Effect of the chest wall on pressure–volume curve analysis of acute respiratory distress syndrome lungs*

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Objective: **Previously published methods to assess the chest wall effect on total respiratory system pressure-volume (P-V) curves in acute respiratory distress syndrome have been performed on the lung and chest wall in isolation. We sought to quantify the effect of the chest wall by considering the chest wall and lung in series.**

Design: **Prospective study.**

Setting: **Academic health center medical and surgical intensive care units.**

Patients: **Twenty-two patients with acute respiratory distress syndrome/acute lung injury.**

Interventions: **Using a sigmoidal equation, we fit the pressure–volume data of the lung alone, and defined for each curve the pressure at the point of maximum compliance increase (P_{mci}), decrease (P_{mcd}), and the point of inflection (P_{inf}). We calculated the pressure to which the total respiratory system must be inflated to achieve a volume that would place the lung at each point of interest. We compared these "corrected" pressures ($P_{mci,c}$, $P_{mcd,c}$, and $P_{inf,c}$) to the measured values of the total respiratory system.**

Measurements and Main Results: **The average difference between P_{mci} and $P_{mci,c}$ was 0.12 cm H_2O on inflation (2SD = 5.6 cm H_2O) and −1.4 cm H_2O on deflation (2SD = 5.0 cm H_2O); between P_{mcd} and $P_{mcd,c}$ was 1.73 cm H_2O on inflation (2SD = 4.5 cm H_2O) and −0.15 cm H_2O on deflation (2SD = 4.9 cm H_2O); and between P_{inf} and $P_{inf,c}$ was 0.14 cm H_2O on inflation (2SD = 6.7 cm H_2O) and −0.35 cm H_2O on deflation (2SD = 5.0 cm H_2O).**

Conclusions: **This method of "correcting" the total respiratory system P-V curve for the chest wall allows for calculation of an airway pressure that would place the lung at a desired volume on its P-V curve. For most patients, the chest wall had little influence on the total respiratory system P-V curve. However, there were patients in whom the chest wall did potentially have clinical significance. (Crit Care Med 2008; 36:2980–2985)**

KEY WORDS: acute respiratory distress syndrome; mechanical ventilation; static pressure–volume curve; chest wall; esophageal balloon; lung mechanics

Quasi-static pressure–volume (P-V) curves of the lung or the respiratory system have been used to guide ventilator management in patients with the acute respiratory distress syndrome (ARDS). These curves are sigmoidal with upward concavity at low inflation pressures and downward concavity at higher inflation pressures. The shape of the curve may reflect the continuous and aggregate process of opening and/or closing of small airways and alveoli (1–4), although other investigators have proposed that this shape may reflect the introduction of air into fluid-filled airways (5). Limited understanding of this process has hampered the ability of investigators to best use information from P-V curves in clinical settings. Despite this, a prospective, randomized study by Amato et al. (6) used respiratory system P-V curves as part of a lung-protective strategy in patients with ARDS, and showed reduced barotrauma, a higher rate of weaning from mechanical ventilation, and improved survival at 28 days compared with standard ventilator management without P-V curves. Two other studies have shown either a reduction in pulmonary and systemic inflammatory cytokines (7) or mortality (8) with the use of the P-V curve as part of a lung-protective strategy.

In all of these studies, positive end-expiratory pressure was set to a pressure above the lower inflection point (LIP) of the total respiratory system inflation P-V curve. However, other authors have argued that the P-V curve generated by the total respiratory system, and thus the derivation of the LIP, may not well represent the mechanical behavior of the lungs. Mergoni et al. (9) showed that the LIP of the total respiratory system can be determined by chest wall rather than lung mechanics. Ranieri et al. (10) reported that analysis of the total respiratory system alone may lead to overestimation of the LIP and, if used to guide ventilator management, inappropriately high positive end-expiratory pressure (PEEP). These same authors also assessed the upper inflection point and noted that on average, analysis of the total respiratory system routinely underestimates the upper inflection point of the lung. A more recent study by Pereira et al. (11) found a lesser contribution to the LIP by the chest wall in a mixed group of ARDS, acute lung injury, and cardiogenic pulmonary edema patients.

However, these previous data presenting values for inflection points for the chest wall and lung have under-emphasized the fact that neither is ventilated in isolation, thus the meaning of these inflection points could be questioned. Because the lung and chest wall are in series, if one identifies the thoracic volume at a point of interest on the P-V curve of the lung, the equivalent pressure applied

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***See also p. 3100.**

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to the total respiratory system for that volume can be determined either graphically, or, if the P-V data are fitted with a continuous equation, calculated exactly. Motivated by these observations, we proceeded to use this new method to evaluate the extent to which the P-V curve of the total respiratory system is influenced by the curvature of the chest wall.

MATERIALS AND METHODS

Patients were enrolled from the medical or surgical intensive care units at Massachusetts General Hospital (Boston, MA) after approval was obtained from the local institutional review board for human studies. Patients were included if a P-V curve was requested by the treating physicians, thus informed consent was waived. All patients fulfilled the American-European Consensus Conference criteria for ARDS/acute lung injury (acute onset, Pa_{O_2}/F_{IO_2} ≤300, bilateral infiltrates on chest radiograph, and no clinical evidence for left atrial hypertension) and were over the age of 18 yrs. Patients were heavily sedated or paralyzed and sedated without spontaneous breaths. Exclusion criteria included those who were too hemodynamically unstable (mean arterial pressure ≤60 mm Hg or pulse >140 beats/min) to be removed from the ventilator, those with leaks around their endotracheal tube or tracheostomy tube, those with pneumothorax or bronchopleural fistulas, and those with recent head injury or cerebral edema. P-V curves were obtained from a total of 52 patients over a 6-yr period from 1997 to 2003. Of those, 25 did not have esophageal balloons and were excluded. Of the remaining 27, five studies were repeated in the same patient and were excluded. The remaining 22 had sufficient inflation and deflation data to analyze. The data for the total respiratory system only from four of these patients have previously been reported (12). Patient ages, underlying diagnosis, Murray Lung Injury Score, and outcome are shown in Table 1. The mean Murray lung injury score was 2.93 ± 0.45 SD. P-V data were obtained at times ranging from 0 to 50 days postintubation (median, 4 days).

Table 1. Patient characteristics

Patient	Age	Sex	BMI (kg/m^2)	Chest Wall Compliance (mL/cm H_2O)	Diagnosis	Lung Injury Score	Outcome
1	54	F	44.0	65	Necrotizing transverse myelitis[a]	3	Died
2	48	F	20.1	34	PNA	3	Lived
3	23	F	22.7	21	Aspiration PNA	2	Lived
4	39	F	25.9	94	Sepsis[a]	3	Lived
5	53	M	23.2	56	PNA, empyema	3	Lived
6	33	M	28.3	409	Acute eosinophilic pneumonitis	3.25	Lived
7	24	F	27.5	50	PNA	3	Lived
8	32	F	31.1	119	Sepsis[a]	3.5	Lived
9	42	M	28.8	354	Duodenal ulcer s/p, Billroth II[a]	3.25	Lived
10	65	M	48.8	29	Ischemic bowel[a]	3.25	Died
11	59	F	41.4	84	Sepsis[a]	2	Died
12	58	F	27.5	41	Arterial thrombosis, urosepsis[a]	3.25	Lived
13	48	F	26.7	82	PNA, sepsis	2.75	Lived
14	76	M	27.3	54	s/p Decortication	2.5	Lived
15	32	F	42.8	68	PNA	3.25	Lived
16	22	M	26.6	50	Aspiration PNA	2.25	Lived
17	77	M	28.4	49	Pancreatitis[a]	3.25	Died
18	22	M	28.5	31	PNA	2.75	Lived
19	71	F	29.7	35	Aspiration PNA	3.5	Died
20	54	F	24.3	462	PNA	3.5	Died
21	30	M	31.2	200	PNA	2.5	Lived
22	36	M	104.0	54	PNA	2.75	Lived

BMI, body mass index; PNA, pneumonia; M, male; F, female.
[a] Indirect lung injury.

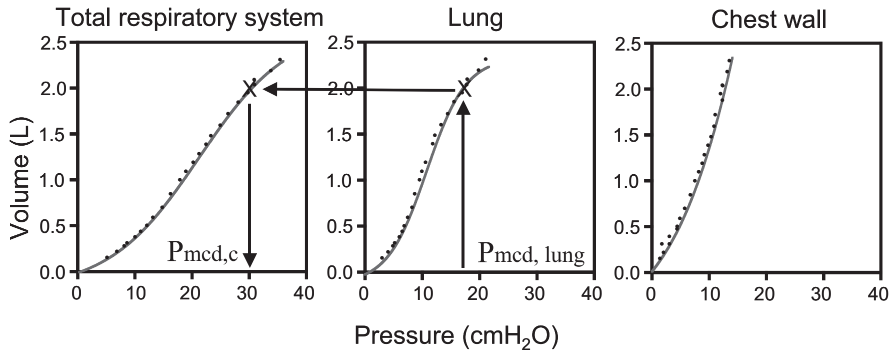

Figure 1. An example of the pressure-volume curves obtained from the total respiratory system, the lung alone, and the chest wall. To obtain a "corrected" point of maximal compliance decrease ($P_{mcd,c}$), the P_{mcd} of the lung is first identified graphically or calculated from the curve-fit equation ($P_{mcd} = 1.317d$). Then the volume at which this occurs is obtained. The pressure to which the total respiratory system would need to be inflated to reach this volume can be calculated or obtained graphically.

Procedure. Once the inclusion and exclusion criteria were met and demographic information obtained, the patient was ventilated with 100% oxygen for approximately 10 mins. Changes in pleural pressure (P_{pl}) were estimated from changes in esophageal pressure (P_{es}), using a thin-walled balloon catheter (Bicore CP 100, Allied Healthcare Products, St. Louis, MO, USA) that was positioned in the esophagus and inflated with 0.6 to 0.8 mL of air. Care was taken to avoid balloon overinflation by withdrawing all air with the syringe before inflation. The validity of the esophageal pressure measurement was assessed by observing for the appearance of cardiac oscillations at an appropriate distance from the airway opening (approximately 40 cm), and, in some instances, appropriate changes in pressure with the occlusion test (13). After allowing the lungs to reach functional residual capacity (approximately 5 secs), the patient's airway was connected to a 3-L calibration syringe previously filled with 100% oxygen. The inflation curve was obtained by sequentially adding 50–100 mL incremental volumes in a stepwise fashion until a pressure of 35 cm H_2O was reached. Four seconds were allowed between steps, allowing equilibrium, so that proximal airway pressure approximated alveolar pressure. The patient was then reconnected to the ventilator at the original settings for approximately 5 mins. Pilot data showed that 5 mins was an adequate time to reestablish the volume history of the lungs in this patient population. The patient was again removed from the ventilator and after allowing the lungs to reach functional residual capacity, the syringe was reconnected to the patient and the lungs rapidly inflated to 35 cm H_2O (approximately 2–3 secs). After a pause of approximately 4 secs, volumes were then withdrawn in 50–100 mL decrements, waiting at each step until the pressure signal reached a quasi-steady-state level (approximately 4 secs). The procedure was stopped when a pressure of 0 cm H_2O was reached.

Measurements. A 3-L calibration syringe was fitted with a linear displacement transducer connected to a personal computer data acquisition system. Also connected were pressure transducers measuring simultaneous airway and esophageal balloon pressures. Dedicated software was developed using LabVIEW™ (National Instruments Corp.) to acquire, display, save, and analyze pressure and volume signals as the syringe was manually inflated in incremental steps. The user manually accepted each data point when the pressure signal had reached a quasi-steady-state level. Volume and pressure values were saved in a spreadsheet file.

Inflation volume data were not corrected for changes in temperature and humidity or oxygen consumption because these effects have been found to offset one another during inflation in the time taken by the procedure (14). For the deflation limb, the volume was corrected for changes in temperature, humidity, and oxygen consumption. Loss of volume because of oxygen consumption was assumed to be 95 mL/min and the average rate of loss in thoracic gas volume was measured in patients by Dall'ava-Santucci et al. (14).

Data Analysis. Only complete data sets including both inflation and deflation were analyzed. Two or three data sets were collected from each patient. One set of data was chosen for analysis based on the best curve-fit (maximum R^2) of the total respiratory system data to the sigmoid equation for inflation and deflation. Transpulmonary pressure was calculated as the difference between the pressure of the total respiratory system and the esophageal pressure. P-V data were obtained for the total respiratory system, the chest wall, and the lung alone from the plot of volume against proximal airway pressure, esophageal pressure, and transpulmonary pressure, respectively. P-V data were then fitted with the equation:

$$V = a + b/(1 + e^{-(P-c)/d}) \qquad [1]$$

that has been previously reported (4, 12). This equation has four fitting variables: *a*, in units of volume, representing the lower asymptote; *b*, in units of volume, representing the distance from *a* to the upper asymptote, or tidal inflation; *c*, in units of pressure, representing the true inflection point (where concavity changes direction); and *d*, in units of pressure, representing the distance from *c* of the zone of high compliance. Using the program DeltaGraph (Red Rock Software, Salt Lake City, UT), the equation was fitted to the P-V data using the Levenberg–Marquardt iterative algorithm to minimize the sum of squared residuals. The algorithm was set to run until the resulting sum of squared residuals changed by <0.0001, yielding estimates of the variables *a*, *b*, *c*, and *d* and the best-fit coefficient R^2. Initial guess coefficients were $a = 0$ L, $b = 3$ L, $c = 20$ cm H_2O, and $d = 10$ cm H_2O. The point of maximum compliance increase (P_{mci}, where the rate of change of upward slope is maximal or where the second derivative of the function has a maximum) is $c - 1.317d$. The point of maximum compliance decrease (P_{mcd}) is $c + 1.317d$. Points of maximum compliance increase or decrease falling outside the range of data collected (<0 cm H_2O or greater than the highest data point collected in cm H_2O) were not included in the analysis.

For both inflation and deflation total respiratory system P-V curves for each patient, the point of maximal compliance increase (P_{mci}), the point of maximal compliance decrease (P_{mcd}), and the point of inflection (P_{inf}) were calculated from the variables obtained after curve-fitting with Equation 1. This process was repeated for both inflation and deflation P-V curves for the lung. The volumes at each pressure of interest (P_{mci}, P_{inf}, and P_{mcd}) were identified on the lung P-V curves and were used to calculate "corrected" pressures ($P_{mci,c}$, $P_{inf,c}$, and $P_{mcd,c}$) for the respiratory system P-V curves corresponding to those volumes (Fig. 1). These "corrected" values were compared with the corresponding values directly calculated from the respiratory system P-V curves by computing Pearson

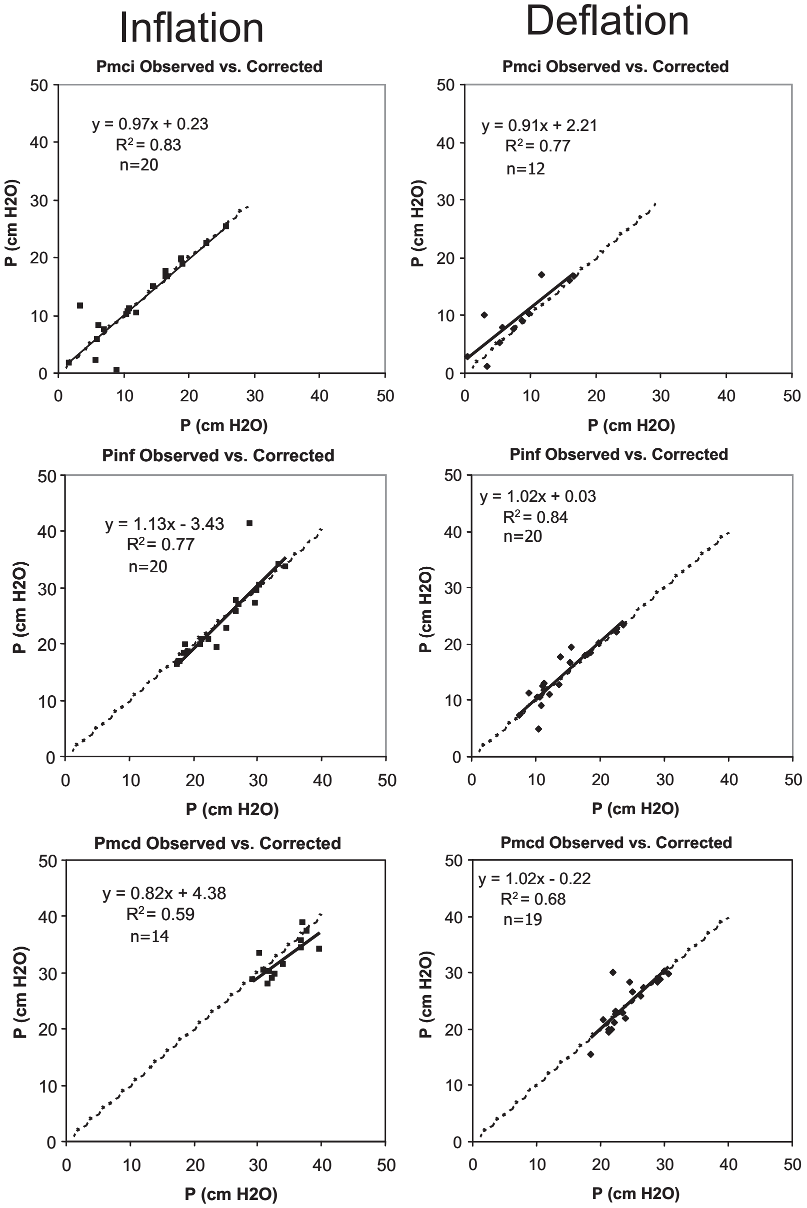

Figure 2. Plots of the corrected inflection points ($P_{mci,c}$, $P_{inf,c}$, and $P_{mcd,c}$) vs. the observed inflection points (P_{mci}, P_{inf}, and P_{mcd}) of the total respiratory system for both inflation (■) and deflation (◆). A regression line (*solid black line*) and the line of identity (*dashed line*) are also shown. Number of data points (n), regression equation, and R^2 value are incorporated for each figure.

correlation coefficient and by Student's t-test with Bonferroni correction for multiple comparisons. In addition, Bland–Altman plots (15) were constructed to quantify the bias and precision of the uncorrected values in relation to those corresponding to the points of interest for the lung ("corrected values").

To examine the relationship between body mass index (BMI) and chest wall influence on the total respiratory system P-V curve, the difference between the corrected and calculated points for P_{mci}, P_{inf}, and P_{mcd} for inflation and deflation were plotted against BMI and Pearson correlation calculated. A similar plot and analysis was done with static cord compliance calculated from 0 to 40 cm H_2O static airway pressure.

RESULTS

Corrected values based on the lung alone measurements were plotted against observed data from the total respiratory system (Fig. 2). For P_{mci}, the regression line for inflation was $y = 0.97x + 0.23$ ($R^2 = .83$) and deflation was $y = 0.91x + 2.21$ ($R^2 = .77$). For P_{inf}, the regression yielded $y = 1.13x - 3.43$ ($R^2 = .77$) for inflation and $y = 1.02x + 0.03$ ($R^2 = .84$) for deflation. For P_{mcd}, the regression line for inflation was $y = 0.82x + 4.38$ ($R^2 = .59$) and deflation was $y = 1.02x - 0.22$ ($R^2 = .68$). Paired Student's t-test analysis of each groups of points (P_{mci}, P_{inf}, or P_{mcd}), either on inflation or deflation, showed that none of the corrected values were statistically different from their respective observed value.

By using the Bland–Altman analysis (Fig. 3), the average difference between P_{mci} and $P_{mci,c}$ was 0.12 cm H_2O on inflation (2SD = 5.6 cm H_2O) and -1.4 cm H_2O on deflation (2SD = 5.0 cm H_2O). The average difference between P_{mcd} and $P_{mcd,c}$ was 1.73 cm H_2O on inflation (2SD = 4.5 cm H_2O) and -0.15 cm H_2O on deflation (2SD = 4.9 cm H_2O). The average difference between P_{inf} and $P_{inf,c}$ was 0.14 cm H_2O on inflation (2SD = 6.7 cm H_2O) and -0.35 cm H_2O on deflation (2SD = 5.0 cm H_2O).

BMI plotted against chord compliance did not yield a linear relationship (Fig. 4). The Pearson coefficient was -0.15. On inflation, the Pearson coefficients for the differences between corrected and calculated measurements of P_{mci}, P_{inf}, and P_{mcd} vs. BMI were -0.07, 0.21, and 0.10, respectively. On deflation, the Pearson coefficients for the differences between corrected and calculated measurements of P_{mci}, P_{inf}, and P_{mcd} vs. BMI were 0.46, 0.10, and 0.06, respectively.

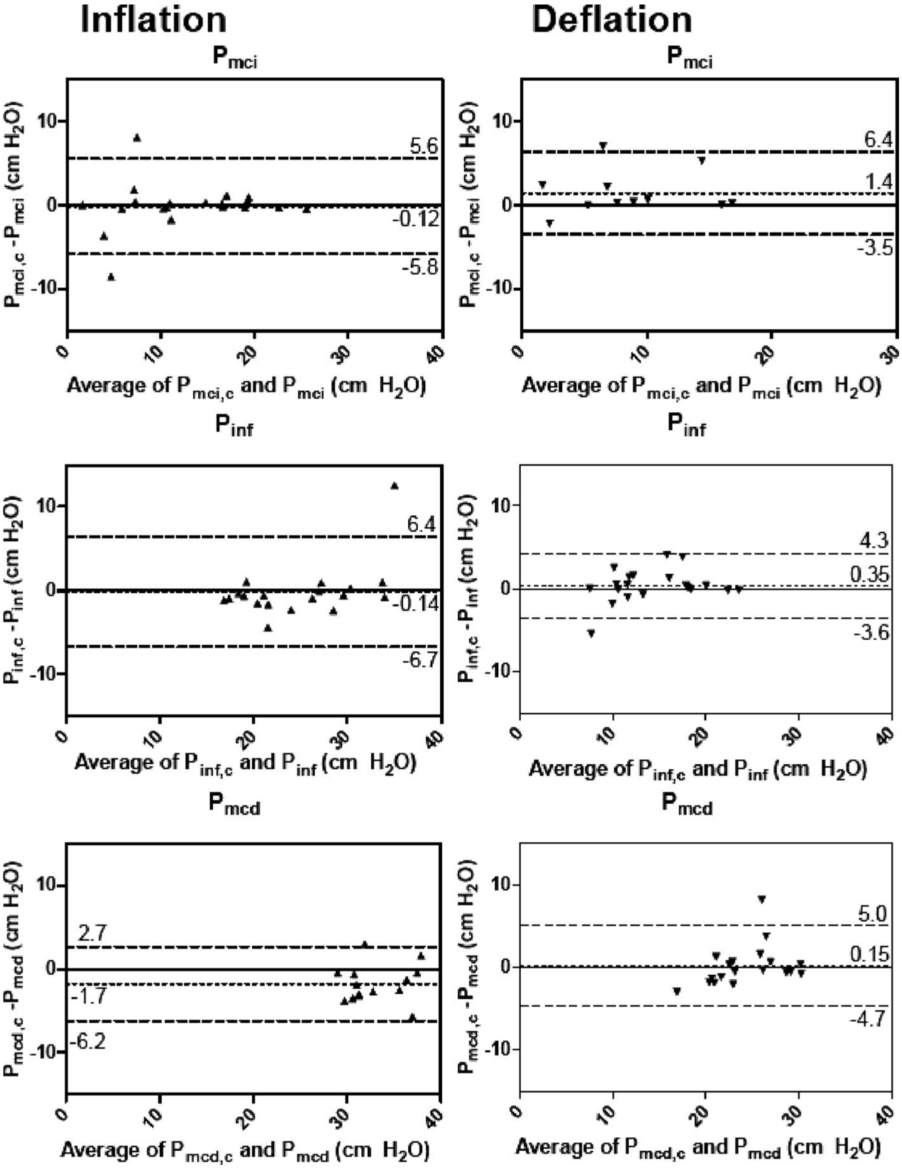

Figure 3. Difference between the corrected inflection points ($P_{mci,c}$, $P_{inf,c}$, and $P_{mcd,c}$) and observed inflection points (P_{mci}, P_{inf}, and P_{mcd}) of the total respiratory system vs. P_{mci}, P_{inf}, and P_{mcd} for both inflation (▼) and deflation (△). The bias and two standard deviations from the bias are represented by the *dashed lines*, with values shown next to the *dashed lines*.

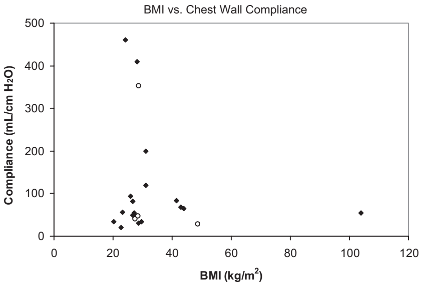

Figure 4. Chest wall compliance vs. body mass index (*BMI*). Chest wall compliance is measured as cord compliance from 0 to 40 cm H_2O pressure. Patients with abdominal pathology (9, 10, 12, and 17) are displayed as *open circles* (○).

DISCUSSION

A major finding of this study was that Equation 1 could be used to correct pressures of interest on the lung P-V curve for the effect of the chest wall P-V curve. In other words, this method allows one to identify the pressure that one would need to inflate the total respiratory system to place the lung at a particular point on its P-V relationship. Furthermore, despite the fact that for most of our subjects P_{mci}, P_{inf}, and P_{mcd} obtained from P-V curves of the total respiratory system are close to those corrected for the effect of the chest wall, there were a number of instances where differences were large. The maximum difference was 12 cm H_2O (inflation P_{inf} vs. $P_{\text{inf,c}}$). Although our sample size was small, patients with the greatest differences between the corrected and uncorrected values could not be identified based upon diagnosis, age, whether or not they had abdominal or thoracic pathology, or other baseline demographics. BMI did not identify patients with a larger degree of chest wall influence despite a large range of BMI (one subject weighed 348 kg with BMI of 104 kg/m^2). No clear relationship was seen between chest wall static cord compliance and BMI. Examination of the chest wall compliance curves of the five subjects with BMI $>$40 kg/m^2 did not reveal obvious differences when compared with those with BMI $<$40 kg/m^2. Similarly, the difference between corrected and calculated points of interest did not vary systematically with BMI. The lack of relationship between BMI and chest wall compliance is consistent with other published literature (16, 17). In those patients whose corrected values were substantially different from the uncorrected ones, careful examination of their P-V curves revealed that the chest wall demonstrated more curvature than the lung alone. For those that had close agreement between the uncorrected and corrected values, the chest wall generally demonstrated less curvature than the lung P-V curve, and thus the lung and total respiratory system had similar curvature. Therefore, although the chest wall compliance affects the total respiratory system compliance, it generally did so in a nearly volume-independent way throughout the range of pressures studied.

This result is similar to findings that have been published in other disease states, such as chronic obstructive pulmonary disease and pulmonary edema, in which abnormalities in total respiratory system mechanics essentially reflect alterations in lung mechanics rather than chest wall mechanics (18). The sigmoidal shape of the P-V curve of the total respiratory system, and hence the points of interest studied, are determined to a greater extent by the characteristics of the lung. Again, while the chest wall may also be affected in patients with ARDS, in most of our patients it did not significantly alter the shape features of the total respiratory system P-V curve. Our results are similar to those of Roupie et al. (19) and Pereira et al. (11) who also used Equation 1 to curve-fit the total respiratory system, the lung, and the chest wall P-V curves. However, in that study, they compared P_{mci} of the lung ($P_{\text{mci,L}}$) to P_{mci} of the total respiratory system ($P_{\text{mci,trs}}$) and determined whether the chest wall had a large contribution to the total respiratory system P_{mci} if the difference was greater than 50%. It is possible to have large differences in $P_{\text{mci,L}}$ and $P_{\text{mci,trs}}$ yet inflating the total respiratory system to $P_{\text{mci,trs}}$ will be equivalent to inflating the lung alone to $P_{\text{mci,L}}$ if the chest wall compliance is nearly linear. In that case, the chest wall will simply displace the $P_{\text{mci,L}}$ to higher pressures, thus creating a larger difference between $P_{\text{mci,trs}}$ and $P_{\text{mci,L}}$.

Our results differ from those of Mergoni et al. (9) who found that the chest wall can contribute to the LIP and Ranieri et al. (10) who found that the chest wall can contribute to the upper inflection point. The discrepancy in findings may be related to the method of determining the contribution of the chest wall to total respiratory system mechanics. In both the prior studies, the inflection points were calculated and compared using the P-V curves of the chest wall, the lung, and the total respiratory system in isolation. Because these structures are not ventilated in isolation, we question the relevance of comparing the inflection points of the lung and chest wall to those from the total respiratory system.

We did not use the LIP or upper inflection point in this analysis because of problems in the definitions and objectivity that have been pointed out elsewhere (11, 12). Although some groups have found the determination of the LIP to be reproducible (20, 21), we (12) and others (22) have not, so we chose objective variables obtained through curve-fitting for our analysis. In those subjects where the chest wall had a significant influence, it was not systematic for all points (P_{mci}, P_{inf}, or P_{mcd}). This finding suggests that the choice of points on the P-V curve is important in determining whether or not the chest wall will have significant influence, but that is not possible to tell *a priori* who or which point will be affected.

We acknowledge some limitations of our study. Our patients were primarily medical patients, and only three had had recent surgery. Therefore, these results may not be generalized to a surgical population. However, when the data from these three patients and one other with abdominal pathology (patients 9, 10, 12, and 17) were examined, no consistent deviation between corrected and uncorrected values was seen, nor was chest wall compliance markedly different in this group compared with the remaining patients (Fig. 4). Another limitation is the use of esophageal pressure measurements as an estimate of pleural pressure. This measurement has been criticized for not being representative of pleural pressure around the lung and also for being affected by the weight of mediastinal contents in a supine patient (23). We eliminated the effect of mediastinal weight by using only the change in pleural pressure, not absolute values. Although esophageal pressure likely reflects the pleural pressure only in the adjacent pleural space at that level, there currently exists no other practical method to estimate local pleural pressure. In our use of esophageal manometry, a further limitation is that we did not consistently perform an occlusion test after esophageal balloon placement. Despite our careful technique to ensure adequate balloon placement, in theory balloon position and/or pressure transmission may not have been accurate. However, our measurements of chest wall compliance were similar to those reported in the literature (24, 25). Because many different authors have reported chest wall compliances above the anticipated normal values (16, 26), we believe that further work is required to determine whether this variance has biological and/or methodologic bases. Despite these limitations, the method of correcting the points of interest on the P-V curve for the chest wall is not dependent on the methods used or the population studied and can be applied to any P-V data using either models to curve-fit the data or using graphical methods.

A prospective, randomized study by Amato et al. (6), where the P-V curve of the total respiratory system was used as part of an “open-lung, low distending pressure” strategy showed reduced barotrauma, a higher weaning rate, and improved survival at 28 days compared with standard ventilator management. In a study by Ranieri et al. (7) in ARDS patients, pulmonary and systemic inflammatory mediators were significantly reduced 36 hrs after randomization to a protective ventilation strategy using the P-V curve to set PEEP and tidal volume compared to control ventilation without P-V curve measurement.

More recently, Villar et al. (8) demonstrated an improved mortality in persistent ARDS patients randomized to an open-lung strategy, where PEEP was set above the LIP of the P-V curve. None of these studies used esophageal pressure measurements to evaluate the effect of the chest wall on the LIP. Based on our data, it is likely that only a small number of patients may have had their PEEP affected if esophageal pressure measurements were made. However, because the measurement is simple to perform and carries minimal risk, we feel that it should be part of the P-V measurement to ensure accurate assessment of lung mechanics. A clinical trial in ARDS is now ongoing using esophageal pressure measurements to set PEEP (27).

In conclusion, in this group of largely medical ARDS, the chest wall had little influence on the total respiratory system P-V curve for most patients. However, for a few subjects the chest wall did have a significant influence and this effect could not be predicted clinically. If the P-V curve of the lung is to be used as a tool to guide ventilator management, an estimation of pleural pressure seems necessary. This method of correcting the respiratory system P-V curve for the effect of the chest wall can be used to obtain precise pressure settings for the lung during mechanical ventilation. It remains to be seen whether or not the P-V curve will be a useful tool for guiding pressure limits during mechanical ventilation.

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