Expiratory flow limitation and intrinsic positive end-expiratory pressure in obesity

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PEEPi was significantly smaller (position (P), kg/m² and six age-matched normal-weight control subjects 0.05) and decreased to 0.20 6

LUNG FUNCTION IN OBESITY is characterized by reduced expiratory reserve volume (ERV) and functional residual capacity (FRC) (2, 21, 36). The effects of obesity on airway caliber and dynamic airway function are less clear. A low ERV has been associated with frequency dependence of compliance, an abnormal distribution of ventilation, and increased closing volume. These factors are suggestive of small airway closure and air trapping (7, 11). In a recent communication, Rubinstein and colleagues (23) described reduced expiratory flow rates in morbidly obese, nonsmoking men. Although the decreased compliance of the respiratory system should tend to prevent dynamic hyperinflation, two earlier studies have observed an increased residual volume-to-total lung capacity ratio (RV/TLC) in subgroups of grossly obese subjects (OS), suggestive of air trapping (21, 24). These observations are in contrast to others, which showed normal forced expiratory flow rates and normal airway conductance (6, 36).

The purpose of the present study was to detect expiratory flow limitation (EFL) in OS with a novel method of using negative expiratory pressure (NEP) (13, 33). This method has the advantage of detecting EFL during tidal breathing without nonphysiological forced-breathing maneuvers, i.e., comparison of maximal and tidal flow-volume curves. The FVC maneuver causes considerable compression of gas within the thorax, which affects the thoracic gas volume (TGv) (12). Therefore, comparison of maximal and tidal flow-volume curves should be performed with a body plethysmograph (12, 13, 22). With the NEP technique, measurements can be performed in different body positions, avoiding the need for a body plethysmograph (13). This point is of particular importance because EFL and air trapping, if present at all, should be enhanced in the supine posture. When OS change their position from upright to supine, lung volumes are further decreased and subjects breathe very close to RV (2, 32). It has been demonstrated earlier that breathing at low lung volumes may lead to airway closure (5, 7, 11) and that closing volumes may exceed FRC even in healthy middle-aged subjects when lung volumes are reduced by adopting the supine posture (14).

A consequence of EFL would be that end-expiratory lung volume is increased, i.e., FRC at end expiration remains above the relaxation volume of the respiratory system. PEEP, is the end-expiratory elastic recoil pressure of the respiratory system due to incomplete expiration, which must be counterbalanced by the inspiratory muscles before inspiratory flow is achieved during the next breathing cycle. PEEP, imposes additional elastic load on the inspiratory muscles and thus increases the work of breathing. Although PEEP, has recently been described in patients with cirrhotic ascites (8), no systematic investigation has been performed concerning PEEP, in obesity.

Thus, the purpose was to investigate 1) whether in OS EFL occurs during breathing at rest, 2) whether obesity is associated with PEEP, and a concomitant increase in respiratory muscle load, and 3) whether these two phenomena are promoted by the supine position.

METHODS

Subjects

The study was performed in eight OS and in six age-matched normal-weight healthy volunteers. Five of the OS had mild obstructive sleep apnea (OSA), and in three subjects OSA was excluded (apnea-hypopnea index < 5/h) by previous polysomnography. The smoking history of the OS revealed that one individual was a smoker, one was an exsmoker, and six were nonsmokers. All had a normal chest radiograph. Two
the control subjects (CS) were smokers, and four were nonsmokers. No subject had clinical signs or complaints of acute or chronic bronchitis. The anthropometric and lung function data are summarized in Table 1. The experimental protocol was approved by the ethics committee of the Philipps University Marburg, and informed written consent was obtained from the subjects.

Measurements

Body plethysmography. At the start of the study, spirometric and body plethysmographic lung function tests (MasterLab; Jaeger, Würzburg, Germany) and blood-gas analysis (ABL 300; Radiometer, Copenhagen, Denmark) were performed in all subjects in the upright position. Tidal flow-volume loops were superimposed on maximal flow-volume envelope. EFL was assumed when the tidal flow-volume loop lay on or outside the maximal flow-volume envelope.

NEP. We also measured EFL with an experimental setup that provides NEP, as described by Koulouris et al. (13). The device consisted of a T tube with a mouthpiece, equipped with a one-way pneumatic valve on one end and open to the atmosphere at the other end (occlusion pressure valve setup 9327; Hans Rudolph, Kansas City, MO). The valve was connected to a manually controlled switch (Hans Rudolph control switch 9301), which allows rapid (30–60 ms) opening or closure of the tube. This end of the T tube was connected to a commercial vacuum cleaner to expose the subjects to NEP.

A variable-orifice pneumotachograph (VarFlex flow transducer, Bicore, Irving, CA) was attached to the mouthpiece of the T tube. The pressure drop across the two ports of the pneumotachograph was measured with a differential piezoelectric pressure transducer (163P01D36, ± 12.7 cmH2O; Microswitch, Freeport, IL). Because the pressure signal across this pneumotachograph is not linear to airflow, linearization must be performed. A microcomputer constructed to transfer the pressure signal into a flow curve was used to perform this procedure (Biscope; Sing Medical, Stäfa, Switzerland). Pressure at the airway opening (Pao) was measured through a side port at the mouthpiece by using a piezoelectric pressure transducer (143P03D, ± 176 cmH2O, Microswitch). Combined resistance of the T tube and the flow sensor during tidal breathing was measured as $R = K_1 + K_2V$, adapted from Rohrer’s equation $P_{res} = K_1V + K_2V^2$, where $P_{res}$ is resistive pressure (kPa), $V$ is airflow (l/s), $K_1$ is the coefficient of linear resistance, and $K_2$ is the coefficient of nonlinear resistance (for inspiration, $K_1 = 0.19$ and $K_2 = 0.03$; for expiration, $K_1 = 0.20$ and $K_2 = 0.05$). The equipment dead space was 150 ml.

Diaphragmatic muscle effort and PEEP. Esophageal (Pes) and gastric (Pga) pressures were measured by using a catheter-mounted transducer (CMT) system with two piezoelectric pressure transducers located on the tip and 20 cm proximal to the tip (Gaeltec; Dunvegan, Isle of Skye, UK). A previous investigation showed that transpulmonary pressure measured with this system correlates well with measurements obtained with balloon catheters (19). Time synchrony of pressure and flow measurements was assessed by placing the flow sensor, the Pao sensor, and the CMT into the tube of a bass-reflex loudspeaker. Over the range of 5–10 Hz, there was a constant time delay ($\Delta t$) of airflow in relation to pressures of 0.03 s. The flow signal was corrected for changes in gas temperature and gas composition. All pressure channels were calibrated by using a water manometer. Before and after each investigation, the CMT was checked for drifts from zero. The flow and pressure signals were amplified and sampled at a rate of 100 Hz by using a computer data-acquisition system with a built-in 12-bit analog-to-digital converter (Topas; constructed by our group). The collected data were stored on optical disk for subsequent analysis. All variables were also recorded on a 16-channel strip-chart recorder (Picker, Munich, Germany) at a paper speed of 10 mm/s.

Experimental procedure and data analysis. All investigations were performed in the morning, at least 2 h after breakfast. The CMT was introduced through the nose, advanced until both transducers were located intraesophagally, and then withdrawn until opposite phase directions appeared during respiratory efforts, indicating placement of the Pes transducer at the gastroesophageal junction. The catheter was then withdrawn another 10–15 cm until minimal cardiac artifact was present and optimal correlations of Pao and Pes within a 10% range were registered in the upright and supine position by means of the occlusion method described by Baydur et al. (3). The CMT was fixed to the nose by elastic tape to prevent dislocation while subjects adopted different body positions. The nostrils were occluded with a nasedip.

<table>
<thead>
<tr>
<th>Table 1. Anthropometric and lung function data</th>
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<tr>
<td><strong>FVC</strong></td>
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<td><strong>Liters</strong></td>
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<tr>
<td><strong>Obese subjects</strong></td>
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<td>Subject 1</td>
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<td>Subject 2</td>
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<td>Subject 3</td>
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<td>Subject 8</td>
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<tr>
<td>Mean ± SD</td>
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<tr>
<td>Normal-weight control subjects</td>
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<tr>
<td>Mean ± SD</td>
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</table>

M, male; F, female; BMI, body mass index; FVC, forced vital capacity; FEV$_1$, forced expiratory volume in 1 s; ERV, expiratory reserve volume; FRC, functional residual capacity; Raw, airway resistance; $P_{aO_2}$, arterial $P_{O_2}$; $P_{aCO_2}$, arterial $P_{CO_2}$; Al, anemia index; HI, hypopnea index. Predicted (Pred) values are adapted from Ref. 35. *P < 0.05 vs. control.
During the first part of the study, subjects were studied while breathing spontaneously for a 10-min period after adopting different body positions in the following order: seated upright, lying supine, and lying on the right lateral decubitus side. Then, the upright position was reassumed. NEP was applied with 0.5 kPa during the beginning of selected expirations and was maintained throughout the ensuing expiration (Fig. 1). Subjects were taught not to interrupt expiration when NEP was applied. At least 10 experiments were performed per subject and per position, first upright and then supine. Because the valve that exposed the subjects to NEP was manually run, not all experiments were satisfactory, i.e., NEP was applied too early or too late. In the further analysis, only those experiments were included during which NEP was applied during the first 15% of expiratory time (TE).

Volume was obtained by numerical integration of the flow signal. Duration of inspiration (TI) and TE were analyzed from the flow tracing. The flow-volume loops obtained during the NEP maneuver and the immediately preceding breaths were superimposed (33). The flow signals were corrected for offsets by using the drift over a segment of at least 1 min. After that, flow gains were not adjusted on a breath-by-breath basis. Differences between expiratory flow with NEP compared with control were determined as Δflow at mid-tidal volume. EFL during tidal breathing was assumed when, during the application of NEP, expiratory flow did not increase in comparison with baseline flow during part of the tidal expiration (13). The flow-limited portion was expressed as a percentage of the tidal volume.

Dynamic PEEPi was measured as Pes change between the beginning of the abrupt pressure decay in Pes, indicating the start of inspiration, and the point where flow reaches the zero line. This method of estimating PEEPi in spontaneously breathing subjects is based on the assumption that the change in pleural pressure required to start inspiratory airflow approximates the end-expiratory elastic recoil pressure of the respiratory system, provided that the expiratory muscles are relaxed during expiration (18). PEEP, was corrected for the 0.03-s Δt of the flow signal (see above), assuming a linear decrease in Pes between the start of inspiratory muscle activity and inspiratory flow. Transdiaphragmatic pressure (Pdi) was calculated as Pga – Pes. Pressure-time product (PTP) for the diaphragm was obtained by electronically integrating Pdi over Ti. Inspiratory muscle effort was then determined as mean Pdi by dividing diaphragmatic PTP by Ti (1).

Comparisons of physiological variables in the different body positions were made by using Wilcoxon's signed rank test. Correlation analysis between PEEPi and Pdi was performed by using Spearman's rank correlation test. Error probability for significant results was determined as P < 0.05. Values are expressed as means ± SD.

RESULTS

In all subjects, NEP was successfully applied after a short training period. Measurements of Pdi were performed in all CS and in six of the eight OS. One subject did not tolerate the catheter, and in one subject the cardiac artifact was too strong to determine PEEPi.

EFL. Two methods were used to detect flow limitation (FL) in the sitting position. In subjects 2–5 and 8, no method was suggestive of EFL. Two subjects (subjects 6 and 7) were assumed to be flow limited with use of both methods. In contrast, in subject 1 only the comparison of the tidal and maximal flow-volume curves indicated FL, whereas there was no EFL with the NEP method. Figure 2 shows tidal flow-volume curves before and during the application of NEP in the sitting and supine position. In the CS, expiratory flow during NEP was always increased compared with unaffected expiration, indicating that CS were not flow limited. In the supine position, however, the NEP-induced increase in expiratory flow was smaller compared with the upright position (Table 2). This demonstrates that the expiratory flow reserve was reduced in recumbency. We found EFL in two of eight sitting and seven of eight supine OS: subject 1 was not flow limited in the upright or supine position, subjects 2–5 and 8 were flow limited in the supine but not in the sitting position, whereas subjects 6 and 7 were flow limited in both positions.

Table 3 gives additional information about the degree of EFL, i.e., the percentage of control tidal volume exhibiting FL. No subject was flow limited in the sitting position only. In most flow-limited subjects, there was an initial flow increase after application of NEP during the first part of expiration. This phenomenon has been described before and is probably related to postinspiratory activity of the inspiratory muscles and to a reduction in volume of the upper airways (13). OS with EFL

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**Fig. 1.** Representative record of flow (V˙) and pressure at airway opening (Pao) during spontaneous breathing. Negative pressure (NEP) was applied at onset of expiration (1st arrow) and was maintained throughout expiration. NEP was stopped after next inspiratory breathing effort was performed (2nd arrow).
in both positions did not differ with respect to age, body mass index, or lung function from those with FL only in the supine position or from subject 1 without FL.

PEEP. Figure 3 shows a representative record of flow, Pao, Pes, Pga, and Pdi in one OS in the supine position. The detection of dynamic PEEPi is based on the \( \Delta t \) of inspiratory flow and Pao in relation to inspiratory effort (1). In CS, \( \Delta t \) was always below 0.05 s (0.02 ± 0.02 s sitting, 0.03 ± 0.02 s supine, and 0.02 ± 0.02 s right lateral position). In contrast, \( \Delta t \) in OS increased from 0.15 ± 0.09 s in the upright position to 0.22 ± 0.07 s in the supine posture (P < 0.05) and decreased again to 0.14 ± 0.06 s in the right lateral position (P < 0.05, for comparison with supine). Individual values of PEEPi in different body positions are shown in Fig. 4. PEEPi in OS increased from 0.14 ± 0.06 kPa in the upright position to 0.41 ± 0.11 kPa in the supine posture (P < 0.05) and decreased to 0.20 ± 0.08 kPa in the right lateral position (P < 0.05, for comparison with supine), whereas values in CS measured with this method were significantly (P < 0.05) lower: 0.01 ± 0.00 kPa sitting, 0.02 ± 0.02 kPa supine, and 0.01 ± 0.01 kPa lateral position. Inspiratory muscle effort (mean Pdi) increased when OS changed their position from upright (1.02 ± 0.32 kPa) to supine (1.26 ± 0.17 kPa; not significant) and decreased again in the right lateral position: 1.06 ± 0.26 kPa (P < 0.05, compared with supine) (Fig. 5). In contrast, in CS mean Pdi in each position was significantly (P < 0.05) lower: 0.65 ± 0.12 kPa in the upright vs. 0.72 ± 0.15 kPa in the supine vs. 0.62 ± 0.16 kPa in the right lateral position (Fig. 5). There was no significant correlation

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Fig. 2. Flow-volume loops of 1 spontaneous (dashed line) and 1 directly following breath (solid line), during which NEP was applied at onset of expiration (down arrow), are superimposed on 1 graph. Graphs are of 1 representative control subject (subj) and all obese subjects in upright and supine position.
Table 2. Influence of NEP on expiratory flow at mid-tidal volume

<table>
<thead>
<tr>
<th></th>
<th>Sitting</th>
<th>Supine</th>
</tr>
</thead>
<tbody>
<tr>
<td>Subject 1</td>
<td>0.60 ± 0.09</td>
<td>0.49 ± 0.10</td>
</tr>
<tr>
<td>Subject 2</td>
<td>0.80 ± 0.13</td>
<td>0.49 ± 0.15</td>
</tr>
<tr>
<td>Subject 3</td>
<td>0.44 ± 0.05</td>
<td>0.39 ± 0.04</td>
</tr>
<tr>
<td>Subject 4</td>
<td>0.57 ± 0.12</td>
<td>0.26 ± 0.11</td>
</tr>
<tr>
<td>Subject 5</td>
<td>0.37 ± 0.04</td>
<td>0.29 ± 0.10</td>
</tr>
<tr>
<td>Subject 6</td>
<td>0.52 ± 0.10</td>
<td>0.28 ± 0.06</td>
</tr>
<tr>
<td>Mean ± SD</td>
<td>0.55 ± 0.15</td>
<td>0.37 ± 0.11*</td>
</tr>
</tbody>
</table>

Values for individual subjects are means ± SD of 5 experiments with negative expiratory pressure (NEP) of 0.5 kPa. ∆V, expiratory flow at mid-tidal volume compared with control breath. *P < 0.05 vs. sitting. †P < 0.01 vs. normal-weight control subjects.

The main findings of the present studies can be summarized in the following three observations. First, calculations are based on changes in Pes rather than absolute values. Because airway pressure changes equally with inspiratory pressure when the airway is occluded during inspiratory efforts, static PEEP, in this situation gives an estimate of pleural pressure. Using this test, Baydur et al. (3) found that amplitudes in Pes were lower than amplitudes in mouth pressure in the majority of 10 volunteers in the supine posture, when the tip of the esophageal catheter was positioned 10 cm proximal from the cardia. However, differences in pressure swings could be reduced by individually placing the tip of the catheter in a position where cardiac artifacts are minimal. By the use of the same approach in our study, Pes changes were within a 10% range, comparable to changes in Pao. Second, the evaluation of dynamic PEEP, during spontaneous respiration is based on a ∆t of airflow in relation to inspiratory effort (1). This ∆t was also increased with postural change. Therefore, limitations of this study due to inaccurate assessment of pleural pressures should not have substantially affected the estimation of PEEP, and inspiratory muscle effort.

EFL. In the seated position, two OS were classified as flow limited with both methods used, whereas results were discrepant in one other OS. The methodological differences between and consequences of a comparison of tidal and maximal flow-volume curves and the NEP method have been discussed earlier (13). The NEP method is probably superior because both breathing cycles being compared are performed at similar breathing maneuvers.

Until now, EFL as a consequence of obesity has been reported only as an isolated observation in one single subject (13), and to our knowledge there are no existing...
data as to whether recumbency further promotes this phenomenon. Several mechanisms could induce EFL in obesity. Because airflow is dependent on lung volume, any decrease in FRC also decreases expiratory flow reserve. Breathing at low lung volumes may promote airway closure and air trapping (20). This has been demonstrated in subjects breathing at low lung volumes due to chest strapping (5) or voluntarily reducing the breathing level to RV (4). Significant closing volumes and decreased expiratory flows near RV have also been found in a subgroup of healthy subjects whose body weight was higher and RV was lower compared with others (31) and in OS (7). In agreement with earlier studies (21, 25, 36), ERV in OS in this investigation was considerably lower compared with ERV in CS. When OS change their body position from upright to supine, ERV is further reduced to very low values and subjects breathe close to RV (2, 32). Whereas most studies have shown that in supine OS, FRC is lower compared with the upright sitting position (2, 26, 32), one recently published investigation has found only minor differences in FRC, whereas resistance of the respiratory system was significantly increased (35). The authors speculated that the position-related increase in specific resistance either might be a consequence of airway narrowing due to the enhanced increase in intrathoracic blood volume or might be induced by extrathoracic airway narrowing. OSA is associated with pharyngeal narrowing in the awake state.

**Fig. 3.** Records of flow, Pao, esophageal pressure (Pes), gastric pressure (Pga), and transdiaphragmatic pressure (Pdi) in 1 obese subject in supine position. Dotted lines, 0 values; 1st vertical line, start of inspiratory effort (pressure decay of Pes and rise in Pdi); 2nd vertical line, start of inspiratory flow. Fall in Pes during time delay of 2nd vertical line compared with 1st line indicates intrinsic positive end-expiratory pressure (PEEP.).

**Fig. 4.** PEEP during different body positions. For obese subjects (n = 6) individual values and mean values (small solid horizontal lines) are displayed, whereas values in normal-weight control subjects (n = 6) are group means ± SD. *P < 0.001, obese subjects vs. controls. **P < 0.05, supine vs. sitting and supine vs. right lateral.

**Fig. 5.** Inspiratory muscle activity (mean Pdi) during different body positions. Values are means ± SD. ◇, Obese subjects (n = 6); ■, normal-weight control subjects (n = 6). *P < 0.001, obese subjects vs. normal-weight control subjects. **P < 0.05, supine vs. right lateral.
pared with weight-matched CS (29). Because obese individuals with and without OSA were part of our study population, we carefully registered this abnormality by performing full-night-sleep studies. Although airway resistance measured by body plethysmography in the upright position was not different between OS with and without OSA in our study, we cannot exclude differences between both groups in the supine position. EFL, however, was also found in OS without OSA. Thus, although the localization of the flow-limiting segment remains unclear, a specific contribution of OSA in addition to obesity is unlikely.

PEEP$_r$, PEEP$_r$ is a direct consequence of FL and was detected in all of the six OS, in whom the quality of the Pes signal allowed detection of the start of inspiratory muscle action. Also, the observation that PEEP$_r$ always increased when subjects adopted the supine position is in agreement with the observation that FL is promoted by recumbency. In the sitting position, minor PEEP$_r$ was also detected in the absence of FL. Therefore, we had to consider several mechanisms, other than FL, which are known to induce positive alveolar pressure at end expiration (16).

First, PEEP$_r$ can occur without EFL if $T_e$ is too short to enable the pressure of the alveoli to equilibrate with the effective downstream pressure, $T_e$ in OS was considerably shorter compared with CS. On the other hand, in obesity the time constant of the respiratory system is decreased as a consequence of the increased lung and chest wall elastance. Therefore, it is unlikely that the reduced $T_e$ contributes to EFL. We did not observe significant position-related differences in breathing pattern, either.

Second, in acute respiratory failure due to exacerbations of chronic obstructive pulmonary disease, several studies have shown that expiratory muscles might contract at end expiration, the initial decrease in Pes being a consequence of a sudden expiratory muscle relaxation (1, 18). PEEP$_r$, in this situation does not reflect the end-expiratory elastic recoil pressure of the respiratory system. In the studies mentioned, Pga was used to give an estimation of expiratory muscle activity because electromyographic activity of abdominal muscles was concordantly registered with positive Pga swings. Although in obese patients with the obesity hypoventilation syndrome we occasionally observed negative deflections of Pga swings during the initial inspiratory phase (unpublished observations), we have no evidence of expiratory muscle activation at end expiration in the OS included in the present investigation, either in the sitting or supine position.

Third, postinspiratory activity of the inspiratory muscles could impair lung deflation to relaxation volume before the next inspiration starts (28). Persistent postinspiratory diaphragmatic activity in OS has been documented before and was significantly longer compared with in normal-weight controls. In some of the heaviest OS, electromyographic activity of the diaphragm persisted throughout the entire first half of expiration (25). Finally, phase differences between airflow and effort could theoretically be related to an

### Table 4. Ventilatory parameters in different body positions

<table>
<thead>
<tr>
<th>VT, liters</th>
<th>SpO$_2$, %</th>
<th>f, breath/min</th>
<th>$V˙E$, l/min</th>
<th>TI, s</th>
<th>TE, s</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting</td>
<td>Obese subjects (n = 6)</td>
<td>Normal-weight control subjects (n = 6)</td>
<td>Sitting</td>
<td>Lateral</td>
<td>Sitting</td>
</tr>
<tr>
<td>Subject 1</td>
<td>0.84</td>
<td>0.60</td>
<td>0.47</td>
<td>0.45</td>
<td>0.39</td>
</tr>
<tr>
<td>Subject 2</td>
<td>0.57</td>
<td>0.66</td>
<td>0.47</td>
<td>0.45</td>
<td>0.39</td>
</tr>
<tr>
<td>Subject 3</td>
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<tr>
<td>Subject 4</td>
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<td>0.53</td>
<td>0.39</td>
<td>0.32</td>
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<tr>
<td>Subject 5</td>
<td>0.42</td>
<td>0.53</td>
<td>0.39</td>
<td>0.32</td>
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<tr>
<td>Subject 6</td>
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<td>0.53</td>
<td>0.39</td>
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<tr>
<td>Subject 7</td>
<td>0.42</td>
<td>0.53</td>
<td>0.39</td>
<td>0.32</td>
<td>0.29</td>
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<tr>
<td>Subject 8</td>
<td>0.42</td>
<td>0.53</td>
<td>0.39</td>
<td>0.32</td>
<td>0.29</td>
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Mean = SD

Mean $\pm$ SD
increased respiratory system iner-{
ance at high frequencies (27), it is unlikely that this mechanism affected the estimation of PEEP, during tidal breathing in our subjects.

Results of our study imply that, in severe obesity, inspiratory muscles are loaded not only by decreased chest wall compliance (17, 26) but also by the task of counterbalancing PEEPi. PEEP represents an additional elastic load that must be overcome during each inspiratory muscle action. To counterbalance PEEP, inspiratory muscles contract isometrically. Isometric muscle action does not induce airflow and, therefore, is not usually included in calculations of work of breathing. Nevertheless, it represents an active and energy-consuming process of the respiratory muscles.

The authors thank A. Geisler for reviewing the English of the manuscript. They also thank Heinrich Becker and Frank Schütter for help in performing the experiments.

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Received 16 September 1997; accepted in final form 25 May 1998.

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