Surface Tension of Upper Airway Mucosal Lining Liquid in Obstructive Sleep Apnea/Hypopnea Syndrome

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Study Objectives: The obstructive sleep apnea hypopnea syndrome (OSAHS) is a disorder characterized by repetitive closure and reopening of the upper airway during sleep. Upper airway luminal patency is influenced by the surface tension (γ) acting within the liquid layer lining the upper airway. The aim of the present study was to examine the γ of upper airway mucosal lining liquid (UAL) in both healthy subjects and patients with OSAHS before and after sleep.

Design: Measurements were performed before (PM) and after (AM) an overnight polysomnographic study.

Setting: Sleep laboratory.

Participants: We studied 11 healthy adults (5 men, 6 women) and 15 patients with OSAHS (14 men, 1 woman).

Interventions: None.

Measurements and Results: The γ of UAL (“pull-off” force technique, pooled PM and AM samples) in patients with OSAHS was increased (59.9 [53.7, 59.5] mN/m; mean [95% confidence interval]) compared with healthy subjects (56.3 [57.7, 62.1] mN/m; linear mixed effects models; P = .05). In both groups there was no significant difference between PM (56.6 [53.7, 59.5] mN/m for healthy subjects, 60.1 [57.9, 62.3] mN/m for the patients with OSAHS) and AM (56.1 [51.8, 60.4] mN/m and 59.6 [57.4, 61.8] mN/m, respectively) samples for γ of UAL and salivary flow rate (5 minutes unstimulated collection; PM =0.53 [0.22, 0.84] mL/minute for healthy subjects, 0.38 [0.22, 0.54] mL/minute for OSAHS; AM=0.39 [0.23, 0.55] mL/minute and 0.32 [0.2, 0.44] mL/minute). However, the occurrence of nasal breathing during sleep was associated with a fall in γ of UAL overnight (r² = 0.15, P < .05).

Conclusion: Patients with OSAHS have normal salivary flow rate but an increased γ of UAL. In both healthy subjects and OSAHS patients, nasal breathing during sleep was associated with an overnight fall in the γ of UAL.

Key Words: Obstructive sleep apnea, upper airway mechanics, surface tension

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INTRODUCTION

THE OBSTRUCTIVE SLEEP APNEA HYPOPNEA SYNDROME (OSAHS) IS CHARACTERIZED BY REPETITIVE CLOSURE AND REOPENING OF THE UPPER AIRWAY during sleep. A widely accepted analysis of the control of upper airway patency is based on the concept that upper airway luminal size will be dependent on the balance of forces acting across the upper airway walls.1 While the role played by intraluminal pressure and the action of upper airway dilator muscles in determining this balance of forces has been extensively studied, little attention has been paid to other forces that may be in operation. One such force is exerted by the surface tension (γ) acting within the liquid layer lining the upper airway. The aim of the present study was to examine the γ of upper airway mucosal lining liquid (UAL) in both healthy subjects and patients with OSAHS before and after sleep.

One such force is exerted by the surface tension (γ) acting within the liquid layer lining the upper airway. The aim of the present study was to examine the γ of upper airway mucosal lining liquid (UAL) in both healthy subjects and patients with OSAHS before and after sleep.

In a previous study, we found that the γ of UAL (~57 mN/m) in healthy subjects was similar to that of saliva.8 During sleep, salivary flow rates are depressed,9 providing a situation in which salivary coating of the pharyngeal mucosal wall may be reduced. A reduction of saliva may potentially contribute to a relatively high γ of UAL and rendering the collapsible segments (retropalatal, retroglossal) of the upper airway more vulnerable to narrowing and closure. In the present study, we measured the γ of UAL and the rate of production of saliva before and after sleep in both healthy subjects and patients with OSAHS. In addition, we monitored breathing route (nasal versus oral breathing) during sleep in order to test the hypothesis that mouth breathing (with attendant potential dehydration of the UAL) may influence γ of UAL.
METHODS

Subjects

Healthy volunteers were screened via questionnaire and excluded if they were smokers, had recent or current upper respiratory tract infections or previous upper airway surgery, or gave a history of habitual snoring. Patients referred to a sleep laboratory for a diagnostic polysomnography (PSG) study because of suspected OSAHS were also recruited. Subjects were retained in the healthy and OSAHS groups based on the level of sleep-disordered breathing detected during overnight PSG. Healthy subjects with an apnea-hypopnea index (number of apneas plus hypopneas per hour; AHI) < 5 per hour remained in the healthy group, while patients with suspected obstructive sleep apnea required an AHI > 5 per hour to remain in the OSAHS group. Subjects who did not meet these criteria were excluded from the study. In all, 11 healthy volunteers (5 men, 6 women; age: 30 years [20, 40; 95% confidence interval]; body mass index: 23.6 kg/m² [21.6, 25.6]) and 15 patients with OSAHS (14 men, 1 woman; age: 36 years [25, 45]; body mass index: 33.0 kg/m² [31.6, 35.4]) were included in the study. No patients with OSAHS had undergone treatment for their sleep-disordered breathing at the time of study. Informed written consent was obtained from each participant, and the protocols were approved by the Western Sydney Area Health Service Human Ethics Committee.

MEASUREMENTS

The \( \gamma \) of UAL

The \( \gamma \) of UAL was measured using the “pull-off” force technique, a methodology we have described previously. Briefly, the “pull-off” technique measures \( \gamma \) as the force required to separate 2 silica surfaces bridged by a droplet of the liquid under study. Once the droplet is in place, a motor-driven translation stage bends a double cantilever spring that applies a force to separate the 2 silica surfaces. When this force overcomes the \( \gamma \) of the liquid sample, the 2 surfaces separate. A micrometer measures the amount of spring bending (“jump distance”). The device is calibrated with liquids of known \( \gamma \). Using this approach, we determined \( \gamma \) with a surface-force measurement device as the force required to overcome the adhesion of 1 silica surfaces bridged by a sample (~0.2 µL) of the test liquid.

UAL Sampling

The UAL was sampled by advancing polyethylene tubing (internal diameter = 0.5 mm; external diameter = 0.8 mm) via the mouth to the posterior pharyngeal wall. An attached 1-mL syringe (1 mL, Terumo Medical Corporation, Elkerton, MD) was then used to draw a small quantity (5-10 µL) of UAL into the tubing. Samples were then transferred with a 1-µL syringe (7000.5 N, Hamilton Company, Reno, Nevada) to the surface-force measurement device.

Salivary Flow Rate

Salivary flow rate was determined using a standard nonstimulated saliva-collection method, as previously described. Subjects were seated, leaning forward with their mouths open, breathing via the nose, and were asked to not swallow. Saliva was collected (dribbled) in a container for a 5-minute period and salivary flow rate expressed in mL/minute.

Polysomnography

Nocturnal PSG was performed in a sleep laboratory. Signals recorded included left and right electrooculogram, submental electromyogram, electroencephalogram, arterial oxygen saturation, nasal pressure (Salter Adult Nasal Cannulas, Salter Labs, Arvin, Calif), chest and abdominal wall movement (impedance plethysmography), and body position. All monitored parameters were recorded directly on a computer software system (S Series, Compumedics, Melbourne, VIC, Australia). Sleep staging was performed manually according to Rechtschaffen and Kales. Arousals were identified according to American Academy of Sleep Medicine criteria, and respiratory events using the “Chicago” criteria.

Breathing Route During Sleep

Breathing route during sleep was monitored using a dual-channel thermocouple device (F-ONT2A, Grass Telefactor, West Warwick, RI) with separate oral and nasal probes. The device was placed on the subject’s upper lip with the thermocouple leads supported by hooking them behind the ears and taping them to the subject’s cheeks. The absence of cross-contamination was verified at the commencement of each study by having the subject voluntarily breathe via the nasal and oral routes separately and ensuring that no signal was detected for the opposite route.

Protocol

Subjects had nothing to eat or drink for 1.5 to 2 hours prior to sleep. Immediately before “lights out” (~10 PM) a salivary flow-rate collection was performed with the subject seated. A single sample of UAL (PM sample) was then obtained with the subject supine on the bed. Subjects then slept throughout the night until awakened at 5:30 to 6:00 AM. Immediately upon waking, a second single UAL sample (AM sample) was obtained while each subject was still supine. This was followed by a salivary flow-rate collection in the seated position.

Data Analysis

All data was analyzed by 1 or more of the authors, none of whom were blind to the study.

Sleep Parameters

The total sleep time (TST) for each subject was calculated from the number of 30-second sleep (total non-rapid eye movement stages 1 to 4 plus rapid eye movement) epochs for the duration of the study. An arousal index (AI) was calculated as the total number of arousals detected divided by the TST. The respiratory disturbance index was calculated as the total number of apneas and hypopneas divided by TST. The duration of non-rapid eye movement and rapid eye movement sleep was expressed as a percentage of TST. Oxyhemoglobin saturation was expressed as the minimum overnight value (MinSaO2).
Breathing Route

The nasal and oral thermocouple signals were used to classify the breathing route during sleep, assessed in each 30-second epoch as either a nasal, oral, or oronasal breathing epoch. Nasal breathing epochs were defined as epochs containing ≥ 3 consecutive phasic signals on the nasal thermocouple channel only. Oral breathing epochs were defined as epochs containing ≥ 3 consecutive phasic signals on the oral thermocouple channel only. Oronasal breathing epochs contained ≥ 3 consecutive phase-linked signals on both the nasal and oral thermocouple channels. The occurrence rate for nasal, oral, and oronasal breathing epochs was expressed as a percentage of the total sleep epochs (TSE) analyzed.

Statistical Analysis

Data are expressed as mean [95% confidence interval]. Student unpaired t tests were used to examine group mean differences for body mass index, sleep parameters, and breathing route. The effect of sleep period (PM versus AM) and subject group (healthy vs OSAHS) on γ of UAL and salivary flow rate was examined by including both AM and PM data in a linear mixed-effects model. Both linear and logistic regression analyses were used to examine the relationship between breathing route during sleep, and γ of UAL. P < .05 was considered significant.

RESULTS

Subjects

There was no significant difference in group mean age between the healthy subjects and patients with OSAHS (P = .5). The groups, however, were not matched for sex or body mass index (P < .001).

Sleep-Disordered Breathing

There was no significant difference for group mean TST between the healthy subjects (320 minutes [272, 368]) and the patients with OSAHS (293 minutes [258, 328]; Table 1; P = .4). However, the AHI and AI were greater and the MinSaO2 and rapid eye movement sleep time lower in the patients with OSAHS (Table 1, all P < .05).

γ of UAL

For PM samples, the γ of UAL was 56.6 [53.7, 59.5] mN/m for the healthy subjects and 60.1 [57.9, 62.3] mN/m for the patients with OSAHS. Corresponding values for AM samples were 56.1 [51.8, 60.4] mN/m and 59.6 [57.4, 61.8] mN/m, respectively. When both PM and AM values were considered, the γ of UAL in healthy subjects was 3.5 [0, 7.1] mN/m lower than in the patients with OSAHS (56.3 [53.8, 58.8] mN/m versus 59.9 [57.7, 62.1] mN/m, respectively; P = .05; Figure 1). Overnight, a decrease in γ of UAL was observed in 5 healthy subjects and 7 patients with OSAHS and an increase in 6 healthy subjects and 7 patients with OSAHS, while γ of UAL was unchanged overnight in the remaining patients with OSAHS. There was no significant difference

Table 1—Sleep Parameters in Healthy Subjects and Subjects with Obstructive Sleep Apnea-Hypopnea Syndrome

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Healthy</th>
<th>OSAHS</th>
<th>P value</th>
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<tbody>
<tr>
<td>TST, min</td>
<td>320 [272, 368]</td>
<td>293 [258, 328]</td>
<td>.4</td>
</tr>
<tr>
<td>AHI, events/h</td>
<td>2 [1, 3]</td>
<td>44 [34, 54]</td>
<td>.0001</td>
</tr>
<tr>
<td>AI, arousals/h</td>
<td>10 [7, 13]</td>
<td>33 [23, 43]</td>
<td>.0002</td>
</tr>
<tr>
<td>Min SaO2, %</td>
<td>98 [96, 100]</td>
<td>87 [78, 96]</td>
<td>.001</td>
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</table>

Data are mean [95% confidence interval]. OSAHS refers to obstructive sleep apnea-hypopnea syndrome; TST, total sleep time; AHI, apnea-hypopnea index; AI, Arousal index; REM, rapid eye movement sleep time; Min SaO2, = overnight minimum oxygen saturation.

Figure 1—Surface tension (γ) of upper airway mucosal lining liquid (UAL) in healthy subjects and patients with obstructive sleep apnea/hypopnea syndrome (OSAHS). Plots showing γ of UAL in (A) healthy subjects and (B) patients with OSAHS before (PM) and after (AM) overnight polysomnography. Symbols and lines denote individual subject values, thick bars are the group mean data. There was no significant difference between PM and AM values for γ of UAL in either group. However, overall (ie, PM plus AM data), the γ of UAL was significantly higher in the OSAHS group than in the healthy subjects (P = .05).
between the group mean PM and AM values for either group (P > .6; Figure 1). There was no significant relationship between subject body mass index values and γ of UAL values for either the PM or AM samples or for the difference between the PM and AM values (all P > .1).

Salivary Flow Rate

For healthy subjects, the group mean PM salivary flow rate was 0.53 [0.22, 0.84] mL/minute, while the AM rate was 0.39 [0.23, 0.55] mL/minute. Corresponding values for patients with OSAHS were 0.38 [0.22, 0.54] mL/minute and 0.32 [0.2, 0.44] mL/minute, respectively. The AM salivary flow rates were less than PM flow rates in 6 healthy subjects and 9 patients with OSAHS, while the reverse occurred for the remaining subjects (Figure 2). However, there was no significant difference between group mean PM and AM values or between patients with OSAHS and healthy control values (all P > .1).

Breathing Route During Sleep

Nasal and oronasal breathing occurred in approximately equal amounts in both healthy subjects and patients with OSAHS (Table 2). Pure mouth breathing did not occur in the healthy subjects and was rare in the patients with OSAHS. There was no significant difference in the occurrence rate for nasal and oronasal breathing during sleep (P > .8) for both healthy individuals and patients with OSAHS and also no difference between the OSAHS and healthy groups (P > .9).

Breathing Route and Change in γ of UAL

For the total group (healthy + OSAHS), there was a significant correlation between the magnitude of an overnight fall in γ of UAL (Δγ, AM-PM values) and the occurrence rate for nasal-only breathing epochs during sleep (linear regression, \( r^2 = 0.15, P < .05 \); Figure 3). Logistic regression analysis confirmed that an occurrence rate of > 50% TSE for nasal breathing was a significant predictor for an overnight decrease in γ of UAL of > 5 mN/m (P = .02, odds ratio = 16.8 [1.6, 176]). For the subjects with > 50% TSE for nasal breathing, there was a significant overnight decrease of 3.1±4.2 mN/m in the γ of UAL (P < .02, unpaired t test), whereas for those subjects with < 50% TSE for nasal breathing, there was no significant overnight change in the γ of UAL (P = .16). Moreover, the AHI for patients with OSAHS with > 50% TSE for nasal breathing was significantly lower than for those with < 50% TSE for nasal breathing (26 ± 14 events/hour vs 55 ± 16 events/hour, mean ± SEM, P < .005).

DISCUSSION

In the present study, there were no differences in sleep salivary flow rates before and after PSG between healthy subjects and patients with OSAHS. However, values for γ of UAL were approximately 3.5 mN/m higher in patients with OSAHS. While there was no group mean difference between PM and AM samples when individual overnight changes in γ of UAL were examined; these individual changes correlated with breathing route during sleep. Moreover, individuals spending more than 50% of the sleep period utilizing nasal-only breathing were approximately 17 times more likely to have an AM γ of UAL value that was > 5 mN/m less than their PM value. Thus, it appears that nasal-only breathing during sleep promoted an overnight fall in γ of UAL, a process that was negated when there was frequent oronasal breathing.

We measured γ of UAL using the “pull-off” technique, a methodology that we have previously described. In our laboratory, the use of this technique is associated with a coefficient of variation for repeated measurements of γ of UAL from the same sample of approximately 5%, while for multiple samples from the same subject under the same conditions, the coefficient of variation is approximately 6%. The values for γ of UAL determined in the present study are similar to those we have reported previously for anesthetized rabbits, anesthetized humans, and a different group of patients with OSAHS. At approximately 57 mN/m in healthy subjects, the γ of posterior pharyngeal wall UAL is substantially less than that for water (71.2 mN/m), reflecting the presence of endogenous surfactants.

In the present study, the γ of UAL was slightly, but significantly, higher in the patients with OSAHS than in the healthy subjects.

<table>
<thead>
<tr>
<th>A) Healthy subjects</th>
<th>B) OSAHS patients</th>
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<td><img src="image1.png" alt="Graph" /></td>
<td><img src="image2.png" alt="Graph" /></td>
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Figure 2—Salivary flow rates in healthy subject and patients with obstructive sleep apnea/hypopnea syndrome (OSAHS). Plots showing the non-transformed salivary flow rates in (A) healthy subjects and (B) patients with OSAHS before (PM) and after (AM) overnight polysomnography. Symbols and lines denote salivary flow rate values for each subject; thick bars are the group mean data. There was no significant difference in the salivary flow rates between patients with OSAHS and healthy subjects or between AM and PM values.
However, this result should be interpreted with caution, since the difference is only small and our sample size is limited. Since we have previously demonstrated in patients with OSAHS that a lower $\gamma$ of UAL is associated with a reduction in the collapsibility of the upper airway during sleep, this higher $\gamma$ would tend to promote increased collapsibility. Using our previous data, a reduction in $\gamma$ of UAL of approximately 3.5 mN/m would be associated with a reduction in AHI of approximately 6 events per hour. The clinical importance of this is obviously different if the base AHI is 10 to 15 events per hour versus say 50 events per hour.

Patients with OSAHS are known to have smaller pharyngeal airway dimensions and increased upper airway mucosal folding, as compared with healthy subjects. Consequently, they may be more vulnerable to the effects of a normal $\gamma$ of UAL, let alone an increased $\gamma$ of UAL. Since the distance separating mucosal surfaces will be small in the recesses of pharyngeal wall mucosal folds, the influence of $\gamma$ of UAL as an adhesive force will be enhanced. Smaller dimensions and increased mucosal folding in patients with OSAHS may serve to amplify the effect of relatively small changes in the $\gamma$ of UAL on upper airway patency, particularly on the process of reopening a closed pharyngeal airway.

We have previously shown that, in healthy subjects, the $\gamma$ of saliva samples from the oral cavity is similar to that for UAL. In the course of swallowing, saliva from the oral cavity is distributed to the mucosal surface of the oropharynx. This process is thought to assist in lubricating the passage of food through the pharynx and to initiate the digestive process. However, saliva is continuously produced and swallowed, even during sleep, thus providing a potential continuing contribution to the $\gamma$ of UAL. Since saliva is known to contain surface-active phospholipids and has a relatively low $\gamma$ (approximately 57 mN/m), this process may result in a relatively low $\gamma$ for the liquid lining the pharyngeal wall. The quantity and quality of saliva produced, its $\gamma$, and the processes that result in the maintenance of a salivary film on the pharyngeal mucosal surfaces (eg, swallowing) may all contribute to the preservation of a low $\gamma$ of UAL. Thus, the presence of a higher $\gamma$ of UAL in patients with OSAHS may be reflective of abnormalities in this pathway. There are no reported measurements of the $\gamma$ of saliva in OSAHS, so its role in these patients is unknown. Other than its association with swallowing, little is known regarding processes that transfer saliva to or remove saliva from, the pharyngeal wall. Further examination of these processes will clearly be required.

### Table 2—Occurrence of Nasal and Oronasal Breathing During Sleep

<table>
<thead>
<tr>
<th></th>
<th>Healthy</th>
<th>OSAHS</th>
<th>$P$ values</th>
</tr>
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<tbody>
<tr>
<td>Nasal, %TSE</td>
<td>48 [35, 61]</td>
<td>49 [32, 66]</td>
<td>.9</td>
</tr>
<tr>
<td>Oronasal, %TSE</td>
<td>52 [38, 64]</td>
<td>51 [35, 67]</td>
<td>.9</td>
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OSAHS refers to obstructive sleep apnea-hypopnea syndrome; %TSE, percentage of total sleep epochs. Data are mean [95% confidence interval]. $P$ values healthy subjects vs subjects with OSAHS.

### Figure 3—Change in surface tension ($\gamma$) of upper airway mucosal lining liquid (UAL) and overnight nasal breathing. Plot showing change in $\gamma$ of UAL ($\Delta \gamma$) from PM to AM samples (negative values represent a decrease in $\gamma$ of UAL with sleep) and nasal-only breathing expressed as a percentage of total sleep epochs (TSE) for both healthy subjects (open symbols) and patients with obstructive sleep apnea-hypopnea syndrome (OSAHS) (closed symbols). Horizontal dashed line represents no change in $\gamma$ of UAL, and vertical dashed line represents 50% nasal breathing TSE. Note that as the amount of nasal breathing increases, there is a decrease in the $\gamma$ of UAL ($r^2 = 0.15$, $P < .05$). Full line = linear regression line, dashed line = no overnight change in $\gamma$ of UAL.
In the present study, salivary flow rates were not different between patients with OSAHS and healthy subjects and were not different before and after sleep. Our measurements of salivary flow were performed before and after sleep and may not be representative of what occurred during sleep. There are no reports in the literature describing salivary flow during sleep in patients with OSAHS. Indeed, there are few data reporting measured salivary flow rates in sleeping healthy subjects. Salivary flow may be reduced during sleep, but experience (eg, drooling of saliva occurs in some individuals during sleep) suggests that it continues at least at some basal rate. Swallowing, presumably of accumulated saliva, certainly does occur during sleep and may play a role in spreading saliva to the pharyngeal wall.

While breathing route during sleep in healthy subjects has been previously investigated, there are no published data concerning breathing route during sleep in patients with OSAHS. We used thermocouple devices to monitor breathing in a manner similar to that which we described previously. While thermocouple devices are thought to have poor sensitivity to changes in airflow, we only used them to detect the presence or absence of nasal and oral airflow. We did not use the thermocouple signals to quantify the amount of airflow present. In the present study, both healthy subjects and patients with OSAHS spent about equal time during sleep breathing via the nose only and via a combination of nose and mouth. Oral-only breathing was rarely encountered, which is similar to our findings in healthy subjects and snorers.

A fascinating finding in the present study was the association between breathing route during sleep and overnight change in \( \gamma \) of UAL. The presence of at least some mouth breathing allows colder, drier inhaled air to reach the pharynx than is the case for nasal breathing. Consequently, evaporative water loss from the upper airway mucosal surfaces is likely to be greater. Loss of water from the mucosal surfaces of the mouth and pharynx may provide a potential mechanism for alteration of \( \gamma \) of UAL, even if salivary flow rates are normal and swallowing occurs. However, we found that the main effect of breathing route on \( \gamma \) of UAL appeared to be mediated more via the amount of nasal-only breathing than via the amount of oral breathing. Thus, subjects with nasal breathing for more than 50% TSE had a significant overnight fall in \( \gamma \) of UAL, while subjects with oral breathing for more than 50% TSE did not change their \( \gamma \) of UAL overnight (See Figure 3). Thus, it would appear that the occurrence of frequent oronasal breathing during sleep acts to prevent the nasal breathing associated with overnight fall in \( \gamma \) of UAL rather than being associated with an increased \( \gamma \) of UAL itself. The mechanisms linking nasal breathing to a lower \( \gamma \) of UAL are not known. It may be that prolonged nasal breathing promotes maintenance of a low \( \gamma \) of UAL layer of saliva on the pharyngeal mucosal wall, whereas oral breathing disrupts this process. Alternative hypotheses include effects of breathing route on the composition of the saliva itself; Whatever the mechanism linking breathing route and \( \gamma \) of UAL, in the present study, patients with OSAHS who spent the majority of the night breathing exclusively via the nose had both less severe sleep-disordered breathing and an overnight fall in \( \gamma \) of UAL. Whether this finding reflects a breathing route or a \( \gamma \) of UAL influence on upper airway mechanics is not clear. However, we have previously demonstrated that acutely reducing the \( \gamma \) of UAL with exogenous surfactant decreases upper airway collapsibility and reduces the severity of sleep-disordered breathing in patients with OSAHS.

In conclusion, we have demonstrated that patients with OSAHS have normal salivary flow rates but a slightly increased \( \gamma \) of UAL. In addition, for both healthy individuals and patients with OSAHS, frequent occurrence of nasal-only breathing during sleep was associated with an overnight fall in the \( \gamma \) of UAL. Preservation of a low \( \gamma \) of the liquid lining the collapsible pharyngeal segments of the upper airway during sleep may contribute to the maintenance of upper airway patency during a period when other protective mechanisms, such as upper airway dilator muscle activity, are reduced.

**ACKNOWLEDGEMENTS**

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