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 26 ± 5 and 80 ± 27 cmH2O·l·s-1 in the nonsnorers and snorers, respectively. On the breath immediately after CPAP, Kinsp did not increase over control in snorers (80 ± 27 cmH2O·l·s-2) for the breath after CPAP) or nonsnorers (26 ± 5 vs. 29 ± 6 cmH2O·l·s-1). These findings held true for Rexp and expiratory flow limitation are not observed in young snorers.

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 (Respirac), 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) 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.
(Statham). The pressure transducers were kept at a fixed point relative to the catheter tip. Supraglottic pressure (Psg) was measured 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 (Rinsp) and expiratory (Rexp)] was measured at peak flow (Psg = peak flow). Inasmuch as the measurement of inspiratory flow resistance at a single point has been shown to be insufficient to characterize upper airway mechanics (3), Rinsp and Rexp were also characterized by the flow-pressure relationship before the point of flow limitation (8). The following equation was used to describe this curve

\[ P/V = K \cdot V \]

where P is pressure, V is flow, and the coefficient K was calculated separately for inspiration (K_{insp}) and expiration (K_{exp}) for each condition. For each individual, K was calculated at a similar flow rate across conditions. The pressure-flow data for all the subjects fit this equation with an average r² 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 was 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 Respirac pressure signal was compared between the breaths immediately after the termination of CPAP and the breaths immediately preceding the termination of CPAP.

**RESULTS**

Rinsp and Rexp. 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., Rinsp (7 ± 1 and 10 ± 2 cmH₂O·l⁻¹·s⁻¹ 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_{insp} and K_{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_{insp} and K_{exp} than in the snorers.

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

CPAP effects. The CPAP was 7–12 cmH₂O. For the snorers, K_{insp} was significantly reduced with CPAP but was unchanged in the nonsnorers (Fig. 1, Table 1). The reduction in Rinsp was also significant in the nonsnorers (10 ± 2 and 4 ± 1 cmH₂O·l⁻¹·s⁻¹ for control and CPAP, respectively) but not in the nonsnorers (7 ± 1 and 6 ± 2 cmH₂O·l⁻¹·s⁻¹ for control and CPAP, respectively). CPAP significantly decreased K_{exp} in the snorers but significantly increased K_{exp} in the nonsnorers. The K values for nasal resistance were not affected by the application of CPAP (29 ± 12 and 47 ± 24 cmH₂O·l⁻¹·s⁻¹ for control and CPAP, respectively).

In the snorers, CPAP significantly reduced peak EMGガ and peak and tonic EMGガ (Fig. 3, Table 2). Peak EMGガ was significantly reduced in the nonsnorers. Phasic EMGガ activity was eliminated by CPAP in
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 EMGgg 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 $5 \pm 1$ cmH$_2$O·l$^{-1}$·s for control and after termination of CPAP, respectively, $P < 0.03$) and the nonsnorers ($7 \pm 1$ and $5 \pm 1$ cmH$_2$O·l$^{-1}$·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

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**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><strong>EMGgg Peak</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>13±6</td>
<td>8±4</td>
</tr>
<tr>
<td>CPAP</td>
<td>7±4†</td>
<td>4±1</td>
</tr>
<tr>
<td><strong>Tonic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>4±2</td>
<td>5±3</td>
</tr>
<tr>
<td>CPAP</td>
<td>4±2</td>
<td>4±2</td>
</tr>
<tr>
<td><strong>EMGan Peak</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>23±12</td>
<td>8±1*</td>
</tr>
<tr>
<td>CPAP</td>
<td>9±6†</td>
<td>3±1</td>
</tr>
<tr>
<td><strong>Tonic</strong></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Control</td>
<td>5±2</td>
<td>4±1</td>
</tr>
<tr>
<td>CPAP</td>
<td>2±1†</td>
<td>3±1</td>
</tr>
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Values are means ± SE. EMGgg and EMGan, genioglossus and alae nasi EMG activity. *Significantly different from snorers $P < 0.05$. †Significantly different from control, $P < 0.05$. 

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**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).

**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.
Also noted in inspiratory resistance. The authors inferred 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 pressure-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 nonsnorers 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, transmural 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 inhibition of upper airway muscle activity in snorers did not cause an increase in either marker of inspiratory resistance 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 end-expiratory lung volume. These findings suggest that increased drive, i.e., increased suction pressure, is important in developing the increased upper airway resistance in snorers. The increased suction on the airway may cause gradual narrowing of the upper airway, which results in hypoventilation and hypercapnia, further increases in drive, and further suction on the airway. Although end-tidal CO2 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 contributed to the increased resistance in these snorers or that the absence of engorgement due to the positive pressure 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 engorgement 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...
(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., nonsnoring) 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

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**Fig. 4.** Top: 6 single-breath pressure-flow loops from period immediately after termination of CPAP. Note progressive increase in slope of curve, indicating a progressive increase in inspiratory resistance. Bottom: tracings of flow, supraglottic pressure (Psg), EMGgg, and EMGan from last few breaths on CPAP and for period immediately after termination of CPAP (arrow). 1–6, 6 breaths corresponding to loops shown at top.

**Fig. 5.** Tracings of flow (\(\dot{V}\)), Psg, EMGgg, and EMGan in a snoring subject. Far left: ambient control conditions. Solid bar, CPAP. With application of CPAP, there is a reduction in Psg and EMG activity and an increase in flow. After CPAP is terminated, Psg and EMG activities increase gradually but remain well below control conditions for at least 3 min. MA, moving average; Inspir, inspiration; Expir, expiration.
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 alae nasi and the genioglossus 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 airway patency during sleep, at least during nonsnoring 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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