Respiratory restriction and elevated pleural and esophageal pressures in morbid obesity

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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 (P_pl) 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 (P Es, P Ga) using a balloon-catheter, airway pressure (P Ao), flow, and volume. We compared P Es to another estimate of P_pl based on P Ao and flow. Reasoning that the lungs would not inflate until P Ao exceeded alveolar and pleural pressures (P Ao > P Alv > P pl), we disconnected subjects from the ventilator for 10–15 s to allow them to reach relaxation volume (V_rel) and then slowly raised P Ao until lung volume increased by 10 ml, indicating the “threshold P Ao” (P Ao-th) for inflation, which we took to be an estimate of the lowest P Alv or P pl to be found in the chest at V_rel. P Ao-th ranged from 0.6 to 14.0 cm H₂O in obese and 0.2 to 0.9 cm H₂O in control subjects. P Es at V_rel was higher in obese than control subjects (12.5 ± 3.9 vs. 6.9 ± 3.1 cm H₂O, means ± SD; P = 0.0002) and correlated with P Ao-th (R² = 0.16, P = 0.0015). Respiratory system compliance (C Es) was lower in obese than control subjects (0.032 ± 0.008 vs. 0.053 ± 0.007 l/cm H₂O) due principally to lower lung compliance (0.043 ± 0.016 vs. 0.084 ± 0.029 l/cm H₂O) rather than chest wall compliance (obese 0.195 ± 0.109, control 0.223 ± 0.132 l/cm H₂O). We conclude that many severely obese supine subjects at relaxation volume have positive P_pl throughout the chest. High P Es suggests high P_pl in such individuals. Lung and respiratory system compliances are low because of breathing at abnormally low lung volumes.

esophageal pressure; compliance; elastance; gastric pressure; pressure-volume curve

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 (P_pl) in morbid obesity.

To date, the studies of respiratory restriction in the obese have focused on changes in P_pl that determine chest wall and lung compliance (Ccw, Cl) rather than estimating P_pl 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 (P Es); none reported actual values of P Es, presumably due to concern about the validity of esophageal manometry in estimating P_pl in the obese. Although esophageal balloon catheters have been used to estimate P_pl in normal subjects (3), their use in the obese has not been validated.

In this study we report values of P_pl as estimated independently by esophageal balloon and by using the relation between the airway pressure (P Ao) and lung volume (Vl) 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 P_pl than normal subjects and that P Es is a useful indicator of P_pl in obese subjects. We also related P Es to estimated P_pl, gastric pressure (P Ga), and BMI and studied the compliance of the respiratory system (C Es), Ccw, and Cl.

HIGHLIGHTED TOPIC | Pulmonary Physiology and Pathophysiology in Obesity

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 ± 2.9 kg/m²) and were aged 21–61 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. P Ga and P Es 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 PaO and then withdrawn to 40 cm to measure PaO. (In 25 subjects in whom it was not possible to advance the balloon into the stomach, PaO was not measured.) The 10-cm-long balloon was inflated with 0.5–1.0 ml of air. PaO 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, PAO-Thr. A major aim of this study was to estimate the distribution of pleural pressure surrounding the lungs. To do this, we inferred Pn from the airway pressure needed to cause lung inflation. We reasoned that in a relaxed intubated subject with open airways, if the highest Pn to be found in the chest is subatmospheric, then inflation should begin immediately as the airway pressure (PaO) is raised above atmospheric pressure. By contrast, if the lowest Pn to be found in the chest is above atmospheric pressure, the lungs would not begin to inflate until PaO is raised above that lowest Pn (where the local transpulmonary pressure would first become positive). Therefore, the PaO at which the lung volume first begins to increase is an approximate indicator of the lowest Pn to be found in the chest at relaxation volume (VRel).

We determined the “threshold PaO” (PaO-Thr), the PaO required to initiate lung inflation during the slow increase in PaO from relaxation volume. To prevent noise in the flow signal from being interpreted as the start of inspiratory flow we needed to use 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, PaO-Thr would be zero when the lowest Pn to be found in the chest at VRel was negative, and PaO-Thr would be equal to the lowest Pn to be found in the chest at VRel when the lowest Pn was positive. Under these theoretical conditions, PaO-Thr would be an upper bound of the lowest Pn to be found in the chest at VRel.

In reality, pulmonary resistance and lung elastance would raise the measured PaO-Thr above atmospheric pressure even with all the intrapulmonary airways open and Pn subatmospheric everywhere in the chest at VRel. To estimate how much PaO-Thr would be increased in such circumstances, we used a computational model to calculate the volume-time course of inflation, assuming that PaO increased at a constant rate, alveolar pressure (PAlv) = ΔV/CRS, and inspiratory flow rate = (PaO – PaO)/RRS, where ΔV is the volume increment above VRel and RRS is respiratory resistance. We assumed a relatively high rate of increase in PaO = 4 cmH2O/s, a high RRS = 15 cmH2O-1·s-1, and a low CRs = 0.02 l/cmH2O to conservatively estimate a high value of PaO-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, PaO-Thr would be <1.3 cmH2O if most of the airways in the lung were open and PaO surounding most of the lung at VRel was at atmospheric pressure or below. Conversely, PaO-Thr would be >1.3 cmH2O if most of the airways in the lung were closed (presumably due to positive pleural pressures at VRel) and/or if PaO surrounding most of the lung was above atmospheric pressure. Higher PaO-Thr values, by inference, would indicate commensurately higher Pn surrounding most of the lung at VRel.

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

Analysis. We measured PAO and Pn and PGa at end expiration (PaO-EE, Pn-EE, PGa-EE) and at end inspiration (PaO-EE, Pn-EE, PGa-EE) averaged over five consecutive tidal breaths. We also measured the Pn at VRel (PaO-Rel). Dynamic compliance of the respiratory system (CRS) was defined as average tidal volume change (ΔV/CRS) divided by the average difference in PaO from end inspiration to end expiration at instants of zero flow: CRS = ΔV/ΔPAO. Similarly, the dynamic compliance of the lung (CLS) was defined as CLS = ΔV/ΔPL, where PL = transpulmonary pressure = PAO – Pn, and dynamic compliance of the chest wall (Ccw) as Ccw = ΔV/ΔPEs.

To explore the mechanism of obesity-related restriction, we compared the elastance of the respiratory system (ERs = 1/CCW) with its additive components, lung and chest wall elastance (EL = 1/CCW). We reasoned that in subjects with low PaO-Thr, the

RESULTS

PaO-Thr ranged from 0.6 to 14.0 cmH2O (3.1 ± 3.0 cmH2O) in the obese group and from 0.2 to 0.9 cmH2O (0.4 ± 0.2 cmH2O) in the control group (P < 0.0001). In most of our obese subjects (21 female and 14 male), PaO-Thr was above 1.3 cmH2O indicating that most of the airways were closed and/or Pn surrounding most of the lung was above atmospheric pressure at VRel. By contrast, all control subjects had PaO-Thr below 1.3 cmH2O, indicating that most of the airways were open and Pn was subatmospheric surrounding most of the lung at VRel.

PaO-Thr ranged from 3.0 to 25.7 cmH2O (12.5 ± 9.2 cmH2O) in the severely obese group and from 0.7 to 12.2 cmH2O (6.9 ± 3.1 cmH2O) in the control group (P < 0.0001). In all subjects, there was a significant correlation between PaO-Thr and PaO-EE (R2 = 0.16, P = 0.0015, Fig. 2) and a weak correlation in the obese group analyzed separately (R2 = 0.086, P = 0.0363).

In the 30 obese subjects in whom it was possible to pass the balloon into the stomach, PaO-EE ranged from 6.7 to 17.0 cmH2O (11.5 ± 2.8 cmH2O). PaO and PGa-EE were well correlated in all subjects at end expiration and end inspiration (R2 = 0.44, P < 0.0001 and R2 = 0.39, P < 0.0001 respectively, Fig. 3) and in the obese group analyzed separately (R2 = 0.17, P = 0.0225 and R2 = 0.13, P = 0.0470).

CRs in the severely obese group was lower than in the controls (P < 0.0001, Table 1). CCW in the obese group was not statistically different from values in the control group (Table 1), whereas EL was substantially lower in obese than controls (P = 0.0016, Table 1). Respiratory system elastance (ERS), which is the sum of EL and ECW, was strongly correlated with EL in all subjects (R2 = 0.91, P < 0.0001, Fig. 4) and in the obese group (R2 = 0.90, P < 0.0001), whereas ERS was not significantly correlated with ECW. In all subjects, neither PaO-Thr nor PaO-EE was correlated with ECW. However, both PaO-Thr and PaO-EE were positively correlated with both EL and ERS (R2 = 0.17, P ≤ 0.0092).

In all subjects, BMI was positively correlated with ERS (R2 = 0.23, P < 0.0001) and EL (R2 = 0.20, P < 0.0003) but not with ECW. In the obese subjects, neither PaO-Thr (Fig. 5) nor PaO-Rel was correlated with BMI. When the BMI range was expanded to include the controls, a weak correlation was found for PaO-Thr (R2 = 0.068, P = 0.0425) and a stronger correlation for PaO-Rel (R2 = 0.26, P < 0.0001).

To explore the mechanism leading to high PaO-Thr, we analyzed data obtained during inflation of the lung with the Ambu bag. We reasoned that in subjects with low PaO-Thr, the
inflation would begin with open airways, and the quasi-static inflation \( P_{AO\cdot VL} \) curve would be relatively linear, whereas in those obese subjects with high \( P_{AO\cdot Thr} \), inflation would begin with many airways closed, and the quasi-static inflation \( P_{AO\cdot VL} \) curve would exhibit the effects of recruitment after prior airway closure. Comparing the quasi-static inflation \( P_{AO\cdot VL} \) curves from the obese subjects with the five highest and five lowest \( P_{AO\cdot Thr} \) values, we found that in the lowest \( P_{AO\cdot Thr} \) group (Fig. 6A), the inflation \( P_{AO\cdot VL} \) curves appear almost linear above \( V_{Rel} \), consistent with open airways in a

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**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 (\( P_{AO\cdot Thr} \)) causes lung inflation. \( P_{AO\cdot Thr} \) is 7.3 cmH2O in this example. (The threshold volume change of 10 ml is not visible at this scale.)
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 base-exhalation. By contrast, in the highest PAO-Thr group (Fig. 6 obese subjects at VRel, whether estimated using PEs or inferred the first report of resting values of PEs in anesthetized, para-normal, often substantially above atmospheric pressure. This is from airway pressure and flow measurements, is greater than

**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-

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. 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 Ctl, whereas Ccw 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 Ctl with low Ccw in obesity. However, more recent studies during muscle paralysis (5) or with confirmed diaphragm relaxation (20) reported normal Ccw in obesity, suggesting that differences in Ccw 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 Ers and Eccw during voluntary relaxation than during paralysis, suggesting that incomplete relaxation in early studies could have contributed to lower Ccw in the obese (23). By exception, Pelosi et al. (13) found both Ccw and Ctl 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 Ctl in the obese group (4). Ctl 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...
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 $P_E$ 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 $P_E$ 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 $P_E$, 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 $P_E$-$V_L$ curve as the positive pressure artifact in $P_E$ decreased during inflation (see rationale and depiction in Ref. 7). We examined the quasi-static inflation curves of chest wall ($P_{E}$-$V_L$ 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 $P_P$ 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 $P_E$ 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

![Fig. 4. Dynamic respiratory system elastance ($E_{RS}$) plotted against dynamic lung elastance ($E_L$). $E_{RS}$ was principally determined by $E_L$ in our severely obese subjects ($P < 0.0001$).](image)

![Fig. 5. $P_{AO}$-$Thr$ plotted against body mass index (BMI). $P_{AO}$-$Thr$ was not correlated with BMI in our obese subjects ($R^2 = 0.0002$) but was weakly correlated when the control group was included in the regression ($R^2 = 0.068$). (There are superimposed symbols in the control group.)](image)
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 PAO-VL curves of the obese subjects with the five highest and five lowest PAO-Thr values (Fig. 6). In the lowest PAO-Thr group, the inflation PAO-VL curves were nearly linear above VRel, consistent with most of the airways being open and PPl 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 PAO-Thr, the PAO-VL curves have a “knee” or region of downward convexity above VRel, consistent with pleural pressure surrounding most of the lung being above atmospheric pressure, causing closure of most small airways at VRel. As PAO increased, initially there was little inflation until the airways opened and positive pleural pressure was exceeded, enabling progressive recruitment of lung regions until the PAO-VL curve followed a linear trajectory similar to those in the low PAO-Thr group.

During spontaneous breathing, positive end-expiratory alveolar pressure, known as intrinsic PEEP (PEEPi), is greater in obese subjects and is increased by the supine posture (12). PEEPi 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 EL and ERS were positively correlated with PEx-Rel and PGA-EE suggest that high pleural pressures in the obese reduce the FRC, lowering expiratory flow rates, increasing PEEPi, and reducing the measured dynamic lung compliance.

As expected, PGA was elevated in most of our obese subjects. Assuming the equivalence of PGA 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 (PGA > 10 cmH2O), and three of them (all female) had IAP in the range of abdominal hypertension (PGA > 16.3 cmH2O).

PGA and PEx 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 PGA and PEx at end expiration, and the position of the gastric mucosa can change with respiration, altering PGA and PEx at the time of measurement.
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.

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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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