The influence of episodic hypoxia on upper airway collapsibility in subjects with obstructive sleep apnea


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Rowley JA, Deebajah I, Parikh S, Najar A, Saha R, Badr MS. The influence of episodic hypoxia on upper airway collapsibility in subjects with obstructive sleep apnea. J Appl Physiol 103: 911–916, 2007. First published June 14, 2007; doi:10.1152/japplphysiol.01117.2006.—We have previously shown that in subjects with obstructive sleep apnea, repetitive hypoxia is associated with long-term facilitation as manifested by decreased upper airway resistance (Rua). Our objective was to study the influence of long-term facilitation on upper airway collapsibility as measured by the critical closing pressure (Pcrit) model and to determine whether changes in Rua correlated with changes in collapsibility. We studied 13 subjects (10 men, 3 women) with a mean apnea-hypopnea index of 43.9 ± 24.0 events/h. In the first protocol with 11 subjects, we measured collapsibility using a Pcrit protocol before and after episodic hypoxia. Brief (3 min) isocapnic hypoxia (inspired O2 fraction = 8%) followed by 5 min of room air was induced 10 times. A sham study without hypoxia was performed on eight subjects. Ventilatory parameters, Rua, and Pcrit before and after episodic hypoxia were measured. At 20 min of recovery, there was no change in minute ventilation but there was a significant decrease in Rua compared with the control period (control, 8.6 ± 4.8 cmH2O·L−1·s−1 vs. recovery, 5.9 ± 3.8 cmH2O·L−1·s−1; P < 0.05). However, there was no change in Pcrit between the control (2.3 ± 1.9 cmH2O) and recovery (2.7 ± 3.2 cmH2O) periods. No changes in Rua or Pcrit were observed in the sham protocol. We conclude that long-term facilitation of upper airway dilators is not associated with changes in upper airway collapsibility in subjects with obstructive sleep apnea. These results corroborate previous evidence that changes in upper airway resistance and caliber can be dissociated from changes in upper airway collapsibility.

METHODS

The experimental protocols described below were approved by the Human Investigation Committee of the Wayne State University School of Medicine and the John D. Dingell Veterans Affairs Medical Center. Informed written consent was obtained from all subjects.

Measurements

The following parameters were measured in all subjects. Standard sleep parameters were recorded using the international 10–20 system of electrode placement. Airflow (V̇) was measured by a pneumotachometer (model 3700A, Hans Rudolph) attached to a nasal mask. Tidal volume (VT) was obtained from the integrated V̇ signal. Supraglottic airway pressures were measured using a pressure-tipped catheter (model TC-500XG, Millar) threaded through the mask and positioned in the oropharynx just below the base of the tongue. Correct placement was verified by visually inspecting the catheter’s position in the oropharynx. Mask pressure was measured in all subjects and used as the surrogate of nasal pressure (Pn). End-tidal PCO2 (PETCO2) was measured by a sensor placed in the nasal mask.

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was measured using air sampled continuously from the nasal mask by an infrared analyzer (model CD-3A, AEI Technologies, Pittsburgh, PA). Arterial oxygen saturation (Sao2) was measured by a pulse oximeter (Biox 3700, Ohmeda). All signals were displayed on a polygraph recorder (Grass/Telefactor, West Warwick, RI) and recorded using data acquisition software (PowerLab, Colorado Springs, CO) for further analysis (see below).

**Experimental Setup**

The subject was connected to the circuit with an airtight silicone rubber mask strapped and glued to the face to prevent leaks (see Fig. 1 in Ref. 1). The mask was connected to a Plateau Exhalation Valve (Respirronics, Pittsburgh, PA) via a heated pneumotachometer. The valve, which provides a continuous leak path in the breathing circuit and serves as an exhaust vent, was connected on the inspiratory line. The pneumotachometer was attached to a Y-shaped circuit with two balloon valves. The first balloon valve, when open, allowed the subject to breathe a gas mixture from three cylinders containing the following gases: 100% N2, 8% O2, or 100% O2. To maintain isocapnia, supplemental CO2 was added to the inspiratory line from an external source, and PETCO2 was maintained at control levels.

The other half of the Y-shaped circuit was connected to a continuous positive pressure generator; the valve for this part of the circuit was open throughout the study protocol. A negative pressure generator (modified REM-Star, Respirronics, Murrysville, PA) was also available to put in series with the Y-shaped circuit if it was determined that the subject had a negative Pcrit. If needed, the negative pressure generator could generate a subatmospheric pressure in the upper airway as indicated by a decrease in Pn. The level of subatmospheric pressure generated could be preset on the modified REM-Star unit.

**Protocol**

All patients were monitored in the supine position and used a U-shaped pillow to maintain head and neck position. Patients were allowed to fall asleep breathing at a continuous positive airway pressure (CPAP) level that eliminated apneas and hypopneas but at which flow limitation was present for >50% of the breaths (Pn-FL). A Pcrit protocol as previously described was performed after the onset of stage 2 sleep (25, 26). During periods of stable stage 2 sleep, Pn was abruptly reduced by decreasing Pn by 1 cmH2O at the end of expiration; the decreased Pn was maintained for two breaths and then raised back to the holding pressure. Pn was subsequently reduced in 1.0-cmH2O decrements at 1- to 2-min intervals (with return to atmospheric after 2 breaths) until airflow ceased. The Pn drop associated with zero flow was repeated to ensure that zero flow was achieved at this pressure. If there was not complete airway closure, at a Pn = 0 cmH2O, the negative pressure generator was attached to the circuit and negative pressure was generated in 1.0-cmH2O decrements. Complete airway collapse was achieved in all subjects.

After the Pcrit was obtained, the LTF protocol was performed with the patient breathing at the Pn-FL. The subjects breathed room air for 5 min (control period), followed by 3 min of hypoxic gas (8% O2); this sequence was repeated 10 times. Hypoxia was rapidly induced by having the subject breathe one or two breaths of 100% N2 followed by continuous 8% O2 for 3 min to maintain hypoxia (O2 saturation <88%). Care was taken to ensure that isocapnia was maintained throughout the hypoxia period by measuring PETCO2, and 5% CO2 was supplemented as needed. Hypoxia was abruptly terminated with one breath of 100% O2. Twenty minutes after the last hypoxic exposure, a repeat Pcrit determination was performed. Note that hypoxia was always initiated when the patient was in stage 2 sleep but was continued for 3 min even if there was a change in sleep stage or return to wakefulness.

Subjects were invited to return for a sham study. During the sham study, a Pcrit determination was performed after the onset of stable non-rapid eye movement (NREM) sleep. The patient was then allowed to sleep for 80 min with no hypoxia interventions with a repeat Pcrit determination at the end of the 80 min.

**Data Analysis**

**Sleep state.** Wakefulness/sleep stage was scored according to standard criteria (20). The subjects were in stable stage 2 or stage 3 sleep during the hypoxic exposures, Pcrit protocols, and data collection.

**Selection of breaths.** The control period consisted of 3 min immediately preceding the first hypoxic exposure. The last 20 breaths were used for measurement of resistance and ventilation. For each hypoxia period, the last 10 breaths were chosen for analysis. Starting at 20 min after the last hypoxia period, 20 breaths were chosen for analysis to represent recovery. For the sham studies, 20 breaths immediately following the Pcrit protocol were chosen for control; 20 breaths were chosen for the recovery period starting at 80 min after the control breaths. All breaths were chosen during periods of stable stage 2 sleep; breaths associated with arousals were not analyzed. The criteria for selecting the recovery segment included a similar sleep state to the control period and similar distribution of various sleep waveforms. An independent observer matched the sleep state between the control and the recovery period without knowledge of the breathing in either segment.

**Ventilation and timing.** Inspired tidal volume (Vt), breathing frequency (f), minute ventilation (Vt), inspiration time (Ti), total time for a breath (Tt), PETCO2, and Sao2 were calculated for each breath chosen for the control, hypoxia, and recovery periods. Rua was measured for the same breaths. Rn was measured on the linear portion of the inspiratory portion of the pressure-flow curve, independent of flow rate, using a polynomial equation-based method that allows for precise, automated measurements (13).

**Determination of Pcrit.** For each reduction in Pn, the second breath was analyzed. Pcrit was defined as the first measured Pn at which flow was zero. If there was more than one Pn at which flow was zero, the largest (most positive) Pn was used as the Pcrit value for the subject. Upstream resistance or Rn was calculated as previously described (Fig. 1) (26). For each trial, Vmax and Pn were plotted and a regression line drawn. Rn was calculated as the inverse of the slope of the regression line.

**Statistical Analysis**

Repeated-measures one-way ANOVA was performed to compare respiratory parameters and PETCO2 between control, hypoxia and recovery. Paired t-test was used to compare Rua, Pcrit, and Rn before and after episodic hypoxia in the experimental protocols and to compare all parameters for the sham studies.

**RESULTS**

We studied 13 subjects; subject demographics are provided in Table 1. Eleven subjects completed the episodic hypoxia study; six of these subjects also completed a sham study. Two additional subjects were unable to sleep during the episodic hypoxia study but completed a sham study, for a total of eight sham studies.

For the episodic hypoxia trials, the mean Sao2 associated with the breaths chosen for analysis was 85.3 ± 3.3%. Mean PETCO2 was 40.8 ± 6.5 Torr during the control period, 38.4 ± 4.1 Torr during the hypoxia periods and 40.7 ± 4.8 Torr during the recovery period (P = not significant). Results of the analysis for the respiratory parameters are shown in Table 2. There was an increase in Vt/Ti during hypoxia but not during the recovery period. There was an increase in Vt and Vr during hypoxia with no difference between control and recovery. There was no difference between control, hypoxia, and recov-
DISCUSSION

The aim of this study was to determine whether LTF secondary to episodic hypoxia is associated with changes in the collapsibility of the upper airway as measured by the Pcrit. In

Table 1. Subject demographics

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Studies</th>
<th>Sex</th>
<th>Age, yr</th>
<th>BMI, kg/m²</th>
<th>NC, cm</th>
<th>AHI, events/h</th>
<th>CPAP, cmH₂O</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>LTF/sham</td>
<td>F</td>
<td>46</td>
<td>25.8</td>
<td>34.5</td>
<td>12.5</td>
<td>5</td>
</tr>
<tr>
<td>2</td>
<td>LTF/sham</td>
<td>M</td>
<td>28</td>
<td>25.8</td>
<td>40.5</td>
<td>57.9</td>
<td>13</td>
</tr>
<tr>
<td>3</td>
<td>LTF/sham</td>
<td>F</td>
<td>47</td>
<td>32.9</td>
<td>43.0</td>
<td>41.7</td>
<td>10</td>
</tr>
<tr>
<td>4</td>
<td>LTF/sham</td>
<td>M</td>
<td>48</td>
<td>42.8</td>
<td>48</td>
<td>38.8</td>
<td>10</td>
</tr>
<tr>
<td>5</td>
<td>LTF/sham</td>
<td>M</td>
<td>44</td>
<td>30.7</td>
<td>44</td>
<td>54.8</td>
<td>12</td>
</tr>
<tr>
<td>6</td>
<td>LTF/sham</td>
<td>M</td>
<td>45</td>
<td>31.0</td>
<td>42.5</td>
<td>47.6</td>
<td>7</td>
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<tr>
<td>7</td>
<td>LTF/sham</td>
<td>M</td>
<td>49</td>
<td>29.3</td>
<td>44.5</td>
<td>78.9</td>
<td>9</td>
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<tr>
<td>8</td>
<td>LTF/sham</td>
<td>F</td>
<td>48</td>
<td>27.7</td>
<td>36</td>
<td>95.8</td>
<td>10</td>
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<tr>
<td>9</td>
<td>LTF/sham</td>
<td>M</td>
<td>51</td>
<td>29.8</td>
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<td>44.2</td>
<td>12</td>
</tr>
<tr>
<td>10</td>
<td>LTF/sham</td>
<td>M</td>
<td>48</td>
<td>35.0</td>
<td>49</td>
<td>32.5</td>
<td>10</td>
</tr>
<tr>
<td>11</td>
<td>LTF/sham</td>
<td>M</td>
<td>44</td>
<td>35.5</td>
<td>42</td>
<td>32.5</td>
<td>15</td>
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<tr>
<td>12</td>
<td>Sham</td>
<td>M</td>
<td>43</td>
<td>22.5</td>
<td>38</td>
<td>16.9</td>
<td>10</td>
</tr>
<tr>
<td>13</td>
<td>Sham</td>
<td>M</td>
<td>38</td>
<td>29.3</td>
<td>44.5</td>
<td>16.6</td>
<td>9</td>
</tr>
</tbody>
</table>

Mean±SE 44.5±6.0 30.6±5.2 42.1±4.3 43.9±24.0 10.5±2.9

BMI, body mass index; NC, neck circumference; AHI, apnea-hypopnea index; CPAP, continuous positive airway pressure; LTF, long-term facilitation; F, female; M, male.

Table 2. Respiratory parameters: episodic hypoxia study

<table>
<thead>
<tr>
<th>Value</th>
<th>Control</th>
<th>Hypoxia</th>
<th>Recovery</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT, l</td>
<td>0.57±0.08</td>
<td>0.46±0.06</td>
<td>0.53±0.08</td>
<td>0.458</td>
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<tr>
<td>Vr, liter</td>
<td>0.576±0.125</td>
<td>0.627±0.130</td>
<td>0.566±0.129</td>
<td>0.03*</td>
</tr>
<tr>
<td>f3, breaths/min</td>
<td>15.7±3.1</td>
<td>15.6±3.7</td>
<td>15.4±3.6</td>
<td>0.776</td>
</tr>
<tr>
<td>VT, l/min</td>
<td>9.0±2.8</td>
<td>10.9±3.2</td>
<td>9.6±3.4</td>
<td>0.013*</td>
</tr>
<tr>
<td>VT/TI, l/s</td>
<td>0.31±0.09</td>
<td>0.35±0.10</td>
<td>0.28±0.08</td>
<td>0.041†</td>
</tr>
</tbody>
</table>

Values are means ± SD. Ti, inspiration time; Tr, total time for each breath; Vr, tidal volume; f3, breathing frequency. *Hypoxia vs. control, recovery, P < 0.05; control vs. recovery, not significant. †Recovery vs. hypoxia, P < 0.05; control vs. hypoxia, recovery, not significant.

\( P = 0.571 \). Rn was also not significant different before (25.5 ± 17.2 cmH₂O·1⁻¹·s) and after (23.7 ± 13.6 cmH₂O·1⁻¹·s; \( P = 0.734 \)) repetitive hypoxia.

For the sham studies, mean PETCO₂ was 40.6 ± 8.1 Torr during the control period and 39.3 ± 8.4 Torr during the sham recovery period (\( P = \) not significant). Ventilatory parameters are presented in Table 3; there were no differences between the control and recovery periods for any parameter, including \( V_l \). Rua did not change over the course of the sham study (control, 6.9 ± 3.3 cmH₂O·1⁻¹·s vs. 80 min, 7.1 ± 3.2 cmH₂O·1⁻¹·s; \( P = 0.85 \); Fig. 3). The results of the Pcrit protocol are presented in Fig. 3. Pcrit at baseline (2.1 ± 0.6 cmH₂O) was not different from the Pcrit after 80 min of stable NREM sleep (2.5 ± 0.7 cmH₂O; \( P = 0.279 \)). There was no change in Rn during the course of the study (baseline, 18.2 ± 6.8 cmH₂O·1⁻¹·s vs. 80 min, 21.3 ± 12.8 cmH₂O·1⁻¹·s; \( P = 0.413 \)).

We performed a repeated-measures two-factor ANOVA of the six subjects who had both episodic hypoxia and sham studies. The factors were night (episodic hypoxia vs. sham) and group (control vs. recovery). There were no differences in Pcrit or Rn between the two nights of study or between control and recovery.

Fig. 1. Maximal inspiratory airflow (\( V_{\text{max}} \)) vs. nasal pressure (\( P_n \)) for 1 representative subject before (top) and after (bottom) episodic hypoxia. Note that the critical closing pressure (Pcrit) did not change after episodic hypoxia. Nasal resistance (Rn) was calculated as the inverse of the slope of the regression line; Rn also did not change after episodic hypoxia.
this study, we demonstrated that episodic hypoxia in patients with sleep apnea evokes LTF, manifesting as decreased Rua. However, decreased Rua was not associated with a change in Pcrit, suggesting that upper airway collapsibility was not altered. In addition, in there was no change in collapsibility after 80 min of sleep without hypoxia intervention in eight subjects who underwent sham studies.

We showed that repetitive hypoxia results in decreased Rua indicative of upper airway dilatation. Decreased Rua without increased Vt or Vt/Tt suggests LTF of the ventilatory motor output to upper airway dilators but not to thoracic pump muscles. These findings confirm our previous work demonstrating reduced Rua, without change in inspiratory thoracic EMG, after episodic hypoxia (1). In addition, we have preliminary data that indicates the genioglossus muscle activity is increased in normal subjects after episodic hypoxia (S. Chowdhuri, personal communication). Our findings corroborate animal studies demonstrating that repetitive hypoxia elicits LTF of ventilatory motor output to upper airway dilators. Mateika and Fregosi (14) showed that repetitive hypoxia in vagotomized animal cat is followed by increased activity of the genioglossus and the alae nasae but not the diaphragm (14).

Upper airway mechanics during sleep have been studied and measured by a variety of measures, including Rua (19, 30) and collapsibility (8, 25, 26). Rua, when measured on the linear portion of the pressure-flow loop, is commonly used as a surrogate of upper airway caliber (2, 4, 27) during sleep. However, the computation of Rua is predicated on a constant relationship between driving pressure and inspiratory flow, which is true only on the linear portion of the pressure-flow loop; in fact, flow limitation develops after 10–15% of Tt (5, 11). Thus Rua as measured in our study likely reflects the behavior of the upper airway only at the beginning of inspiration. In other words, the Rua provides only a partial picture of the dynamic behavior of the pharyngeal airway during sleep. The dynamic behavior of the airway, including its propensity to collapse, can be better characterized by measuring collapsibility using the critical closing pressure methodology as in this study. An advantage of the Pcrit methodology is that closely approximates the inspiratory flow limitation condition; thus Pcrit likely reflects the behavior and properties of the upper airway as inspiratory flow limitation develops later in the inspiratory cycle.

The dissociation between Rua and Pcrit observed in this study could be occurring because these parameters are measuring properties of the upper airway at different points of the inspiratory cycle with the Rua measuring the behavior at the beginning of inspiration and Pcrit at the peak of inspiration. Alternatively, the dissociation could be secondary to the two parameters measuring the behavior of the airway at different locations of the upper airway. Pcrit most likely reflects collapsible segments at either the naso- or oropharynx. However, Rua may reflect the cross-sectional area of the airway at multiple different locations, although not likely the nasal cavity given the lack of change in Rn.

Another advantage of the Pcrit technique is the ability to partition the upper airway into several segments, which allows measurement of the upstream resistance or Rn. In this study, we have found no change in Rn after episodic hypoxia. The lack of change is likely due to the fact that the Rn is primarily determined by the bony and cartilaginous structures of the nasal cavity. However, the lack of change in Rn also indicates that episodic hypoxia is not altering the vascular and properties of the nasal mucosa.

The results of our study indicate that the various measures of upper airway mechanics cannot be used interchangeably. For

![Fig. 2. Group mean upper airway resistance (Rua), Pcrit, and Rn values before and after episodic hypoxia. Values are means ± SD. *There was a significant decrease in the Rua after episodic hypoxia (P = 0.012). There was no difference in the group mean Pcrit or Rn values after episodic hypoxia.](image)

![Fig. 3. Group mean Rua, Pcrit, and Rn values for the sham control and sham recovery periods. Values are means ± SD. There was no difference in the group mean Rua, Pcrit, and Rn values over the course of the sham study.](image)

**Table 3. Respiratory parameters: sham studies**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Control</th>
<th>Recovery</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tt, s</td>
<td>2.04±0.44</td>
<td>2.14±0.47</td>
<td>0.310</td>
</tr>
<tr>
<td>Tr, s</td>
<td>3.81±0.71</td>
<td>3.89±0.79</td>
<td>0.542</td>
</tr>
<tr>
<td>Tt/Tt</td>
<td>0.54±0.12</td>
<td>0.56±0.12</td>
<td>0.351</td>
</tr>
<tr>
<td>Vt, liter</td>
<td>0.55±0.78</td>
<td>0.58±0.10</td>
<td>0.267</td>
</tr>
<tr>
<td>Is, breaths/min</td>
<td>15.1±1.9</td>
<td>14.9±2.9</td>
<td>0.514</td>
</tr>
<tr>
<td>Vi, l/min</td>
<td>8.3±1.5</td>
<td>8.2±2.4</td>
<td>0.912</td>
</tr>
<tr>
<td>Vt/Tt, l/s</td>
<td>0.28±0.07</td>
<td>0.28±0.07</td>
<td>0.976</td>
</tr>
</tbody>
</table>
instance, Rua has frequently been measured as an index of upper airway collapsibility with an increase in resistance between two conditions believed to be indicative of increased upper airway collapsibility (19, 30). However, we found that decreased Rua was not associated with a change in Pcrit, suggesting that upper airway collapsibility was not altered. This finding corroborates our laboratory’s previous studies in sleep apnea patients and in normal snoring subjects demonstrating no change in Vmax despite decreased Rua and increased Vt (1, 28).

Methodological Considerations

Several limitations have to be considered for proper interpretation of our findings. First, changes in sleep state might have caused a misinterpretation of the data. However, we performed the Pcrit protocol only during periods of stage 2 sleep and analyzed the breath by breath data only when sleep was in stable stage 2 or greater, with no evidence of arousal. Thus the data reported here were from periods where there was no change in sleep state. Second, we were unable to maintain precise isocapnia during hypoxia. However, the reduction in PetCO2 was minimal and not significant. Third, the study was conducted while the patients were receiving nasal CPAP therapy. Subjects were studied on CPAP because we have previously shown that manifestations of LTF are most likely observed under conditions of inspiratory flow limitation (3). Fourth, we did not change the CPAP setting during the recovery period. In theory, the decreased Rua during recovery would indicate a larger upper airway caliber; therefore, upper airway collapsibility measurements starting at different airway calibers may not be comparable. To our knowledge, this hypothesis has not been specifically tested. Fifth, we chose to use Rua as an indicator of upper airway caliber. However, there is evidence that changes in upper airway cross-sectional area during sleep does not necessarily correlate with changes in Rua (22, 24).

Our protocol differed from previous Pcrit protocols. First, we did not intend to partition the Pcrit into its active and passive components as previously described (18). However, we likely studied a neurally intact upper airway because passive Pcrit pressure is measured when the upper airway is studied under hypotonic conditions, generally achieved when CPAP is set at a level sufficient to eliminate inspiratory flow limitation. We studied our patients at a CPAP level at which inspiratory flow limitation was present, because manifestations of LTF are most likely observed under conditions of inspiratory flow limitation (3). Second, we measured Pcrit as the pressure at which flow was zero instead of the regression technique that has also been described (16). This could result in “overshoot” of the Pcrit measurement and add noise to the data. However, we decreased Pn in 1-cmH2O increments, which should reduce the possibility of overshoot.

The studies were performed over the course of 2 h which could influence the measurement of Pcrit. However, within subject differences in the Pcrit were generally less than <3 cmH2O for both the experimental and sham studies, and there was no significant difference in either Pcrit or Rn between nights for the six subjects who had both studies. Furthermore, the changes in Pcrit observed on both the experimental and sham studies are consistent with previous studies investigating the stability of the Pcrit measurement over the course of the night (16, 17).

Finally, the studies were performed only in subjects with obstructive sleep apnea. We studied subjects with sleep apnea primarily because previous data indicated that LTF in this group may stabilize respiration in this group (1). In addition, our experience performing Pcrit measurements in normal subjects was associated with frequent arousals during the negative pressure trials (25); given the long length of the present protocol, achieving and maintaining sleep at 80 min was critical to data collection.

In summary, we have shown that LTF manifesting as decreased Rua is not associated with changes in upper airway collapsibility in subjects with obstructive sleep apnea. These results corroborate previous evidence that changes in Rua and caliber can be dissociated from changes in upper airway collapsibility. The results indicate that researchers should be careful when using terminology used to describe upper airway mechanics as the various measurements are likely measuring different upper airway mechanical phenomena. Use of the term “collapsibility” should be reserved for those studies in which collapsibility is specifically measured using an accepted technique such as the Pcrit.

GRANTS

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REFERENCES


