Changes in lung volume and diaphragm muscle activity at sleep onset in obese obstructive sleep apnea patients vs. healthy-weight controls

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Submitted 15 December 2009; accepted in final form 11 August 2010

Obstructive sleep apnea (OSA) is a common sleep disorder characterized by repetitive periods of upper airway (UA) collapse during sleep. Male sex and obesity are two key predictors of OSA (4, 38), but the mechanisms via which these factors contribute to OSA remain unclear. In addition to an anatomically smaller airway (15) and an abrupt fall in UA dilator muscle activity following sleep onset (6, 16, 25), reduced lung volume is also likely to contribute to poor UA function in obese OSA patients (11, 27). A decrease in lung volume potentially reduces the degree of axial tension (caudal traction) exerted on the UA and, therefore, the propensity for UA collapse. Our laboratory recently demonstrated increased UA collapsibility with experimental abdominal compression during sleep in OSA patients (26), strongly supporting mechanical effects of abdominal obesity on UA function. These effects are likely to be most evident in the supine posture and during sleep, particularly at the wake-sleep transition when other compensatory mechanisms become diminished, and may at least partly explain sex and obesity influences in OSA.

Stanchina et al. (27) demonstrated important effects of changes in lung volume on UA function, with a decrease in lung volume during sleep resulting in marked increases in UA collapsibility and pharyngeal resistance. Data from animal studies suggest that changing lung volume may alter UA stiffness by modulating tracheal traction on the UA (21, 31). End-expiratory lung volume (EELV) decreases when healthy-weight individuals move from upright to the supine posture (33, 36), largely due to cranial displacement of the diaphragm (32). Despite increased thoracoabdominal mass loading and intra-abdominal pressure (23), there is little change in EELV following a similar postural transition in obese individuals (33, 36). One possible explanation is that obese individuals may actively defend against a fall in lung volume via increased minimum expiratory (tonic) diaphragm muscle activity (eEMGdia). Muller (17) identified augmented tonic diaphragmatic activity with external abdominal compression (17), consistent with active defense of lung volume, at least during wakefulness. Consequently, obese OSA patients may have augmented tonic diaphragm activity to help preserve lung volume and UA tension, such that transient reductions in diaphragm activity at sleep onset could lead to exaggerated lung volume changes and propensity for UA collapse.

Data concerning the magnitude of lung volume and trans-diaphragmatic pressure changes at sleep onset in obese OSA patients vs. healthy-weight controls are currently lacking. In healthy-weight individuals, EELV has been shown to fall by ~15% during sleep (2, 14) and is profoundly reduced following induction of general anesthesia in the obese (18). Consequently, a fall in EELV at sleep onset, particularly in obese OSA individuals, could importantly contribute to an acute increase in the propensity for UA collapse at sleep onset. While it is difficult to separate effects of obesity per se from other potential pathogenic factors operating in OSA patients, lung volume and tracheal traction effects are likely dominated by thoracoabdominal mass loading effects of obesity itself. Consequently, we elected to first establish if diaphragm muscle activity and lung volume changes occurring in the immediate sleep-onset period are any different in obese OSA patients vs. healthy-weight controls before further separate studies de-
signed to separate obesity from other potential contributory effects in OSA.

The aims of this study were, therefore, to compare eEMG\(_{\text{dia}}\) during wakefulness between obese OSA patients and age-matched, healthy-weight controls and to compare changes in inspiratory diaphragm muscle activity (iEMG\(_{\text{dia}}\)), eEMG\(_{\text{dia}}\), end-expiratory gastric (P\text{ga}) and transdiaphragmatic pressure (P\text{di}), and EELV following sleep onset between these two groups. Given sleep onset is frequently associated with respiratory events in OSA patients, we also aimed to investigate if the magnitude of lung volume, iEMG\(_{\text{dia}}\), eEMG\(_{\text{dia}}\), and end-expiratory P\text{ga}, and P\text{di} changes differ according to the degree of UA collapse following sleep onset. We hypothesized that eEMG\(_{\text{dia}}\) would be higher during wakefulness in obese OSA patients and that there would be a greater fall in lung volume, iEMG\(_{\text{dia}}\), eEMG\(_{\text{dia}}\), and end-expiratory P\text{ga}, and P\text{di} following sleep onset in obese OSA patients compared with healthy-weight controls, with a greater reduction at sleep onset shortly followed by respiratory events.

METHODS

Participant Selection

Obese [body mass index (BMI) 30–40 kg/m\(^2\)] male OSA patients with moderate-to-severe OSA, aged between 18 and 65 yr, and healthy-weight (BMI < 25 kg/m\(^2\)), age-matched male subjects without sleep-disordered breathing participated. Nasal pressure recordings of airflow were undertaken, and OSA diagnosis and severity was based on the consensus criteria (Chicago) [5] using a total sleep out sleep-disordered breathing participated. EEMG\(_{\text{dia}}\) was recorded via a series of nine equally spaced (1-cm interelectrode distance) stainless steel rings situated between the P\text{ga} and P\text{es} balloons of the multilumen catheter. Electrodes were connected in sequentially adjacent pairs [electrodes 1 (most proximal) and 2, 2 and 3, etc.] to an amplifier (model 15LT, Grass Instruments, Quincy, MA) and band-pass filtered (0.3–1 kHz) via eight bipolar EEMG channels with an interpair distance of 1 cm. Once connected, catheter position was further adjusted to achieve maximal iEMG\(_{\text{dia}}\) activity near the center of the electrode array, while maintaining positive P\text{ga} and negative P\text{es} swings during inspiration. The catheter was then secured at the mask using a tight-sealing stainless steel luer (SSA1380, S4J Manufacturing Services, Cape Coral, FL).

Changes in lung volume. Abdominal and thoracic excursions were measured continuously using two pairs of magnetometer coils (Polhemus Liberty, Colchester, VT) placed in the anterior-posterior axis of the chest and abdomen (26). See the online supplement for further details on lung volume measurements.

Data acquisition. All conventional sleep-related signals were recorded on a Compumedics data-acquisition system (E-series, Compumedics, Melbourne, Australia). X, Y, and Z coordinates for each of the four magnetometer sensors were recorded on a computer at a sample rate of 200 Hz (Magnetometer Liberty). The remaining signals were recorded on a 32-channel data-acquisition system at 200 Hz, except for EEMG\(_{\text{dia}}\) channels, which were sampled at 1 kHz (DI-720 DATAQ Instruments). To facilitate accurate time matching (within \(\sim 100\) ms) between the three recording systems, a computer-actuated event mark signal was simultaneously placed on all three acquisition systems approximately every hour.

Protocol

Following instrumentation and while supine, participants underwent a 5-min baseline period in which they were instructed to remain relaxed, with their eyes open, breathing solely through their nose. For recording maximal iEMG\(_{\text{dia}}\) activity, participants then performed a minimum of three maximal Mueller maneuvers by maximally inspir-
A total of nine obese OSA patients and eight control subjects were recruited. Data from one obese OSA patient were excluded due to significant mask leaks. Complete presleep measurements of breath-by-breath ventilatory data were obtained in eight patients and eight controls. One control subject could not tolerate the multilumen catheter, and $R_{UA}$ measurements could not be assessed in one obese OSA patient and one control subject due to catheter blockage. Thus end-expiratory Pga and Pdi measurements were obtained in eight obese OSA patients and seven control subjects, while $R_{UA}$ measurements were obtained in seven obese OSA patients and seven control subjects. Two obese OSA patients and one control subject could not reliably perform Mueller maneuvers. Consequently, between-group comparisons in iEMGdia and eEMGdia (% maximum) data are represented by six obese OSA and six control individuals.

Anthropometric variables in each group are presented in Table 1. Both groups were matched for age and had healthy lung function, while OSA patients were significantly heavier than controls and had severe OSA by design. Maximal EMDgia was not significantly different between groups (OSA vs. controls, 29.7 ± 4.0 vs. 23.6 ± 4.3 μV, $P = 0.27$).

Average presleep onset (sleep onset categories combined) data are shown in Table 2. Vi, $R_{UA}$, end-expiratory Pga, Pdi, iEMGdia (% maximum), and iEMG (μV) were all significantly higher in the obese OSA group before sleep onset [Vi, $P = 0.007$; $R_{UA}$, $P < 0.001$; Pga, $P = 0.001$; Pdi, $P = 0.008$; iEMGdia (% maximum), $P = 0.019$; and iEMG (μV), $P = 0.006$]. There was a trend for increased eEMGdia (μV) in the OSA group ($P = 0.063$).

### Changes at Sleep Onset

There were, on average, 20.9 ± 2.9 (range 13–33) and 22.9 ± 2.6 (range 14–35) sleep onsets in obese OSA patients and control participants respectively, that met the inclusion criteria for analysis. Sleep onsets with respiratory events were significantly more frequent in obese OSA patients than controls with 78/167 (46.7%), 48/167 (28.7%), and 41/167 (24.6%) of sleep
Table 2. Average presleep onset (breaths −5 to −2) data in obese OSA patients and controls

<table>
<thead>
<tr>
<th></th>
<th>OSA</th>
<th>Controls</th>
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<tbody>
<tr>
<td>Vt, l/min</td>
<td>8.1 ± 0.5§</td>
<td>6.4 ± 0.2</td>
</tr>
<tr>
<td>Vt, liter</td>
<td>0.53 ± 0.04</td>
<td>0.46 ± 0.03</td>
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<tr>
<td>F_s, breaths/min</td>
<td>16.0 ± 1.0</td>
<td>14.4 ± 0.3</td>
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<tr>
<td>PRCO₂, Torr</td>
<td>41.9 ± 0.6</td>
<td>44.1 ± 1.3</td>
</tr>
<tr>
<td>R_LA, cmH₂O·1·s⁻¹</td>
<td>15.3 ± 1.0§</td>
<td>9.0 ± 0.6</td>
</tr>
<tr>
<td>Pga, cmH₂O</td>
<td>12.3 ± 0.8§</td>
<td>6.6 ± 1.5</td>
</tr>
<tr>
<td>Pdi, cmH₂O</td>
<td>5.3 ± 0.5§</td>
<td>0.2 ± 1.8</td>
</tr>
<tr>
<td>iEMG_dia, %maximum</td>
<td>20.6 ± 1.6§</td>
<td>14.6 ± 1.7</td>
</tr>
<tr>
<td>eEMG_dia, %maximum</td>
<td>10.1 ± 0.6</td>
<td>9.2 ± 0.8</td>
</tr>
<tr>
<td>iEMG_dia, µV</td>
<td>5.9 ± 0.5§</td>
<td>3.3 ± 0.5</td>
</tr>
<tr>
<td>eEMG_dia, µV</td>
<td>2.9 ± 0.3</td>
<td>2.0 ± 0.3</td>
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Values are means ± SE; N = 8 obese OSA patients and 8 controls, *N = 7 obese OSA patients and 7 controls, †N = 8 obese OSA patients and 7 controls, ‡N = 8 obese OSA patients and 6 controls. Minute ventilation (Vt), tidal volume (Vt), breathing frequency (F_s), end-tidal PCO₂ (PRCO₂), upper airway resistance (R_LA), end-expiratory gastric (Pga) and transdiaphragmatic pressures (Pdi), and average inspiratory (iEMG_dia) and tonic expiratory diaphragm activity (eEMG_dia) are shown. $P < 0.05$, obese OSA patients vs. controls.

onsets classified as stable, hypopnea, and apnea wake-sleep transitions, respectively, in the obese OSA patients vs. 149/179 (83.2%), 30/179 (16.8%), and 0/179 in the control participants ($χ^2, P < 0.001$).

Figure 1 shows changes in $V_t$ across the wake-sleep transition in the obese OSA patients and control subjects for all sleep onsets combined (Fig. 1A) and with stable sleep, hypopnea, and apnea transitions shown separately for the obese OSA patients (Fig. 1C) and stable sleep and hypopnea transitions in the control subjects (Fig. 1E). There was a significantly greater and more rapid reduction in $V_t$ in obese OSA patients (Fig. 1A, group, $P = 0.009$; group × breath, $P < 0.001$). By analytic design, the fall in $V_t$ following sleep onset was dependent on the degree of subsequent airway obstruction (Fig. 1, C and E, category, $P < 0.001$).

EELV significantly decreased by 61.4 ± 8.0 and 34.0 ± 4.2 ml by the third sleep onset breath from wakefulness in obese OSA patients and control subjects, respectively (Fig. 1B, wake vs. postsleep onset breath 3, $P < 0.001$). While there were no between-group differences in the overall reduction in EELV following sleep onset ($P = 0.528$), there was a greater decline in EELV over time following sleep onset in the obese OSA patients (Fig. 1B, group × breath, $P = 0.007$), with this group experiencing greater decrements in EELV over time in apnea and hypopnea transitions (Fig. 1D, category × breath, $P < 0.001$). While there was no category × breath interaction effect in the control group, the overall decrease in EELV was significantly greater in hypopnea compared with stable breathing transitions (Fig. 1F, category, $P = 0.01$). By the third breath following sleep onset in the obese OSA group, EELV had statistically significantly fallen in each transition category (stable, 25.9 ± 10.8 ml, $P = 0.017$; hypopnea, 84.7 ± 16.3 ml, $P < 0.001$, and apnea, 118.9 ± 16.4 ml, $P < 0.001$, Fig. 1D).

Similar decrements in lung volume by the third postsleep onset breath were evident for each transition category in the controls (stable, 34.2 ± 4.3 ml, $P < 0.001$; hypopnea, 41.0 ± 13.1 ml, $P = 0.002$, Fig. 1F).

Changes in iEMG_dia and eEMG_dia activity following sleep onset are shown in Fig. 2. iEMG_dia and eEMG_dia showed significantly greater (Fig. 2A, $P = 0.02$, and Fig. 2B, $P = 0.017$, respectively) and more rapid (Fig. 2A, group × breath, $P < 0.001$, and Fig. 2B, group × breath, $P < 0.001$, respectively) reductions following sleep onset in the obese OSA.

Fig. 1. Breath-by-breath changes in minute ventilation ($V_t$; left) and end-expiratory lung volume (EELV; right) across the wake-sleep transition (vertical line). Changes in $V_t$ are expressed as a percentage of the stable presleep onset baseline period, while EELV changes are expressed as milliliter difference ($Δ$) from baseline. A and B: obese obstructive sleep apnea (OSA) patients and controls (categories combined). C and D: obese OSA patients (stable sleep, hypopnea, and apnea categories separated). E and F: control subjects (stable sleep and hypopnea categories separated). Average apnea (OSA patients) and hypopnea (OSA and control participants) onset times relative to sleep onset are shown as isolated points above baseline. Values are means ± SE; $N = 8$ OSA and 8 controls.
group, with activity significantly decreasing by a maximum of 14.5 ± 1.6% of baseline \((P < 0.001)\) and 8.3 ± 1.2% of baseline \((P < 0.001)\) from wakefulness, respectively. In the obese OSA group, decreases in iEMG\(_{dia}\) and eEMG\(_{dia}\) were significantly greater and more rapid when accompanied by respiratory events (Fig. 2, C and D, category, \(P < 0.001\)). iEMG\(_{dia}\) and eEMG\(_{dia}\) decreased by a maximum of 28.3 ± 2.7% of baseline \((P < 0.001)\) and by 13.2 ± 2.6% of baseline \((P < 0.001)\), respectively, for apnea transitions. In contrast, the control group showed no significant sleep onset category or sleep onset category \(\times\) breath-dependent effects.

Relationships between changes in EELV and eEMG\(_{dia}\) and changes in EELV and PIF from baseline to the third postsleep onset breath within each patient and category of sleep onset response are shown in Fig. 3, A and B, respectively. There was no significant relationship between eEMG\(_{dia}\) and EELV when data from OSA patients and controls were combined (Fig. 3A; \(r^2 = 0.06, P = 0.19\)). Similarly, there was no significant relationship in either the obese OSA group (Fig. 3A; \(r^2 = 0.06, P = 0.3\)) or the control group (Fig. 3A; \(r^2 = 0.04, P = 0.5\)). Greater falls in EELV were associated with larger decrements in PIF for all group data combined (Fig. 3B; \(r^2 = 0.27, P = 0.003\)). While not statistically significant, there was a trend for a relationship in the obese OSA group (Fig. 3B; \(r^2 = 0.18, P = 0.07\)). A strong relationship was evident in the control group (Fig. 3B; \(r^2 = 0.78, P < 0.001\)).

Changes in end-expiratory Pga and Pdi following sleep onset are shown in Fig. 4. There were no group or interaction effects in changes in end-expiratory Pga following sleep onset (Fig. 4A). Similarly, there were no sleep onset category or sleep onset category \(\times\) breath-dependent effects in changes in end-expiratory Pga in either the obese OSA group or the control group (Fig. 4A). However, there was a greater increase in end-expiratory Pdi over time following the wake-sleep transition in the control group (Fig. 4B, group \(\times\) breath, \(P = \))
Changes Preceding Respiratory Events

Figure 5 shows changes in $V_i$ preceding the onset of hypopneas and apneas in obese OSA patients (Fig. 5A) and preceding the onset of hypopneas in the control group (Fig. 5B). $V_i$ significantly decreased preceding respiratory events in both groups (breath effect, all $P < 0.001$), with $V_i$ decreasing by 51.0 ± 4.2 and 35.7 ± 8.1% below wakefulness levels. EELV significantly decreased by 29.4 ± 12.3 ml ($P = 0.018$) and 89.6 ± 14.2 ml ($P < 0.001$) below wake levels by the first hypopneic breath and apneic inspiratory effort, respectively, in the obese OSA group (Fig. 5C). However, there were no sleep onset category or sleep onset category × breath dependent effects.

DISCUSSION

The key findings of this study were that acute decrements in EELV accompany sleep onset in both obese OSA patients and healthy-weight controls, and that greater decrements in EELV and end-expiratory Pdi occur during wake-sleep transitions accompanied by respiratory events. However, despite obesity and substantially raised intra-abdominal pressure, consistent with previous reports (23, 29), there was no evidence for a greater overall sleep onset-related reduction in lung volume, end-expiratory Pga, or end-expiratory Pdi in obese OSA patients compared with healthy-weight controls. While some caution is warranted, given the small sample size, we estimate that lung volume changes of this magnitude were detected accompanying apnea events following sleep onset, it appears unlikely that a similar magnitude or larger systematic effects of obesity on average lung volume responses at sleep onset in OSA patients, compared with healthy-weight controls for the same event severity, would have been missed due to type II error. Given more frequent hypopnea and apnea events and greater lung...
volume decrements over time in OSA patients compared with controls, more substantial lung volume changes were, nevertheless, a more common outcome following sleep onset in OSA patients compared with healthy-weight controls.

EELV significantly decreased in the order of \(-100\) ml at the onset of complete UA obstruction in the obese OSA group. The contribution of this decrement to the development of obstruction in the immediate postsleep onset period is not clear and cannot be separated from other potential effects of reduced respiratory drive on UA function. Nevertheless, this finding highlights that systematic lung volume changes with a similar time course to decrements in ventilatory drive do accompany respiratory events. This study also demonstrated a significant relationship between changes in EELV and PIF, supporting previous reports demonstrating significant modulating effects of EELV on UA function (10, 11, 30). While these reports typically investigated the influence of EELV changes in the order of 500 ml above normal EELV, the effects of lung volume changes below EELV are unknown. Longitudinal traction effects on UA compliance could be alinear at the lower range of tension, rendering UA function more sensitive to lung volume changes below EELV, particularly in obese patients with already reduced lung volume (33, 36) and UA size during wakefulness (24). Consequently, modest lung volume changes at sleep onset in obese OSA patients, at least equivalent to those seen in normal-weight controls, may have greater effects on UA function. Further studies are needed to elucidate the impact of experimentally induced lung volume decrements in the order of 100–150 ml below EELV on UA function in obese vs. nonobese controls.

A similar pattern and time course of changes in \(\dot{V}_t\), iEMG\(_{dia}\) and eEMG\(_{dia}\) activity at sleep onset support that ventilatory and lung volume decrements reflect an acute and widespread reduction in phasic and tonic respiratory muscle tone at the wake-sleep transition, with periods of greater reduction associated with more severe respiratory events. The greater decline in end-expiratory Pdi over time following sleep onsets accompanied by respiratory events in obese OSA patients is consistent with diaphragm relaxation and ascent. As reported by a previous study (28), iEMG\(_{dia}\) was found to be significantly higher in the obese group during wakefulness, likely reflecting increased inspiratory work required to offset mass loading.
effects on the respiratory system. In contrast, we found little evidence of increased eEMGdia to support active defense of lung volume during wakefulness, although postsleep onset falls in activity did appear to be greater and more rapid in obese OSA patients. While these findings do not support a diaphragm neurocompensatory reflex, caution is warranted, given the small sample size. We estimate that wakefulness eEMGdia in the order of −3% of maximal activity could have been detected between groups with 80% power and a two-tailed significance level of 0.05. Therefore, it appears unlikely that larger systematic effects of obesity on tonic diaphragm activity in OSA patients would have been missed due to Type II error.

If the reduction in eEMGdia at sleep onset is a key factor contributing to a cranial displacement of the diaphragm and, consequently, fall in EELV, temporal relationships and associations between changes in eEMGdia, changes in EELV, and the degree of UA obstruction would be expected. While changes in eEMGdia, EELV, and ventilation occurred over a similar time course, and with greater changes in more severe obstructive events, eEMGdia decreases appeared more abrupt and to plateau while EELV continued to decrease. In addition, there was no correlation between the degree of change in eEMGdia and EELV by the third postsleep onset breath. While cause-and-effect relationships cannot be discerned from these data, these findings are perhaps consistent with an abrupt reduction in muscle tone contributing to decrements in lung volume, but with further modulation by other factors, such as reduced drive to other muscles, abdominal muscle contraction (12), development of atelectasis, and potentially intrinsic positive end-expiratory pressure similar to that seen under general anesthesia (18) and greater expiratory vs. inspiratory tidal volume (37).

The larger decline in ventilation and iEMGdia following sleep onset in OSA patients supports previous findings (6). In addition to decrements in diaphragm muscle activity and EELV observed in this study, reduced ventilation in OSA patients is known to be importantly influenced by reduced UA dilator muscle activity at sleep onset (6, 16). In a follow-up protocol, Fogel et al. (6) found that the decline in diaphragm muscle activity at sleep onset was not significantly different between OSA patients and controls when RUA was matched between groups via application of continuous positive airway pressure, but that overall ventilation and genioglossus muscle activity continued to fall to a lower level in OSA patients. In addition, while continuous positive airway pressure led to a significant reduction in phasic activation of the genioglossus during wakefulness in obese OSA patients, activity remained substantially higher than in control individuals. These findings support the presence of a heightened centrally mediated respiratory drive output beyond the negative neurocompensatory reflex component of UA dilator muscle activation. Combined with the present findings, these data support that OSA patients exhibit an increased and widespread wakefulness-dependent drive to the respiratory system that is not limited to the UA. This may be an adaptation necessary to compensate for respiratory system mass loading effects.

Methodological Considerations

There are several methodological issues to consider in this study. Greater decreases in lung volume and diaphragm muscle activity with more severe respiratory events at sleep onset clearly do not establish that causal relationships with UA collapse necessarily exist. Nevertheless, these observations are consistent with, and do not discount, the presence of causal relationships and are in contrast to ruling out lung volume influences had we found no changes at sleep onset. That lung volume/caudal traction changes with obesity per se contribute to increased UA collapse and the strength of such effects remain to be established.

We did not attempt to recruit an obese non-OSA comparator group for several reasons. The primary intent of the study was to investigate the impact of obesity, a known major factor in OSA risk, on ventilatory parameters potentially contributing to UA collapse in the context of OSA. Obese non-OSA individuals are difficult to recruit, may be skewed toward abnormally robust UA function, despite obesity effects, and/or exhibit a different pattern of obesity than captured by conventional BMI measurements. Consequently, we elected to first establish the nature and magnitude of lung volume changes at sleep onset before further studies designed to examine these mechanisms in more detail.

We used conventional criteria using manual scoring of sleep recordings (blinded to group allocation and independent from the respiratory analysis) to identify sleep onset as α EEG activity followed by three breaths or more of θ EEG activity. We did not employ a breath-by-breath α-to-θ criterion ratio as has been performed by others (6, 35). While it is not clear what impact this may have on the measured outcomes, we speculate that manually scored sleep onset periods would include fewer and more prolonged transitions into sleep, and potentially underestimate transitory effects identified from shorter episodes of sleep in closer proximity to prior respiratory and arousal events.

In contrast to previous studies in which diaphragm activity was assessed by surface electrodes (6, 35), the present study measured diaphragm activity via intraesophageal recordings over a larger area of the crural diaphragm (3). Surface recordings are likely to be highly influenced by changes in lung volume/diaphragm position (8) and must be interpreted with considerable caution, as apparent decreases in activity may reflect diaphragm movement away from the recording electrodes. Although intraesophageal recordings must also be interpreted with caution, by averaging across all electrode pairs over a wide distance, these measures are more robust to confounding by diaphragm movement with respiration and lung volume changes. Consequently, the apparent loss in diaphragm activity at sleep onset is unlikely a recording artifact and was consistent with simultaneous decrements in lung volume and ventilatory output. It is also unclear to what extent sleep onset-related changes in lung volume and potentially caudal tracheal traction are influenced by costal vs. caudal diaphragmatic displacement. Two studies have shown significant cranial movement of the most posterior regions of the diaphragm apposed to the lung following the induction of anesthesia in patients in the supine position (7, 19). Given that −30% of the diaphragm muscle, i.e., the domes of the diaphragm, is lung apposed at functional residual capacity (9) and caudal traction forces are importantly modulated by the action of the diaphragm acting on mediastinal structures, we would suggest that the crural diaphragm may have a greater effect (via caudal traction) on the UA than does the costal diaphragm.
Our method of quantifying diaphragm EMG activity is somewhat of a departure from more conventional methods (6, 34) and was chosen on the basis of superior signal-to-noise characteristics via integration over the full period of inspiration. In six subjects from a previous study undertaken in our laboratory, the coefficient of variation in \(i\)EMG\(_{\text{dia}}\) and \(e\)EMG\(_{\text{dia}}\) was 7.8 ± 0.5 and 6.2 ± 0.9% compared with 12.9 ± 2.4 and 6.4 ± 1.1% derived from conventional peak and tonic activity measurements, respectively.

**Summary and Conclusions**

Despite increased intragastric pressure and Pdi, we found no significant difference in \(e\)EMG\(_{\text{dia}}\) during wakefulness between obese OSA patients vs. healthy-weight controls. Sleep onset was accompanied by significant decrements in ventilation and respiratory muscle activity, with greater decrements potentially in OSA patients. Muscle activity and EELV significantly was accompanied by significant decrements in ventilation and obstructive sleep apnea. Am J Respir Crit Care Med 172: 114–117, 2005.


REFERENCES


