Expiratory Flow Limitation and Orthopnea in Massively Obese Subjects*

Anna Ferretti, MD; Pietro Giampiccolo, MD; Alberto Cavalli, MD; Joseph Milic-Emili, MD; and Claudio Tantucci, MD

Background: Morbidly obese subjects, who often complain about breathlessness when lying down, breathe at low lung volume with a reduced expiratory reserve volume (ERV). Therefore, during tidal breathing the expiratory flow reserve is decreased, promoting expiratory flow limitation (EFL), which is more likely to occur in the supine position, when the relaxation volume of the respiratory system, and hence the functional residual capacity (FRC), decrease because of the gravitational effect of the abdominal contents.

Purpose: The aim of the study was to assess EFL and orthopnea in massively obese subjects and to evaluate whether orthopnea was associated with the development of supine EFL.

Methods: In 46 healthy obese subjects (18 men) with a mean (± SD) age of 44 ± 11 years and a mean body mass index (BMI) of 51 ± 9 kg/m², we assessed EFL in both the seated and the supine positions by the negative expiratory pressure method and assessed postural changes in FRC by measuring the variations in the inspiratory capacity (IC) with recumbency. Simultaneously, dyspnea was evaluated in either position using the Borg scale dyspnea index (BSDI) to determine the presence of orthopnea, which was defined as any increase of the BSDL in the supine position.

Results: Partial EFL was detected in 22% and 59%, respectively, of the overall population in seated and supine position. The mean increase in the supine IC amounted to 120 ± 200 mL (4.1 ± 6.4%), indicating a limited decrease in FRC with recumbency in these subjects. Orthopnea, although mild (mean BSDI, 1.7 ± 1.3), was claimed by 20 subjects, and in 15 of them EFL occurred or worsened in the supine position. Orthopnea was associated with lower values of seated ERV (p < 0.05) and was marginally related to supine EFL values (p = 0.07). No significant effect of age, BMI, obstructive sleep apnea-hypopnea syndrome, FEV₁, and forced expiratory flow at 75% of vital capacity was found on either orthopnea or EFL.

Conclusion: In morbidly obese subjects, EFL and dyspnea frequently occur with the subject in the supine position, and both supine EFL and low-seated ERV values are related to orthopnea, suggesting that dynamic pulmonary hyperinflation and intrinsic positive end-expiratory pressure may be partly responsible for orthopnea in massively obese subjects.

(CHEST 2001; 119:1401–1408)

Key words: body posture; flow limitation; lung function; negative expiratory pressure; orthopnea

Abbreviations: BMI = body mass index; BSDL = Borg scale dyspnea index; EELV = end-expiratory lung volume; EFL = expiratory flow limitation; ERV = expiratory reserve volume; f = respiratory frequency; FEF₇₅ = forced expiratory flow at 75% of vital capacity; FL = flow-limited; FRC = functional residual capacity; IC = inspiratory capacity; MRC = Medical Research Council; NEP = negative expiratory pressure; NFL = non-flow-limited; OSAHS = obstructive sleep apnea-hypopnea syndrome; Pao = pressure measured at the airway opening; PEEPi = intrinsic positive end-expiratory pressure; Rs = respiratory system resistance; RV = residual volume; TE = expiratory time; Ttot = total breathing cycle time; V̇e = minute ventilation; Vr = relaxation volume of the respiratory system; Vt = tidal volume

In healthy obese subjects, the relaxation volume of the respiratory system (Vr) is reduced because the equilibrium between the elastic forces of the lung and chest wall occurs at a lower thoracic gas volume due to alterations in the pressure-volume curve of the chest wall.¹–⁷ Accordingly, the functional residual capacity (FRC) usually is decreased with the patient in the sitting position.⁸–¹⁰ Since, in contrast, the residual volume (RV) either is normal or slightly increased, at least in middle-aged obese subjects,⁸,¹⁰,¹¹ the expiratory reserve volume (ERV) typically is reduced in these subjects.⁸–¹¹

For related article see page 1409
Because maximal expiratory flow rates decrease progressively with decreasing lung volume, breathing at a low lung volume necessarily is associated with a reduction in the expiratory flow reserve, which can further diminish in the presence of airway obstruction.

Although overt airflow obstruction is not a characteristic feature in simple obesity, reduced expiratory flow rates have been found in grossly obese nonsmoking male subjects. Furthermore, a subset of dyspneic obese subjects has been shown to have reduced forced expiratory flow at 75% of vital capacity (FEF 75%).

Thus, morbidly obese subjects may develop tidal expiratory flow limitation (EFL) that reaches the maximal expiratory flow rate during quiet breathing. This phenomenon is more likely to occur with the subject in the supine position, in which Vr, and hence FRC, are lower than in the sitting position due to the gravitational effect of the abdominal contents. Therefore, ERV is further reduced because RV does not change substantially with body posture.

Tidal EFL promotes dynamic pulmonary hyperinflation and intrinsic positive end-expiratory pressure (PEEPi), which imposes a threshold load on the inspiratory muscles. This hypothesis has been tested in a small number of male obese subjects who were found to exhibit EFL and PEEPi mainly in the supine posture with a concomitant increase in diaphragmatic activity that was, in part, related to PEEPi.

The imbalance between the reduced strength of the inspiratory muscles and the increased mechanical load due to PEEPi, which was caused by dynamic pulmonary hyperinflation, has been related to the dyspnea sensation in patients with COPD. Similarly, in supine severely obese subjects who have a mild degree of inspiratory muscle weakness, which probably is related to an overstretching of the diaphragm, and an increased work of breathing because of reduced chest wall and lung compliance and increased respiratory system resistance (Rrs), the presence of EFL might contribute to the occurrence or worsening of dyspnea with recumbency, which frequently is observed in clinical practice.

Therefore, the aim of this study was to assess in a large population of healthy grossly obese subjects either the presence of EFL, in both the seated and the supine positions, or the occurrence of orthopnea and to evaluate whether orthopnea was associated with the development of supine EFL. A relationship between these two phenomena recently has been reported in patients with acute left-sided heart failure.

Materials and Methods

Subjects

Forty-six obese subjects (18 men) with a mean body mass index (BMI) of 40 kg/m² were investigated in a prospective open study at the respiratory division of Sant’Orsola-Malpighi Hospital in Bologna, Italy. The mean (±SD) age and BMI were 42 ± 11 years and 50 ± 8 kg/m², respectively, for men and 45 ± 10 years and 51 ± 9 kg/m², respectively, for women. Five men and eight women were current smokers, while four men and five women were previous smokers. None of the subjects had acute or chronic cardiopulmonary or neuromuscular diseases. No electrolytic or metabolic abnormalities were found in routine laboratory evaluations. All subjects had normal radiographs. Fourteen men and 7 women had obstructive sleep apnea-hypopnea syndrome (OSAHS), with an apnea-hypopnea index of > 10, as determined by previous full-night polysomnography monitoring. These subjects were treated with nocturnal continuous positive airway pressure that was withdrawn 1 week before the study began. The experimental protocol was approved by the local ethics committee, and informed consent was obtained from all subjects.

Measurements

All subjects underwent routine pulmonary function tests in the sitting position while breathing through a mouthpiece and wearing a noseclip. Spirometry was performed with a pneumotachograph using a computerized system (P.K. Morgan; Gillingham, UK). After the measurement of slow vital capacity, three acceptable and reproducible maximal flow/volume curves were obtained. The best values of FEV₁ and FVC and flows from the flow/volume curve with the highest sum of FEV₁ and FVC were considered for analysis. RV was determined using the helium dilution method, and FRC was calculated as the sum of the RV and the ERV. The predicted values were those reported by the European Community for Coal and Steel. Arterial blood gas analysis was performed on blood samples obtained from subjects in the seated position (Stat Profile; Nova Biomedical; Wallingford, MA).

EFL was assessed by the negative expiratory pressure (NEP) technique both in the seated and the supine position. Flow was measured with a pneumotachograph (model 3813; Hans-Rudolph; Kansas City, MO) with a ± 13.3 L/s linearity range connected to the mouthpiece and a differential pressure transducer (model DP55 [± 3 cm H₂O]; Raytech Instruments; Vancouver, BC, Canada). Pressure was measured at the airway opening (Pao) via a rigid polyethylene tube (internal diameter = 1.7 mm), connected to a differential pressure transducer (DP55 [± 100 cm H₂O]; Raytech Instruments). The system used to measure mouth pressure had no appreciable shift or alteration in amplitude up to 20 Hz.

A Venturi device (Aeromech Devices Ltd; Almonte, ON, Canada) that was capable of rapidly generating a negative pressure was connected in series with the cone of the pneumotachograph. The dead space of the assembly was about 150 mL. A side orifice on the Venturi device was attached via an electrically operated solenoid valve to a tank of compressed air. A pressure regulator between the tank and the valve was used to obtain the desired level of negative pressure at Pao. The electrical valve (model 8262G208; Ascolectric; Brantford, ON, Canada) was driven by a computer (Direc Physiologic Recording System; Raytech Instruments) and had an opening time of 28 ms. The opening valve was activated when the expiratory flow reached a threshold level of 20 mL/s with an optional delay that was empirically predetermined for each subject in order to apply NEP immediately after the peak of the tidal expiratory flow. In all
instances, the NEP application was timed to last until the lung volume corresponding to the end-expiratory lung volume (EELV) of the previous control breath was reached.

The volume and Pao signals were amplified (AC Bridge Amplifier-ABC module; Raytech Instruments), were filtered through a low-pass filter at 50 Hz, were sent to a 16-bit A/d converter (Direc Physiologic Recording System; Raytech Instruments) connected to a personal computer (486DX, 66Mhz processor), and were sampled at 200 Hz. Both digitized signals were displayed in real time on the computer screen together with the volume signal obtained by numerical integration of the flow signal. The tracings were continuously monitored both with respect to time and as flow/volume curves. The recordings were stored on the hard drive of the computer (Direc format) and were used for subsequent analysis. Data analysis was performed using data analysis software (Direc NEP, version 3.1; Raytech Instruments; or Anadat, version 5.2; RHT-InfoDat; Montreal, Quebec, Canada).

Chronic dyspnea was assessed using the modified Medical Research Council (MRC) dyspnea scale27 with verbal descriptors starting from 0 (ie, not troubled by dyspnea) to a maximum value of 5 (ie, dyspnea for minimal effort).

Orthopnea was defined as any increment in the modified Borg dyspnea scale28 after recumbency and, if present, was quantified according to the score difference in this category scale (range, 0 to 10) between the seated and the supine positions.

Procedure and Data Analysis

During the study, the subjects were placed in a comfortable dentist’s chair with their necks fixed in a neutral position, while they breathed through a rigid mouthpiece with their nostrils occluded by a noseclip. Initially, the subjects were studied sitting upright and then, by rotating the chair, in the supine position without changing the experimental setup. In both positions, after an initial period of adaptation, at least 1 min of quiet breathing was recorded for the analysis of the breathing pattern parameters, such as inspiratory time (Ti), expiratory time (Te), total breathing cycle time (TTtot), Ti/TTe ratio, tidal volume (VT), respiratory frequency (f), minute ventilation (Ve), mean inspiratory flow (ie, VT/Ti ratio), and mean expiratory flow (ie, VT/Te ratio). Subsequently, five or more NEP breath tests were performed by applying pressure of ~5 cm H2O, or less if needed, at the onset of expiration. At least four regular breaths were allowed between two subsequent NEP breath tests.

Complete or partial EFL was detected when, following the NEP application, the expiratory flow did not increase relative to that of the preceding control expiration throughout the whole tidal expiration or part of it (ie, < 50% of VT).29 In that case, the extent of EFL was expressed as the percentage of control VT (Fig 1).

Finally, with a stable EELV level during tidal breathing, the subjects were asked to perform twice a slow maximal inspiration applying pressure of 2.86 cm H2O, or less if needed, at the onset of inspiration. At least four regular breaths were allowed between two subsequent NEP breath tests.

Complete or partial EFL was detected when, following the NEP application, the expiratory flow did not increase relative to that of the preceding control expiration throughout the whole tidal expiration or part of it (ie, < 50% of VT).29 In that case, the extent of EFL was expressed as the percentage of control VT (Fig 1).

The MRC dyspnea scale was administered before experiments. After adequate instruction, the subjects were asked to point at the level of their dyspnea sensation on the modified Borg scale, first in the seated position and next in the supine posture, always 15 min after assuming either position and before starting the respective NEP test procedure. The number selected by the subjects was referred to as the Borg scale dyspnea index (BSDI).

An unpaired Student’s t test was used to compare the anthropometric and functional characteristics of subjects with and without EFL, as well as those of subjects with and without orthopnea. We used the Fisher’s Exact Test to compare the distribution of either supine EFL or orthopnea according to sex and OSAHS and also to compare the distribution of orthopnea according to supine EFL. Data were expressed as mean ± SEM, unless otherwise specified.

Results

The anthropometric characteristics and lung function parameters of the subjects are given in Table 1. According to the MRC dyspnea scale, the chronic dyspnea score as reported by our subjects was mild, amounting to 1.1 ± 0.1. Twenty-one subjects (14 men) had OSAHS, with a markedly higher prevalence in men (78%) than in women (25%).

None of the subjects exhibited complete EFL in either position. Partial EFL was detected in 10 subjects (4 men) when seated, and in 27 subjects (10 men) when supine, corresponding to 22% and 59%, respectively, of the overall population (Fig 2). In the supine position, 56% of the men (10 of 18 men) and 61% of the women (17 of 28) exhibited partial EFL.

In the whole population, IC was 4 ± 1 L higher in the supine position than in the sitting position, increasing from 2.89 ± 0.12 L to 3.01 ± 0.13 L (p < 0.001). In men in the supine position, the IC increased from 3.58 ± 0.13 L to 3.73 ± 0.17 L (p < 0.02) and in women from 2.41 ± 0.10 L to 2.51 ± 0.11 L (p < 0.01).

No significant differences were found among several anatomic and functional parameters, except for BSDI, between obese subjects who were partially flow-limited (FL) and those who were non-flow-limited (NFL) in the supine position (Table 2). With recumbency, IC increased from 2.85 ± 0.16 L to 3.02 ± 0.18 L in the supine FL group and from 2.86 ± 0.18 L to 2.98 ± 0.20 L in the supine NFL group. None of the breathing pattern variables was different between the seated and supine position in the whole population and also in the subgroups of NFL and FL obese subjects when they were in the supine position (Table 3).

Orthopnea was claimed by 20 subjects (9 men), corresponding to 44% of the overall sample (Fig 2). Therefore, 50% of the men and 39% of the women had greater dyspnea when supine. However, according to the BSDI increment (∆BSDI), the increase in dyspnea in the supine position was mild, amounting to 1.7 ± 1.3 ∆BSDI (range, 0.5 to 4) [Fig 3]. Partial EFL occurred or worsened for subjects in the supine position in 15 of the 20 subjects claiming orthopnea (75%; 6 men) [Fig 2, 3].

No significant influence of sex, age, smoking habit, BMI, OSAHS, FEV1, and FEF75% was found on the presence of either orthopnea or EFL in subjects in the supine position. Orthopnea was associated with significantly lower values of ERV (61 ± 4 vs
72 ± 3% predicted) measured in the sitting position (p < 0.05) and was marginally related to the presence of EFL measured in the supine position (p = 0.07; relative risk, 2.11; 95% confidence interval, 0.92 to 4.81).

**DISCUSSION**

The main findings of this study of healthy, massively obese subjects are the following: (1) partial EFL is common when subjects are supine, while it is seldom observed when they are seated; (2) almost half of these subjects experienced orthopnea, although to a mild degree; and (3) in most subjects orthopnea was associated with the occurrence or worsening of EFL in the supine position.

Before discussing these results, some aspects inherent in the NEP method chosen to detect EFL need to be addressed. Most of our morbidly obese subjects (about 70% of whom were in the supine position) exhibited a drop in expiratory flow below the control tidal expiration immediately after the application of NEP, as shown in Figure 1. This phenomenon is likely due to a temporary increase in upper airways resistance in the face of NEP, as was demonstrated in healthy, snoring, nonobese subjects. Indeed, almost all of these obese subjects were heavy snorers, making it difficult to establish whether this finding was related to the obesity or to the snoring habit. When the reduction in the expiratory flow following the NEP is transient, the NEP method is still able to detect EFL (Fig 1).

**Figure 1.** Tidal flow-volume loop during resting breathing with the corresponding NEP test breath in two representative healthy obese subjects. In the first subject (top), partial EFL is detected only in the supine position, while in the second subject (bottom), partial EFL is present in the seated position and worsens by adopting the supine posture. Note that in all instances of the application of NEP (pressure, –5 cm H2O), the expiratory flow shows a transient drop below the control flow, which likely reflects a temporary increase of upper airway resistance. After this transient decrease in flow, the expiratory flow during NEP exceeds the control flow, showing that there is no EFL until true intrathoracic EFL occurs (arrows).
This is because, once the upper airways narrowing has been effectively reversed by the reflex activation of the upper airway dilating muscles, the subsequent behavior of the expiratory flow during NEP reflects again only the intrathoracic airways mechanics (Fig 1). In contrast, if this drop below the control expiratory flow is prolonged, the NEP method can no longer be valid to assess EFL, as the persistent narrowing or collapse of the upper airways markedly reduces the expiratory flow rate, preventing the detection of any possible EFL. In this case, however, valid measurements may be obtained with repeated NEP breath tests using lower levels of NEP (eg, from 23 to 21 cm H₂O). In our study, this was necessary only a few times and, mainly, for subjects in the supine position to avoid a prolonged fall in the expiratory flow, thereby achieving clear information about the presence or absence of intrathoracic EFL in all subjects.

In line with an investigation performed with a small group of obese male subjects using the NEP method, this larger study on subjects with extreme obesity of both sexes shows that partial EFL is uncommon for subjects in the seated position, whereas it is frequently encountered in subjects who are in the supine position. Despite a relatively high mean expiratory flow (ie, Vt/Te during resting breathing; Table 3), normal values of FEF₇₅% prevented EFL from developing in most obese subjects when seated, even if tidal breathing is taking place near to the RV.

In our population of massively obese subjects, the Ti/Ttot ratio was abnormally high (.0.5) due to Te being shorter than Ti. This finding, which accounts for the increase in mean expiratory flow, has been reported previously in eucapnic morbidly obese subjects.

### Table 1—Anthropometric and Lung Function Data for the Subjects*

<table>
<thead>
<tr>
<th>Age, yr</th>
<th>BMI, kg/m²</th>
<th>FEV₁, % pred</th>
<th>FEV₁/FVC, %</th>
<th>FEF₇₅%, % pred</th>
<th>TLC, % pred</th>
<th>VC, % pred</th>
<th>IC, % pred</th>
<th>FRC, % pred</th>
<th>ERV, % pred</th>
<th>RV, % pred</th>
<th>Pao₂, mm Hg</th>
<th>Paco₂, mm Hg</th>
</tr>
</thead>
<tbody>
<tr>
<td>44 ± 11</td>
<td>51 ± 9</td>
<td>95 ± 14</td>
<td>86 ± 6</td>
<td>103 ± 22</td>
<td>93 ± 8</td>
<td>95 ± 12</td>
<td>105 ± 16</td>
<td>83 ± 12</td>
<td>66 ± 19</td>
<td>93 ± 17</td>
<td>77 ± 11</td>
<td>42 ± 4</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. TLC = total lung capacity; VC = vital capacity; % pred = percent predicted.

### Table 2—Anatomic and Functional Data in Obese Subjects With and Without EFL in the Supine Position*

<table>
<thead>
<tr>
<th>Data</th>
<th>NFL (n = 19)</th>
<th>FL (n = 27)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sex</td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>51 ± 9</td>
<td>50 ± 10</td>
</tr>
<tr>
<td>MRC score</td>
<td>0.9 ± 0.9</td>
<td>1.2 ± 0.9</td>
</tr>
<tr>
<td>TLC, % pred</td>
<td>93 ± 8</td>
<td>93 ± 8</td>
</tr>
<tr>
<td>VC, % pred</td>
<td>95 ± 15</td>
<td>95 ± 10</td>
</tr>
<tr>
<td>FRC, % pred</td>
<td>84 ± 10</td>
<td>82 ± 13</td>
</tr>
<tr>
<td>ERV, % pred</td>
<td>69 ± 18</td>
<td>65 ± 20</td>
</tr>
<tr>
<td>RV, % pred</td>
<td>94 ± 19</td>
<td>93 ± 15</td>
</tr>
<tr>
<td>FEF₁, % pred</td>
<td>95 ± 17</td>
<td>95 ± 12</td>
</tr>
<tr>
<td>FEF₁/FVC, %</td>
<td>86 ± 6</td>
<td>85 ± 6</td>
</tr>
<tr>
<td>FEF₁₆₅%, % pred</td>
<td>105 ± 38</td>
<td>95 ± 24</td>
</tr>
<tr>
<td>FEF₁₆₅%, % pred</td>
<td>102 ± 25</td>
<td>105 ± 19</td>
</tr>
<tr>
<td>Pao₂, mm Hg</td>
<td>77 ± 12</td>
<td>78 ± 9</td>
</tr>
<tr>
<td>Paco₂, mm Hg</td>
<td>42 ± 4</td>
<td>42 ± 4</td>
</tr>
<tr>
<td>BSDI</td>
<td>0.1 ± 0.2</td>
<td>0.4 ± 0.7</td>
</tr>
<tr>
<td>BSDI supine</td>
<td>0.5 ± 1.0</td>
<td>1.3 ± 1.7</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD. FEF₁₆₅% = forced expiratory flow at 50% of vital capacity. See Table 1 for abbreviations not used in the text.

†p < 0.05.
subjects and is related to an alteration in neural timing. However, the precise mechanisms that lead to the shortening of Te (eg, elevated recoil pressure of the respiratory system, decreased postinspiratory activity of inspiratory muscles, or expiratory muscle activity during expiration) are not clear in obese subjects and need further investigation. In this respect, it should be noted that the persistence of the diaphragmatic activity during expiration that has been demonstrated in obese subjects was significantly longer than that in control subjects of normal weight.32 Ancedotal observations also have shown no evidence of expiratory muscle activity at end-expiration in grossly obese subjects in both the seated and supine positions.19

Tidal EFL was detected in about 60% of the subjects when supine, with similar prevalence in men and women. Three factors might account for supine EFL in massive obesity, as discussed below.

Substantial reductions in ERV are consistently found in seated obese subjects. In our population, the ERV amounted to 0.79 ± 0.04 L in the seated position, corresponding to 66 ± 3% of that predicted. Hence, in many obese subjects the ERV may not be large enough to accommodate the posture-related decrease in Vt, obliging the EELV to occur very close to the RV and necessarily causing the development of partial EFL. This mechanism is substantiated by the relatively small increase (4 ± 1%) in IC exhibited by our obese subjects on shifting to the supine posture. Similar results have been found previously in obese subjects when supine8,33 compared to control subjects, reflecting a smaller decrease in FRC with recumbency.8,33,34 In this respect, it is worthwhile to note that the absolute value of seated ERV was smaller in the FL than NFL subjects in the supine position, amounting to 750 ± 60 mL vs 840 ± 50 mL, respectively.

In our subjects, as well as in the subgroup with supine EFL, the breathing pattern did not change between the sitting and supine positions (Table 3). This information was consistent with that from a previous report19 showing no postural variations of the Vt, f, and Ve in a small sample of massively obese subjects. It follows that changes in the breathing pattern could not be invoked as the mechanism able to induce supine EFL under these circumstances. It should be stressed, however, that the high Vt and mean expiratory flow, such as those exhibited by these obese subjects, may favor EFL in the presence of other facilitating factors.

Increased airway resistance and decreased maximal expiratory flow rates at low lung volume with recumbency, in excess of those accountable for by the reduction in FRC, could represent an important mechanism in eliciting supine EFL by further limiting the expiratory flow reserve. In a 1995 article,33

### Table 3—Breathing Pattern Variables in the Seated and the Supine Positions*

<table>
<thead>
<tr>
<th>Variables</th>
<th>Vt, L</th>
<th>f, breaths/min</th>
<th>Ve, L/min</th>
<th>Ti, s</th>
<th>Te, s</th>
<th>Ti/Ttot, %</th>
<th>Vt/Ti, L/s</th>
<th>Vt/Te, L/s</th>
</tr>
</thead>
<tbody>
<tr>
<td>All</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seated</td>
<td>0.96 ± 0.34</td>
<td>16.8 ± 5.4</td>
<td>14.9 ± 4.2</td>
<td>2.36 ± 1.26</td>
<td>1.82 ± 0.97</td>
<td>0.56 ± 0.03</td>
<td>0.44 ± 0.14</td>
<td>0.57 ± 0.14</td>
</tr>
<tr>
<td>Supine</td>
<td>0.95 ± 0.35</td>
<td>16.3 ± 5.6</td>
<td>13.9 ± 2.8</td>
<td>2.47 ± 1.35</td>
<td>1.92 ± 1.08</td>
<td>0.56 ± 0.04</td>
<td>0.41 ± 0.09</td>
<td>0.53 ± 0.11</td>
</tr>
<tr>
<td>NFL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seated</td>
<td>1.03 ± 0.35</td>
<td>16.2 ± 3.9</td>
<td>15.9 ± 5.0</td>
<td>2.26 ± 0.78</td>
<td>1.77 ± 0.63</td>
<td>0.56 ± 0.03</td>
<td>0.47 ± 0.17</td>
<td>0.60 ± 0.16</td>
</tr>
<tr>
<td>Supine</td>
<td>0.99 ± 0.37</td>
<td>16.2 ± 4.3</td>
<td>14.9 ± 2.8</td>
<td>2.32 ± 0.93</td>
<td>1.77 ± 0.75</td>
<td>0.57 ± 0.03</td>
<td>0.44 ± 0.10</td>
<td>0.57 ± 0.12</td>
</tr>
<tr>
<td>FL</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Seated</td>
<td>0.92 ± 0.33</td>
<td>17.2 ± 6.3</td>
<td>14.1 ± 3.3</td>
<td>2.43 ± 1.52</td>
<td>1.86 ± 1.16</td>
<td>0.57 ± 0.03</td>
<td>0.42 ± 0.10</td>
<td>0.54 ± 0.13</td>
</tr>
<tr>
<td>Supine</td>
<td>0.92 ± 0.34</td>
<td>16.5 ± 6.5</td>
<td>13.2 ± 2.7</td>
<td>2.57 ± 1.59</td>
<td>2.02 ± 1.26</td>
<td>0.56 ± 0.04</td>
<td>0.39 ± 0.09</td>
<td>0.50 ± 0.11</td>
</tr>
</tbody>
</table>

*Values given as mean ± SD.

---

Figure 3. Changes in dyspnea according to the BSDI on shifting from the seated to the supine position in both NFL and partially FL obese subjects when supine. Orthopnea, defined as any increase in dyspnea with recumbency (closed symbols), was more frequent and severe in obese subjects who had EFL in the supine position (FL).
the increase in supine Rrs, which was not explained by the reduction in mid-tidal lung volume, was markedly greater in healthy obese subjects than in normal control subjects. Although this increase in Rrs might be related to the airway narrowing due to increased intrapulmonary blood volume in obese subjects when supine, the small decrease in the forced expiratory flows at low lung volumes exhibited by this group of subjects in the presence of a larger fall in peak flow favors an extrathoracic airway narrowing as the main site of increased Rrs in the supine position. However, since we did not measure airway resistance or maximal expiratory flows in the supine position, the role of the recumbent obstruction of intrathoracic airways in promoting supine EFL, although likely to be modest, cannot exactly be defined in our subjects.

Orthopnea, although mild, was claimed by almost half of the subjects, with a substantially greater prevalence in men. Most of the subjects with orthopnea developed EFL or worsened the extent of EFL (from 15 ± 2% to 28 ± 3% of Vr; p < 0.01) when supine (Fig 1, 2).

In the presence of predisposing conditions such as the shortening of TE due to rapid breathing, augmented Vr because of high ventilatory demand, and increased airway resistance caused by airway obstruction or reduced lung volumes, EFL easily can induce dynamic hyperinflation and PEEPi by preventing the respiratory system from reaching its Vr during expiration. Thus, EFL in the supine position may increase the inspiratory work of breathing because of PEEPi, as has been documented in supine obese subjects. Indeed, this could be an important mechanism leading to orthopnea in several obese subjects, especially those with higher levels of Vr and Vr/TE. However, 12 of the 27 subjects who were partially FL in the supine position had no orthopnea (Fig 2, 3), suggesting a little increase in dynamic hyperinflation with recumbency, a higher threshold of dyspnea, or both in these individuals.

On the other hand, dynamic hyperinflation and PEEPi also could occur in massively obese subjects when supine simply because of the severe reduction in ERV. In fact, according to previous reports, many of our subjects should have their Vr below RV in the supine position due to the markedly decreased ERV. Since RV represents an absolute lower limit to EELV, it is implicit that these subjects, when supine, breathe tidally above the Vr, become dynamically hyperinflated, develop PEEPi, and possibly experience orthopnea. Under these conditions, supine EFL would be merely an associated phenomenon. This is supported by the fact that significantly lower values of ERV were found in the obese subjects with orthopnea (p < 0.05).

Finally, 25% of the subjects who claimed to have orthopnea were not FL in the supine position. Conceivably, other factors besides the development of dynamic hyperinflation and PEEPi may cause or worsen dyspnea in these subjects when they are recumbent. A marked increment in upper and lower airway resistance has been observed in seated obese subjects who have increasing apnea-hypopnea index scores, and a further significant increase in Rrs was found with recumbency in obese, healthy subjects. Since in our population orthopnea was reported mostly by men who exhibited a striking prevalence of OSAHS compared to women, it is likely that an increase in mechanical resistive load may play a role in eliciting dyspnea in some of these individuals when supine.

In conclusion, our results indicate the following: (1) in healthy, massively obese subjects the occurrence or worsening of partial EFL and dyspnea are frequent when they are in the supine position; (2) in these subjects both EFL and reduced ERV, which are related to orthopnea, could explain the dynamic hyperinflation and PEEPi previously observed in obese subjects when they were supine; and (3) this mechanism may partly account for orthopnea in obese subjects.

REFERENCES

13 Crapo RO, Kelly TM, Elliott CG, et al. Spirometry as a
preoperative screening test in morbidly obese patients. Surgery 1986; 99:763–768
18 Pepe PE, Marinii JJ. Occult positive end-expiratory pressure in mechanically ventilated patients with airflow obstruction: the auto-PEEP effect. Am Rev Respir Dis 1982; 126:166–170