exp}})\) for each condition. For each individual, \(K_{\text{exp}}\) was calculated at a similar flow rate across conditions. The pressure-flow data for all the subjects fit this equation with an average \(r^2\) of 0.921.

Data were recorded on a computerized sleep system (CNS) and simultaneously on videotape (Neurodata) for later processing.

Experimental methods. The effects of decreased upper airway muscle activity on upper airway resistance were examined by inhibiting the activity of these muscles and observing the effect on the resistance across the supraglottic airway. It has been demonstrated that CPAP inhibits upper airway muscle activity \((1, 10, 18)\). It was reasoned that if CPAP were applied to the upper airway of sleeping subjects until there was a reduction in upper airway EMG activity and then the CPAP was abruptly removed, the resistance measured would be that of the airway with minimal influence of active muscles. The first breath after this return to ambient pressure was measured, inasmuch as it has been shown that, during sleep, there is no or very little immediate EMG response to an increase in upper airway resistance in humans \((2)\). The response to upper airway loading during sleep is largely dependent on increases in chemical drive, which would not be sensed during this first breath. A series of breaths after the termination of CPAP was also measured in a subgroup of subjects to observe the time course of the recovery of EMG activity and upper airway resistance.

After instrumentation and calibrations, subjects were allowed to fall asleep. All subjects slept in the supine position. A curved pillow and the manner in which the apparatus was fixed maintained head position constant throughout the study.

Once subjects were in a steady state of stage 3/4 sleep for at least 3 min, data collection began. Control (ambient pressure) data were collected for 1 min, then CPAP was increased until phasic EMG activity was eliminated or the CPAP was as high as the subject could tolerate without arousal. Data were collected for 1 min, then during expiration CPAP was rapidly decreased to ambient and data collection continued for the subsequent period. After at least 3 min of stable breathing at ambient pressure, the process was repeated.

Analysis. Ten breaths before and during CPAP administration were analyzed, and the means were calculated for each variable. The average mean from all trials in each individual was then calculated. Data from the first ambient breath immediately after withdrawal of CPAP were analyzed separately. Linear regressions were performed to determine the relationship between the parameters. Nonparametric \(t\)-tests were performed on the EMG and resistance data because of the between-subject variability. \(P < 0.05\) was considered to be significantly different. To determine whether CPAP affected end-expiratory lung volume, the end-expiratory level of the Respitrace signal was compared between the breaths immediately after the termination of CPAP and the breaths immediately preceding the termination of CPAP.

RESULTS

\(R_{\text{insp}}\) and \(R_{\text{exp}}\). Control \(K\) data during slow-wave sleep for inspiration and expiration are shown in Table 1. Although there was a trend for \(K\) to be higher in the snorers, the difference was not significant. This was also true for resistance measured at peak flow, i.e., \(R_{\text{insp}}\) \((7 \pm 1 \text{ and } 10 \pm 2 \text{ cmH}_2\text{O} \cdot \text{L}^{-1} \cdot \text{s}^{-1} \text{ for nonsnorers and snorers, respectively})\). Flow limitation, however, was observed only in the snorers and only during inspiration. Figure 1 shows representative pressure-flow curves for a snorer and a nonsnorer. Hysteresis in the inspiratory pressure-flow curve was observed in snorers but not in nonsnorers. For both groups, there was a significant relationship between \(K_{\text{insp}}\) and \(K_{\text{exp}}\) (Fig. 2). However, as shown in Fig. 2, the regression for the nonsnorers lies close to the line of identity, indicating a greater similarity between \(K_{\text{insp}}\) and \(K_{\text{exp}}\) than in the snorers.

Thirteen subjects had phasic EMGg and 13 had phasic EMGan during ambient control recording in slow-wave sleep. All snorers had phasic EMGg and EMGan. Table 2 shows average phasic and tonic EMG activity for both groups. Only peak EMGan was significantly different between the snorers and nonsnorers.

CPAP effects. The CPAP was 7–12 cmH\(_2\)O. For the snorers, \(K_{\text{insp}}\) was significantly reduced with CPAP but was unchanged in the nonsnorers (Fig. 1, Table 1). The reduction in \(R_{\text{insp}}\) was also significant in the snorers \((10 \pm 2 \text{ and } 4 \pm 1 \text{ cmH}_2\text{O} \cdot \text{L}^{-1} \cdot \text{s}^{-1} \text{ for control and CPAP, respectively})\) but not in the nonsnorers \((7 \pm 1 \text{ and } 6 \pm 2 \text{ cmH}_2\text{O} \cdot \text{L}^{-1} \cdot \text{s}^{-1} \text{ for control and CPAP, respectively})\). CPAP significantly decreased \(K_{\text{exp}}\) in the snorers but significantly increased \(K_{\text{exp}}\) in the nonsnorers. The \(K\) values for nasal resistance were not affected by the application of CPAP \((29 \pm 12 \text{ and } 47 \pm 24 \text{ cmH}_2\text{O} \cdot \text{L}^{-1} \cdot \text{s}^{-1} \text{ for control and CPAP, respectively})\).

In the snorers, CPAP significantly reduced peak EMGg and peak and tonic EMGan (Fig. 3, Table 2). Peak EMGan was significantly reduced in the nonsnorers. Phasic EMGg activity was eliminated by CPAP in

<table>
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<th>Table 1. K data for snorers and nonsnorers</th>
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<tr>
<td>(K_{\text{insp}})</td>
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<td>-----</td>
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<tr>
<td>Control</td>
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<td>CPAP</td>
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Values are means ± SE in cmH\(_2\)O \cdot \text{L}^{-1} \cdot \text{s}^{-1}. \(K_{\text{insp}}\) and \(K_{\text{exp}}\) coefficients of inspiratory and expiratory resistance. CPAP, continuous positive airway pressure; breath 1, 1st breath after termination of CPAP. *Significantly different from control, \(P < 0.05\).
7 of 11 subjects in whom phasic activity was observed. Six of the 13 subjects with phasic EMGan had no phasic activity during CPAP.

Effects of a hypotonic airway. The termination of CPAP did not result in any consistent change in end-expiratory lung volume for at least 30 s after the end of CPAP. There were no changes in EMGg and EMGan during the first breath after termination of CPAP (Table 2). When $K_{\text{insp}}$ was compared between ambient control (active muscles) and the first breath after termination of CPAP (hypotonic), there were no significant differences for snorers or nonsnorers. $R_{\text{insp}}$ was actually lower during the first breath after termination of CPAP in the snorers ($10 \pm 2$ and $5 \pm 1 \text{cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$ for control and after termination of CPAP, respectively, $P < 0.03$) and the nonsnorers ($7 \pm 1$ and $5 \pm 1 \text{cmH}_2\text{O} \cdot \text{l}^{-1} \cdot \text{s}$ for control and after CPAP, respectively, $P < 0.004$). $K_{\text{exp}}$ also did not change from ambient control for the snorers or nonsnorers during the first hypotonic breath. There were no correlations between any control EMG value (phasic or tonic) and the effects of the hypotonic airway on $K_{\text{insp}}$ or $K_{\text{exp}}$ during the first breath after termination of CPAP.

In the snorers, there was a tendency for resistance and $K_{\text{insp}}$ to be less during the first hypotonic breath than during control with the muscles activated. Generally, 4–18 breaths were required for resistance and $K_{\text{insp}}$ to return to the pre-CPAP level. Snoring often took much longer to reoccur. Figures 4 and 5 illustrate this gradual increase in resistance in two individual snorers after CPAP was discontinued.

**DISCUSSION**

The purpose of this study was to examine the role of the upper airway muscles in determining upper airway resistance, both inspiratory and expiratory, during sleep in young snorers and nonsnorers. The findings demonstrate that, during sleep, 1) inspiratory and expiratory resistances, as characterized by the coefficient $K$, are related in nonsnorers and snorers. In snorers, however, $K_{\text{insp}}$ can be substantially greater than $K_{\text{exp}}$. 2) CPAP has differential effects on $K_{\text{exp}}$ in snorers vs. nonsnorers; and 3) phasic upper airway

![Fig. 1. Single-breath pressure-flow loops from a nonsnoring (A) and a snoring subject (B) during ambient control, during continuous positive airway pressure (CPAP), and at 1st breath after termination of CPAP (Br 1).](image1)

![Fig. 2. Coefficients of inspiratory vs. expiratory resistance ($K_{\text{insp}}$ vs. $K_{\text{exp}}$) for all subjects (nonsnoring (○) and snoring (●)). Solid lines, regression lines for each group; dashed line, line of identity. Regression for nonsnoring subjects lies close to line of identity, in contrast to regression line for snoring subjects.](image2)

**Table 2. Average EMG data for snorers and nonsnorers**

<table>
<thead>
<tr>
<th></th>
<th>Snorers</th>
<th>Nonsnorers</th>
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<tbody>
<tr>
<td>EMGg Peak</td>
<td>13 ± 6</td>
<td>8 ± 4</td>
</tr>
<tr>
<td></td>
<td>CPAP 7 ± 4†</td>
<td>4 ± 1</td>
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<tr>
<td>Tonic</td>
<td>4 ± 2</td>
<td>5 ± 3</td>
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<tr>
<td></td>
<td>CPAP 4 ± 2</td>
<td>4 ± 2</td>
</tr>
<tr>
<td>EMGAn Peak</td>
<td>23 ± 12</td>
<td>8 ± 1*</td>
</tr>
<tr>
<td></td>
<td>CPAP 9 ± 6†</td>
<td>3 ± 1</td>
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<tr>
<td>Tonic</td>
<td>5 ± 2</td>
<td>4 ± 1</td>
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<tr>
<td></td>
<td>CPAP 2 ± 1†</td>
<td>3 ± 1</td>
</tr>
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</table>

Values are means ± SE. EMGg and EMGan, genioglossus and alae nasi EMG activity. *Significantly different from snorers $P < 0.05$. †Significantly different from control, $P < 0.05$. 

these findings may relate to the differences in measure-
resistance in nonsnorers. The difference in
found no difference between inspiratory and expiratory
suciently greater than
K
insp was also higher than K
exp in the snorers, and in
some snorers this difference was substantial. This
difference between inspiratory and expiratory resist-
tance was not observed in OSA patients in a study by
Sanders and Moore (17). These investigators found that
when inspiratory resistance increased in the breath
immediately preceding an apnea, a similar change was
also noted in expiratory resistance. The authors in-
ferred from this that the airway was narrowing during
expiration as airflow decreased and that inspiratory
effort was not required to cause airway closure in OSA.
Expiratory flow limitation, as evidenced from the pres-
ure-flow curve, was not observed in any subject in the
present study. The difference between these two studies
is most likely due to the differences in the subject
groups. Diagnostic sleep studies were performed on all
but one subject, and none had an apnea/hypopnea
index of more than two events per hour of sleep. Despite
evidence of inspiratory airway narrowing in this and other
studies (11), airway closing pressure is
subatmospheric (4, 7). In contrast, individuals with
OSA have closing pressures above atmospheric, so that,
as flow rate decreases toward end expiration, transmu-
ratal pressure becomes positive and airway narrowing or
collapse can occur. This would not be expected to occur
in nonapneic snorers, who have more negative critical
or closing pressure.

An important finding of this study is that the inhibi-
tion of upper airway muscle activity in snorers did not
cause an increase in either marker of inspiratory resist-
ance in the first breath after discontinuation of
CPAP. It also was a consistent finding of this study that
snoring also did not occur immediately after the return
to ambient pressure from CPAP. Snoring and/or flow
limitation took several breaths to several minutes to
develop and required a substantial increase in Psg.

During this time there were no measurable changes in
date-expiratory lung volume. These findings suggest
that increased drive, i.e., increased suction pressure, is
important in developing the increased upper airway resist-
tance in snorers. The increased suction on the airway may
cause gradual narrowing of the upper airway, which
results in hypventilation and hypercapnia, further
creases in drive, and further suction on the airway.
Although end-tidal CO
2 was not measured during this
study, it has previously been demonstrated that
increased upper airway resistance during sleep results in
hypercapnia and that this hypercapnia can be reversed
when inspiratory resistance is reduced with CPAP (6).

It has been suggested that one mechanism by which
upper airway resistance increases is through vascular
engorgement due to suction on the airway (24). It is
unlikely, however, that vascular engorgement contrib-
uted to the increased resistance in these snorers or that
the absence of engorgement due to the positive pres-
sure of CPAP explains the relatively low resistance
immediately after termination of CPAP. The effects of
vascular engorgement would most likely be observed in
the measurement of nasal resistance (24). CPAP did not
decrease nasal resistance in the six subjects in whom it
was measured; thus it is unlikely that a gradual
increase in nasal resistance due to increasing engorge-
ment could explain the slow increase in upper airway
resistance in this group of subjects.

The finding that reducing upper airway muscle activity
did not result in an increase in upper airway
resistance in the snorers also suggests that, despite the
increased collapsibility of the upper airway in snorers

![Integrated genioglossus and alae nasi EMG activity](http://jap.physiology.org/DownloadedFrom)
(7), the upper airway muscle activity that was observed is not required to maintain upper airway patency, at least during normocapnic, normal drive (i.e., nonsnor- ing) conditions. That is, with reduced muscle activity, neither measure of upper airway resistance was elevated in the first breaths after discontinuation of CPAP. The activity of these muscles has been shown to increase in response to hypercapnia (5, 23) and increased inspiratory resistance (2). These upper airway muscles, then, are most likely not recruited to compensate for a smaller airway per se but are recruited incrementally as the airway is further narrowed by increasingly negative intraluminal pressures.

Limitations. The purpose of this study was to examine upper airway resistance in the hypotonic airway. CPAP did not completely inhibit phasic EMG activity in all subjects, a finding that has been reported by other investigators (10, 18). The maintenance of phasic EMG activity, although reduced, may have resulted in an underestimation of the susceptibility of the airway to...
collapse when CPAP was removed. In addition, only two upper airway muscles were studied. It is possible that other upper airway muscles were not inhibited by CPAP and continued to have a significant effect on upper airway patency. The genioglossus and the alae nasi were chosen because they continue to exhibit phasic inspiratory activity during non-rapid-eye-movement sleep (25–27) and because CPAP has been shown to reduce their activity (1, 10, 14, 18). Muscles with only tonic activity have been shown to significantly decrease their activity during sleep (20), and CPAP would not be expected to have any further effect on these already hypotonic muscles. Although CPAP did not fully inhibit phasic activity in all subjects, there were significant reductions in phasic and tonic activity with CPAP. Although the airway was not atonic, the reduction in activity should have had an impact on upper airway resistance if this activity were essential for maintaining patency. Finally, it should be noted that the snorers included in this study were young and of normal weight for height. They would not have been classified as severe snorers, and none gave a history of witnessed apneas. These findings, then, may not be applicable to obese snorers or snorers with witnessed apneas.

In summary, young snorers and nonsnorers are not dependent on phasic upper airway muscle activity to maintain upper airway patency during sleep, at least during nonsnorning conditions. The genioglossus and alae nasi rather appear to be recruited in response to the increased suction on the airway and resultant hypercapnia, which gradually develop during sleep. Finally, the marked sleep-induced increases in inspiratory resistance that were observed in some snorers were not accompanied by a similar increase in inspiratory resistance. This may reflect the subatmospheric critical pressure that has been reported in snorers.

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