

Static pressure–volume curves of the respiratory system: were they just a passing fad?

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Purpose of review

The aim of this article is to describe the physiologic utility, correlation with lung morphology, difficulties in interpretation and current clinical applications of static respiratory system pressure–volume curves at the bedside in patients with acute lung injury or acute respiratory distress syndrome.

Recent findings

Complex interpretation of pressure–volume curves indicates that alveolar reopening continues past the lower inflection point on the linear part of the curve and suggests the presence of homogeneous lung disease in which recruitment is still possible by positive end-expiratory pressure application. Setting positive end-expiratory pressure above the lower inflection point and tidal ventilation (approximately 6 ml/kg) in the linear portion of the respiratory system pressure–volume curve improved mortality and ameliorated lung and plasma inflammatory mediators compared with ventilation with the lowest positive end-expiratory pressure at traditional tidal volumes. Recent studies have found that regular use of pressure–volume curves provides useful physiological data that help to optimize mechanical ventilation at the bedside.

Summary

The physiologic data obtained by measuring the static pressure–volume curves have helped clinicians to better understand the behavior of the respiratory system when positive-pressure ventilation is applied. The advanced technology incorporated into modern ventilators allows routine measurement of pressure–volume curves under sedation without paralysis, with acceptable variability and no serious adverse effects.

Keywords

acute lung injury, acute respiratory distress syndrome, lung recruitment, mechanical ventilation, pressure volume curve

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Introduction

Pressure and volume are measured to characterize the mechanical properties of the lungs and chest wall in static conditions in patients with acute lung injury or acute respiratory distress syndrome (ARDS) receiving mechanical ventilation. Pressure–volume curves describe the optimal ventilator settings for maximum recruitment without end-inspiratory overdistension. Although bedside measurement of pressure–volume curves of the respiratory system is not a routine or even widely used diagnostic method, it can help clinicians understand physiological data and decide how best to set the ventilator.

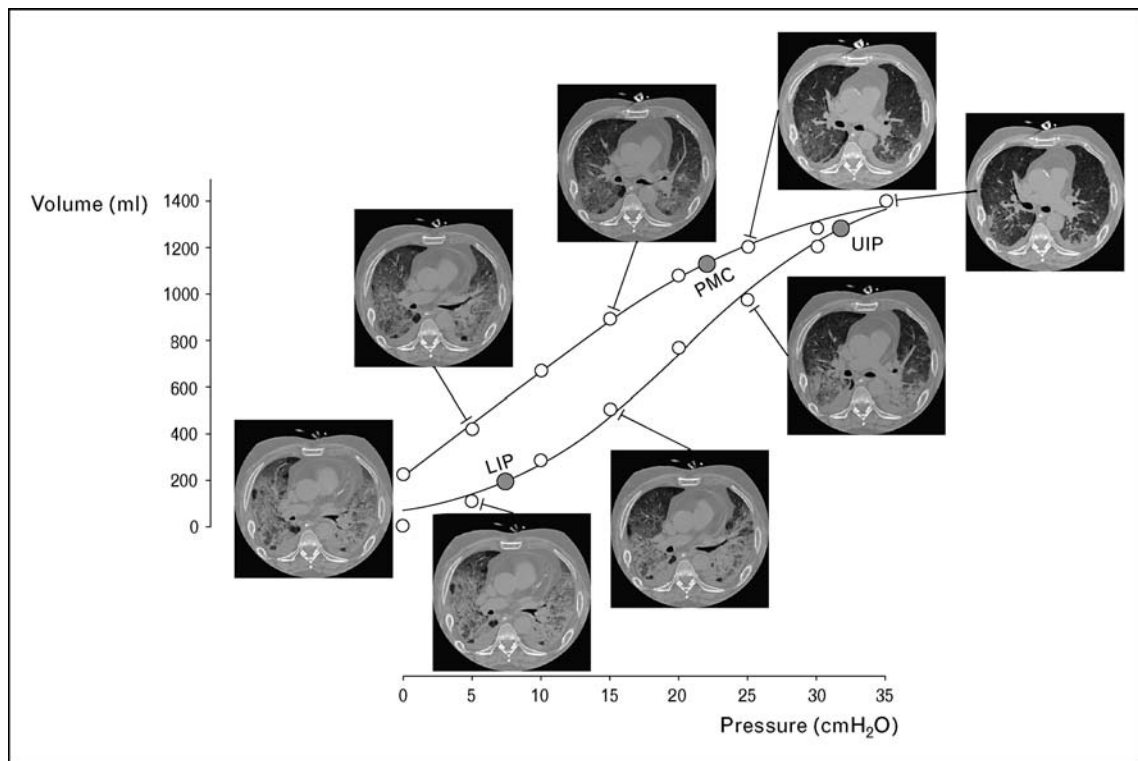
Physiology behind pressure–volume curves

The static respiratory pressure–volume curve usually assumes a characteristic sigmoidal shape, with a central

linear part and two inflection points respectively located at the beginning and at the end of the curve. In the classical interpretation of pressure–volume curves, the upper inflection point (UIP) indicates alveolar overdistension, while the lower inflection point (LIP) represents the point at which the airways or alveoli reopen. The physiologic meaning of the LIP can be understood by considering that, at the beginning of lung inflation, alveoli are less distensible and require high insufflation pressures to open. Respiratory system compliance (C_{rs}) is low and remains low until a critical pressure value is reached, and above this point the pressure–volume curve becomes linear (Fig. 1). During insufflation, the critical pressure point can be exceeded but at end-expiration the system returns to the resting volume below the critical point. This implies that in each act of breathing, the alveoli cyclically open and close, creating a shear stress trauma. The application of external positive end-expiratory pressure (PEEP) slightly above the

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Figure 1 Lung recruitment and derecruitment along the pressure-volume curve in a patient with early acute respiratory distress syndrome



Computed tomographic scan images were obtained while tracing the curve in static conditions. Note that recruitment only starts when airway pressure is higher than the lower inflection point (LIP) of the inspiratory limb and continues up to the maximum pressure reached, even above the upper inflection point (UIP). There is, however, no derecruitment when airway pressure decreases from this level to the expiratory point of maximum curvature (PMC). With pressures below this point, derecruitment starts and continues along the rest of the expiratory limb of the curve.

LIP prevents end-expiratory alveolar collapse and improves intrapulmonary shunt and gas exchange, probably as a result of effective lung recruitment. However, PEEP titration is not possible when the LIP is not identifiable on the pressure–volume curve, as occurs in late-stage ARDS patients, presumably because fibrosis replaces edema.

In the last decade, several studies have suggested a more complex interpretation of pressure–volume curves showing that alveolar reopening continues past the LIP on the linear part of the curve. Moreover, the LIP indicates the presence of homogeneous lung disease in which recruitment is still possible by PEEP application, even if the optimal level of PEEP cannot be deduced [1–3]. Finally, the UIP may indicate the end of recruitment and not necessarily the beginning of overdistension [4].

Correlation between pressure–volume curves and lung morphology

Static pressure–volume curves are a surrogate marker of the changes in lung parenchyma during inspiration

and expiration. This has been demonstrated by studying the correlation between pressure–volume curves and changes in lung aeration measured by computed tomography.

The hallmark of acute lung injury in tomographic studies is the loss of aeration (by alveolar collapse, fluid-filled airways, or a combination of both) [5]. This phenomenon is reflected in CT scans as an increase in density, and lung tissue has been divided in four compartments (hyper-aerated, normally, poorly and nonaerated) according to the proportions of gas and water in a given volume. From this point of view, we can define aeration as the increase in gas volume in previously aerated areas and recruitment as the increase of gas volume in previously nonaerated areas.

Several studies have demonstrated that the inspiratory limb of the pressure–volume curve is parallel to both aeration and recruitment curves [6,7]. For inspiratory pressures between 0 cmH₂O and the inspiratory LIP, the increase in lung volume is mainly due to aeration of normally and poorly aerated areas. When airway

pressure increases above the LIP, recruitment starts. Therefore, the LIP reflects the onset of recruitment of nonaerated areas in the lung [8]. It has been reported that the presence of an LIP in the curve indicates significant potential for recruitment [9]. As detecting this potential could have important consequences for patient management [10^{••}], it is tempting to speculate that pressure–volume curves could be a useful tool at the bedside.

Recruitment pressures have a Gaussian distribution that is responsible for the steeper part of the curve [11], with the inspiratory UIP reflecting the end of this massive recruitment. Some recruitment, however, takes place at pressures higher than this point [7,12[•]]. The amount and relevance of this ‘marginal’ recruitment is a matter of debate [13].

The overall correlation between the expiratory limb of the pressure–volume curve and derecruitment is not so good [11,14], probably due to the complex phenomena that occur during expiration, especially airway closure. The expiratory point of maximum curvature (the expiratory equivalent to the UIP), however, is a good marker of the onset of derecruitment [7]. If PEEP is aimed to completely avoid derecruitment, this point should be the target, although this practice is associated to hyperinflation of healthy areas [15]. In some cases, an expiratory lower inflection point is seen, but its significance remains unclear. Figure 1 illustrates the significance of the inflection points on the pressure–volume curve.

As pressure–volume curves are a global measurement, it is uncertain whether these findings are representative of what happens in single alveolar units. In an interesting in-vivo microscopy study, DiRocco and coworkers [16[•]] found that only the expiratory point of maximum curvature is a good marker of alveolar derecruitment. No correlation between the LIP and alveolar recruitment was found, however. Although this animal model is different from clinical acute lung injury, this experiment reinforces the idea that expiratory pressure–volume curves are a good method to study derecruitment and could therefore have some use in a PEEP setting.

Hyperinflation is the tomographic marker of lung overstretching [17], although these two phenomena can be different. In terms of volume, the overall contribution of hyperinflation to the shape of the pressure–volume curve is small, and it is masked in the bulk of aeration and recruitment. Some authors have advocated the use of the inspiratory UIP as a marker of the onset of overstretching [18], but no studies using CT scans have demonstrated this point. Therefore, the usefulness of pressure–volume curves to detect hyperinflation and overstretching in the injured lung is not clear.

Reproducibility of pressure–volume curves

It has been documented that the different techniques available to trace pressure–volume curves yield quite reproducible results. Using the syringe method, Mehta and coworkers [19] report a good reproducibility of the inspiratory curves, with a difference lower than 3 cmH₂O in the LIP in three successive curves. Automated low-flow methods incorporated in recent mechanical ventilators probably result in an even better reliability of the curves.

The interpretation of the results, in terms of determination of the inflection points, however, has a substantially lower reliability. Inflection points are identified by eye fitting by drawing two or more straight lines that fit to the data points. The intersections of these lines represent the inflection points. This method [20] has a high inter-observer variability (up to 11 cmH₂O), although other authors have reported better concordance [19]. This variability is dependent on the data points available, and their scatter (or the range of values explored during the maneuver) influences the results [21].

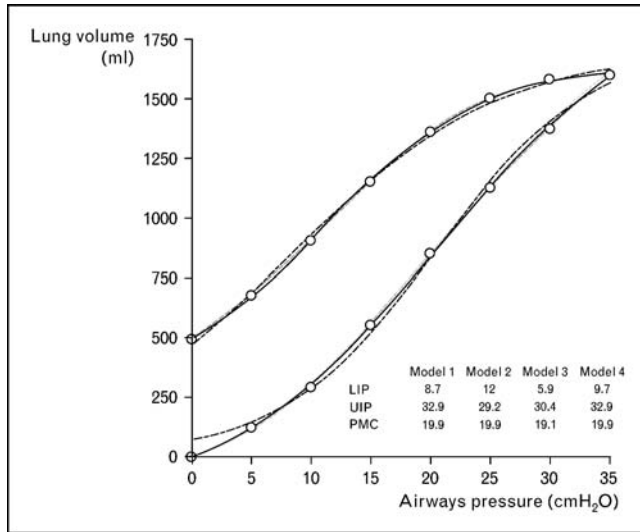
To avoid these limitations, several mathematical models have been developed to fit the experimental data and calculate inflection points objectively [22–24]. These models overcome interobserver variability and the problems derived from data scattering. Different mathematical models, however, can also yield disparate results. It has been reported [25[•]] that the differences between models for a concrete inflection point can be as high as 6 cmH₂O (for the same curve fitted to two different equations), despite excellent goodness-of-fit (Fig. 2). This is probably because these models were developed to fit the data with no ‘mechanistic’ reasoning behind the formula. Additionally, inflection points probably represent ‘transition zones’ (i.e. pressure ranges for which abrupt changes in the lung parenchyma occur) rather than single points.

Therefore, a mathematical model should be used to extract results from pressure–volume curves, and the nature of this model should be carefully reported and taken into account, especially when comparing different studies.

Airway pressure as a function of time as surrogate of pressure–volume curve

The inspiratory portion of the dynamic airway pressure as a function of time (P/t curve) can be used to determine alveolar behavior, since equal incremental amounts of volume per time unit are obtained during constant flow inflation in volume-controlled ventilation. At airway opening, the rate of changes in pressure is related to

Figure 2 Mathematical models of pressure-volume curves

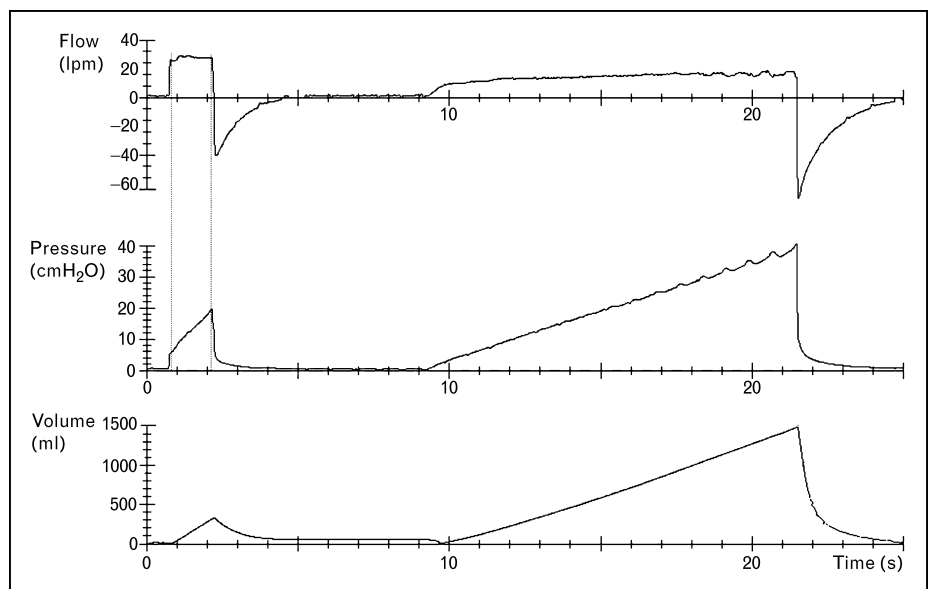


The same data points were adjusted to four mathematical models, and the inflection points were calculated. Note that, for the same curve, the values of the inflection points can be substantially different, even though the curves are mostly superimposed. These differences are more important for points on the inspiratory limb of the curve. LIP, lower inflection point; PMC, point of maximum curvature; UIP, upper inflection point Modified with permission [25*].

changes in alveolar compliance, if resistances remain constant. Airways opening pressure increases linearly when compliance does not change with increasing lung volume. Otherwise, the *P/t* profile can indicate alveolar recruitment or overinflation, depending on whether its shape is convex or concave, respectively [26].

Figure 3 A normal breath in volume-controlled ventilation with constant flow is followed by a pressure–volume curve maneuver using the constant low-flow method

Dotted lines define constant flow inflation and highlight the portion of the airways-pressure/time profile (*P/t*) that has to be considered to detect alveolar recruitment or overdistension.



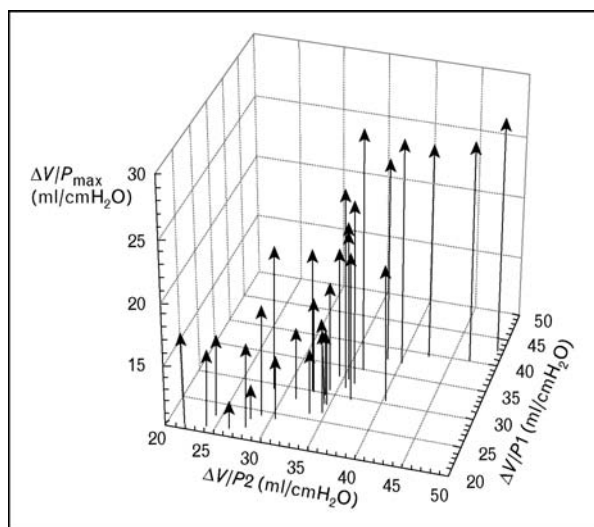
A *P/t* curve obtained with constant low-flow inflation can also be used as a surrogate pressure–volume curve at the bedside (Fig. 3) [27,28]. Analyzing *P/t* curves using a classical sigmoidal equation [22] enables the shape, LIP, and UIP to be accurately determined and yields results comparable to those obtained with traditional pressure–volume curves. This simple method makes important clinical information readily available at the bedside without the technical support required to perform the standard pressure–volume curve.

Static versus dynamic compliance

From a clinical point of view, it is important to make a distinction between static ($C_{rs,stat}$) and dynamic respiratory system compliance ($C_{rs,dyn}$) [29].

In mechanically ventilated patients, C_{rs} is easily obtained at the bedside by the rapid airway occlusion technique during constant inspiratory flow. C_{rs} is calculated as the change in volume (ΔV) divided by the change in airway pressure (ΔP) under the assumption of zero airflow. When flow becomes null, the airway pressure curve is characterized by a rapid decrease from a maximum (P_{max}) to $P1$, followed by a slow decrease until a plateau pressure level ($P2$) is reached. The differences between $P1$ and $P2$ are due to pendelluft and viscoelastic lung and chest wall rearrangement. In a healthy subject, this difference is negligible; however, in patients with acute and chronic lung disease, this disparity is clinically relevant. For this reason, while $P1$ could be used instead of $P2$ for the calculation of the C_{rs} in healthy patients, the $P2$ value must be used in patients with suspected lung pathology.

Figure 4 Three-dimensional correlation of $\Delta V/P_{\max}$, $\Delta V/P1$, and $\Delta V/P2$ in 32 mechanically ventilated patients



$\Delta V/P_{\max}$ is markedly different from $\Delta V/P2$, highlighting inequality between static and dynamic compliance. Less disparity between $\Delta V/P1$ and $\Delta V/P2$ is noted in healthy lung.

To avoid misunderstandings, it is useful to consider the true alveolar pressure ($P2$) to calculate $C_{rs,stat}$ ($\Delta V/P2$) and to keep in mind that lung distensibility measured employing $P1$ does not account for postocclusive phenomena.

Moreover, $C_{rs,dyn}$, which considers P_{\max} ($\Delta V/P_{\max}$), is a spurious estimation of alveolar distensibility because its value is strongly affected by flow-dependent resistances. $C_{rs,dyn}$ is actually a measure of respiratory system impedance because it considers the sum of elastic and resistive pressures applied to the patient. At present, the utility of $C_{rs,dyn}$ to assess recruitment and derecruitment after full lung inflation is confined to very recruitable experimental models of ARDS [30*].

Three simultaneous measurements, the ratio of ΔV to P_{\max} , ΔV to $P1$, and ΔV to $P2$, in 32 mechanically ventilated patients are reported in Fig. 4. All patients show a marked discordance between $\Delta V/P_{\max}$ and the other two ratios. In patients with homogeneous lung parenchyma, the ratios $\Delta V/P1$ and $\Delta V/P2$ have similar values, while in those with lung disease the difference is amplified.

Contribution of hysteresis and chest wall to the shape of pressure–volume curve

Pulmonary hysteresis is a phenomenon in which the pressure–volume relationship is different during inspiration and expiration. The energy applied to the lung in inspiration is not recovered in expiration because alveolar recruitment is an energy-dissipating process. Hysteresis

is expressed in terms of surface area between ascending and descending portions of the pressure–volume loop. When a pressure–volume curve maneuver is performed, hysteresis can be produced by the following possible mechanisms: recruitment and derecruitment of lung units; volume and time-dependent molecular reorganization of the surface-active material that coats air–liquid interfaces in alveoli and conducting airways; stress relaxation and stress recovery of airways and lung parenchyma; spurious changes in lung volume caused by gas adsorption or artifacts due to thermodynamics or gas exchange, which is a well described problem of the super syringe technique [31,32].

During a typical clinical interpretation of a pressure–volume curve, the elastic contribution of the chest wall is assumed to be negligible. It is well known, however, that chest wall compliance is reduced in mechanically ventilated acute lung injury patients and in obese patients undergoing mechanical ventilation. Partitioning the pressure–volume curve into chest wall and lung components allows the separate influence of each constituent on pressure–volume morphology to be analyzed. The presence of an LIP on a pressure–volume curve can be mainly due to the chest wall per se rather than lung characteristics in acute lung injury patients [33].

Current clinical applications of pressure–volume curves at the bedside

Three randomized studies in which tidal volume and PEEP were selected on the basis of the findings obtained from pressure–volume curves drawn in zero PEEP conditions have contributed interesting data [34,35,36**]. Setting PEEP above the LIP and tidal ventilation (approximately 6 ml/kg) in the linear portion of the respiratory system pressure–volume curve (i.e. between the lower and the upper inflection points) improved mortality and reduced plasma and bronchoalveolar lavage concentrations of inflammatory mediators compared with ventilation with the lowest PEEP at tidal volumes in the range of 10–12 ml/kg.

Moreover, recent studies have found that regular use of pressure–volume curves provides useful physiological data that help to optimize mechanical ventilation at the bedside. Empirical settings of PEEP and tidal volume frequently fail to induce alveolar recruitment and may increase alveolar overdistension [37]. Aboab *et al.* [38**] examined whether ventilation with 100% oxygen induces derecruitment in patients with ARDS. Recruited volume at two PEEP levels was computed from two pressure–volume curves, recorded from PEEP and from zero end-expiratory pressure, using the sinusoidal flow modulation method. Interestingly, they found that pure oxygen leads to derecruitment, but that

applying high PEEP prevented this derecruitment. Patients with ARDS present collapse of the lung basis adjacent to the diaphragm, and it is difficult to recruit those areas to improve lung function. Richard *et al.* [39*] measured pressure–volume curves of the respiratory system recorded from PEEP and changes in lung volume to assess time-dependent increase in recruitment associated with vertical position (trunk elevated at 45° and legs down at 45°). Compared with supine positioning, vertical positioning increased Pa_{O_2} significantly from 94 ± 33 to 142 ± 49 mmHg, with an increase higher than 40% in 11 responders. The volume at 20 cmH₂O of airway pressure measured on the pressure–volume curve from PEEP increased using the vertical position only in responders. Demoule *et al.* [40] found a correlation between chord compliance measured on pressure–volume curves obtained at two levels of PEEP (0 and 10 cmH₂O) and biological markers of collagen turnover or surfactant degradation in bronchoalveolar lavage fluid obtained simultaneously in the early phase of the ARDS. This suggests that when chord compliance is severely impaired (here below 28 ml/cmH₂O), the reduction in compliance may be associated with an intense ongoing fibroproliferative and inflammatory process characterized by inextensible collagen fiber deposition. Finally, Terragni *et al.* [41**] found that limiting tidal volume to 6 ml/kg of predicted body weight and plateau pressure to 30 cmH₂O may not be sufficient to avoid superimposed lung injury in ARDS patients, characterized by a larger nonaerated compartment. Therefore, lung-protective ventilation might not be enough to fully prevent ventilator-induced lung injury.

Fine measurement of pulmonary mechanics using pressure–volume curves has been performed at the bedside for decades. Although these measurements have not directly influenced outcome in patients with ARDS, the physiologic data obtained has helped clinicians to better understand the behavior of the respiratory system when positive-pressure ventilation is applied and has thus influenced the design of treatment protocols to improve survival in ARDS.

The advanced technology incorporated into modern ventilators allows routine measurement of pressure–volume curves under sedation without paralysis, with acceptable variability and no serious adverse effects [27,42*,43]. Pressure–volume measurements could be useful for fine tuning the ventilator, even when lung-protective ventilation strategies are applied in patients with severe ARDS.

Conclusion

Static pressure–volume curves correlate with the changes in the morphology of lung parenchyma during passive

inflation and deflation in patients with ARDS. The different techniques available to trace static pressure–volume curves at the bedside yield quite reproducible results. The assessment of the different inflection points, however, has a high interobserver variability. Nowadays, physiologists and clinicians consider that inflection points probably represent pressure ranges or transition zones where abrupt changes in the lung parenchyma occur rather than single points. Recent studies have found that regular use of pressure–volume curves provides useful physiological data that help to optimize mechanical ventilation at the bedside and more interestingly to improve outcome.

References and recommended reading

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

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- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 110–111).

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