Pulmonary Physiology and Pathophysiology in Obesity

Respiratory restriction and elevated pleural and esophageal pressures in morbid obesity

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Submitted 10 October 2008; accepted in final form 11 November 2009

Behazin N, Jones SB, Cohen RI, Loring SH. Respiratory restriction and elevated pleural and esophageal pressures in morbid obesity. J Appl Physiol 108: 212–218, 2010. First published November 12, 2009; doi:10.1152/japplphysiol.91356.2008.—To explore mechanisms of restrictive respiratory physiology and high pleural pressure (PPl) in severe obesity, we studied 51 obese subjects (body mass index = 38–80.7 kg/m²) and 10 nonobese subjects, both groups without lung disease, anesthetized, and paralyzed for surgery. We measured esophageal and gastric pressures (PEs, PGa) using a balloon-catheter, airway pressure (PAO), flow, and volume. We compared PEs to another estimate of PPI based on PAO and flow. Reasoning that the lungs would not inflate until PAO exceeded alveolar and pleural pressures (PAO > PAI > PPI), we disconnected subjects from the ventilator for 10–15 s to allow them to reach relaxation volume (VRel) and then slowly raised PAO until lung volume increased by 10 ml, indicating the “threshold PAO” (PAO-Thr) for inflation, which we took to be an estimate of the lowest PAI or PPI to be found in the chest at VRel. PAO-Thr ranged from 0.6 to 14.0 cmH₂O in obese and 0.2 to 0.9 cmH₂O in control subjects. PES at VRel was higher in obese than control subjects (12.5 ± 3.9 vs. 6.9 ± 3.1 cmH₂O means ± SD; P = 0.0002) and correlated with PAO-Thr (R² = 0.16, P = 0.0015). Respiratory system compliance (CCw) was lower in obese than control (0.032 ± 0.008 vs. 0.053 ± 0.007 l/cmH₂O) due principally to lower lung compliance (0.043 ± 0.016 vs. 0.084 ± 0.029 l/cmH₂O) rather than chest wall compliance (obese 0.195 ± 0.109, control 0.223 ± 0.132 l/cmH₂O). We conclude that many severely obese supine subjects at relaxation volume have positive PPI throughout the chest.

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Obesity is a known cause of restrictive respiratory physiology and low lung volumes. Morbidly obese individuals [body mass index (BMI) ≥ 40 kg/m²] have reduced total lung capacity (TLC), functional residual capacity (FRC), and vital capacity (VC) (6, 26). These subjects often have FRC reduced to near residual volume (RV); the reduction of FRC is even greater in the supine position (26). Such reductions in lung volume suggest elevation of pleural pressure (PPI) in morbid obesity.

To date, the studies of respiratory restriction in the obese have focused on changes in PPI that determine chest wall and lung compliance (CCw, CL) rather than estimating PPI itself. Naimark and Cherniack (11) and later Sharp et al. (16) reported that chest wall compliance was significantly reduced in obesity while lung compliance was normal. Subsequent studies by Hedenstierna and Santesson (5) and Suratt et al. (20) reported contrary findings of normal CCw and decreased CL in the obese. More recently, Pelosi et al. (13) found both CCw and CL to be reduced in morbidly obese subjects.

All the aforementioned studies reported changes in esophageal pressures (PES); none reported actual values of PES, presumably due to concern about the validity of esophageal manometry in estimating PPI in the obese. Although esophageal balloon catheters have been used to estimate PPI in normal subjects (3), their use in the obese has not been validated.

In this study we report values of PII as estimated independently by esophageal balloon and by using the relation between the airway pressure (PAO) and lung volume (Vₜ) during passive inflation of the lungs. We studied severely obese subjects and normal-weight controls, anesthetized and paralyzed, and tested the hypotheses that our severely obese subjects had higher PPl than normal subjects and that PES is a useful indicator of PPl in obese subjects. We also related PES to estimated PPI, gastric pressure (PGa), and BMI and studied the compliance of the respiratory system (CCw, CCw, CL).

SUBJECTS AND METHODS

Subjects. Fifty morbidly obese subjects and one moderately obese subject, which we will collectively call “severely obese subjects,” and 10 nonobese subjects (BMI < 30), all with healthy lungs, were studied immediately after induction of general anesthesia for elective surgery at Beth Israel Deaconess Medical Center. The severely obese subjects had BMI ranging from 38.0 to 80.7 kg/m² (48.5 ± 8.9, mean ± SD) and were aged 20 to 69 yr (43.8 ± 11.9 yr), and 33 were female (see Supplement available with the online version of this article). Ten control subjects had BMI ranging from 19.2 to 29 kg/m² (25.2 ± 2.8 kg/m²) and were aged 21 to 61 yr (44.7 ± 11.4 yr), and 8 were female. Two additional subjects originally recruited as controls were found to have mild obesity, BMI slightly above 30 kg/m²; they were not included in the analysis but are shown in the figures with distinct symbols. The research was approved by the Committee on Clinical Investigations, and subjects gave informed written consent before study.

Measurements. Subjects were studied before surgical incision and immediately after induction of general anesthesia including complete muscle relaxation using vecuronium with or without succinylcholine. Subjects were supine and mechanically ventilated with 100% O₂ with tidal volumes of 0.64 ± 0.11 liters (Supplemental Table S-1, available with the online version of this article) and no preset positive end-expiratory pressure (PEEP), although a small PEEP was present in all subjects due to ventilator characteristics. PGa and PES were measured using an adult esophageal balloon catheter (Ackrad Laboratories), passed by mouth or nose to position the balloon’s tip at 60 cm from...
the incisors to measure $P_{es}$ and then withdrawn to 40 cm to measure $P_{es}$. (In 25 subjects in whom it was not possible to advance the balloon into the stomach, $P_{es}$ was not measured.) The 10-cm-long balloon was inflated with 0.5–1.0 ml of air. $P_{ao}$ was measured at the endotracheal tube, and airflow was measured with a pneumotachometer (Fleisch pneumotachograph type 1, Metabo SA). Signals were digitized, displayed, and recorded using Windaq software and hardware (Dataq Instruments, Akron, OH).

Threshold inflation airway pressure, $P_{ao-thr}$. A major aim of this study was to estimate the distribution of pleural pressure surrounding the lungs. To do this, we inferred $P_{pl}$ from the airflow pressure needed to cause lung inflation. We reasoned that in a relaxed intubated subject with open airways, if the highest $P_{pl}$ to be found in the chest is subatmospheric, then inflation should begin immediately as the airway pressure ($P_{ao}$) is raised above atmospheric pressure. By contrast, if the lowest $P_{pl}$ to be found in the chest is above atmospheric pressure, the lungs would not begin to inflate until $P_{ao}$ is raised above that lowest $P_{pl}$ (where the local transpulmonary pressure would first become positive). Therefore, the $P_{ao}$ at which the lung volume first begins to increase is an approximate indicator of the lowest $P_{pl}$ to be found in the chest at relaxation volume ($V_{rel}$).

We determined the “threshold $P_{ao}$” ($P_{ao-thr}$), the $P_{ao}$ required to initiate lung inflation during the slow increase in $P_{ao}$ from relaxation volume. To prevent noise in the flow signal from being interpreted as the start of inspiratory flow we used to needed a 10 ml increase in lung volume as our criterion for the start of inflation (Fig. 1). If there were no pulmonary resistive or elastic pressures to be overcome during a 10 ml inflation, $P_{ao-thr}$ would be zero when the lowest $P_{pl}$ to be found in the chest at $V_{rel}$ was negative, and $P_{ao-thr}$ would be equal to the lowest $P_{pl}$ to be found in the chest at $V_{rel}$ when the lowest $P_{pl}$ was positive. Under these theoretical conditions, $P_{ao-thr}$ would be an upper bound of the lowest $P_{pl}$ to be found in the chest at $V_{rel}$.

In reality, pulmonary resistance and lung elastance would raise the measured $P_{ao-thr}$ above atmospheric pressure even with all the intrapulmonary airways open and $P_{pl}$ subatmospheric everywhere in the chest at $V_{rel}$. To estimate how much $P_{ao-thr}$ would be increased in such circumstances, we used a computational model to calculate the volume-time course of inflation, assuming that $P_{ao}$ increased at a constant rate, alveolar pressure ($P_{alv}$) = $\Delta V/C_{rs}$, and inspiratory flow rate = ($P_{ao} - P_{alv}$)/$R_{rs}$ resistance. We assumed a relatively high rate of increase in $P_{ao}$ of 4 cmH2O/s, a high $R_{rs}$ of 15 cmH2O·1·s$^{-1}$, and a low $C_{rs}$ of 0.02 l/cmH2O to conservatively estimate a high value of $P_{ao-thr}$ due to resistance and elastance, namely 1.3 cmH2O (See Supplement, Fig S-1, available with the online version of this article). With these assumptions, $P_{ao-thr}$ would be <1.3 cmH2O if most of the airways in the lung were open and $P_{pl}$ surrounding most of the lung were positive and/or if $P_{pl}$ surrounding most of the lung was above atmospheric pressure. Higher $P_{ao-thr}$ values, by inference, would indicate commensurately higher $P_{pl}$ surrounding most of the lung at $V_{rel}$.

Protocol. The protocol is illustrated in Fig. 1A. We measured $P_{ga}$ and $P_{es}$ during five or more tidal mechanical breaths. Then we disconnected the subject from the ventilator for 10–15 s to allow deflation to $V_{rel}$, connected an Ambu bag to the endotracheal tube, and squeezed it to increase $P_{ao}$ gradually to 1–3 cmH2O (mean 1.5 cmH2O) until the bag was nearly empty. The deflation to $V_{rel}$ and subsequent quasi-static inflation for determination of $P_{ao-thr}$ were repeated three times with at least one intervening minute of mechanical ventilation (Fig. 1A).

Analysis. We measured $P_{ao}$ and $P_{es}$ and $P_{ga}$ at end expiration ($P_{ao-ee}$, $P_{es-ee}$, $P_{ga-ee}$) and at end inspiration ($P_{ao-ei}$, $P_{es-ei}$, $P_{ga-ei}$) averaged over five consecutive tidal breaths. We also measured the $P_{es}$ at $V_{rel}$ ($P_{es-rel}$). Dynamic compliance of the respiratory system ($C_{rs}$) was defined as average tidal volume change ($\Delta V$) divided by the average difference in $P_{ao}$ from end inspiration to end expiration at instants of zero flow: $C_{rs} = \Delta V/\Delta P_{ao}$. Similarly, the dynamic compliance of the lung ($C_{L}$) was defined as $C_{L} = \Delta V/\Delta P_{L}$, where $P_{L}$ = transpulmonary pressure = $P_{ao} - P_{pl}$, and dynamic compliance of the chest wall ($C_{cw}$) as $C_{cw} = \Delta V/\Delta P_{cw}$.

To explore the mechanism leading to high $P_{ao-thr}$, we analyzed data obtained during inflation of the lung with the Ambu bag. We reasoned that in subjects with low $P_{ao-thr}$, the
Fig. 1. A: typical data from one experiment, illustrating the protocol (see text for explanation), showing airway pressure (P_AO), gastric pressure (P_Ga), and esophageal pressure (P_es). Note the pressure rise when the balloon is withdrawn from stomach into the lower esophagus. B: third quasi-static inflation in A with expanded time scale and volume channel added. The gradual rise in P_AO above the threshold airway pressure (P_AO-Thr) causes lung inflation. P_AO-Thr is 7.3 cmH2O in this example. (The threshold volume change of 10 ml is not visible at this scale.)

Inflation would begin with open airways, and the quasi-static inflation P_AO-V_L curve would be relatively linear, whereas in those obese subjects with high P_AO-Thr, inflation would begin with many airways closed, and the quasi-static inflation P_AO-V_L curve would exhibit the effects of recruitment after prior airway closure. Comparing the quasi-static inflation P_AO-V_L curves from the obese subjects with the five highest and five lowest P_AO-Thr values, we found that in the lowest P_AO-Thr group (Fig. 6A), the inflation P_AO-V_L curves appear almost linear above V_Rel, consistent with open airways in a
of PEs in supine subjects have ignored the resting “baseline” PEs-Rel and PAO-Thr suggests that both measurements can be line PEs in conscious obese subjects, but most previous studies analyzed obese subjects. Steier et al. (18) reported elevated baseline exhalation. By contrast, in the highest PAO-Thr group (Fig. 6b), obese subjects at VRel, whether estimated using PEs or inferred the first report of resting values of PEs in anesthetized, paralyzed obese subjects. Steier et al. (18) reported elevated baseline PEs in conscious obese subjects, but most previous studies of PEs in supine subjects have ignored the resting “baseline” values of PEs, reporting only changes in PEs.

This is also the first study to estimate PPl in obese subjects by a method independent of PEs. The correlation between PEs-Rel and PAO-Thr suggests that both measurements can be used to estimate the likelihood of high effective PPl in obese supine subjects. However, although PEs and PAO-Thr are correlated, they differ substantially and variably. What could explain this difference? First, PEs is thought to be higher than PPl in the supine posture (7, 25). The esophagus is roughly midway between the ventral and dorsal boundaries of the thoracic cavity, and in the supine posture, there is a hydrostatic increase in pressure within the mediastinum due to the weight of the heart and mediastinal tissues above. Therefore, PEs is probably higher than PPl surrounding the lung at the same height in thorax, a discrepancy estimated to be ~5 cmH2O (25). Second, the hydrostatic pressure gradient in the pleural space could cause PAO-Thr (i.e., the lowest pleural pressure surrounding a substantial portion of the lung at VRel plus any opening pressure. In summary, PAO-Thr and PEs-Rel are correlated, although hydrostatic pressure gradients in the mediastinum and pleural space and the opening pressure of the collapsed airways weaken the correlation.

The second major finding is that, compared with control subjects, our severely obese subjects are characterized by lower CRS and CL, whereas C CW is usually normal. This finding differs from those of Naimark and Cherniak (11) and Sharp et al. (16), who measured compliance while subjects breathed spontaneously or relaxed respiratory muscles voluntarily. Both studies had reported normal CL with low C CW in obesity. However, more recent studies during muscle paralysis (5) or with confirmed diaphragm relaxation (20) reported normal C CW in obesity, suggesting that differences in C CW were due to the experimental method. Van Lith et al. (23) investigated this possibility by comparing elastance in six healthy obese subjects both during voluntary muscle relaxation and during anesthesia with paralysis. They found higher E RS and E C W during voluntary relaxation than during paralysis, suggesting that incomplete relaxation in early studies could have contributed to lower C CW in the obese (23). By exception, Pelosi et al. (13) found both C CW and CL to be significantly lower in anesthetized, paralyzed, morbidly obese subjects studied after major abdominal surgery.

It is possible that ventilation with 100% oxygen in our study could have contributed to development of atelectasis that reduced CL in the obese group (4). CL was not reduced in the control group, suggesting high PPl and consequent small airway closure would be required for the formation of atelectasis in obese subjects. Furthermore, the short duration of ventilation required to open small airways closed by a meniscus (2). The opening pressure of healthy human airways is unknown, but opening pressure has been estimated to be in the range of 3 cmH2O in excised rat lungs (2), and it may not be much higher in healthy lungs that have not developed widespread atelectasis. In this study, when PAO-Thr is >1.3 cmH2O, PAO-Thr is an estimate of the minimum pleural pressure surrounding a substantial portion of the lung at VRel plus any opening pressure.

DISCUSSION

The major finding of this study is that pleural pressure in obese subjects at VRel, whether estimated using PEs or inferred from airway pressure and flow measurements, is greater than normal, often substantially above atmospheric pressure. This is the first report of resting values of PEs in anesthetized, paralyzed obese subjects. Steier et al. (18) reported elevated baseline PEs in conscious obese subjects, but most previous studies of PEs in supine subjects have ignored the resting “baseline” values of PEs, reporting only changes in PEs.

This is also the first study to estimate PPl in obese subjects by a method independent of PEs. The correlation between PEs-Rel and PAO-Thr suggests that both measurements can be used to estimate the likelihood of high effective PPl in obese supine subjects. However, although PEs and PAO-Thr are correlated, they differ substantially and variably. What could explain this difference? First, PEs is thought to be higher than PPl in the supine posture (7, 25). The esophagus is roughly midway between the ventral and dorsal boundaries of the thoracic cavity, and in the supine posture, there is a hydrostatic increase in pressure within the mediastinum due to the weight of the heart and mediastinal tissues above. Therefore, PEs is probably higher than PPl surrounding the lung at the same height in thorax, a discrepancy estimated to be ~5 cmH2O (25). Second, the hydrostatic pressure gradient in the pleural space could cause PAO-Thr (i.e., the lowest pleural pressure surrounding a large part of the lung) to be lower than PPl at mid-lung height. Even when PPl at mid-lung height is positive (presumably causing airway closure and positive pressures in the dependent alveoli), PPl may be negative in nondependent lung regions, where open airways and atmospheric alveolar pressures at VRel enable PAO-Thr values to be low. Third, when widespread positive PPl at VRel causes closure of most of the small airways, PAO-Thr will be higher than PPl by the opening pressure re-

Fig. 2. PEs at relaxation volume (PEs-Rel) plotted against PAO-Thr, with line of regression ($R^2 = 0.158$).

Fig. 3. PPl at end expiration and end inspiration plotted against the corresponding PEs, with regression lines showing correlations ($R^2 = 0.39$, $P < 0.0001$, and $R^2 = 0.39$, $P < 0.0001$, respectively). The majority of subjects have above-normal intra-abdominal pressure (IAP) (PPl at end expiration ≥ 10 cmH2O).
on 100% oxygen (~10 min) argues against the primary role of atelectasis in reducing $C_L$ in the obese.

Another possibility is that $C_L$ is lower in obese subjects because the positional artifact that increases $P_{Es}$ in supine subjects is volume dependent (7). The importance of this artifact is said to decrease during inflation because upward movement of the ventral thorax lifts the heart and mediastinal contents, which no longer rest as heavily on the esophagus and balloon (7). However, the artifactual reduction in $P_{Es}$ during inflation would cause the apparent elastance of the lung to increase and that of the chest wall to decrease by the same amount, so the sum of these elastances, which is $E_{RS}$, would not be altered. Therefore, the strong correlation of respiratory system elastance, which is independent of $P_{Es}$, with lung elastance ($R^2 = 0.8962$) argues against this explanation and suggests that the increase in $E_{RS}$ is caused by a real increase in $E_L$. In addition, the above artifact could cause a downward concavity of the $P_{Es}-V_L$ curve as the positive pressure artifact in $P_{Es}$ decreased during inflation (see rationale and depiction in Ref. 7). We examined the quasi-static inflation curves of chest wall ($P_{Es}-V_L$ curves) in those subjects pictured in Fig. 6B and found no hint of the decreasing compliance that might be predicted by this mechanism (7).

The fact that $C_{CW}$ is normal in obese subjects is consistent with “mass loading” of the chest wall rather than “stiffening of the chest” by elastic loading, as demonstrated experimentally by Sharp et al. (15).

**Model of P-V curves in obesity.** Another mechanism whereby high $P_{PT}$ could cause low compliance of the lung and respiratory system is shown schematically in Fig. 7. In Fig. 7A, pressure-volume (P-V) curves are drawn for a normal lung and chest wall, and the respiratory system P-V curve was constructed by adding the pressures across lung and chest wall. FRC (taken as equal to $V_{Rel}$) is the volume at which lung and chest wall recoil pressures are equal and opposite. In this depiction, the normal lung is shown as having a region of low compliance and stiffening at low volumes where airway closure occurs at negative transpulmonary pressures. This region of the inflation P-V curve of normal lungs is seldom observed in vivo, because normally transpulmonary pressures are not statically negative, especially in upright subjects (14). However, this behavior is observed routinely in experimental preparations, for example when excised lungs are exposed to sustained negative $P_{AO}$ (24). Stiffening of the lungs at very low volumes has also been demonstrated in healthy breath-hold divers after they empty their lungs below residual volume by employing glossopharyngeal exsufflation (8).

Figure 7B illustrates the possible effects of an increase in extrapulmonary volume, for example fat, within the chest wall or “mass loading” (15). The chest wall P-V curve as drawn exhibits higher pressures at any lung volume without being stiffer (i.e., without a decrease in slope), consistent with our observations of normal $C_{CW}$ and high $P_{Es}$ in many obese subjects. The volumes of intra-abdominal and mediastinal fat increase in obesity (9), which could cause the chest wall to contain more volume and the lung to be smaller than normal at $V_{Rel}$, making the pleural pressure more positive. Since the normal chest wall curve stiffens at low volumes, largely due to developing tension in the diaphragm (17), the chest wall curve becomes more compliant as chest wall volume increases. Thus increasing the volume of fat within the chest wall could

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**Table 1. Range and mean values of $C_{RS}$, $C_{CW}$, and $C_L$ in severely obese and control subjects**

<table>
<thead>
<tr>
<th></th>
<th>$C_{RS}$, l/cmH$_2$O</th>
<th>$C_{CW}$, l/cmH$_2$O</th>
<th>$C_L$, l/cmH$_2$O</th>
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<tbody>
<tr>
<td>Control group (BMI &lt; 30 kg/m$^2$)</td>
<td>0.041–0.063 (0.053 ± 0.007)</td>
<td>0.086–0.539 (0.223 ± 0.132)</td>
<td>0.044–0.142 (0.0.084 ± 0.029)</td>
</tr>
<tr>
<td>Severely obese group</td>
<td>0.017–0.054 (0.032 ± 0.008)</td>
<td>0.072–0.554 (0.195 ± 0.109)</td>
<td>0.018–0.090 (0.043 ± 0.016)</td>
</tr>
<tr>
<td>$P$ value (unpaired t-test)</td>
<td>$P &lt; 0.00001$</td>
<td>$P = 0.5284$</td>
<td>$P &lt; 0.0016$</td>
</tr>
</tbody>
</table>

Values are ranges (means ± SD). $C_{RS}$, respiratory system compliance; $C_{CW}$, chest wall compliance; $C_L$, lung compliance; BMI, body mass index.
actually increase its compliance over much of the vital capacity.

In the resulting respiratory system P-V curve in Fig. 7B, FRC has decreased to a lung volume near the former RV, where the lung’s P-V curve is stiffened by small airway closure, a phenomenon that causes the respiratory P-V curve to exhibit a downward convexity (24) similar to that observed by Pelosi et al. (13) in anesthetized obese subjects.

This theoretical analysis is consistent with the quasi-static inflation $P_{AO}$-V$_L$ curves of the obese subjects with the five highest and five lowest $P_{AO}$-Thr values (Fig. 6). In the lowest $P_{AO}$-Thr group, the inflation $P_{AO}$-V$_L$ curves were nearly linear above $V_{Rel}$, consistent with most of the airways being open and $P_{Pl}$ surrounding most of the lung being at or below atmospheric pressure (Fig. 6A). Increasing airway pressure caused inflation. By contrast, in the group with highest $P_{AO}$-Thr, the $P_{AO}$-V$_L$ curves have a “knee” or region of downward convexity above $V_{Rel}$, consistent with pleural pressure surrounding most of the lung being above atmospheric pressure, causing closure of most small airways at $V_{Rel}$. As $P_{AO}$ increased, initially there was little inflation until the airways opened and positive pleural pressure was exceeded, enabling progressive recruitment of lung regions until the $P_{AO}$-V$_L$ curve followed a linear trajectory similar to those in the low $P_{AO}$-Thr group.

During spontaneous breathing, positive end-expiratory alveolar pressure, known as intrinsic PEEP (PEEP$_i$), is greater in obese subjects and is increased by the supine posture (12). PEEP$_i$ constitutes a threshold load for inspiration, increasing the work of breathing and increasing the dynamic elastance of the lung during tidal ventilation. Our findings that dynamic $E_L$ and $E_{RS}$ were positively correlated with $P_{Es-Rel}$ and $P_{Ga-EE}$ suggest that high pleural pressures in the obese reduce the FRC, lowering expiratory flow rates, increasing PEEP$_i$, and reducing the measured dynamic lung compliance.

As expected, P$_{Ga}$ was elevated in most of our obese subjects. Assuming the equivalence of P$_{Ga}$ and bladder pressure in assessing intra-abdominal pressure (IAP) (1), and using reference values of the World Congress on Abdominal Compartment Syndrome (10), we found 23 of 30 obese subjects (76%) had IAP greater than normal (P$_{Ga}$ = 10 cmH$_2$O), and three of them (all female) had IAP in the range of abdominal hypertension (P$_{Ga}$ = 16.3 cmH$_2$O).

P$_{Ga}$ and P$_{Es}$ were correlated at end expiration and end inspiration in all subjects (Fig. 3) and in the obese group, as was reported in ventilated patients by Talmor et al. (21). However, the correlation is not very strong for several reasons. The distal esophagus is lower than the gastric fundus in supine subjects, causing a gravitational pressure gradient that raises...
P_{Es} relative to P_{Ga} to differing degrees among subjects. In addition, there may be passive tension in the diaphragm in some subjects reducing P_{Es} relative to P_{Ga}. Finally, the aforementioned effects of the mediastinal contents pressing on the esophagus contribute variability and weaken the correlation between P_{Es} and P_{Ga}.

In obese and control subjects combined, there was a weak correlation of P_{AO-Thr} with BMI and a stronger correlation of P_{Es} with BMI. However, contrary to expectation, we found no correlation between BMI and P_{AO-Thr} (Fig. 5) or P_{Es} in our severely obese subjects, suggesting that obesity, per se, is not predictive of high pleural pressure. The question whether the pattern of obesity (abdominal vs. peripheral) might affect the elevation of P_{Pl} was tested in the final 13 obese subjects in whom we measured waist circumference (WC) and sagittal abdominal diameter (SAD), the anthropometric correlates of intra-abdominal fat volume and the intra-abdominal pressure (19, 22). Neither WC nor SAD was correlated with P_{AO-Thr} or P_{Es-Rel} in this cohort, nor was there any sex-based difference in the levels of P_{AO-Thr} and/or P_{Es-Rel} in obese subjects.

In conclusion many morbidly obese supine subjects appear to have high, often positive pleural pressure surrounding most of the lung while relaxed at V_{Rel}. High pleural pressure would cause tidal breathing to be initiated from low end-expiratory volumes where the lungs are less compliant and airways are prone to close on exhalation. This finding is variable and not predictable from BMI, WC, or SAD. High esophageal pressure is a useful, if imperfect, indicator of the likelihood of high pleural pressure in these subjects.

ACKNOWLEDGMENTS

We thank Lauren Marquis, Dr. Carlos Puyo, Dr. Vimal Akhouri, and Beth Israel Deaconess Medical Center surgeons for help in recruiting subjects and acquiring data.

GRANTS

This work was supported by grant HL-52586 from the National Institutes of Health.

DISCLOSURES

None of the authors has any financial interest that could be affected by the subject of this report. No conflicts of interest are declared by the authors.

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