

The chest wall in acute lung injury/acute respiratory distress syndrome

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Purpose of review

There has recently been renewed interest in the chest wall during mechanical ventilation, related to lung-protective ventilation strategies, as well as in the role of abdominal pressure in many facets of critical illness. The purpose of this review is to address relevant issues related to the chest wall and mechanical ventilation, particularly in patients with acute lung injury/acute respiratory distress syndrome.

Recent findings

In mechanically ventilated patients with acute lung injury, intra-abdominal pressure is an important determinant of chest wall compliance. With elevated intra-abdominal pressure, the compliance of the chest wall and total respiratory system is decreased, with a relatively normal compliance of the lungs. The lung compression effects of increased intra-abdominal pressure may lead to a loss of lung volume with atelectasis. An appropriate level of positive end-expiratory pressure is necessary to counterbalance this collapsing effect on the lungs. Also, the stiff chest wall results in a lower transpulmonary pressure during positive-pressure ventilation.

Summary

As chest wall compliance may have important clinical implications during positive-pressure ventilation, the physiology of this effect should be considered, particularly in patients with acute lung injury and increased abdominal pressure.

Keywords

abdominal compartment syndrome, acute lung injury, chest wall compliance, lung compliance, positive-pressure ventilation, respiratory system compliance

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Introduction

The respiratory system includes the lungs and the chest wall. The mechanics of the respiratory system depend on the mechanics of the lungs and the chest wall, and their interactions [1]. There is interest in the chest wall during mechanical ventilation related to lung-protective ventilation strategies and the role of abdominal pressure in critically ill patients. When assessing respiratory mechanics during positive-pressure ventilation, it is important to separate the effects of the lungs from those of the chest wall. In this review, we explore aspects of chest wall compliance related to its physiology, measurement and effects during positive-pressure ventilation.

Physiology of the chest wall

The chest wall, like the lungs, is elastic. At normal resting conditions (i.e. functional residual capacity), the chest wall is pulled inward and the lungs are pulled outward. The pressure between the chest wall and the lungs, pleural pressure, is thus negative relative to atmospheric pressure. If the lungs are open to the atmosphere and the

intrapleural pressure increases, as is the case with a tension pneumothorax or abdominal distension, the lungs will collapse inward and the chest wall will expand outward [1].

The interaction between the lungs and the chest wall can be illustrated from the relaxation pressure–volume (P/V) curves of each [2,3]. The relaxation pressure of the chest wall is negative except at very high volume. In other words, the chest wall tends to expand outward. The relaxation pressure of the lungs is always positive. In other words, the lungs tend to collapse. The sum of the P/V curves of the lungs and the chest wall produces the respiratory system P/V curve, which can be measured at the bedside in mechanically ventilated patients. The sigmoid shape of the normal respiratory system P/V is determined by the chest wall at low lung volumes and the lungs at high lung volumes. At any volume, the relaxation pressure for the lungs and the chest wall is the sum of the pressures for the lungs and the chest wall separately. Thus, the elastance (i.e. $\Delta P/\Delta V$) of the respiratory system is the sum of the elastances of the lungs and the chest

wall. As compliance is the inverse of elastance, it follows that:

$$1/C_{RS} = 1/C_L + 1/C_{CW} \quad (1)$$

where C_{RS} is respiratory system compliance, C_L is lung compliance and C_{CW} is chest wall compliance.

The pressure on the outer surface of the chest wall is atmospheric pressure (P_{ATM}) and the pressure on the inner surface of the chest wall is pleural pressure (P_{PL}). The pressure across the chest wall (P_{CW}) is, therefore, the difference between P_{PL} and P_{ATM} . With the exception of the unusual circumstance of negative-pressure ventilation (e.g. iron lung), P_{ATM} is zero. Thus, the distending pressure on the chest wall is P_{PL} . The volume changes of the lungs and chest wall are normally equal. If 1 l of volume is inhaled, the corresponding change in P_{PL} is about 5 cm H₂O. In other words, normal chest wall elastance ($\Delta P/\Delta V$) is 5 cm H₂O/l and normal chest wall compliance ($\Delta V/\Delta P$) is 0.2 l/cm H₂O. Normal C_L is also 0.2 l/cm H₂O and thus normal C_{RS} is 0.1 l/cm H₂O.

A common clinical question relates to the effect of positive-pressure ventilation on P_{PL} . The pleural pressure is determined by lung volume [tidal volume (V_T) or lung volume change with the addition of positive end-expiratory pressure] and chest wall compliance, and is independent of lung compliance:

$$P_{PL} = \text{volume}/C_{CW} \quad (2)$$

Thus, it is not entirely correct to refer to the amount of pressure *transmitted* to the pleural space because pleural pressure is determined by volume and C_{CW} . P_{PL} as a fraction of alveolar pressure (P_{ALV}) is determined by the relationship [1]:

$$\Delta P_{PL}/\Delta P_{ALV} = C_L/(C_L + C_{CW}) \quad (3)$$

or

$$\Delta P_{PL}/\Delta P_{ALV} = C_{RS}/C_{CW} \quad (4)$$

Under normal conditions where C_L and C_{CW} are equal, ΔP_{PL} will be half of ΔP_{ALV} . When C_{CW} is reduced relative to C_L , ΔP_{PL} will be a greater fraction of ΔP_{ALV} . On the other hand, when C_L is reduced relative to C_{CW} , ΔP_{PL} will be a lower fraction of ΔP_{ALV} .

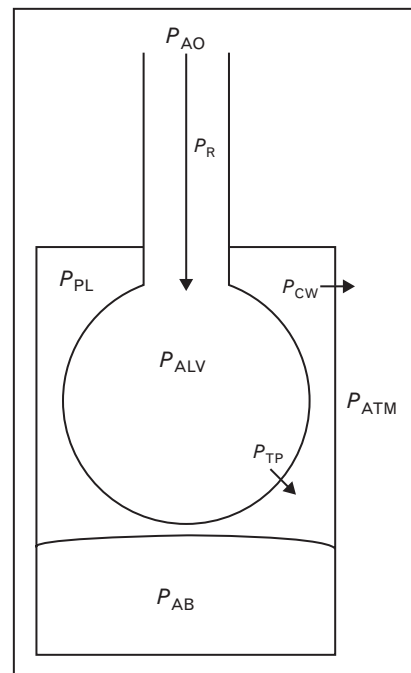
The chest wall is comprised of the rib cage and the abdomen. Abnormalities affecting chest wall compliance include skeletal deformity, burns with eschar, morbid obesity, chest wall edema, ascites, pregnancy and abdominal compartment syndrome (ACS). A relaxed diaphragm transmits pressure from the abdomen. In normal persons, C_{CW} decreases by 25% in a supine position compared to an

upright position. A decrease in C_{CW} results in a lower lung volume for the same P_{PL} . If lung volume is restored with the addition of PEEP, however, the increase in P_{ALV} will result in an increase in ΔP_{PL} , dependent on the C_L and C_{CW} as discussed above.

In mechanically ventilated patients with acute lung injury (ALI), intra-abdominal pressure (IAP) is an important determinant of chest wall compliance [1,4^{••},5]. With elevated IAP, there is flattening and rightward shift of the P/V curve of the chest wall and total respiratory system, with a relatively normal P/V curve of the lungs. The impact of IAP on C_{CW} is variable, and may depend upon the tone of the diaphragm and body position. In mechanically ventilated patients with chronic obstructive lung disease who have auto-PEEP and dynamic hyperinflation due to expiratory flow limitation, the relative contribution of the chest wall to total respiratory system auto-PEEP is small [6]. Application of PEEP beyond the level that counterbalances auto-PEEP causes an important worsening in the elastic properties of the lungs and chest wall.

The determinants of airway pressure required for passive inflation of the lungs during positive-pressure ventilation are illustrated in Fig. 1. The pressure applied must overcome airways resistance, lung compliance and chest wall compliance. P_{ALV} is determined by V_T and C_L ,

Figure 1 A cartoon of the respiratory system illustrating respiratory mechanics



The diagram illustrates the relationships between airway opening pressure (P_{AO}), alveolar pressure (P_{ALV}), pleural pressure (P_{PL}), abdominal pressure (P_{AB}), atmospheric pressure (P_{ATM}), transpulmonary pressure (P_{TP}) and pressure across the chest wall (P_{CW}).

whereas P_{PL} is determined by V_T and C_{CW} . P_{AB} affects C_{CW} due to its effect on the diaphragm.

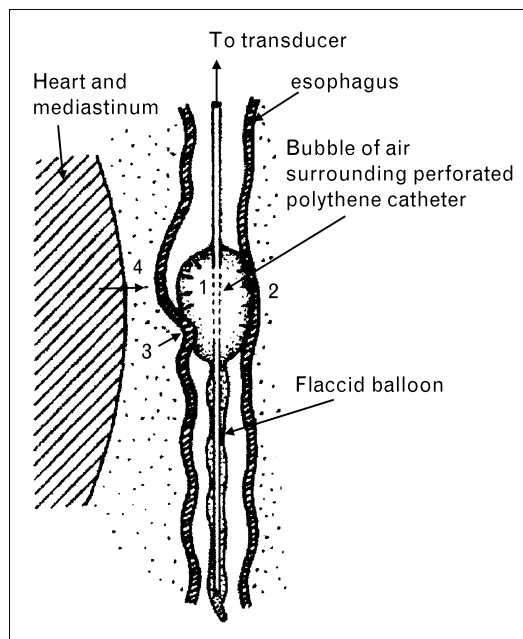
Measurement of chest wall compliance

Calculation of C_{CW} requires measurements of V_T and P_{PL} . V_T is easy to measure at the bedside and is displayed on all modern ventilators. P_{PL} cannot be easily measured directly. The traditional approach to assess P_{PL} is the use of an esophageal balloon, which consists of a thin catheter with multiple small holes in the distal 5–7 cm of its length [7]. A 10-cm balloon is placed over the distal end of the catheter to prevent the holes in the catheter from being occluded by esophageal tissue and secretions, and the balloon is inflated with a small amount of air (0.5–1 ml). The proximal end of the catheter is attached to a pressure transducer.

Correct positioning of the esophageal balloon is necessary to ensure accurate esophageal pressure (P_{ES}) measurements. The classic technique used to validate the balloon's position requires the subject to perform static Valsalva and Mueller maneuvers with the glottis open [8]. In patients unable to cooperate, changes in P_{ES} and changes in airway opening pressure (P_{AO}) are assessed during a spontaneous breathing effort against a closed airway. When changes in P_{ES} are equal to P_{AO} , it is assumed that transmission of P_{PL} to P_{ES} is unimpeded and P_{ES} accurately reflects P_{PL} ; however, this approach is not possible in patients who do not make respiratory efforts due to sedation or paralysis. In this case, the catheter tip should be about 35–45 cm from the nares and the presence of cardiac oscillations should be present on the P_{ES} waveform.

The proximity of the esophagus to the pleural space allows use of P_{ES} as an estimate of P_{PL} ; however, P_{ES} accurately reflects P_{PL} only if the pressure measured in the balloon is an accurate measure of the pressure in the esophagus, the transmural pressure of the esophagus is zero, there is no compression of the esophagus by the heart or other intrathoracic structures and the pressure in the mediastinum surrounding the esophagus is equal to P_{PL} (Fig. 2) [9]. P_{ES} may be affected by measurement artifacts such as the elastic recoil of balloon (worsened by balloon over-inflation), elastic recoil of the esophagus, active esophageal contraction or pressure transmitted from surrounding structures. Moreover, P_{ES} varies with lung volume and body position. Upright-to-supine differences in P_{PL} are attributed to artifacts caused by direct compression of the esophagus by mediastinal contents such as the heart. In healthy subjects, the average increase in P_{ES} from upright to supine is about 3 cm H₂O when adjusted for decreased lung volume (95% confidence interval: -1 to +7 cm H₂O) [10^{*}]. Without adjustment for lung volume changes, P_{ES} is 7.0 cm H₂O higher

Figure 2 Sources of error in pleural pressure estimation



Esophageal pressure may be affected by (1) elastic recoil of balloon, (2) elastic recoil of esophagus, (3) active esophageal contraction or (4) transmitted pressure from surrounding structures. Reproduced from [8].

in the supine than in the upright position in normal individuals. It follows that absolute values of P_{ES} are unpredictable and of questionable value for clinical purposes [11^{*},12^{**}]. In the upright position, values of P_{ES} closely reflect both absolute static values and dynamic changes of P_{PL} , whereas in the supine position, absolute P_{ES} measurements provide a good estimate of the P_{PL} only in the mid-chest, but overestimate P_{PL} in the nondependent and underestimate P_{PL} in the dependent lung. Changes in P_{ES} closely reflect changes in P_{PL} , however, and hence changes in P_{ES} are useful when assessing respiratory system mechanics.

Measurement of P_{ES} is invasive, requires special equipment and is not commonly available for the care of mechanically ventilated patients. While P_{ES} is the standard method of estimating P_{PL} , pleural pressure changes during lung inflation are transmitted to other structures in the mediastinum. In 1965, Comroe [13] suggested that an intra-thoracic vein with its thin wall is capable of transmitting pleural pressure and might therefore be an acceptable alternative to the esophagus for P_{PL} measurement [13]. In healthy adults, it has been shown that valid measurements of P_{PL} can be obtained from P_{ES} or central venous pressure (P_{CVP}) [14]. In one study, it was suggested that P_{CVP} was more reliable than P_{ES} in reflecting a change in P_{PL} in anesthetized supine patients [15]. Respiratory variation in P_{CVP} is easily detected at the bedside during mechanical

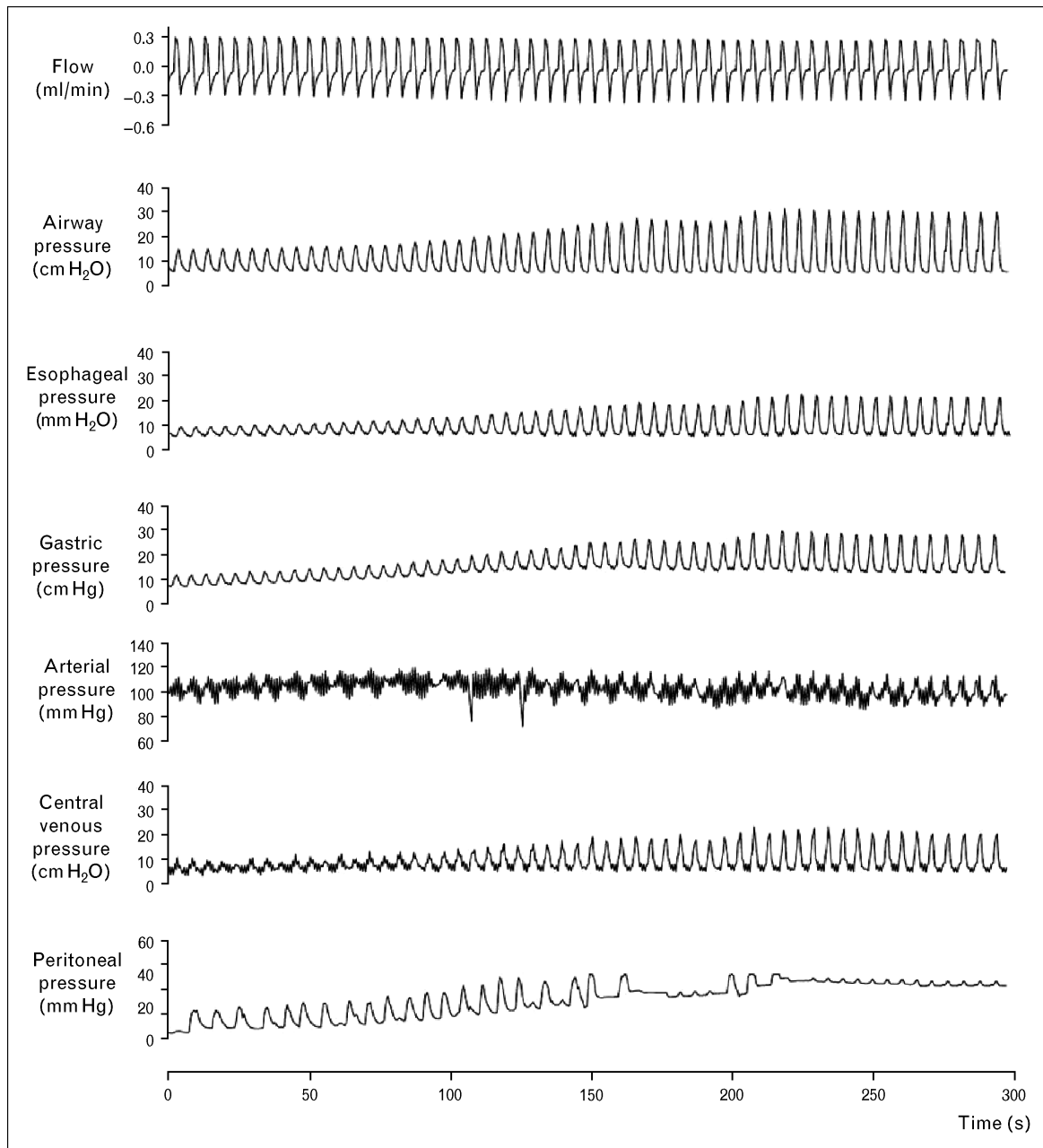
ventilation and varies from a positive deflection when the respiratory muscles are completely inactive to negative swings during large inspiratory efforts. Chievey-Williams *et al.* [16] recently compared ΔP_{ES} to ΔP_{CVP} and reported that useful information can be obtained from ΔP_{CVP} during assisted mechanical ventilation. In an experimental model, Valenza *et al.* [17**] reported that ΔP_{CVP} was similar to ΔP_{ES} when animals' abdomens were inflated with helium (Fig. 3). It is also interesting to note that end-expiratory P_{CVP} did not increase unless PEEP was added.

Chest wall compliance is calculated from V_T and the corresponding ΔP_{PL} :

$$C_{CW} = V_T / \Delta P_{PL} \tag{5}$$

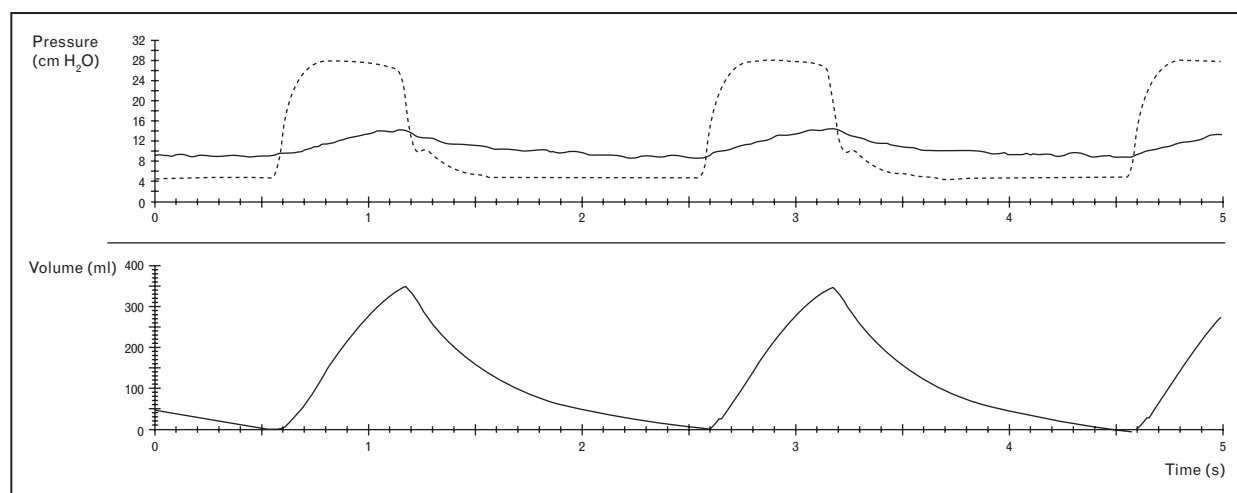
ΔP_{PL} is estimated from ΔP_{ES} (Fig. 4) or ΔP_{CVP} (Fig. 3). Note that estimates of ΔP_{PL} to calculate C_{CW} require that the patient is relaxed and breathing in synchrony with the ventilator. Estimates of ΔP_{PL} to calculate C_{CW} are not valid if the patient is making active breathing efforts.

Figure 3 Effects of the insufflation of helium into the peritoneum of a pig over time



From top to bottom, tracings of flow, airway, esophageal, gastric, arterial, central venous and peritoneal pressure are represented. The static and dynamic components of esophageal and central venous pressure are clearly identified. From [17**].

Figure 4 Airway pressure (upper panel, dotted line), esophageal pressure (upper panel, solid line) and tidal volume measurements (lower panel) in a patient receiving positive-pressure ventilation



Note that the esophageal pressure increases by 5 cm H₂O as a tidal volume of 350 ml is delivered. Thus, the chest wall compliance is 70 ml/cm H₂O.

Obesity and chest wall compliance

Patients with morbid obesity are increasingly seen in the ICU. These patients occasionally develop ALI and require mechanical ventilation. The respiratory function of these patients can be affected in several ways. The increase in IAP can displace the diaphragm cephalad (particularly in the supine position), reducing lung volume, causing small airway closure and atelectasis, and producing gas exchange abnormalities. The chest wall compliance may be reduced, resulting in an increased P_{PL} when positive pressure is applied to increase lung volume.

Pelosi *et al.* [18] evaluated respiratory system mechanics in eight sedated, paralyzed, morbidly obese patients. Compared with nonobese patients, obese patients had a markedly lower functional residual capacity and a higher IAP. The P/V curve of the respiratory system of the obese patients was curvilinear, with an inflection point in the low part of the curve that was not seen in the nonobese patients. Obese patients also had increased airways resistance (R_{AW}) and decreased C_{RS} . The authors suggested that the reduction in lung volume due to atelectasis formation and/or airway closure may explain the observed decreased compliance of the respiratory system as well as the development of a lower inflection point in the P/V curve. Moreover, the lung volume reduction was associated with a reduction in airway caliber, resulting in increased airways resistance. These data suggest that the main cause of the mechanical alterations of the respiratory system in obese subjects during sedation and paralysis is a reduction in lung volume due to excessive unopposed IAP.

In another study by Pelosi *et al.* [19], respiratory system mechanics, lung mechanics and chest wall mechanics were compared in 10 patients with morbid obesity and another 10 nonobese patients during sedation and paralysis. In the obese patients, there was a reduction in C_{RS} and an increase in R_{AW} . The reduction in C_{RS} was due to reductions in both C_L and C_{CW} . The relative contribution of the lungs and the chest wall to the C_{RS} was, however, similar in nonobese and obese patients. Moreover, C_L was reduced to a greater extent than C_{CW} in both the obese patients and the nonobese patients. Functional residual capacity was reduced in the obese patients, which may explain the decreased C_L secondary to alveolar collapse.

There are several implications for the application of positive-pressure ventilation for patients with morbid obesity. Generous amounts of PEEP may be necessary to counterbalance the lung-collapsing effects of IAP. Measures of IAP (e.g. urinary bladder pressure) may be useful to estimate the amount of IAP applied to the lungs. As the reduced C_{RS} may be the result of C_L more so than C_{CW} , the effect of positive-pressure ventilation on P_{PL} may be less than expected. In other words, the chest wall may not have a substantial effect on P_{TP} in many obese patients.

Intra-abdominal pressure

Interactions between the abdominal compartment and the thoracic compartment are important considerations in the critically ill patient. Both compartments are linked by the diaphragm. If the diaphragm is allowed to freely shift upward into the thorax under increased abdominal pressure, lung volume will be reduced. If lung volume is restored with PEEP, however, then the abdominal

pressure can result in an increase in intra-thoracic pressure. On average, half of the pressure in the intra-abdominal compartment (range 25–80%) has been noted to be present in the intra-thoracic space in humans and animals [5]. This wide range may be related to the amount of PEEP that has been applied to restore lung volume in the setting of increased abdominal pressure.

IAP is the steady state pressure in the abdominal cavity [4^{••},5]. Normal IAP is about 5 mmHg and increases during inhalation with diaphragmatic contraction. Direct measurement of intraperitoneal pressure is the gold standard for determination of IAP; however, this is not practical and thus the bladder method is most commonly used for intermittent IAP measurement [20,21[•]]. The bladder acts as a passive structure, transmitting IAP after infusion of saline volumes between 50 and 100 ml at body temperature [21[•]]. IAP should be measured at end-exhalation in the complete supine position, ensuring that abdominal muscle contractions are absent and the transducer is zeroed at the mid-axillary line. Intra-abdominal hypertension (IAH) is defined as a consistent increased IAP value above 12 mmHg recorded a minimum of 3 times over 4 to 6 h [5]. ACS is defined as IAH value above 20 mmHg recorded during a minimum of three standardized measurements performed 1–6 h apart and associated with single or multiple organ system failure that was not previously present. The prevalence of IAH has been reported to range between 54% in medical ICU patients and 65% in surgical ICU patients [22]. Of these patients, 8.2% overall were classified as having ACS. Thus, the prevalence of IAH and ACS may be under-appreciated in critically ill patients.

In mechanically ventilated patients, abdominal compression results in decreased C_{RS} with flattening and rightward shift of the P/V curve of the respiratory system [5]. These changes are due to decreased C_{CW} , while C_L remains unchanged. A strong positive correlation between IAP and the lower inflection point of the respiratory system P/V curve has also been reported in conditions with increased IAP. This suggests that the IAP may be correlated with the best PEEP in ventilated patients with ARDS and IAH [5].

In deeply sedated patients with ARDS, the diaphragm behaves as a passive structure, and thus moves upward in the rib cage, transmits increased IAP to the lower lobes of the lung and causes compression atelectasis [23]. Surgical abdominal decompression has been shown to recruit lung volume and to increase the quotient of arterial partial pressure of oxygen to fraction of inspired oxygen (PaO_2/FiO_2) [24]. In animal models, high PEEP has been shown to improve gas exchange during carbon dioxide-induced pneumoperitoneum, resulting in increased

oxygenation and carbon dioxide elimination [25,26]. Putting the patient in an upright position increases IAP and decreases respiratory system compliance, suggesting that putting a patient upright may result in a deterioration of respiratory function in patients with IAH [5].

Using an experimental model, Valenza *et al.* [17^{••}] recently investigated the effects of IAH on P_{ES} and P_{CVP} obtained at end-exhalation (i.e. in static conditions) and during positive-pressure ventilation (i.e. in dynamic conditions). During anesthesia and paralysis, animals' abdomens were inflated with helium. IAP was measured directly by an intraperitoneal catheter and compared to simultaneous P_{ES} and P_{CVP} measurements. The dynamic component of the P_{ES} increased significantly after IAH and was correlated with peritoneal pressure. P_{ES} at end-exhalation (i.e. static) was, however, unaffected by abdominal inflation (Fig. 4). PEEP significantly influenced static but not dynamic P_{ES} during IAH. Similar to P_{ES} , the dynamic component of P_{CVP} rose with IAH, but P_{CVP} at end-exhalation was unaffected. Mean values of P_{CVP} increased significantly with IAH, whereas transmural P_{CVP} and intra-thoracic blood volume did not change significantly. The authors concluded that dynamic P_{ES} changes occurred during IAH, whereas the end-expiratory P_{ES} was affected only when high PEEP levels were applied. The absolute P_{CVP} value itself cannot be relied on to guide resuscitation in patients with IAH, particularly when abdominal pressures are changing over short periods of time.

Altered chest wall compliance in acute lung injury/acute respiratory distress syndrome

The most common causes of decreased chest wall compliance in ALI/ARDS patients are abdominal diseases such as bowel distension, ascites and hemoperitoneum. Differences have been reported in chest wall mechanics between patients with pulmonary ARDS, usually due to diffuse pneumonia, and in those with extrapulmonary ARDS, usually due to abdominal diseases [27]. Although C_{RS} is similar in patients with pulmonary and extrapulmonary ARDS, P_{PL} is normal in patients with pulmonary ARDS, but abnormally high in patients with extrapulmonary ARDS. There is a linear correlation between the increase of IAP and the increase in P_{PL} [28]. In patients with ALI/ARDS, IAH contributes to a reduction in lung volume and gas-exchange abnormalities. Patients with ACS often develop extrapulmonary ARDS. In patients with ACS and extrapulmonary ARDS, generous levels of PEEP may be necessary to counterbalance the collapsing effects on the lungs. Owing to the reduced C_{CW} , positive-pressure ventilation may result in a substantial increase in pleural pressure and hence a decrease in P_{TP} for a given plateau pressure (P_{PLAT}).

Using estimates of pleural pressure to set the ventilator

Ventilator-induced lung injury (VILI) can be reduced by avoiding injurious ventilator settings. The key elements of such a strategy are minimization of overdistension by limiting V_T and the use of sufficient PEEP to prevent cyclic alveolar collapse. In some clinical studies, lung-protective mechanical ventilation strategies were associated with lower mortality [29]. P_{TP} is an important consideration in the context of VILI [30,31]. P_{TP} is the distending pressure on the lungs:

$$P_{TP} = P_{ALV} - P_{PLP} \quad (6)$$

P_{ALV} is easily estimated at the bedside using an end-inspiratory breath-hold maneuver, i.e. P_{PLAT} . ΔP_{PL} is estimated from ΔP_{ES} or ΔP_{CVP} . For example, if P_{PLAT} (P_{ALV}) is 30 cm H₂O and P_{PL} increases by 5 cm H₂O as the lungs are inflated, the P_{TP} will be about 25 cm H₂O at end-inhalation. Note that a stiffer chest wall will result in a greater ΔP_{PL} and thus a lower P_{TP} . In other words, a stiffer chest wall will decrease alveolar distension. This may be lung protective, as it has been demonstrated in experimental animals where the degree of lung injury was ameliorated during positive-pressure ventilation by strapping the chest wall to reduce C_{CW} [32]. It also follows that a higher P_{PLAT} may be acceptable in the presence of a stiff chest wall. Moreover, in cases of severely reduced chest wall compliance (e.g. ACS), a higher P_{PLAT} may be necessary to allow adequate alveolar ventilation. It should also be noted that P_{TP} increases with active inspiratory muscle activity, which decreases P_{PL} , when P_{ALV} is held constant such as with pressure support or pressure-controlled ventilation. In this case, the decreased P_{PL} results in an increased P_{TP} which might contribute to alveolar overdistension [32].

The P/V curve of the respiratory system describes the mechanical behavior of the lungs and chest wall during inflation and deflation [33]. It has been applied in patients with ARDS in the hope that it might allow clinicians to customize ventilator settings according to a patient's individual respiratory mechanics and thus protect the patient from VILI. In ARDS, the P/V curve appears sigmoidal in the volume range between end-expiratory and end-inspiratory lung volumes. The lower inflection point (P_{FLEX}) has been used to set PEEP = $P_{FLEX} + 2$ cm H₂O. The upper inflection point is thought to represent alveolar overdistension and thus it has been recommended that the V_T should be set such that the P_{PLAT} is lower than the upper inflection point.

Abnormalities in the P/V curve in patients with ARDS have been generally attributed to alteration in lung

mechanics rather than to chest wall mechanics. It has, however, been shown that abnormalities in the chest wall affect the P/V curve of the respiratory system [23,34]. Ranieri *et al.* [24] reported that, in mechanically ventilated patients with ARDS, impairment of the P/V curve of the respiratory system varies greatly with the underlying disease responsible for ARDS. In patients in whom ARDS was related to major abdominal surgery (i.e. surgical ARDS), the inspiratory P/V curve of the respiratory system and lungs showed an upward convexity, indicating that compliance decreased with inflation, causing alveolar overdistension with inflating volume. Patients with ARDS not consequent to major surgery (i.e. medical ARDS) had an upward concavity on the inspiratory P/V curves of the respiratory system and lungs, indicating an increase in compliance and alveolar recruitment with lung inflation. This suggests that the flattening of the P/V curves of the respiratory system observed in some patients with ARDS may be due to a decrease in chest wall compliance related to abdominal distension. If chest wall effects are not considered, unnecessary restriction of V_T might occur in patients with surgical ARDS and overestimation of the PEEP requirement might occur in patients with medical ARDS (Fig. 5). Mergoni *et al.* [34] also studied the effects of chest wall mechanics on the respiratory system P/V curve in patients with acute respiratory failure. They found that the improvement in PaO₂ with PEEP is significant only in patients in whom the lower inflection point is on the lung P/V curve and not on the chest wall curve.

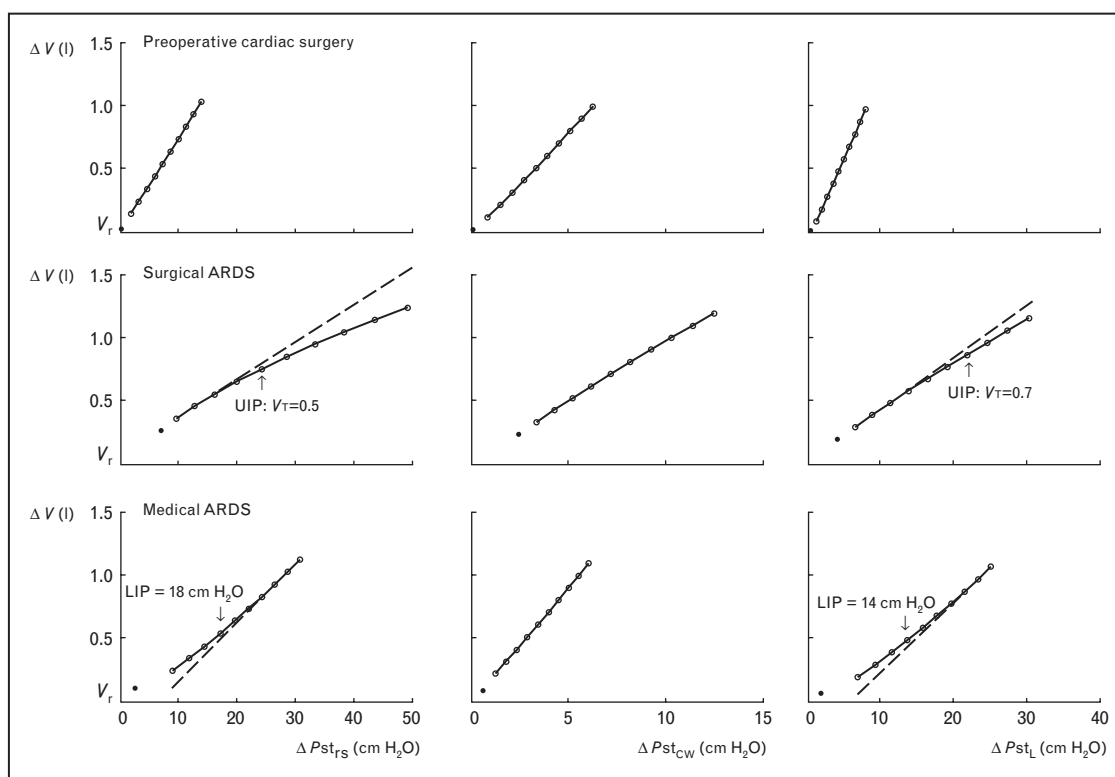
Talmor *et al.* [35**] used P_{ES} to estimate the influence of the chest wall on P_{PL} and P_{TP} in patients with acute respiratory failure. They reasoned that PEEP should be applied to achieve a positive P_{TP} . They subtracted 5 cm H₂O from each value of P_{ES} to correct for artifacts attributable to body position and balloon pressure, and then calculated P_{TP} from the difference between P_{AO} and corrected P_{ES} :

$$P_{TP} = P_{AO} - P_{ES} + 5 \text{ cm H}_2\text{O} \quad (7)$$

They reported a wide range of calculated P_{TP} at all values of P_{AO} , supporting the concern that P_{AO} is influenced by chest wall mechanics. They suggested that, by using this approach, ventilator settings could be more appropriately customized to accommodate inter-individual variations in lung and chest wall mechanical characteristics. Although this approach is attractive, it is potentially flawed by the assumption that corrected P_{ES} accurately reflects P_{PL} [11*,12**].

The effects of the prone position on respiratory mechanics and gas exchange during ALI have been

Figure 5 Static inflation P/V curves of the respiratory system (rs), chest wall (cw) and lung (L) in three representative preoperative cardiac surgery (top), surgical acute respiratory distress syndrome (ARDS) (middle) and medical ARDS (bottom) patients



Intrinsic positive end-expiratory pressure and the corresponding increase in end-expiratory lung volume relative to the elastic equilibrium volume of the respiratory system (V_r) are indicated on the P/V curves (closed symbols). The dotted line indicates the zone of lowest elastance determined by a step-by-step regression analysis on samples of four consecutive experimental points. ΔPst , changes in end-inspiratory static pressure; ΔV , changes in lung volume relative to V_r ; UIP, upper inflexion point; LIP, lower inflexion point. Reproduced from [24].

reported by Pelosi *et al.* [36]. In the prone position, PaO_2 increased without significant changes of C_{RS} and end-expiratory lung volume; however, C_{CW} decreased and the decrease was correlated with the oxygenation increase. Furthermore, the greater the baseline supine C_{CW} , the greater its decrease in the prone position and the oxygenation changes in the prone position were predictable from baseline supine C_{CW} . Returning to the supine position, C_{RS} increased compared with baseline, mainly because of C_L . It was concluded that baseline C_{CW} and its changes may play a role in determining the oxygenation response in the prone position.

Conclusion

Chest wall compliance may have important clinical implications during positive-pressure ventilation. The physiology of this effect should be considered during mechanical ventilation, particularly in patients with ALI and IAH.

References and recommended reading

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (p. 125).

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