

# A Simple Bedside Approach to Measurement of Respiratory Mechanics in Critically Ill Patients

A. D. BERSTEN

*Department of Critical Care Medicine, Flinders Medical Centre, Adelaide, SOUTH AUSTRALIA*

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## **ABSTRACT**

**Objective:** *To describe and evaluate clinically applicable approaches to measurement of respiratory mechanics in critically ill patients.*

**Data sources:** *Methodological and evaluation studies of respiratory mechanics in critically ill patients from relevant MEDLINE searches.*

**Summary of review:** *In ventilated subjects clinically important respiratory system mechanics can be measured using airway pressure and flow data. However, since the respiratory system consists of the lung and chest wall, and chest wall mechanics can markedly alter respiratory system mechanics, it is preferable to compartmentalise these parameters with concurrent measurement of oesophageal, and preferably gastric pressure. Additional care must be taken with interpretation of these data since elastance and resistance may be influenced by frequency, volume, volume history and flow. Tissue viscoelasticity and non-homogeneity of regional time constants are responsible for stress adaptation, which can be measured simply, and accounts for some of these effects on elastance and resistance, and for a systematic difference between static and dynamic intrinsic PEEP. Elastance can be measured using the end-inspiratory occlusion technique, or from either static or dynamic volume-pressure curves. PEEP-mediated recruitment can be measured following referencing of these curves to FRC. Similarly, resistance can be measured from either end-inspiratory occlusion or dynamic pressure and flow data.*

**Conclusions:** *Some of this information is available on modern ventilators, but greater insight requires measurement and manipulation of flow and pressure data using a pneumotachograph and pressure transducers. Given the importance of respiratory mechanics in the management of many critically ill patients, and given how poorly the respiratory system is monitored compared with the cardiovascular system, it is worth considering making this simple but additional effort. (Critical Care and Resuscitation 1999; 1: 74-84)*

**Key words:** Respiratory mechanics, chest wall, viscoelastic, elastance, resistance, intrinsic PEEP

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## **Introduction**

During normal spontaneous breathing, contraction of the respiratory muscles leads to expansion of the chest wall. A fall in regional pleural pressure results in alveolar inflation as gas flows in under the resultant pressure gradient. During inspiration the respiratory muscles and/or mechanical ventilator provide this

pressure gradient, and during expiration the expiratory muscles may assist the elastic recoil of the respiratory system.

Although both respiratory muscle work and the interaction of mechanical ventilation with the respiratory system are germane to care of critically ill patients, quantitation and understanding of these parameters can

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*Correspondence to:* Associate Professor A. D. Bersten, Department of Critical Care Medicine, Flinders Medical Centre, Bedford Park, South Australia 5042. (e-mail: [andrew.bersten@flinders.edu.au](mailto:andrew.bersten@flinders.edu.au))

be complex and is generally not part of routine practice. The aim of this article is to provide a basic framework for measurement and interpretation of respiratory mechanics from often readily available pressure, flow and volume waveforms.

### The respiratory system and its pressure gradients

The respiratory system (RS) is composed of the lung (L), and the rib cage and abdomen which function as the chest wall (CW). Although it is often convenient to consider respiratory system mechanics as implying information about the lung, abnormal chest wall compliance can markedly influence these measurements<sup>1-4</sup>. Abdominal distension may alter both the slope and the upper and lower inflection points of the respiratory system volume-pressure (V-P) curve, independent of changes in the lung V-P curve. However, due to differences in the measurement techniques and patient populations used between studies it is not yet possible to generalise these effects. Consequently care must be taken in the interpretation of respiratory system mechanics in patients with abnormal chest wall mechanics.

The pressure gradient across the lung ( $P_L$ ) that generates gas flow, is equal to the difference between the pressure at the airway opening ( $P_{ao}$ ) and the oesophageal pressure ( $P_{es}$ ), i.e.,

$$P_L = P_{ao} - P_{es}$$

Although the oesophageal pressure is not always an accurate measure of the absolute pleural pressure, the change in the oesophageal pressure does reflect the change in pleural pressure. However, this requires an appropriately positioned and functioning oesophageal balloon. In spontaneously breathing subjects a thin latex balloon sealed over a catheter introduced into the lower third of the oesophagus, allows both  $P_{es}$  and  $P_{ao}$  to be measured simultaneously during an end-expiratory airway occlusion. A well-positioned oesophageal balloon will have a ratio of  $\Delta P_{es}/\Delta P_{ao}$  of  $\sim 1$ .<sup>5</sup> This technique is also reliable in supine, intubated and spontaneously breathing patients,<sup>6</sup> and in paralysed subjects it appears that a similar pressure change, induced by manual rib cage pressure,<sup>7</sup> can be used to verify oesophageal balloon function.

Chest wall mechanics are derived from  $P_{es}$  referenced to atmospheric pressure, and in ventilated relaxed subjects respiratory system mechanics are derived from  $P_{ao}$  referenced to atmospheric pressure. It is not surprising then that  $P_{RS} = P_L + P_{CW}$ . Finally, abdominal mechanics can be measured using an intragastric balloon.

Despite the undoubted importance of chest wall mechanics, useful information can be readily gained from respiratory system mechanics in ventilated patients, particularly as  $P_{ao}$  is immediately available.

### Assessment of respiratory mechanics in mechanically ventilated subjects

In a passively ventilated subject  $P_{ao}$  is the sum of i) the pressure required to overcome airway, endotracheal tube and circuit resistance ( $P_{res}$ ), ii) the elastic pressure required to expand the lung and chest wall ( $P_{el}$ ), iii) the elastic recoil pressure at end-expiration or total PEEP ( $P_o$ ), and iv) the inertial pressure required to generate gas flow ( $P_{inert}$ ), i.e.:

$$P_{ao} = P_{el} + P_{res} + P_o + P_{inert}$$

Since the elastance (E), which is the inverse of compliance, is equal to  $\Delta P/\Delta V$ , with the resistance (R) equal to  $\Delta P/\dot{V}$ , and ignoring the inertance,<sup>8</sup> this equation can be rewritten as the single-compartment equation of motion, i.e.:

$$P_{ao} = E_{rs} V + R_{rs} \dot{V} + P_o$$

Many methods have been used to assess respiratory mechanics. For example:

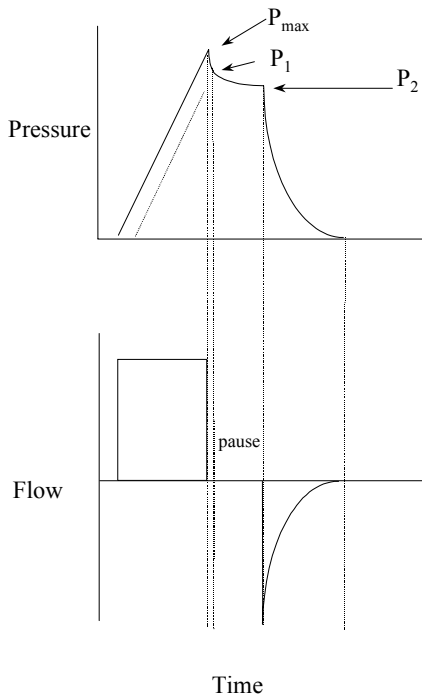
#### (a) End-inspiratory occlusion method

Using this method the values required are estimated by a rapid end-inspiratory airway occlusion during a constant flow breath, provided that there is no muscle activity (Figure 1). If a plateau is introduced at end-inspiration there is a sudden initial pressure drop due to dissipation of flow resistance ( $P_{max} - P_1$ ) followed by a slower, secondary pressure drop to an apparent plateau ( $P_{dif} = P_1 - P_2$ ) due to stress relaxation. This can be represented by a model of the respiratory system (Figure 2) comprising a dashpot representing airways resistance ( $R_{aw}$ ), a spring representing static elastance ( $E_1$ ) and a Maxwell body, comprised of a dashpot ( $R_{ti}$ ) and spring ( $E_2$ ) in parallel. In other words, a simple model can be used to explain most of the behaviour of the respiratory system using static, 'single alveolus' mechanics combined with a non-Newtonian effect, the Maxwell body.

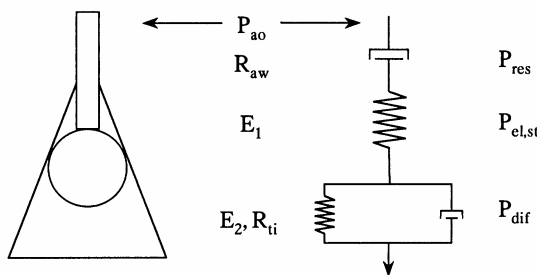
#### Stress adaptation

The respiratory system does not normally behave in a simple Newtonian fashion, and this is further exaggerated in disease states. Elastance and resistance are frequency dependent, and respiratory mechanics depend upon the volume and volume history of the

lung.<sup>9</sup> Consequently these factors must be taken into account when interpreting respiratory mechanics.



**Figure 1.** A schematic diagram of airway pressure and gas flow during a constant flow inflation, with a period of no-flow prior to expiration (a pause), followed by passive expiration. During the pause the airway pressure rapidly drops from its maximum value ( $P_{max}$ ) to  $P_1$ , this is then followed by a slower decrease to an apparent plateau ( $P_2$ ).



**Figure 2.** A schematic diagram of the respiratory system, including a two-compartment model. The alveolus and airways are represented by a single elastance ( $E_1$ ), represented as a spring, and a single resistance ( $R_{aw}$ ) represented as a dashpot, with a corresponding resistive pressure drop ( $P_{res}$ ) and static elastance ( $P_{el,st}$ ). Stress adaptation is represented as a Maxwell body with an elastance ( $E_2$ ) and resistance ( $R_{ti}$ ) in parallel. Airway pressure ( $P_{ao}$ ) is the sum of  $P_{res}$ ,  $P_{el,st}$  and the pressure due to stress adaptation ( $P_{dif}$ ).

With increasing frequency of breathing total respiratory system resistance falls and elastance increases. This is particularly obvious in patients with

airflow obstruction,<sup>10,11</sup> and can be explained using the Maxwell body. When the respiratory rate is slow the dashpot  $R_{ti}$  is given time to dissipate and relieve the stretch in  $E_2$ . Consequently, the elastance now approaches  $E_1$  from  $E_1 + E_2$ , and this describes the alveolar volume change exactly. In turn the total resistance approaches the sum of  $R_{aw}$  and  $R_{ti}$  ( $R_L$ ). As the respiratory rate increases insufficient time is allowed for this energy dissipation in  $R_{ti}$ , and  $E_2$  is also forced to follow the changes in alveolar volume. The elastance approaches  $E_1 + E_2$  and the resistance approaches  $R_{aw}$ .

Stress relaxation of the respiratory system is due to both tissue viscoelasticity and time constant inequalities of the respiratory system (i.e. pendelluft). In the normal lung pendelluft has a minimal contribution to stress relaxation,<sup>12</sup> however, heterogeneity of regional resistance and elastance can markedly influence stress relaxation.<sup>13</sup> Pulmonary surfactant and its contribution to changes in surface tension, parenchymal factors including elastic fibres in the lung, contractile elements such as the alveolar duct muscle and changes in pulmonary blood volume have all been implicated in the viscoelastic properties of the lung. However, it is not possible to separate these factors or the role of pendelluft to stress adaptation.

*Calculation of respiratory mechanics*

Returning to Figure 1, it is now simple to estimate respiratory system resistance and elastance from  $P_{ao}$ . The static elastance ( $E_{rs,st}$ ) and the dynamic elastance ( $E_{rs,dyn}$ ) are calculated as

$$E_{rs,st} = (P_2 - P_o)/V_T$$

and,

$$E_{rs,dyn} = (P_1 - P_o)/V_T$$

respectively, where  $P_o$  is the total PEEP (i.e. extrinsic plus intrinsic PEEP). This embodies the concept that  $P_{el,dyn}$  and not  $P_{el,st}$  is the effective recoil pressure of the respiratory system during mechanical ventilation. Consequently, during inspiration, additional work is performed to overcome stress adaptation, and this is stored and dissipated during expiration. This contributes to the hysteresis seen in dynamic volume-pressure curves during mechanical ventilation, and to the generation of expiratory flow. This latter component may be important in patients with airflow obstruction since the imposition of a pause at the end of inspiration results in a 32% dissipation of the total energy loss within the respiratory system.<sup>14</sup> Although there are no clinical studies that examine whether this has any clinical ramifications, this would argue against routinely

including a pause at the end of inspiratory flow, particularly in patients with severe airflow obstruction.

The total airways resistance including the endotracheal tube and associated ventilatory apparatus can be calculated as:

$$R_{aw} = (P_{max} - P_1) / \dot{V}$$

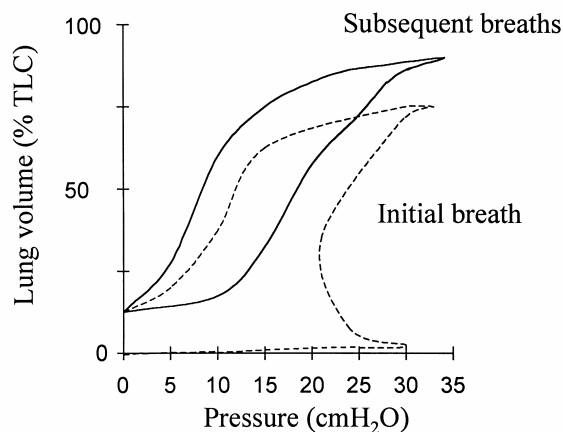
and  $R_L$  is calculated by using  $P_2$  instead of  $P_1$ . Since the endotracheal tube and apparatus will make a significant contribution to  $R_{aw}$  it is best to measure  $P_{ao}$  distal to the endotracheal tube with an intra-tracheal catheter. An alternative approach is to calculate and subtract endotracheal tube resistance when  $P_{ao}$  is measured just proximal to the endotracheal tube, and this is now automatically included in some ventilators. However, in-vivo endotracheal tube resistance is often greater than in-vitro resistance<sup>15</sup> due to the effect of secretions and interaction with the tracheal wall, so that these corrections may be inaccurate. Furthermore, this technique only estimates inspiratory resistance, and measurements need to be made at a constant lung volume as this also influences  $R_{aw}$ . Despite these considerations, this simple measure of resistance can be clinically useful both in the diagnosis and monitoring of airflow obstruction.<sup>16</sup>

#### (b) The static volume-pressure curve

The quasistatic volume-pressure (V-P) curve has become the defacto 'gold standard' for the measurement of respiratory elastance. However, it is infrequently performed, and the relevance of a measurement performed on a single occasion is questionable when lung mechanics are not constant. Various techniques have been described, but the overall concept is that incremental volume and pressure points are made after a sufficient period of no-flow has allowed  $P_{res}$  to be dissipated. This allows definition of a sigmoidal shaped curve with upper and lower inflection points, and a mid-section with relatively linear V-P relations, allowing inflation elastance to be measured as this slope at a given lung volume (Figure 3). If similar measures are made during deflation, a deflation curve and its hysteresis can also be described.

The V-P curve immediately provides an advantage over an occlusion-derived elastance, because with the latter, it is not possible to know which part of the V-P curve is being measured. Consequently this 'chord' elastance may span either inflection point, yielding a falsely high figure. The upper inflection point represents a sudden decrease in elastance with increasing volume, and this has been interpreted as lung overinflation. The lower inflection point represents a sudden decrease in elastance with increased volume, and this has been

interpreted as recruitment of atelectatic airspaces. Ventilation between these two inflection points should minimise both shearing forces secondary to repetitive collapse and reopening of alveoli, and overstretch of alveoli.



**Figure 3.** A schematic volume-pressure curve of the isolated lung following inflation from the degassed state. A high opening pressure is required to inflate collapsed alveoli at the initial breath. Subsequently, small airway closure occurs prior to alveolar collapse and lower airway opening pressures are required.

Conventionally, the static V-P curve has been measured in ventilated patients with the 'super-syringe' method.<sup>17</sup> In a paralysed patient the respiratory system is progressively inflated from functional residual capacity (i.e. the lung volume at 0 cmH<sub>2</sub>O PEEP) in 100 ml steps up to 1700 ml or a predefined pressure limit. After each step, sufficient time is taken for a well defined plateau to become apparent (using a pause of 3-6 seconds). The effects of temperature, humidity, gas compression and ongoing gas exchange during the maneuver need to be taken into account,<sup>18,19</sup> and the volume history standardised before it is performed. As this technique is cumbersome and because many patients become hypoxemic following disconnection from the ventilator (and the 60 seconds or so without PEEP) other techniques have been developed.

The static V-P curve can also be measured by randomly inserting a range of single volume inflations, followed by a prolonged pause,<sup>20</sup> during normal mechanical ventilation. This is performed in the paralysed state at 0 cmH<sub>2</sub>O PEEP. This technique has a number of advantages including its simplicity, the patient is not disconnected from the ventilator, the volume history is the same for each measurement and the gas exchange during the measurement is negligible. Nevertheless, many patients will still become hypoxemic due to prolonged periods without PEEP (this procedure may take 15 minutes), particularly following a small volume breath. Finally, an automated low flow V-P

curve method allowing subtraction of  $P_{res}$  has been described which takes about 20 seconds to perform, and this seems to correlate well with the static occlusion technique.<sup>21,22</sup>

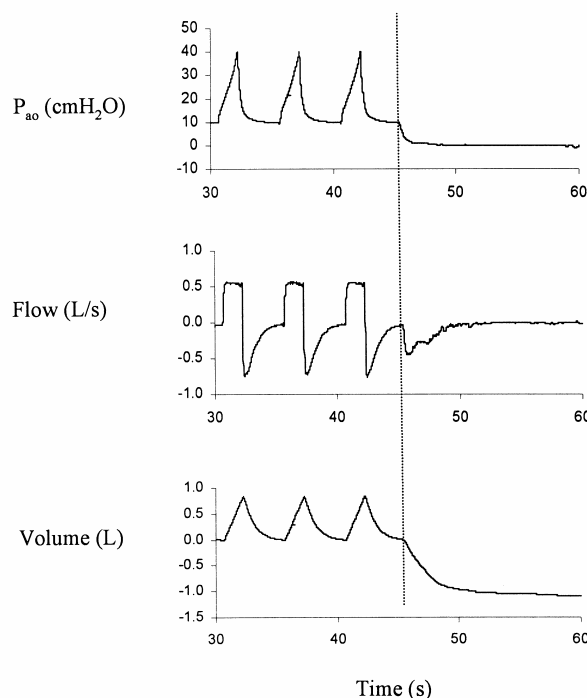
The interpretation of the V-P curve also needs to be reconsidered. In a degassed lung a large opening pressure is required to inflate atelectatic alveoli (e.g. the initial breath in Figure 3). On deflation the small airways collapse before the alveoli, perhaps due to the erectile nature of the distended pulmonary capillaries, so that much lower opening pressures are required for subsequent breaths. It is also clear that airspace recruitment may occur both during a tidal breath,<sup>23</sup> and following a volume recruitment maneuver such as PEEP. When recruitment occurs during tidal ventilation, this will alter both the lower and upper inflection points and the slope of the quasilinear portion of the V-P curve.<sup>24</sup> Finally, PEEP-mediated recruitment is only 90% complete after 4.6 breaths at the higher level of PEEP.<sup>25</sup> Consequently, the lower inflection point may not be an appropriate estimate of the PEEP required to ensure airspace recruitment. A simple alternative is to measure the expired volume at end-expiration during withdrawal of PEEP (Figure 4).<sup>26,27</sup>

This can be performed at different levels of PEEP and the recruited lung volume measured as a vertical displacement of the now referenced static or dynamic V-P curve (Figure 5). The major provisos of this technique are that it does not prevent overinflation, and recruitment can be a stepwise phenomena so that it is worth exploring a reasonable range of PEEP before concluding that an optimal PEEP level has been achieved.

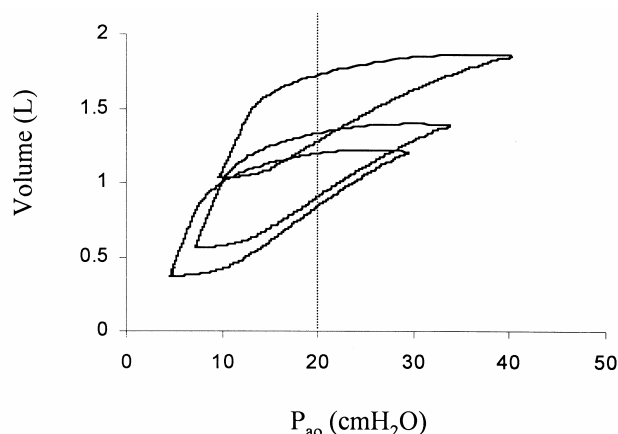
**(c) The dynamic V-P curve**

Dynamic  $P_{ao}$ ,  $\dot{V}$  and tidal volume can be continuously available at the bedside. Although many ventilators now display these variables, the volume signal is not referenced to functional residual capacity (FRC) and the signals are not readily available for quantitative analysis. However,  $\dot{V}$  is readily measured with a heated pneumotachograph, and volume can then be derived by simple integration, if the signal is collected after analogue-to-digital conversion. If  $P_{ao}$  is also collected it is relatively simple to measure dynamic mechanics.

The dynamic V-P curve will always show hysteresis, mainly due to the effects of airway and tissue resistance. Indeed, it is unlikely that hysteresis of the static V-P occurs during tidal breathing.<sup>28</sup> In contrast to static V-P relations, dynamic mechanics are collected during normal ventilation so they do not interfere with patient care, and provide a 'functional' description of respiratory mechanics. Indeed the 'effective' alveolar



**Figure 4.** The change in the end-expiratory volume following the removal of PEEP is shown in this typical example from a 70 year old woman with ARDS. Following 30 min of stable ventilation 10 cmH<sub>2</sub>O of PEEP is removed and the patient allowed to passively expire to FRC. Airway pressure ( $P_{ao}$ ) falls to 0 cmH<sub>2</sub>O in conjunction with additional expiratory flow for the following 5-6 seconds. The integral of flow and the bottom graph demonstrates that the end-expiratory volume was 1.03 L above FRC.



**Figure 5.** Using the expired volume, as described in Figure 4, dynamic V-P curves can now be referenced to FRC and recruitment measured following a PEEP trial, by comparing the vertical displacement of volume at a constant pressure (20 cmH<sub>2</sub>O in this example). These data, measured at random PEEP levels of 5, 7.5 and 10 cmH<sub>2</sub>O, demonstrate little recruitment between 5 and 7.5 cmH<sub>2</sub>O, but 320 ml is suddenly recruited at 10 cmH<sub>2</sub>O. Further PEEP increments did not reveal any more recruitment in this patient.

distending pressure is more accurately the  $P_{el,dyn}$  and not  $P_{el,st}$ . As dynamic mechanics are continuous they can be used to servo-control ventilatory strategies

There are a number of ways to analyse dynamic V-P data. A line can be drawn between no-flow points at end-inspiration and end-expiration to determine elastance. However, this is relatively inaccurate as it is based on two points that can be hard to exactly identify. Multiple linear regression analysis is now the technique most commonly employed. This technique employs a weighted average, using the equation of motion to calculate elastance, resistance and  $P_o$ . This is usually done with a computer following on-line analogue-to-digital signal conversion of a number of typical breaths. The patient does not need to be paralysed<sup>29</sup> provided the respiratory muscles are not active during ventilation, and the signal can be split to allow analysis of inspiratory and expiratory mechanics.

Provided the model fit is acceptable, elastance and resistance are accurately and reproducibly calculated. Generally, the single compartment equation of motion, i.e.:

$$P_{ao} = E_{rs} V + R_{rs} \dot{V} + P_o$$

appropriately approximates the respiratory system with the model explaining in excess of 95% of the measured data ( $R^2 > 0.95$ ). Under these conditions static intrinsic PEEP ( $PEEP_i$ ) is accurately calculated, as  $P_o - PEEP_e$  (extrinsic PEEP), when compared with either an end-expiratory airway occlusion method,<sup>30</sup> or by direct measurement of end-expiratory alveolar pressure.<sup>31</sup> However, the single compartment model only approximates the respiratory system and frequency and volume dependence of the derived mechanics are observed. Provided these factors are recognised and accounted for, it is sound to compare data within and between patients.

Addition of a volume-dependent term to the equation of motion, i.e.:

$$E_{rs} = E_1 + E_2 V$$

and therefore,

$$P_{ao} = (E_1 + E_2 V)V + R_{rs} \dot{V} + P_o$$

improves model fit in some patients ( $R^2 \sim 99\%$ ), and allows conceptual placement of tidal breathing on a dynamic V-P curve.<sup>32</sup>

If the  $\%E_2$  is calculated as:

$$\%E_2 = 100E_2 V / E_{rs}$$

then a  $\%E_2$  greater than 30% quantitates overinflation, and a negative volume dependence (negative  $\%E_2$ ) suggests atelectasis. Combined with referencing of the V-P curve, respiratory system mechanics during ventilation are now easily accessible at the bedside. However, this still requires use of a pneumotachograph and analogue-to-digital conversion. During constant  $\dot{V}$  ventilation, and provided the tidal volume is also constant, the change in either  $P_{max}$  - total PEEP or  $P_1$  - total PEEP (the delta pressure;  $\Delta P$ ) 20-30 minutes following a change in PEEP is highly correlated with  $\%E_2$ .<sup>27</sup> Using the 95% predictive interval for this data, an increase in  $\Delta P > 2$  cmH<sub>2</sub>O indicates overinflation, a  $\%E_2 > 30\%$ .

Alternatives to this approach include polynomial analysis of V and  $P_{ao}$  data or visual analysis of  $P_{ao}$  - time data.<sup>33</sup> However, this requires analysis of that part of inspiration where  $\dot{V}$  is constant, which does not occur throughout the whole of inspiration. Even when this is selected on the ventilator, inspiratory  $\dot{V}$  takes a finite time to be achieved and cessation of  $\dot{V}$  is never instantaneous. As  $\dot{V}$  is measured with each set of V and  $P_{ao}$  (a typical sampling rate is 100Hz) the multiple linear regression analysis technique can be performed independent of  $\dot{V}$  pattern.

Measurements of volume-dependent elastance in patients with ARDS can provide useful management and pathophysiological insights. Given that lung CT's in ARDS demonstrate dependent lung collapse leading to ventilation of the ventral portion of the lung, it is not surprising to find that incremental PEEP may result in concurrent recruitment and overinflation.<sup>27</sup> Similar conclusions have also been drawn from CT studies.<sup>23,34</sup>

It is also noteworthy that both volume-dependent elastance<sup>27</sup> and static V-P curves<sup>35,36</sup> demonstrate that overinflation commonly occurs at alveolar distending pressures less than 30 cmH<sub>2</sub>O. While this does not discount pressure limited approaches to ventilatory management of ARDS, it does highlight the need for a more direct measure of overinflation. Again, chest wall mechanics can influence 'measurements' of overinflation, however, many of the patients with overinflation at low distending pressures appear to have normal chest wall mechanics.

Pressure-limited strategies are based on the notion that total lung capacity is normally achieved with an alveolar distending pressure of  $\sim 30$  cmH<sub>2</sub>O. In addition to the influence of the chest wall and a decrease in the rate of recruitment<sup>24</sup> abnormal surfactant function may contribute to overinflation at low distending pressures.

Surfactant allows surface tension to vary with alveolar size, so normally surface tension increases in large alveoli. Composition and function of surfactant is

abnormal in ARDS and contributes to alveolar collapse, impaired oxygenation and the abnormal respiratory mechanics.<sup>37</sup> An indirect relationship between surfactant composition and volume-dependent elastance in ARDS,<sup>38</sup> suggests that surfactant dysfunction contributes to both alveolar collapse and overinflation. Consequently the finding of overinflation at low distending pressures is not unexpected.

**(d) Resistance measurements**

A number of different techniques can be used to quantitate resistance at the bedside. However, while the relationship described by the equation:

$$R_{aw} = \Delta P / \dot{V}$$

is straightforward, none of the techniques are ideal. Resistance is flow, volume and frequency dependent. Since  $R_L = R_{aw} + R_{ti}$ ,  $R_L$  will decrease as the respiratory frequency increases, particularly when there is small airways disease.

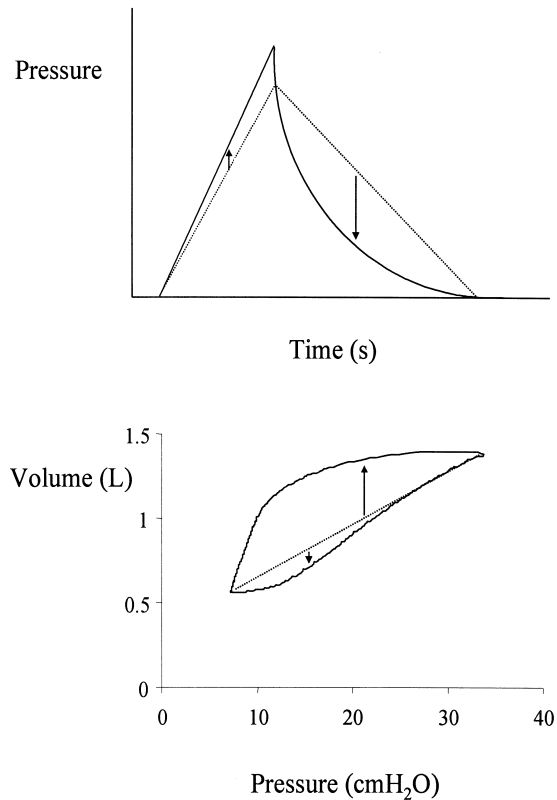
It is also important to compare measurements at similar lung volumes as there is a hyperbolic relation between lung volume and R. This is particularly obvious in patients with ARDS where the incremental administration of PEEP can result in a decrease in  $R_{aw}$  due to concurrent recruitment and an increase in lung volume. Indeed, although the absolute values are increased, when corrected for end-expiratory lung volume  $R_L$ ,  $R_{aw} + R_{ti}$  are unchanged in ARDS.<sup>1</sup> Finally, as gas flow may be a mixture of laminar and turbulent flow, resistance is often flow dependent. The manipulated Rohrer equation, i.e.:

$$R = K_1 + K_2 \dot{V}$$

accounts for non-linear resistance due to turbulent flow. This needs to be applied to endotracheal tube<sup>39</sup> and apparatus resistance. While flow resistance of the respiratory system distal to the endotracheal tube is essentially linear in normal subjects,<sup>40</sup> this is not the case in patients with severe airflow obstruction, who often have flow limitation during tidal expiration with curvilinear  $P_{res} - \dot{V}$  relations.<sup>41</sup> Consequently, linear approximations of R must also be compared at similar  $\dot{V}$  rates.

The end-inspiratory occlusion technique is the simplest way to measure R in ventilated subjects, and allows calculation of inspiratory  $R_L$ ,  $R_{aw}$  and  $R_{ti}$ . Dynamic measures of total, inspiratory and expiratory R can also be made, using either multiple linear regression analysis or from linear interpolation of the V-P curve at a constant volume. However, this latter technique

assumes a constant elastance during tidal inflation, and may be inaccurate as it relies on only two measurements (Figure 6).



**Figure 6.** Measurement of resistance from dynamic pressure, flow and volume data. In the upper diagram a dotted straight line is drawn from the alveolar pressure at end-expiration (extrinsic plus intrinsic PEEP) to the alveolar pressure at end-inspiration, which can be derived using the end-inspiratory occlusion technique. At a given volume, approximately 50% of tidal volume during inspiration or expiration in the diagram, the resistance may be calculated from the concurrent gas flow rate and pressure gradient. In the lower diagram a straight line is drawn between no-flow points on a V-P curve (the middle V-P curve in figure 5), and the resistance calculated in a similar manner. Apart from inaccuracies due to the reliance on two data points, this technique assumes a linear interpolation of elastance across the tidal volume. Examining the V-P curve, which has a %E<sub>2</sub> of 45% indicating overinflation, this may not be an appropriate assumption. Finally, although these diagrams demonstrate inspiratory and expiratory measures, it is common to average these data at a constant volume.

Finally, an average expiratory R can be calculated from the time constant ( $\tau$ ) derived from passive expiration, if the E is known, as:

$$\tau = R/E$$

However, the lung does not empty as a single compartment in patients with airflow obstruction, and E is assumed to be constant over the tidal expiration.

Despite these important provisos, measurement of  $R$  at a given  $\dot{V}$ , volume above FRC and respiratory frequency can be performed at the bedside and does provide clinically useful information.

#### (e) Other measurement techniques

Numerous other techniques have been described for the measurement of respiratory mechanics. Many of these are either modifications of the techniques described or they require equipment that is rarely available at the bedside. However, it is worth briefly mentioning the interrupter and forced oscillation techniques, because they have been frequently used as research tools in critically ill patients, and they provide advantages over some of the currently available techniques.

The interrupter technique consists of a series of short (100-200 ms) interruptions to relaxed expiration by a pneumatic valve.<sup>41</sup> This results in an expiratory plateau in  $P_{ao}$  following equilibration with alveolar pressure. From the  $V$ ,  $P$  and  $\dot{V}$  data, expiratory elastance and the expiratory  $P - \dot{V}$  relationship is measured. This technique does not make assumptions about the behaviour of the respiratory system and can identify dynamic airflow limitation.

Assuming that the respiratory system behaves linearly, it can be analysed following a forced flow oscillation at the airway opening.<sup>42</sup> The resultant pressure waveform depends upon the impedance of the respiratory system, which can be analysed following Fourier analysis of the  $P$  and  $\dot{V}$  waveforms into resistance and reactance. This will then allow measurement of  $R_{aw}$ ,  $R_{ti}$ ,  $E_{rs}$  and inertance, and information can be gained regarding small airways disease by examining  $R_{aw}$  at different oscillatory frequencies.

#### Measurement of intrinsic PEEP

Dynamic hyperinflation occurs when the expiratory time is too short for the respiratory system to reach its static equilibrium at end-expiration. The resultant, average end-expiratory pressure has been termed intrinsic PEEP ( $PEEP_i$ ), to differentiate it from applied (extrinsic) PEEP ( $PEEP_e$ ). This is an important respiratory measurement since  $PEEP_i$  may, a) have unrecognised haemodynamic consequences,<sup>43</sup> b) adds a threshold load to inspiratory work during spontaneous, supported and assisted modes of breathing which may be reduced by application of small amounts of  $PEEP_e$ ,<sup>44</sup> and c) reflects dynamic hyperinflation with the consequent risks of barotrauma<sup>45</sup> and right heart failure. If  $PEEP_i$  is not taken into account during calculation of chord compliance an incorrect denominator is used

which may markedly alter the result.<sup>46</sup>

The two most commonly described techniques for measuring  $PEEP_i$  are end-expiratory airway occlusion in a relaxed subject and the fall in oesophageal pressure during inspiration prior to initiation of inspiratory  $\dot{V}$ . However, these are not really comparable measures, as static and dynamic  $PEEP_i$  respectively are measured.

Static  $PEEP_i$  is measured as the plateau  $P_{ao}$  that is reached after  $\sim 5$  seconds following end-expiratory occlusion. With the cessation of gas flow, alveolar pressure equilibrates with  $P_{ao}$ . When the end-expiratory lung volume exceeds FRC, PEEP is present and  $PEEP_i$  is identified as that PEEP not due to  $PEEP_e$ . As the lung is composed of non-homogeneous units this will represent the average static  $PEEP_i$ . All respiratory effort must be absent as it may independently influence end-expiratory  $P_{ao}$ , and end-expiration must be accurately identified. This is most easily done by the ventilator itself, either using an end-expiratory hold maneuver or by using the next inspiration, the onset of which is concurrent with expiratory valve closure, to close a valve that directs inspiratory flow to atmosphere and seals the circuit. Static  $PEEP_i$  is a surrogate measure of dynamic hyperinflation, and this volume may be directly measured using a spirometer<sup>45</sup> or a pneumotachograph<sup>26,27</sup> during a prolonged expiration. However,  $PEEP_i$  is simply measured by the end-inspiratory occlusion technique, and this is far more commonly performed.

Dynamic  $PEEP_i$  is measured as the pressure change required to initiate inflation. In ventilated subjects this will be the change in  $P_{ao}$  prior to initiation of inspiratory  $\dot{V}$ ,<sup>46</sup> and in spontaneously breathing subjects the change in oesophageal<sup>44</sup> or trans-diaphragmatic<sup>47</sup> pressure from their end-expiratory relaxation values prior to inspiratory  $\dot{V}$ . Measurement of dynamic  $PEEP_i$  in spontaneously breathing subjects is not particularly straightforward. The changes in pressure are small and influenced by cardiogenic oscillations which are preferably filtered out.<sup>48</sup> Furthermore, dynamic  $PEEP_i$  is not constant, with breath-to-breath variation probably due to variation in the extent of dynamic hyperinflation, and many patients with airflow obstruction have an active expiration. This 'falsely' increases  $PEEP_i$ , at least with respect to its threshold load, because cessation of active expiration does not require work, with part of the measured threshold load suddenly dissipated.<sup>49</sup> Consequently, it is preferable to concurrently measure intra-gastric pressure as a measure of active expiration.

Finally,  $PEEP_i$  can be measured as  $P_o$  from dynamic  $P$ ,  $V$  and  $\dot{V}$  data in ventilated subjects. This is thought to be a measure of dynamic  $PEEP_i$  as static  $PEEP_i$  systematically yields a slightly greater result,<sup>30</sup> with



similar discrepancies reported between other dynamic measures of  $PEEP_i$  and static  $PEEP_i$ .<sup>50,51</sup> This systematic difference is thought to be due to the viscoelastic properties and regional time constant inequalities of the respiratory system.<sup>50</sup> This has great clinical significance, because while matching dynamic  $PEEP_i$  with  $PEEP_e$  reduces respiratory work through a decrease in threshold inspiratory load,<sup>44</sup> it does not counterbalance these forces which represent an additional threshold load to inspiration.

### Conclusion

Within the limitations of each technique it is possible to gain some knowledge of respiratory system mechanics in every ventilated patient. Many modern ventilators come equipped with respiratory waveform displays and some respiratory mechanics tools. However, it is crucial that the limitations of these measurements are understood. The visual appearance of a dynamic V-P curve is a poor indicator of overinflation because resistance and stress adaptation also determine its shape. Furthermore, none of these commercially available tools reference the V-P curve to FRC, which allows assessment of  $PEEP$ -induced recruitment. Consequently, it is worth considering the simple but additional effort of measuring flow with a pneumotachograph and at least airway pressure to allow more useful quantitation of respiratory system mechanics.

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