

Review

Overdistension in ventilated children

Véronique Nève, Francis Leclerc, Eric Dumas de la Roque, Stéphane Leteurtre and Yvon Riou

Service de Réanimation Pédiatrique, Centre Hospitalier et Universitaire de Lille, Lille, France

Correspondence: Francis Leclerc, fleclerc@chru-lille.fr

Published online: 13 July 2001

Critical Care 2001, 5:196–203

© 2001 BioMed Central Ltd (Print ISSN 1364-8535; Online ISSN 1466-609X)

Abstract

Ventilating patients with acute respiratory failure according to standardized recommendations can lead to varying volume–pressure (V-P) relationships and overdistension. Young children may be more susceptible than adults to overdistension, and individual evaluation of the effects of ventilator settings is therefore required. Three studies have applied indices for the detection of overdistension to dynamic V-P curves in ventilated children. Two of those studies compared these indices to those obtained using a reference technique ([quasi]-static V-P curves), and suggested that the c coefficient of a second order polynomial equation (SOPE) and the ratio of the volume-dependent elastance to total dynamic elastance ($\%E_2$) were suitable indices for estimating overdistension.

Keywords constant flow insufflation, low flow inflation, overdistension, ventilated children, volume–pressure curve

Analysis of respiratory V-P curves is increasingly being included in the evaluation of whether ventilator settings are suited to the mechanical properties of the respiratory system of the patient. In dynamic conditions, resistive forces, the resistance of the conducting airway and the endotracheal tube (ETT), and viscoelastic or inertial forces, adaptation to stress of units within the lung and the chest wall tissues, are measured. In order to obtain information regarding lung elastic forces, dynamic forces must be eliminated and respiratory muscles relaxed.

The static V-P curve obtained in normal persons has a sigmoidal shape, with a linear segment above functional residual capacity (FRC) where tidal ventilation takes place, two inflection points, and a hysteresis curve. The lower inflection point (LIP) is observed at lung volume lower than FRC, and is therefore not observed in lungs of normal persons above relaxation volume. The upper inflection point (UIP) is observed at near total lung capacity.

In patients with the acute respiratory distress syndrome (ARDS), the classical shape of the V-P curve is altered. Abnormalities include appearance of a LIP above FRC, of a UIP at lower lung volume than in normal persons, decreased slope in the linear portion, and increased hysteresis. The mechanical abnormalities are dependent on the stage of disease [1].

Animal studies [2–6] have shown that mechanical ventilation can initiate or worsen pre-existing lung injury. Ventilator-induced lung injury (VILI) appears to result from repetitive closing and opening of collapsed alveolar units, or from pulmonary overdistension. Both of these exacerbate or initiate significant lung injury and inflammation [4,5,7]. In animal studies [5], VILI is reduced by setting the positive end-expiratory pressure (PEEP) level slightly higher than the LIP of the thoracopulmonary V-P curve, thus avoiding repetitive closing and opening of collapsed alveolar units and keeping the lung open. Also,

ARDS = acute respiratory distress syndrome; $\%E_2$ = ratio of the volume-dependent elastance to total dynamic elastance; EELV = end-expiratory lung volume; ETT = endotracheal tube; FRC = functional residual capacity; LFI = low flow inflation; LIP = lower inflection point; Pao = airway opening pressure; PEEP = positive end-expiratory pressure; SOPE = second order polynomial equation; UIP = upper inflection point; V' = constant inspiratory flow; VDSCM = volume-dependent single compartment model; VILI = ventilator-induced lung injury; V-P = volume–pressure; V_T = tidal volume.

an appropriate tidal volume (V_T) helps to avoid end-inspiratory overdistension.

The results of randomized prospective trials conducted in ARDS patients that compared protective ventilation strategies with more conventional strategies [8–11] suggested that VILI may have a clinical counterpart known as ventilator-associated lung injury. In one study [9], a strategy that combined recruitment manoeuvres, lower V_T and higher PEEP (adjusted to maintain the tidal ventilation above the LIP of the static V-P curve) improved survival and decreased the incidence of barotrauma. In another study, conducted in 861 patients [10], a 25% decrease in mortality was achieved by reducing the stress on the diseased lung by reducing the V_T . In a third study [11] lower concentrations of inflammatory mediators in bronchoalveolar lavage fluid and blood were observed in a group of patients ventilated with lower V_T and higher PEEP (set according to the LIP and the UIP of the V-P curve of the respiratory system) as compared with patients ventilated according to conventional criteria.

Therefore, in ARDS it has been proposed [12] that PEEP should be set above the LIP. The UIP has been suggested [13,14] to be the upper limit of adaptation of V_T . It correlates with pressure and volume values above which a decrease in compliance is observed, and with an increased risk of overdistension.

Young children have a more compliant chest wall than do adults [15]. They may therefore be more prone to lung overdistension, because at a given airway pressure the relative degree of lung distension is greater than that in adults [16]. Many analyses of respiratory V-P curves in ventilated adults, including detection of overdistension on (quasi)-static and dynamic V-P curves, have been reported (for review [17]). However, very few studies have described results regarding detection of overdistension on (quasi)-static and dynamic V-P curves in ventilated children. The present review therefore summarizes the use of V-P curves in the paediatric population as a guide to ventilatory therapy, with particular focus on detection of overdistension.

Techniques to measure the static or quasi-static properties of the respiratory system

The super syringe technique, which is considered the reference technique, allows a V-P curve to be constructed by insufflating increasing gas volumes while recording airway pressure during a period of constant volume. This technique has been abandoned because it necessitates disconnection of the patient from the ventilator, and because gas exchange during measurement complicates interpretation.

The occlusion technique allows V-P curves to be constructed in the volume-controlled mode of ventilation, by

changing respiratory frequency in order to obtain various V_{Ts} and by using the end-inspiratory button of the ventilator to identify the corresponding plateau pressure. As with the super syringe technique, however, this technique is time consuming, and its use is limited in clinical practice.

The easiest way to obtain a complete inspiratory V-P curve during one slow insufflation and without disconnecting the patient from the ventilator is to use the low flow inflation (LFI) technique. This method is rapid, and it can be performed using a modern ventilator; the ventilator ensures a constant inspiratory flow (V'), and the software and the screen allow the V-P curve to be observed and analyzed. Provided that the insufflation V' is sufficiently low (<9 l/min), a quasi-static state can be reached [18]. Higher V' increases the resistive pressure, and leads to a slight displacement of the V-P curve to the right, although this does not appear clinically relevant; the mean increase in resistive pressure induced by constant inspiratory V' of 3 and 9 l/min are 1 ± 1 and 1.8 ± 2.1 cmH₂O, respectively [18]. The LFI technique has been compared to reference techniques, with good results [19].

Detection of overdistension on dynamic respiratory volume–pressure curves in ventilated children

Analysis of the V-P curve obtained under dynamic conditions takes resistive, viscoelastic and elastic pressures into account. Because dynamic V-P curves are obtained during mechanical ventilation without modification of ventilator settings or artificial pauses, they are more relevant to the evaluation of potential lung trauma generated in such circumstances.

To our knowledge, only three studies regarding indices of dynamic V-P curves in ventilated children [20–22] have quantified the decrease in slope (ie compliance) that is observed at the limit of V_T in case of overdistension.

Fisher *et al* [20] quantified the decrease in compliance at the end of tidal inspiration using the C_{20}/C ratio, which was calculated on the inspiratory part of the V-P curve obtained during mechanical ventilation. This index represents the ratio of the compliance calculated from the last 20% of V-P curve (C_{20}) to the total compliance calculated from the entire slope of the curve (C). The equations for C and C_{20} are as follows.

$$C_{20} = (V_T - V_{0.80 P_{max}}) / (P_{max} - 0.80 P_{max}) \quad (1)$$

Where V_T is inspiratory V_T ; $V_{0.80 P_{max}}$ is the volume at 80% of maximum inspiratory pressure; P_{max} is the airway opening pressure at the zero point flow corresponding to end inspiration; and $0.80 P_{max}$ is 80% of maximum inspiratory pressure.

$$C = V_T / P_{\max} - P_{\min} \quad (2)$$

Where P_{\min} is airway opening pressure at the zero flow point, corresponding to the beginning of inspiration.

Those investigators analyzed the V-P curves of neonates ventilated in volume-controlled mode. They used the shape of the V-P curve as the 'gold standard', identifying overdistension by visual inspection. They observed that all patients with evidence of overdistension on the V-P curve had a C_{20}/C ratio below 0.80, and that all those with a normal-appearing loop had a C_{20}/C ratio greater than 1.00. They did not observe any overlapping of the C_{20}/C ratios calculated in nonoverdistended and in overdistended curves. However, these findings obtained in dynamic V-P curves were not compared with results obtained using a static V-P curve reference technique.

Kano *et al* [21] compared the C_{20}/C ratio with $\%E_2$ obtained in dynamic V-P curves in children aged 3–66 months, ventilated in the volume-controlled or the pressure-controlled mode. Those investigators calculated respiratory mechanics using multiple linear regression to fit a volume-dependent single compartment model (VDSCM), expressed as follows.

$$P_{ao} = (E_1 + E_2V)V + R_{rs} V' + EEP \quad (3)$$

Where P_{ao} represents airway opening pressure; $E_1 + E_2V$ represents total dynamic elastance of the respiratory system (E_1 is the volume-independent elastance, and E_2V is the volume-dependent elastance); R_{rs} is the dynamic respiratory system resistance; and EEP is the alveolar pressure at end-expiration. $\%E_2$ reflects the contribution of the volume-dependent elastance (E_2V_T) to total dynamic elastance of the respiratory system for a given V_T ($E_1 + E_2V_T$):

$$\%E_2 = (E_2V_T / [E_1 + E_2V_T]) \times 100 \quad (4)$$

Different degrees of inflation were obtained by changing the level of PEEP. However, no static V-P curves were obtained for reference purposes, which would have allowed the V-P curves to be classified into two groups: overdistended and nonoverdistended. Increasing PEEP decreased the C_{20}/C ratio and increased $\%E_2$. That study had the advantage of showing the effect of ETT on these two indices. Subtraction of the resistive pressure drop across the ETT from P_{ao} induced a systematic decrease in C_{20}/C , being most marked with the smallest ETT (3.0–4.0 mm internal diameter), with no apparent change in $\%E_2$. The investigators also emphasized the influence of the mode of ventilation on the C_{20}/C ratio. Indeed, in the pressure-controlled mode of ventilation, the last part of the V-P curve may not reflect the respiratory system compliance. This is because, once the predetermined pressure is approached, the inspiratory V' is decreased to maintain

pressure and to allow the V_T to be delivered during the inspiratory period. Kano *et al* concluded that no clear threshold value corresponding to overdistension could be determined for the C_{20}/C ratio, which was influenced by ETT size, airway resistance and mode of ventilation.

Finally, the C_{20}/C ratio may be compared with the sign of the nonlinear coefficient c of a SOPE, which is fitted to the V-P data obtained during a period of constant V' in children ventilated in the volume-controlled mode [22]. The governing equation is as follows:

$$\Delta V = a + bP + cP^2 \quad (5)$$

Where a , b , c are constants, c being the dynamic nonlinear coefficient.

Ranieri *et al* [23], in adult patients with ARDS, showed that the sign of this coefficient c describes the curvature of the V-P curve. When a convex shape with a progressive decrease in slope with increasing inflation volume was observed, the coefficient c was negative, indicating overdistension. When a concave shape with a progressive increase in slope with increasing inflation volume was observed, the coefficient c was positive. Figure 1 illustrates the detection of overdistension using the C_{20}/C ratio and coefficient c in two representative patients.

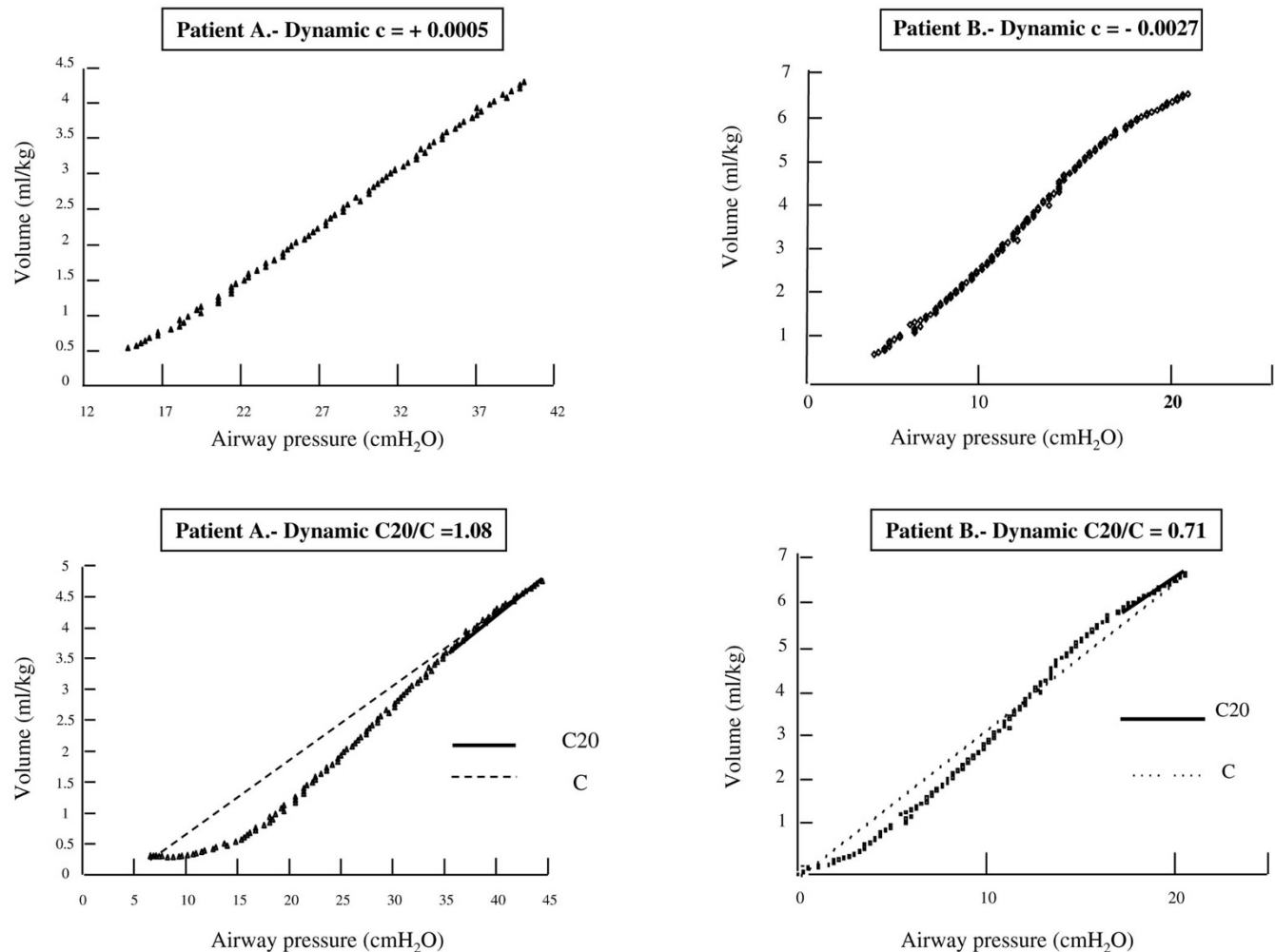
Validation of the detection of overdistension on dynamic volume–pressure curves by comparison with a reference method

Two of the studies referred to above [21,22] are interesting because they aimed to validate indices of overdistension obtained in dynamic conditions in comparison with an index of overdistension obtained using a reference method, such as (quasi)-static V-P curves.

In mongrel puppies, Kano *et al* [21] compared the C_{20}/C ratio and $\%E_2$ calculated from dynamic V-P data with detection of overdistension in static expiratory V-P curves. Dynamic V-P curves that showed various degrees of lung inflation were obtained by increasing V_T or PEEP. Respiratory system overdistension was judged by superimposing each dynamic V-P curve on the static V-P curve for that animal. Dynamic V-P curves were classified as nonoverdistended if they fell entirely within the linear portion, and as overdistended if they extended into the nonlinear region of the static curve. Respiratory mechanics were calculated using multiple linear regression to fit different models.

The best fit, especially in the overdistended group, was with a VDSCM (see Equation 3, above). Overdistension was quantified using $\%E_2$. Overdistended dynamic V-P curves showed higher $\%E_2$ ($43\% \pm 15\%$ versus $0.51\% \pm 18\%$) and lower C_{20}/C ratio (0.71 ± 0.1 versus 0.92 ± 0.16) than did nonoverdistended dynamic V-P curves. C_{20}/C ratio and

Figure 1



Detection of overdistension using the C_{20}/C ratio and c coefficient on the same dynamic volume–pressure curves of two representative patients. The C_{20}/C ratio was calculated on the inspiratory part of the V–P loop obtained during mechanical ventilation. It is the ratio of the compliance calculated from the last 20% of the inspiratory V–P curve (C_{20}) to the total compliance calculated from the entire slope of the inspiratory curve (C) between zero flow points. A C_{20}/C ratio below 0.80 is indicative of overdistension [20]. The nonlinear coefficient c of a SOPE (see Equation 5) was fitted to the V–P data obtained during a period of constant flow. The sign of this coefficient c describes the curvature of the V–P curve. When a convex shape with a progressive decrease in slope with increasing inflation volume is observed, the coefficient c is negative, indicating overdistension; when a concave shape with a progressive increase in slope with increasing inflation volume is observed, the coefficient c is positive [23]. For patient A, the positive value of coefficient c and the C_{20}/C ratio in excess of 0.80 were not indicative of overdistension. For patient B, the negative value of coefficient c and the C_{20}/C ratio below 0.80 were both indicative of overdistension.

$\%E_2$ were inversely correlated. Compared with a linear single compartment model ($P_{ao} = E_{rs}V + R_{rs}V' + EEP$, where E_{rs} is total dynamic elastance of the respiratory system), the volume-dependent elastance (E_2V) should only become significant if the lung is either overventilated (overdistension of some lung units) or underventilated (atelectasis). Comparison of the fit of the linear single compartment model with that of the VDSCM suggested that overdistension could be assumed if the $\%E_2$ was found to be greater than 30%.

The second study [22] compared the value of an overdistension index obtained under dynamic conditions with the detection of overdistension using a reference method, such as quasi-static V–P curves. This is the only study of this type conducted in ventilated children. Data from dynamic V–P curves were analyzed during constant V' ventilation (because the rate of change in pressure during constant V' insufflation indicates a change in compliance, provided that resistive and viscoelastic contributions to pressure remain relatively constant over the range of V_T

[23,24]) and by calculating the C_{20}/C ratio between zero flow points corresponding to the beginning and end of inspiration.

Dynamic V-P data were obtained at the airway opening in the volume-controlled mode of ventilation [22]. Then, quasi-static V-P curves (reference technique) were obtained using a very low constant insufflation V' (the LFI technique of Servillo *et al* [19]) in order to minimize resistive pressure and to achieve quasi-static conditions. The LFI technique allows quasi-static V-P curves to be constructed easily and rapidly, and without disconnecting the patient from the ventilator. It provides a continuous volume recording, comprising an ordinary tidal breath and the LFI. It also allows determination of whether the current V_T is within the optimal segment of the V-P curve. Finally, this approach allows results for the detection of overdistension on dynamic V-P curves to be compared with those with the quasi-static LFI technique for the same insufflated volumes, by using the C_{20}/C ratio or the c coefficient.

Two analyses were performed on the same data to compare detection of overdistension on dynamic with that on LFI curves, the latter being considered the reference technique. First, a mathematical analysis of the curvature of the V-P curve was conducted. A SOPE (see Equation 5, above) was fitted to the dynamic V-P data points obtained during constant V' inflation, and to the LFI V-P data corresponding to the dynamic V_T range. The sign of the nonlinear coefficient c obtained under dynamic conditions (dynamic c coefficient) was compared with the sign of the c coefficient obtained under LFI conditions (LFI c , reference technique). V-P data points obtained during constant V' inflation, after the initial step change in airway pressure due to the resistive component of the respiratory system, were analyzed. They provided the same information as did the static V-P curve regarding the elastic properties of the respiratory system in paralyzed patients. Second, a graphic analysis was conducted. The dynamic C_{20}/C ratio was compared with determination of the UIP on the LFI curve. Overdistension was defined as a negative c value [23], a C_{20}/C ratio below 0.80 [20], and a UIP that fell within the V_T range for that child during regular ventilation.

Comparing the results of the dynamic with the quasi-static V-P curve (reference technique), the C_{20}/C ratio did not appear to be a suitable index for estimating overdistension on dynamic V-P curves, because it failed to detect overdistension in three out of four children. However, the SOPE applied to the period of constant V' of the dynamic V-P curve appeared a more adequate and sensitive index of overdistension, because assessment of the coefficient c in dynamic V-P curves detected all cases of overdistension that were detected on the LFI quasi-static V-P curves.

Particularities of the measurements of volume–pressure data in ventilated children

In adult patients, P and V' measurements may be obtained using the standard sensors of the ventilator, and may be assessed using a ventilator screen, such as the Cesar ventilator screen (Taema, Paris, France) [25]. In young children measurements must be performed at the Y piece of the circuit, because the volume as measured by the sensors of the ventilator may be overestimated compared with the volume measured at the airway opening [26]. Circuit compression volume and compliance of the circuit have an important impact on the pressure and V' delivered at the Y piece. Circuit compression volume is that part of V_T set on the ventilator that remains within the ventilator and inspiratory circuit, and does not reach the airways of the patient. As the pressure in the circuit rises, the system tubing elongates and distends, causing compression of the gas within the inspiratory circuit. During exhalation, compression volume is released and measured by the exhalation valve.

Compression volume depends on the internal volume of the ventilator and humidifier, and on the volume and compliance of the circuit, but also on inflation pressure, which is influenced by lung resistance and compliance. Compression volume and circuit compliance (ratio of compression volume to maximal inspiratory pressure at which that compression volume is measured) increase as lung compliance decreases, reflecting a greater gas compression at higher maximal inspiratory pressure [27]. Because the dynamic respiratory system resistance in young children is higher than in adults, and their thoracopulmonary compliance is lower, especially in those with the most severe respiratory disease, the compression volume represents a larger portion of the V_T that is set on the ventilator.

In addition, pressure reaching the airways of the child may differ from the pressure that is set on the ventilator, because of a pressure drop between the two sites of measurement (inspiratory part and expiratory part of the circuit or Y piece) [28]: the maximal inspiratory pressure that is measured by the sensors of the ventilator may be overestimated in the volume-controlled mode of ventilation [29], and underestimated in pressure-controlled mode [30].

Quasi-static V-P curves can be constructed in children who have received an injection of muscle relaxant (eg vecuronium 0.2 mg/kg) using the LFI technique [19]. In such a procedure, the following must be selected: a low V' insufflation performed in the volume-controlled mode of ventilation, set with an insufflation to total time ratio of 0.5; a prolonged low V' inspiration of 6 s, obtained by setting the minimum value of frequency of the ventilator, ie five breaths/min; and a V_T that results in an expected maximal inspiratory pressure of approximately 40 cmH₂O. For example, in a child with a compliance of 5 ml/cmH₂O, the V_T would be $5 \times 40 = 200$ ml, and would be delivered at a

V' rate of 200 ml/6 s = 33 ml/s. In small children, however, the V' rate ($V_T/6$ s) and corresponding volume set on the ventilator should be corrected for gas compression in ventilator, humidifier and tubing (because compression volume represents a larger part of their V_T), and especially in those with low compliance.

This LFI technique can be performed at any PEEP level, and can be combined with calculation of recruited volume [31]. The gas volume of collapsed lung units recruited using PEEP (recruited volume) can be calculated as the difference in lung volume between zero end-expiratory pressure and the selected PEEP level. Evaluating the aspect of the V-P curve with the PEEP that corresponds to the actual ventilator settings has several advantages. Firstly, it provides a more realistic individual evaluation of the effects of the ventilator settings on the lung. In addition, keeping a sufficient level of PEEP may be particularly important in young paralyzed children, in order to maintain their end-expiratory lung volume (EELV). During spontaneous ventilation, children aged up to 6–12 months dynamically maintain their EELV above the volume determined by the mechanical properties of the system, by using diaphragmatic activity to retard expiratory flow [32]. With muscle paralysis, ventilated children can no longer use this strategy, and PEEP is required to maintain their EELV.

Difference between (quasi)-static and dynamic volume–pressure curves

The importance of dynamic pressure recordings has recently been emphasized [33], because the goal of the measurement is to avoid lung trauma during mechanical ventilation. Under dynamic conditions, the time course of applied pressure during constant V' is characterized by an immediate steady change in P_{ao} , caused by the resistive component of the respiratory system, and abruptly followed by a progressive increase in P_{ao} (steady-state portion of the dynamic V-P curve), that reflects the elastance of the respiratory system. This implies a linear model of the respiratory system, characterized by a single resistance and a single elastance (ie a single time constant). Discrepancies between theoretical and clinical applications of the constant V' technique, which preclude analysis of the initial pressure and volume changes, are due to time delays. These are caused by inability of the ventilator to provide constant V' during the onset of inspiration; non-linear behaviour of the resistive component (especially the ETT, at very low V' in the initial part of the curve); and presence of inhomogeneities in the respiratory system caused by different time constants and/or the presence of viscoelastic behaviour in pulmonary tissues. These factors may impact on the measurement of overdistension.

Because the initial part of the V-P curve reflects the pressure needed to overcome resistive pressures, including the resistance associated with an ETT, it contributes to

the widening of the dynamic V-P loop. It increases the C_{20}/C ratio. The total inspiratory compliance of the C_{20}/C ratio is obtained by fitting a line to the zero flow points at the beginning of inspiration and at end-inspiration. It therefore includes, in the initial part of the V-P curve, the pressure required to overcome the resistive pressure drop across the ETT, airways and tissues of the respiratory system. In this initial part, the pressure increase is not associated with a volume increase. Increase in this resistive component decreases total inspiratory compliance and increases the C_{20}/C ratio. However, it does not modify the value of the c coefficient that is calculated from V-P data obtained after the initial step change in P_{ao} caused by the resistive component of the respiratory system [23], and has no influence on $\%E_2$.

In addition, change in airway pressure under dynamic conditions reflects an average response of the respiratory system to change in lung volume, and the contributions of heterogeneous time constants to lung overdistension. At a given respiratory rate, lung units with a shorter time constant receive a greater proportion of ventilation than do lung units with a relatively long time constant, and may contribute to regional overinflation [34], which is reflected in dynamic indices of overdistension. On the contrary, under static conditions, equalization of pressure between lung units with different time constants is achieved.

Finally, viscoelastic pressure is recorded under dynamic, but not under static conditions. It may increase disproportionately at high pressure, and may contribute to a clearer decrease in compliance at high airway pressure, and a more consistent UIP under dynamic conditions, as observed in acute lung injury and ARDS [35,36].

Limitations of the use of respiratory volume–pressure curves in general, and for the detection of overdistension

A mathematical model of the ARDS lung, incorporating simulated gravitational superimposed pressure and alveolar opening and closing pressure, suggested that the LIP may not be closely related to open-lung PEEP (minimum PEEP preventing end-expiratory collapse of 97.5% of alveoli inflated at end-inspiration). It has also been suggested that recruitment of previously collapsed lung units could continue in the linear portion of the V-P curve well above the LIP, and that an UIP at a relatively low pressure could occur as recruitment stops or diminishes without alveolar overdistension [14]. The slope of the inflation V-P curve and the tidal V-P plot were greatly affected by continuing recruitment, and did not indicate the amount of aerated lung well. That study is supported by a clinical study [33], which indicates that neither the mean tidal V-P slope during an incremental PEEP trial, nor the LIP of the V-P curve is likely to indicate the minimum PEEP required to prevent end-expiratory collapse of most alveoli. Conversely,

the maximum V-P slope during a decremental PEEP trial with a low V_T may be a useful method to identify the optimal open-lung PEEP in ARDS, and should be studied in clinical practice [37].

The respiratory system V-P curve is influenced by the mechanics of the lung and chest wall. Although ARDS is primarily a lung problem, chest wall compliance may be decreased in patients with severe respiratory failure [38] or intra-abdominal disease [39]. However, studies that measured lung or chest wall compliance, together with total respiratory compliance, showed that reduction in total respiratory compliance measured at high V_T was fully attributable to change in lung compliance. That is, the determination of the UIP, and hence the detection of overdistension on the respiratory V-P curve, was valid [13]. Conversely, the LIP may be influenced by chest wall characteristics [40].

Conclusion

Monitoring respiratory mechanics is an important aspect of the management of the ventilated patient. It may reduce ventilator-associated lung injury [8], as suggested by the results of randomized prospective clinical trials that compared protective ventilatory strategies with more conventional strategies [9–11]. It provides a guide for the selection of the level of PEEP and for the upper limit of V_T . This individual evaluation is required to avoid overdistension, especially in infants, even if standardized recommendations are applied [41].

Overdistension can be detected on quasi-static V-P curves constructed using the LFI technique, which can be performed using a modern ventilator and does not necessitate disconnection of the patient from the ventilator. Future developments in ventilator technology for use in paediatric intensive care units should include the measurement of pressure and V' at the Y piece. Ventilator technology should also be refined so that ventilators can be used to control performance of the automated LFI technique, with a screen that has greater definition in order to allow immediate analysis of the (quasi)-static V-P curve and determination of co-ordinates of UIP and LIP. Dynamic indices of overdistension could be provided on-line by such ventilators.

Further studies are required to define which dynamic index of overdistension is the most valid. On the basis of studies conducted thus far, $\%E_2$ or coefficient c of SOPE should be chosen rather than the C_{20}/C ratio. Ranieri *et al* [42], using an isolated nonperfused lavaged model of acute lung injury, recently showed that the pressure–time curve during constant V' ventilation can be used to prime a non-injurious ventilatory strategy. The pressure–time curve was fitted to an equation (pressure = $a \times t^b + c$, where b describes the shape of the curve). The threshold value for

coefficient b that discriminated best between lungs with and without histologic and inflammatory evidence of VILI ranged from 0.90 to 1.10. Further clinical studies are required to evaluate the utility of analysis of the pressure–time curve during constant V' ventilation, in order to identify noninjurious ventilatory strategies.

References

1. Matamis D, Lemaire F, Harf A, Brun-Buisson C, Ansquer JC, Atlan G: **Total respiratory pressure–volume curves in the adult respiratory distress syndrome.** *Chest* 1984, **86**:58–66.
2. Tremblay L, Valenza F, Ribeiro SP, Li J, Slutsky AS: **Injurious ventilatory strategies increase cytokines and c-fos m-RNA expression in an isolated rat lung model.** *J Clin Invest* 1997, **99**:944–952.
3. Laffey JG, Engelberts D, Kavanagh BP: **Buffering hypercapnic acidosis worsens acute lung injury.** *Am J Respir Crit Care Med* 2000, **6**:141–146.
4. Slutsky AS, Tremblay LN: **Multiple system organ failure: is mechanical ventilation a contributing factor?** *Am J Respir Crit Care Med* 1998, **157**:1721–1725.
5. Muscedere JG, Mullen JB, Gan K, Slutsky AS: **Tidal ventilation at low airway pressure can augment lung injury.** *Am J Respir Crit Care Med* 1994, **149**:1327–1334.
6. Dreyfuss D, Saumon G: **Role of tidal volume, FRC and end-expiratory volume in the development of pulmonary oedema following mechanical ventilation.** *Am Rev Respir Dis* 1993, **148**:1194–1203.
7. Slutsky AS: **Lung injury caused by mechanical ventilation.** *Chest* 1999, **116**(suppl):9S–15S.
8. International Consensus Conferences in Intensive Care Medicine: **Ventilator-associated lung injury in ARDS.** *Am J Respir Crit Care Med* 1999, **160**:2118–2124.
9. Amato MB, Barbas CS, Medeiros DM, Magaldi RB, Schettino GP, Lorenzi-Filho G, Kairalla RA, Deheinzelin D, Munoz C, Oliveira R, Takagaki TY, Carvalho CR: **Effect of a protective-ventilation strategy on mortality in the acute respiratory distress syndrome.** *N Engl J Med* 1998, **338**:347–354.
10. The Acute Respiratory Distress Syndrome Network: **Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome.** *N Engl J Med* 2000, **342**:1301–1308.
11. Ranieri VM, Suter PM, Tortorella C, De Tullio R, Dayer JM, Brienza A, Bruno F, Slutsky AS: **Effect of mechanical ventilation on inflammatory mediators in patients with acute respiratory distress syndrome: a randomised controlled trial.** *JAMA* 1999, **282**:54–61.
12. Mancebo J: **PEEP, ARDS, and alveolar recruitment.** *Intensive Care Med* 1992, **18**:383–385.
13. Roupie E, Dambrosio M, Servillo G, Mentec H, el Atrous S, Beydon L, Brun-Buisson C, Lemaire F, Brochard L: **Titration of tidal volume and induced hypercapnia in acute respiratory distress syndrome.** *Am J Respir Crit Care Med* 1995, **152**:121–128.
14. Hickling KG: **The pressure–volume curve is greatly modified by recruitment. A mathematical model of ARDS lungs.** *Am J Respir Crit Care Med* 1998, **158**:194–202.
15. Papastamelos C, Panitch HB, England SE, Allen JL: **Developmental changes in chest wall compliance in infancy and early childhood.** *J Appl Physiol* 1995, **78**:179–184.
16. Clark RH, Slutsky AS, Gertsman DR: **Commentaries. Lung protective strategies of ventilation in the neonate: what are they?** *Pediatrics* 2000, **105**:112–114.
17. Brochard L: **Respiratory pressure–volume curves.** In: *Principle and Practice of Intensive Care Monitoring*. Edited by Tobin MJ. New York: McGraw-Hill, Inc; 1998:597–605.
18. Lu Q, Vieira SR, Richecoeur J, Puybasset L, Kalfon P, Coriat P, Rouby JJ: **A simple automated method for measuring pressure–volume curves during mechanical ventilation.** *Am J Respir Crit Care Med* 1999, **159**:275–282.
19. Servillo G, Svantesson C, Beydon L, Roupie E, Brochard L, Lemaire F, Jonson B: **Pressure–volume curves in acute respiratory failure. Automated low flow inflation versus occlusion.** *Am J Respir Crit Care Med* 1997, **155**:1629–1636.

20. Fisher JB, Mammel MC, Coleman JM, Bing DR, Boros SJ: **Identifying lung overdistension during mechanical ventilation by using volume-pressure loops.** *Pediatr Pulmonol* 1988, **5**: 10-14.
21. Kano S, Lanteri CJ, Duncan AW, Sly PD: **Influence of nonlinearities on estimates of respiratory mechanics using multilinear regression analysis.** *J Appl Physiol* 1994, **77**:1185-1197.
22. Nève V, de la Roque ED, Leclerc F, Leteurtre S, Dorkenoo A, Sadik A, Cremer R, Logier R: **Ventilator-induced overdistension in children: dynamic versus low-flow inflation volume-pressure curves.** *Am J Respir Crit Care Med* 2000, **162**:139-147.
23. Ranieri VM, Giuliani R, Fiore T, Dambrosio M, Milic-Emili J: **Volume-pressure curve of the respiratory system predicts effects of PEEP in ARDS: 'occlusion' versus 'constant flow' technique.** *Am J Respir Crit Care Med* 1994, **149**:19-27.
24. Surrat PM, Owens D: **A pulse method of measuring respiratory system compliance in ventilated patient.** *Chest* 1981, **80**: 34-38.
25. Rodriguez L, Marquer B, Mardrus P, Molenat F, Le Grand JL, Reboul M, Garrigues B: **A new simple method to perform pressure-volume curves obtained under quasi-static conditions during mechanical ventilation.** *Intensive Care Med* 1999, **25**: 173-179.
26. Kacmarek RM, Hess D: **Airway pressure, flow and volume waveforms, and lung mechanics during mechanical ventilation.** In: *Monitoring in Respiratory Care*. Edited by Kacmarek RM, Hess D, Stoller JK. St Louis: Mosby-Year Book, Inc; 1993: 497-543.
27. Bartel LP, Bazik JR, Powner DJ: **Compression volume during mechanical ventilation: comparison of ventilators and tubing circuits.** *Crit Care Med* 1985, **13**:851-854.
28. Guerin C, Viale JP, Chambrin MC, Annat G: **Definition and reliability of monitored parameters: pressures, flows and volumes [in French].** *Réan Urg* 2000, **9**:413-420.
29. Leclerc F, Riou Y, Fourier C, Flurin V, Martinot A, Hue V, Deschilde A: **Measurement of respiratory mechanics in mechanically ventilated children with the Siemens Servoventilator 900C [in French].** *Réan Urg* 1995, **2**:161-167.
30. Sola A, Farina D, Rodriguez S, Kurlat I: **Lack of relationship between the true airway pressure and the pressure displayed with an infant ventilator.** *Crit Care Med* 1992, **20**:778-781.
31. Ranieri VM, Eissa NT, Corbeil C, Chasse M, Braidy J, Matar N, Milic-Emili J: **Effects of positive end-expiratory pressure on alveolar recruitment and gas exchange in patients with the adult respiratory distress syndrome.** *Am Rev Respir Dis* 1991, **144**:544-551.
32. Kosch PC, Hutchison AA, Wozniak JA, Waldemar AC, Stark AR: **Posterior cricoarytenoid and diaphragm activities during tidal breathing in neonates.** *J Appl Physiol* 1988, **64**:1968-1978.
33. Jonson B, Svantesson C: **Elastic pressure-volume curve: what information do they convey?** *Thorax* 1999, **54**:82-87.
34. Otis AB, McKerrow CB, Bartlett RA, Mead J, mcllroy MD, Selvestone NJ, Radford EP: **Mechanical factors in distribution of pulmonary ventilation.** *J Appl Physiol* 1956, **8**:427-443.
35. Svantesson C, John J, Taskar V, Evander E, Jonson B: **Respiratory mechanics in rabbits ventilated with different tidal volumes.** *Respir Physiol* 1996, **106**:307-316.
36. Beydon L, Svantesson C, Brauer K, Lemaire F, Jonson B: **Respiratory mechanics in patients ventilated for critical lung disease.** *Eur Respir J* 1996, **9**:262-273.
37. Hickling KG: **Best compliance during a decremental but not incremental positive end-expiratory pressure trial is related to open lung positive end-expiratory pressure. A mathematical model of acute respiratory distress syndrome lungs.** *Am J Respir Crit Care Med* 2001, **163**:69-78.
38. Pelosi P, Cereda M, Foti G, Giacomini M, Pesenti A: **Alteration of lung and chest wall mechanics in patients with acute lung injury: effect of positive end-expiratory pressure.** *Am J Respir Crit Care Med* 1995, **152**:531-537.
39. Gattinoni L, Pelosi P, Suter PM, Pedoto A, Vercesi P, Lissoni A: **Acute respiratory distress syndrome caused by pulmonary and extra pulmonary disease. Different syndromes?** *Am J Respir Crit Care Med* 1998, **158**:3-11.
40. Mergoni M, Martelli A, Volpi A, Primavera S, Zuccoli P, Rossi A: **Impact of positive end-expiratory pressure on chest wall and lung pressure-volume curve in acute respiratory failure.** *Am J Respir Crit Care Med* 1997, **156**:846-854.
41. Mols G, Brandes I, Kessler V, Lichtwarck-Aschoff M, Loop T, Geiger K, Guttman J: **Volume-dependent compliance in ARDS: proposal of a new diagnostic concept.** *Intensive Care Med* 1999, **25**:1084-1091.
42. Ranieri VM, Zhang HAIBO, Mascia L, Aubin M, Lin CY, Mullen JB, Grasso S, Binnie M, Volgyesi GA, Eng P, Slutsky AS: **Pressure-time curve predicts minimally injurious ventilatory strategy in an isolated rat lung model.** *Anesthesiology* 2000, **93**:1320-1328.