Heavy snoring with upper airway resistance syndrome may induce intrinsic positive end-expiratory pressure

Lofaso, Frédéric, Anne Marie Lorino, Redouane Fodil, Marie Pia D’Ortho, Daniel Isabey, Hubert Lorino, Françoise Goldenberg, and Alain Harf. Heavy snoring with upper airway resistance syndrome may induce intrinsic positive end-expiratory pressure. J. Appl. Physiol. 85(3): 860–866, 1998.—We studied eight heavy snorers with upper airway resistance syndrome to investigate potential effects of sleep on expiratory airflow and lung resistance, intrinsic positive end-expiratory pressure, hyperinflation, and elastic inspiratory work of breathing (WOB). Wakefulness and non-rapid-eye-movement sleep with high- and with low-resistance inspiratory effort (H-RIE and L-RIE, respectively) were compared. No differences in breathing pattern were seen across the three conditions. In contrast, we found increases in expiratory airflow and lung resistance during H-RIE compared with L-RIE and wakefulness (56 ± 24, 16 ± 4, and 11 ± 4 cmH2O·l−1·s−1, respectively), with attendant increases in intrinsic positive end-expiratory pressure (5.4 ± 1.8, 1.4 ± 0.5, and 1.3 ± 1.3 cmH2O, respectively) and elastic WOB (6.1 ± 2.2, 3.7 ± 1.2, and 3.4 ± 0.7 J/min, respectively). The increase in WOB during H-RIE is partly caused by the effects of dynamic pulmonary hyperinflation produced by the increased expiratory resistance. Contrary to the Starling model, a multiple-element compliance model that takes into account the heterogeneity of the pharynx may explain flow limitation during expiration.

METHODS

Entry criteria. All subjects included were patients with suspected upper airway resistance syndrome and were taken from a group of heavy snorers who complained of daytime tiredness and/or daytime sleepiness (7). Two polysomnographic evaluations were required for the diagnosis of upper airway resistance syndrome (7).

A home polysomnography study was done first to identify a subgroup of snorers who had abnormal sleep fragmentation (arousal index >10/h), according to the reference value for our laboratory (12), but did not have OSAS or periodic leg movements to explain this finding. This polysomnography included electroencephalography (C4-A1, C3-A2), electrooculography, chin electromyography, electromyography of the tibialis anterior muscle of both legs, thermistor oronasal airflow assessment, rib cage movements (Multi-Parameter Analysis recorder 2/Medilog 9200; Oxford Medical Instrument, Abingdon, UK), and arterial pulse oximetry (Nellcor BS; Nellcor, Hayward, CA). During this home polysomnography, according to the clinical criteria commonly used for thermistor signals, an abnormal breathing event during sleep was defined as either a complete cessation of airflow lasting at least 10 s (apnea) or a reduction in the oronasal airflow lasting at least 10 s and accompanied with hypopnea (a drop of at least 3% in arterial O2 saturation vs. baseline; see Ref. 16). OSAS was ruled out on the basis of an apnea-hypopnea index value of <5/h of sleep.
A second polysomnography study was then done in the laboratory to demonstrate that the sleep fragmentation was caused by an increase in respiratory effort in response to an increase in upper airway resistance (7). It included respiratory effort evaluation by measurement of esophageal pressure (Pes) and quantitative assessment of ventilation by using a pneumotachometer. For this polysomnography study, sustained high-resistance inspiratory effort (H-RIE) was defined as association of flow limitation (11, 27) and Pes swings lasting at least 1 min and greater than twice the mean value measured during quiet breathing in the awake state (25). The mean value in the awake state was calculated at the beginning of the polysomnography study (during the 3 min before the light was switched off) and at the end of the polysomnography study (during the 3 min after the light was switched on). Only polygraphic recordings with sustained labored inspiratory effort during >10% of the total sleep time were further analyzed in this study.

Clinical study. The clinical study consisted of a further analysis of the second polysomnography study, which included electroencephalography (C4-A1, C3-A2), electrocardiography, chin electromyography, thoracic and abdominal movements assessed by inductive plethysmography (Respiracite; Ambulatory Monitoring, Ardsley, NY) calibrated according to the isovolume maneuvers (9), and arterial pulse oximetry (Nellcor BS; Nellcor). During the study night, oronasal airflow was quantified, via a tightly fitting facial mask, by using a Fleisch no. 2 pneumotachograph (Lausanne, Switzerland) connected to a differential pressure transducer (± 5 cmH2O; Validyne MP45, Northridge, CA). The mask and the pneumotachograph were maintained by a crosspiece, which also maintained the patients in the supine position.

In addition, respiratory effort was monitored by measuring Pes (Gaelect, Dunvegan, Isle of Skye, UK). All signals were recorded by using a 14-channel paper recorder (electroencephalograph; Nihon Kohen, Tokyo, Japan), digitized at 128 Hz, and sampled by using an analog-to-digital system (MP100; Biopac System, Goleta, CA) for subsequent analysis.

Analysis. All records selected for study were visually inspected by two observers. Sleep and sleep stages were analyzed by using the international criteria of Rechtschaffen and Kales (20).

For each patient, six 1-min data sets, characterized by stable H-RIE and recorded during non-rapid-eye-movement (non-REM) sleep, were randomly selected and compared with 1) six data sets characterized by normal breathing with low-resistance inspiratory effort (L-RIE), characterized by no flow limitation and no Pes swings greater than twice the mean value measured during quiet breathing in the awake state and recorded during the same sleep stage, and 2) six data sets with normal breathing recorded during wakefulness (Wake). For each of these three ventilatory groups, inspiratory and expiratory airflow and lung resistances, PEEPi, and inspiratory WOB were calculated. In addition, to assess hyperinflation during labored inspiratory effort, changes in EELV (assessed by inductive plethysmography) and in PEEPi were analyzed in each patient when H-RIE was abruptly stopped by an arousal.

Airway and lung resistance (R) was evaluated according to the formula based on the technique of Mead and Whittenberger (14): R airway and lung = [(Pes0 − Pes)/(V/C)]/V, where Pes0 is the Pes value at the start of inspiratory flow, V is the instantaneous volume of the breath integrated from airflow, C is the dynamic lung compliance calculated for the same breath as the ratio of tidal volume (Vt) to the Pes difference between the beginning and the end of inspiration, and V is the instantaneous airflow. Mean values over the inspiration and the expiration were used as estimates of inspiratory and expiratory airway and lung resistance, respectively. In addition, because there is a nonlinear relationship between airflow and pressure, inspiratory and expiratory airflow and lung resistances were calculated at 200 m/s.

PEEPi was evaluated as the decrease in Pes immediately before the point of zero flow (4).

Inspiratory WOB was computed from the Pes-V loops, as previously described (4). WOB was calculated from the Campbell diagram (3) by computing the area located between the recorded Pes-V curve during inspiration and the static Pes-V curve of the chest wall. The Pes values at the zero-flow points were taken as the beginning and the end of inspiration. The theoretical value of chest wall compliance (C), which theoretically represents ~4% of the predicted value of the vital capacity per cmH2O, was used to obtain the static Pes-V curve of the chest wall (4). This curve meets the value for elastic recoil pressure of the chest wall at end expiration, which was assessed by measuring PEEPi on the Pes tracing. The beginning of inspiration was thus separated from the elastic recoil pressure by an amount equal to the PEEPi on the Campbell diagram, as usually performed (3). In addition, WOB was partitioned into its elastic and resistive components by taking the line joining the two zero-flow points as the boundary between these two components. This method of analysis of the Pes-V curve was valid because, in our study, airway pressure was equal to Patm.

Statistics. Data are given as means ± SD. Comparisons were made by using analysis of variance for repeated measurements. When appropriate (F test with a P value < 0.05), a two-by-two comparison was performed by using a Fisher least

Table 1. Characteristics of 8 snorers with upper airway resistance syndrome

<table>
<thead>
<tr>
<th>Subject No.</th>
<th>Age, yr</th>
<th>BMI, kg/m²</th>
<th>%Pred</th>
<th>Per Hour of Sleep</th>
<th>%Pred</th>
<th>%Pred</th>
<th>%Pred</th>
<th>%Pred</th>
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<td></td>
<td></td>
<td></td>
<td></td>
<td>AI</td>
<td>AHI</td>
<td>AI</td>
<td>AI</td>
<td>AHI</td>
</tr>
<tr>
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<td>48</td>
<td>26</td>
<td>6</td>
<td>0</td>
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<td>92</td>
<td>97</td>
</tr>
<tr>
<td>2</td>
<td>46</td>
<td>28</td>
<td>5</td>
<td>0</td>
<td>4</td>
<td>20</td>
<td>97</td>
<td>99</td>
</tr>
<tr>
<td>3</td>
<td>44</td>
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<td>88</td>
</tr>
<tr>
<td>4</td>
<td>38</td>
<td>27</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>15</td>
<td>97</td>
<td>88</td>
</tr>
<tr>
<td>5</td>
<td>65</td>
<td>22</td>
<td>3</td>
<td>0</td>
<td>3</td>
<td>11</td>
<td>98</td>
<td>90</td>
</tr>
<tr>
<td>6</td>
<td>49</td>
<td>28</td>
<td>4</td>
<td>0</td>
<td>4</td>
<td>12</td>
<td>97</td>
<td>98</td>
</tr>
<tr>
<td>7</td>
<td>43</td>
<td>26</td>
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<td>0</td>
<td>2</td>
<td>15</td>
<td>96</td>
<td>82</td>
</tr>
<tr>
<td>8</td>
<td>51</td>
<td>26</td>
<td>0</td>
<td>0</td>
<td>0</td>
<td>15</td>
<td>96</td>
<td>78</td>
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<tr>
<td>Mean ± SD</td>
<td>48 ± 8</td>
<td>26 ± 2</td>
<td>0 ± 0</td>
<td>3 ± 2</td>
<td>17 ± 6</td>
<td>97 ± 1</td>
<td>88 ± 8</td>
<td>40 ± 2</td>
</tr>
</tbody>
</table>

Values are those measured except for means ± SD (n = 8 subjects). BMI, body mass index; AI, apnea index; AHI, apnea-hypopnea index; AIr, arousal index; S0, baseline arterial oxygen saturation; PaO2, arterial PO2; PaCO2, arterial PCO2; TLC, total lung capacity; VC, vital capacity; FEV1, forced expiratory volume in 1 s; %pred, %predicted value [predicted values were those of the European Community (19)].

For each of these three ventilatory groups, inspiratory and expiratory airflow and lung resistances, PEEPi, and inspiratory WOB were calculated. In addition, to assess hyperinflation during labored inspiratory effort, changes in EELV (assessed by inductive plethysmography) and in PEEPi were analyzed in each patient when H-RIE was abruptly stopped by an arousal.

Airway and lung resistance (R) was evaluated according to the formula based on the technique of Mead and Whittenberger (14): R airway and lung = [(Pes0 − Pes)/(V/C)]/V, where Pes0 is the Pes value at the start of inspiratory flow, V is the instantaneous volume of the breath integrated from airflow, C is the dynamic lung compliance calculated for the same breath as the ratio of tidal volume (Vt) to the Pes difference between the beginning and the end of inspiration, and V is the instantaneous airflow. Mean values over the inspiration and the expiration were used as estimates of inspiratory and expiratory airway and lung resistance, respectively. In addition, because there is a nonlinear relationship between airflow and pressure, inspiratory and expiratory airflow and lung resistances were calculated at 200 m/s.

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Statistics. Data are given as means ± SD. Comparisons were made by using analysis of variance for repeated measurements. When appropriate (F test with a P value < 0.05), a two-by-two comparison was performed by using a Fisher least
statistical difference test. The level of significance was set at \( P = 0.05 \).

RESULTS

During 1996, we studied 422 snorers who underwent home polysomnography. Among these patients, 35 met our criteria for further polysomnographic investigation, including respiratory effort evaluation by Pes measurement and quantitative assessment of ventilation by a pneumotachometer. Of these 35, 12 agreed to undergo the second polysomnography study. Eight of the 12 persons studied displayed sustained labored inspiratory effort during >10% of the total sleep time; these persons were the eight subjects in this study.

Physical, respiratory, and home polysomnography characteristics of the eight patients studied are listed in Table 1. No patients had evidence of airway or lung disease according to European Community reference values (19).

Ventilatory patterns, dynamic lung compliance, inspiratory and expiratory airway and lung resistances, PEEPi, and WOB observed in each condition are listed in Table 2. Figure 1 shows typical signals observed in a representative patient. Figure 2 illustrates changes in resistive pressure and flow in the same patient during each condition, thus giving an index of the changes in inspiratory and expiratory airway and lung resistances that occurred across the three conditions. There were no differences in breathing pattern across the three conditions, but, as expected, total inspiratory WOB increased during H-RIE. Interestingly, during H-RIE, expiratory airway and lung resistance was increased to the same extent as inspiratory airway and lung resistance. Because PEEPi was also increased during H-RIE, the increase in WOB was due not only to an increase in

### Table 2. Breathing pattern and respiratory muscle work indexes in 3 conditions: wakefulness period, and non-REM sleep periods without and with laborious breathing

<table>
<thead>
<tr>
<th></th>
<th>Wake</th>
<th>Lab−</th>
<th>Lab+</th>
<th>Statistical Analysis</th>
</tr>
</thead>
<tbody>
<tr>
<td>VT, ml</td>
<td>515 ± 92</td>
<td>511 ± 92</td>
<td>500 ± 100</td>
<td>NS</td>
</tr>
<tr>
<td>Respiratory rate, breaths/min</td>
<td>14 ± 3</td>
<td>15 ± 3</td>
<td>15 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>Ti/Ttot, %</td>
<td>41 ± 6</td>
<td>40 ± 5</td>
<td>40 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>Esophageal pressure swing, cmH₂O</td>
<td>9 ± 2</td>
<td>11 ± 4</td>
<td>28 ± 7</td>
<td>††</td>
</tr>
<tr>
<td>Dynamic lung compliance, ml/cmH₂O</td>
<td>134 ± 71</td>
<td>128 ± 56</td>
<td>130 ± 57</td>
<td>NS</td>
</tr>
<tr>
<td>Mean inspiratory airway and lung resistance, cmH₂O·l⁻¹·s⁻¹</td>
<td>11 ± 4</td>
<td>16 ± 5</td>
<td>63 ± 27</td>
<td>*††</td>
</tr>
<tr>
<td>Mean expiratory airway and lung resistance, cmH₂O·l⁻¹·s⁻¹</td>
<td>11 ± 4</td>
<td>16 ± 4</td>
<td>56 ± 24</td>
<td>*††</td>
</tr>
<tr>
<td>Inspiratory airway and lung resistance at 200 ml/s, cmH₂O·l⁻¹·s⁻¹</td>
<td>10 ± 4</td>
<td>15 ± 5</td>
<td>64 ± 22</td>
<td>*††</td>
</tr>
<tr>
<td>Expiratory airway and lung resistance at 200 ml/s, cmH₂O·l⁻¹·s⁻¹</td>
<td>11 ± 3</td>
<td>16 ± 3</td>
<td>45 ± 14</td>
<td>*††</td>
</tr>
<tr>
<td>PEEPi, cmH₂O</td>
<td>1.3 ± 1.3</td>
<td>1.4 ± 0.5</td>
<td>5.4 ± 1.8</td>
<td>††</td>
</tr>
<tr>
<td>Total WOB, J/min</td>
<td>6.7 ± 1.4</td>
<td>6.8 ± 2.1</td>
<td>15.4 ± 2.1</td>
<td>††</td>
</tr>
<tr>
<td>Resistive WOB, J/min</td>
<td>3.4 ± 1.3</td>
<td>3.1 ± 1.8</td>
<td>9.3 ± 3.5</td>
<td>††</td>
</tr>
<tr>
<td>Elastic WOB, J/min</td>
<td>3.4 ± 0.7</td>
<td>3.7 ± 1.2</td>
<td>6.1 ± 2.2</td>
<td>††</td>
</tr>
</tbody>
</table>

Values are means ± SD; n = 8 subjects. Wake, wakefulness period; Lab−, Lab+, non-rapid-eye-movement sleep period without and with laborious breathing; PEEPi, intrinsic positive end-expiratory pressure; Ti/Ttot, inspiratory time-to-total cycle time; VT, tidal volume; WOB, work of breathing; NS, not significant. * \( P < 0.05 \) Wake vs. Lab−; † \( P < 0.05 \) Wake vs. Lab+; †† \( P < 0.05 \) Lab− vs. Lab+.
resistive work, but also to an increase in the eWOB (see Table 2 and Fig. 1).

Analysis of the periods in which abnormal Pes swings abruptly returned to normal as a result of arousal showed significant decreases both in EELV (of 74 ± 29 ml) and in PEEPi (1.9 ± 1.3 after the arousal vs. 6.3 ± 1.7 cmH\(_2\)O before the arousal). An example of these changes is given in Fig. 3, top. Figure 3, bottom, depicts changes in resistive pressure and flow during an arousal, thus providing an indication of changes in inspiratory and expiratory airway and lung resistances produced by arousal.

**DISCUSSION**

Previous studies have demonstrated that narrowing of the upper airway in OSAS in snorers and normal subjects can occur not only during inspiration but also during expiration (13, 15, 23, 24, 29). For example, it has been clearly demonstrated in OSAS that expiratory resistance is already increased during the breath preceding the initial occluded inspiratory effort of occlusive apnea (23). In addition, this increase in expiratory resistance may induce intermittent interruptions and/or a prolongation of expiration (24). More recently, it has also been demonstrated that expiratory flow limitation can occur during sleep in snorers, and that, consequently, upper airway obstruction can be present even during expiration (29). Our data corroborate these observations and demonstrate that the increase in inspiratory resistance is of similar magnitude as the increase in expiratory resistance. Because intraluminal pressure is positive during expiration, models that use collapsible tubes do not explain this increase in expiratory resistance. Conversely, the effect of gravity on upper airway structures (2), together with relaxation of pharyngeal dilator muscles, such as the tensor palatini and the genioglossus (21), may promote local upper airway narrowing during expiration. We recently developed a model that consists of a series of successive individualized mass-spring components, each of which has its own compliance on the basis of local differences in anatomic and physiological properties across pharyngeal regions. When we applied gravity and Bernoulli’s law to this model, we observed a flow limitation during inspiration associated with segmental narrowing of the upper airway (6). We also tested this model during expiration and observed that, contrary to the Starling resistor model, it may induce an expiratory-flow-limitation phenomenon (see APPENDIX). Expiratory-flow limitation has been recently observed in snorers in whom the pressure drop in the upper airway was directly detected by the measurement of supraglottic pressure (29). This method is more appropriate than ours for detecting expiratory-flow limitation and/or increase in inspiratory resistance. However, this method cannot detect the dynamic pulmonary hyperinflation resulting from the increased expiratory resistance and its repercussion on inspiratory WOB. The best way to test these phenomena during expiration and to validate the multiple-element compliance model is probably to record Pes and supraglottic pressure simultaneously at different sites along the upper airway. Unfortunately, this study has not yet been performed.

Because gravity facilitates upper airway narrowing (2), body position is an important determinant of upper airway caliber. It is likely that the upper airway is narrower in the supine position than in the lateral position. Consequently, the supine position adopted in our study is probably the position most likely to allow detection of differences across conditions.

Combined lung and upper airway resistances were used in the present study. Previous studies demonstrated that most of the increase in airway resistance occurring during sleep and snoring was caused by an increase in upper airway resistance (27–29). Therefore, we are confident that our findings reflect changes in resistance to air flow in the upper airway.

Conditions that increase expiratory resistance and/or shorten expiratory time may promote dynamic hyperinflation, i.e., cause the EELV to rise above the relaxation volume (30). This leads to a positive elastic recoil of the
respiratory system at the end of expiration. The inspiratory muscles begin to contract before the start of inspiratory flow in an effort to counteract the elastic recoil pressure of the total respiratory system (22). This elastic recoil pressure shared by the inspiratory muscles corresponds to the PEEPi, measured as the amplitude of the negative deflection of Pes between the onset of inspiratory effort and the onset of inspiratory flow (22). This additional inspiratory WOB markedly increases the elWOB (22). In our study, total WOB increased more than twofold in the H-RIE situation compared with the L-RIE situation (Table 2), reaching a level (10 J/min) at which diaphragmatic electromyography suggested excessive loading (4). About one-third of this increase was caused by the increase in elWOB resulting from the dynamic pulmonary hyperinflation produced by the increased expiratory resistance, whereas the remaining two-thirds were caused by the increase in inspiratory resistive WOB induced by the increase in inspiratory airway and lung resistance (see Table 2).

In addition to this increase in expiratory resistance, other mechanical factors may have contributed to the PEEPi increase. The PEEPi increase may have been caused in part by expiratory muscle activity in response to upper airway narrowing (18). Although no clinical expiratory activity was detectable in our patients, this possibility cannot be ruled out, because we did not perform objective assessments of expiratory muscle activity such as electromyography or gastric pressure measurements. Because EELV is generally lower than the functional residual capacity (FRC) when PEEPi is induced solely by an increase in expiratory resistance (22), we analyzed the concomitant changes in PEEPi and in EELV.

As expected, we observed an abrupt decrease in EELV when inspiratory effort and PEEPi returned to normal after an arousal. However, this decrease in EELV was smaller than the one previously observed in COPD (22). This difference may be caused in part by
the lower lung compliance of our patients compared with COPD patients. Another explanation may involve the effects of arousal on FRC. In previous studies involving careful measurement of FRC in normal subjects, FRC clearly decreased during sleep (1, 8). This phenomenon was ascribed to loss of postural tone of inspiratory muscles (17). By contrast, we observed an EELV decrease of \(-100\) ml when H-RIE abruptly returned to normal after an arousal. This suggests that the marked reduction in EELV that can be expected to occur with a reduction in PEEPi was partly counterbalanced, in our patients, by an increase in FRC caused by the sleep disruption induced by the awakening. Assuming normal respiratory system compliance, a 4-cmH\(_2\)O decrease in PEEPi would result in an EELV decrease of \(-400\) ml. The EELV decrease in our study was only \(-100\) ml; this result suggests that the FRC increase caused by sleep disruption was \(-300\) ml. Interestingly, a similar magnitude of change in FRC was observed from Wake to sleep in normal subjects (1, 8). However, abrupt arousal may not represent resting Wake baseline condition. Nevertheless, because FRC increases are commonly observed during awakening in normal subjects, the EELV decrease in our patients during awakening strongly suggests that pulmonary hyperinflation occurred during sleep and H-RIE.

Morrell et al. (15) used a method similar to that used in our study to calculate airway and lung resistance in normal nonsnoring subjects during Wake and sleep. They observed, from Wake to sleep, a nonsignificant increase in expiratory resistance of \(-150\%\). A similar increase was observed in our patients when we compared Wake and periods of sleep with L-RIE. Morrell et al. did not evaluate PEEPi, but our results obtained during L-RIE suggest that this parameter was probably \(<2\) cmH\(_2\)O. However, we found a sixfold increase in mean expiratory resistance during sleep with H-RIE compared with Wake (see Table 2). This also explains the differences in lung volume changes from sleep to Wake between normal subjects and snorers.

When no expiratory activity is present, alveolar expiratory pressure can be mathematically described by a single exponential time function, which equals PEEPi at the end of expiration and can be expressed as

\[
\text{PEEPi} = \frac{VT}{C} e^{-\frac{Te}{RC}}
\]

where Te is the expiratory time, and R, C, and RC are the resistance, compliance, and time constant of the respiratory system, respectively.

Tuxen and Lane (30) validated this mathematical model in mechanically ventilated patients with severe airflow obstruction and also found clear evidence that the risk of dynamic hyperventilation was greater when the volume delivered by the ventilator (VT) was increased and the Te reduced. The fact that VT and Te did not vary across the different conditions in our study suggests that the increase in PEEPi resulted entirely from an increase in the time constant of the respiratory system, which was perhaps mainly caused by an increase in expiratory resistance (Table 2).

In conclusion, our data corroborate previous observations that upper airway narrowing during sleep can persist during expiration (13, 15, 23, 24, 29). In addition, our study demonstrates that the increase in WOB during H-RIE compared with L-RIE is partly caused by the effects of dynamic pulmonary hyperinflation produced by the increased expiratory resistance. A model different from the Starling resistor model of upper airway narrowing is needed to explain the increase in expiratory resistance during sleep and snoring. A two-element compliance model, which takes into account the anatomic and physiological heterogeneities of the pharynx, is therefore proposed to explain the occurrence of flow limitation.

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**Fig. A1.** A: 2-element compliance model proposed by Fodil et al. (6). \(\tilde{A}_1\) and \(\tilde{A}_2\): cross-sectional areas of oropharyngeal and nasopharyngeal segments, respectively. Pexp, expiratory supraglottic pressure; Patm, atmospheric pressure; Pint\(_1\) and Pint\(_2\): internal lateral pressures of oropharyngeal and nasopharyngeal segments, respectively. B: expiratory flow (V)-Pexp relationship predicted by Fodil model. V is normalized for product of initial cross-sectional area of oropharyngeal segment by distensibility-dependent velocity, associated to zero transmural pressure conditions at this segment. Note flow-limitation phenomenon. C: area-Pexp relationships predicted for \(\tilde{A}_1\) and \(\tilde{A}_2\) by model in A. \(\tilde{A}_1\) and \(\tilde{A}_2\) are normalized for their respective initial values. Note narrowing process at oropharyngeal level.
APPENDIX

The single-element compliance model, often referred to as the Starling resistor, predicts an expiratory flow-pressure relationship typical of a nonlinear resistive behavior resulting from the simultaneous increases in cross-sectional area and expiratory flow with increasing expiratory pressure (Pexp). Therefore, this model fails in describing the expiratory-flow alteration observed during the patient’s expiratory effort.

The two-element compliance model proposed by Fodil et al. (6) is represented in Fig. A1A. This model consists of 1) an upstream element which represents the oropharyngeal segment and is submitted to an upstream pressure equal to the supraglottic Pexp, and 2) a downstream element which represents the nasopharyngeal segment and is submitted to a downstream pressure equal to the Patm. The model represented in Fig. A1A is characterized, at the beginning of expiration, by an initial cross-sectional area of the oropharyngeal segment (A1) smaller than the initial cross-sectional area of the nasopharyngeal segment (A2). This difference between A1 and A2 is in accordance with the assumption of a narrowing of the oropharyngeal segment induced by the flow structure coupling that occurred during the preceding inspiration. The Fodil model of Fig. A1A predicts 1) the occurrence of expiratory-flow limitation (Fig. A1B), which can explain the plateau observed for the flow signal during the first part of expiration, and 2) the progressive narrowing of the oropharyngeal segment and the unchanged caliber of the nasopharyngeal segment during expiration (Fig. A1C). Flow limitation, which is specific for expiration, results from the coupling that occurs between expiratory flow and each of the pharyngeal segments. The oropharyngeal segment, which is the site of flow acceleration as Pexp increases, progressively narrows as a results of the decrease in its internal lateral pressure, whereas the nasopharyngeal segment, which is submitted to an internal pressure close to Patm, keeps a roughly constant cross-sectional area.

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