Clinical applications of pulmonary function and graphics

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Pulmonary function and mechanics testing are emerging as a valuable tool to aid clinical decision making in the management of ventilated infants. Although there are as yet no published randomized controlled trials to suggest that pulmonary mechanics testing reduces mortality or morbidity, it has – in conjunction with clinical, radiological, and blood gas monitoring – changed neonatal ventilation from ‘good judgement’ to ‘informed judgement’. It is not surprising that pulmonary graphics are increasingly being used as a tool for assessment of patient status, therapeutic evaluation, and management guidance of infants who become dependent on ventilator.

A working knowledge of pulmonary graphics also improves understanding of pulmonary physiology and pathophysiology, and their responses to mechanical ventilatory support. Recent advances in microprocessor technology for on-line analysis of pulmonary mechanics have made such evaluations easily available for bedside clinical application.

Key words: pulmonary mechanics, pulmonary graphics, pressure-volume loops, flow-volume loops

Introduction

Availability of tidal volume measurements and on-line visualization of pulmonary graphics has led to opportunities that facilitate bedside assessment of neonatal pulmonary functions. Earlier limitations in technology prevented continuous visualization of real-time data and clinicians had to cope with ‘spot’ measured values of respiratory vectors and with calculated measures of dynamic pulmonary mechanics. The current use of microprocessors and sensor technologies has allowed manufacturers and designers of ventilatory equipment to provide clinicians with continuous and real-time display and analysis of ventilatory functions. Interpretation of these data to achieve clinically useful and relevant measures and enhance decision analysis requires an understanding of known physiological principles and clinical judgment to achieve adequate gas exchange (Table 1). A clinician can gain access to this rich source of previously untapped information on the respiratory status of the sick newborn.

Pulmonary graphics refers to the direct and on-line visualization of the three fundamental parameters of the respiratory system namely driving pressure, tidal-airflow and tidal volume. These are continuously displayed on a monitor either as scalar waveforms (Fig. 1) along a time axis or as two-dimensional X–Y plots (Fig. 2). Graphic displays may be accompanied by running average calculated values of pulmonary function parameters such as compliance and resistance as well as the more basic respiratory measurements such as tidal

Table 1. How do you define adequate gas exchange?

- Normal values as defined for a population
- Normal values as defined for individuals
- Clinical discretion, as per:
  - disease state
  - equipment capability
  - physiologic considerations
- Limitations by availability of technology

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volume and minute ventilation. Visualization of pulmonary gas exchange by a graphic assessment of blood gases and their relationship to respiratory support settings may also be considered an extension of pulmonary graphics. These new data sources, adjunctive to traditional blood gas data, chest radiographs and clinical respiratory parameters can be useful in decision-making process for determining ventilator settings and overall ventilatory management of neonates with respiratory failure. In this paper, theoretical basis for clinical applications are reviewed such that real time data can be interpreted in terms of vectors that have a direct impact on clinical decisions. The reader is also referred to classical descriptions of physiologic principles that have led to our understanding of neonatal pulmonary function and graphics [1–4].

Clinical evaluation of respiratory signals

Evaluation of pulmonary oxygenation

The alveolar ‘algebra’ for achieving systemic oxygenation is dependent on the alveolar gas equation (see Table 2). Details of applications of this equation have been extensively described and are well established to determine the alveolar oxygen tension, the alveolar-arterial gradients and how these are impacted by barometric pressure, altitude and body temperature.

Figure 1. Scalar monitoring of pressure, flow and volume signals during spontaneous breathing. The pressure signal has been divided (as demarcated by a straight line connecting points of zero flow) to differentiate the elastic pressure from the resistive pressure (shaded portion).

Figure 2. (a) Pressure-volume loops in a neonate at 2 and then 12 days age during mechanical ventilation (driving pressure = 13 cm H2O) shows a progressive increase in expiratory resistance due to increased resistive load. (b) Tidal flow-volume loop from a normal preterm term neonate and a preterm neonate with high expiratory resistance, thus the ‘ski-slope’ effect during expiration.
Evaluation of pulmonary ventilation

The alveolar ‘algebra’ for achieving ventilation is based on pulmonary gas law (see Table 3). This relationship has had a limited exposure in neonatal practice and has immense scope for clinical care. Tidal volume measurement is an essential index of alveolar ventilation. The three fundamental respiratory signals: driving pressure (P), volume (V) and flow (V/time) are measured as a function of time. Pressure and airflow signals are the ones actually measured, whereas the volume signal is derived from integration of the flow signal (volume/time).

Evaluation of synchronous and asynchronous breathing

Real-time evaluation of synchronous respiratory cycles allows for visualization of successive P-V and flow-volume loops as they superimpose neatly over each preceding loop. Asynchrony of respiratory cycles may be evident during airway obstruction (secretions, bronchospasm etc.), ‘bucking’ (during mechanical ventilation or involuntary valsalva manoeuvres) and during agitation (pain, excessive handling, impaired gas exchange). Objective evaluation of asynchrony is difficult to quantify. On the other hand, synchronous ventilation is easily observed on-line.

Evaluation of pulmonary mechanics

The alveolar ‘algebra’ to determine the least baro-trauma and optimal driving pressure is best defined

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**Table 2. Alveolar algebra: oxygenation**

<table>
<thead>
<tr>
<th>Relationship of inspired oxygen (FiO₂) and arterial oxygen tension (PaO₂)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂ = FiO₂ - (PACO₂/R) + PACO₂ × FiO₂ × (1 - R)/R</td>
</tr>
<tr>
<td>PaO₂ = [FiO₂ (P₀ - 47)] - (PACO₂/R) + PACO₂ × FiO₂ × (1 - R)/R</td>
</tr>
<tr>
<td>PaO₂ = 150 - 44 × 2 = 108 mmHg</td>
</tr>
<tr>
<td>PaO₂ = PaO₂ - PaO₂ = (A - a diff), or, PaO₂ = PaO₂ - (A - a gradient)</td>
</tr>
</tbody>
</table>

**Table 3. Alveolar algebra: ventilation**

| Alveolar ventilation inversely correlated to PaCO₂ |
| Alveolar ventilation is (Vₖ - V₀) × rate |
| Provided dead space is constant: proportional changes in Vₖ or rate will lead to proportional changes in alveolar ventilation and PaCO₂ |

Based on Pulmonary Gas Law: in a steady state, when inspired CO₂ is negligible, then PACO₂ = 863 (VCO₂/V̅A), where, VCO₂ is CO₂ production; PACO₂ is partial pressure of carbon dioxide; 863 is body temp. × std pressure/standard temperature (310 × 760/273 = 863).

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**Evaluation of minute ventilation**

The summation of individual tidal volumes over a 1 min period gives us the minute ventilation. This can also be expressed as the product of tidal volume and respiratory rate. Respiratory rates of most preterm and term infants are 20 to 60 breaths/min. A normal full-term newborn infant at rest breathing at a rate of 40 breaths/min and with a tidal volume of 8 ml/kg has, from an algebraic perspective, a minute ventilation of 320 (ml/kg)/min. Usual normal values range from 240 to 480 (ml/kg)/min [12]. Minute ventilation minus ventilated dead space equals alveolar ventilation. [13] Alveolar ventilation has a direct inverse relationship to alveolar PCO₂ or arterial carbon dioxide tension [14]. Thus, at extremes of infant tachypnea (~100 breaths/min or more), tidal volume is reduced while dead space is unchanged and effective minute ventilation is lost, as in untreated respiratory distress syndrome.
by assuming a linear respiratory model (see Table 4). Knowledge of the delivered driving pressure, use of an interpretative analysis of pulmonary compliance and resistances provides a clinician with strategies to optimize and determine appropriate driving pressure.

**Tidal volume signal**

The tidal volume is evident from placement of a pneumotachometer. The digital readout provides the variability that is evident between spontaneous, mechanical and augmented breaths. In fact, the optimal peak inflating pressure can be ascertained by adjusting to appropriate tidal volume (thereby providing a more objective assessment to auscultation). In a clinical condition, when the baby is breathing synchronously, or when spontaneous breathing have been diminished or abolished, the steady measures of tidal volume provide clinically useful information. Firstly, when the tidal volume value is between 5 to 8 ml/kg and there are no signs of pressure-volume over-distension, the clinician may ascertain that ventilation is at optimal FRC. Incremental changes (by 1 cm H₂O) in PEEP may ascertain that ventilation is at optimal signs of pressure-volume over-distension, the clinician may ascertain that ventilation is at optimal.

**Driving pressure signal**

This is the net pressure change required to overcome elastic, airflow-resistive and inertial properties of the respiratory system during inspiration. Breathing requires the generation of a driving pressure. For inspiration to occur, alveolar pressure must be less than the pressure at the mouth. For expiration to occur, alveolar pressure must be higher than the mouth pressure. During spontaneous breathing this pressure gradient is generated by the respiratory muscles, this gradient between the mouth and intrapleural pressures is defined as the transpulmonary pressure. In mechanical ventilation the ventilator produces the driving pressure that is usually measured as the gradient between the peak inflating pressure (PiP) and the positive end-expiratory pressure (PEEP). The driving pressure at the end-inspiratory portion of the ventilation cycle provides a close estimate of elastic pressure. It is in this context that the ratio of tidal volume to driving pressure provides an indirect approximation of total respiratory compliance. In the absence of over or under inflation and when ventilation is being administered in the most linear portion of the respiratory pressure-volume relationship, this value of respiratory compliance (in ml/cm H₂O) provides an estimation of the volume change (in millilitres) per 1 cm H₂O change in driving pressure (Table 4). These estimated data can provide a crucial understanding of the anticipated change in tidal volume for a given incremental change in ventilator driving pressure. Based on this observation the clinician can differentiate between the effects of over-distending or under-distending the lung. As demonstrated in Figure 2a, when ventilation is shifting away from the linear portion of the P-V loop, actual tidal volume becomes smaller than anticipated. Likewise, when ventilation is moving into the linear portion of the P-V loop, actual tidal volume becomes larger than anticipated. The driving pressure should be distinguished from the mean airway pressure, which is a function of inspiratory time, end-distending pressure and respiratory rate. Mean airway pressure has a direct relationship to oxygenation, whereas, driving pressure provides an insight to respiratory elastic status.

**Pulmonary mechanics and lung compliance**

The elastic behaviour of the lungs is characterized by this pressure-volume curve. More specifically,
the ratio of change in lung volume to change in distending pressure defines the compliance of the lungs. Although the pressure-volume relationship of the lung is not linear over the entire range, the compliance (of slope $\Delta V/\Delta P$) is linear over the normal range of tidal volumes beginning at functional residual capacity. Thus, for a given change in driving pressure, tidal volume will increase in proportion to lung compliance or, $\Delta V = C \Delta P$. As lung compliance is decreased, the lungs are stiffer and more difficult to expand. When lung compliance is increased, the lung becomes easier to distend, and is more compliant. Lung compliance and pressure-volume relationships are determined by the interdependence of elastic tissue elements and alveolar surface tension. Tissue elasticity is dependent on elastin and collagen content of the lung. A typical value for lung compliance in a young healthy newborn is 1.5 to 2.0 ml/cm H$_2$O/kg (based on average tidal volumes of 6 to 8 ml/kg and driving pressures of 4 to 5 cm H$_2$O). Estimation of the effective respiratory compliance is a poor measurement of the ‘stiffness’ of lung and not an objective index of its elasticity. Its clinical usefulness is that of providing the clinician an index of the volume change that is likely to occur for every 1 cm H$_2$O change in driving pressure provided the lungs are operating in a linear pressure-volume relationship. This assumption should be valid and anticipated during tidal breathing at optimal FRC.

**Pulmonary mechanics and resistive properties**

Non-elastic properties of the respiratory system characterize its resistance to motion. Since motion between two surfaces in contact usually involves friction or loss of energy, resistance to breathing occurs in any moving part of the respiratory system. These resistances would include frictional resistance to airflow, tissue resistance, and inertial forces. Lung resistance is predominantly (80%) attributed to frictional resistance to inspiratory and expiratory airflow in the larger airways. Tissue resistance (19%) and inertial forces (1%) also influence lung resistance. Airflow through the airways requires a driving pressure resulting from changes in alveolar pressure. When alveolar pressure is less than atmospheric pressure (during spontaneous inspiration), air flows into the lung; when alveolar pressure is greater than atmospheric pressure, air flows out of the lung. By definition, resistance to airflow is equal to the resistive component of driving pressure ($P_r$) divided by airflow ($V/t$).

**Pulmonary graphic representation of inspiratory and expiratory tidal airflow**

Airflow increases at the initiation of the respiratory cycles, reach a maximum usually at mid-cycle and returns to zero flow at the end of each phase (see Fig. 2b). The location of the peak value depends on the site of maximal airway resistance, such that airflow is measured at its peak rather than at mid-respiratory cycles. It is important to differentiate ventilator circuit airflow from the inspiratory and expiratory airflow that traverses the endotracheal tube.

**Clinical applications of pulmonary functions for bedside ventilator management**

Bedside evaluation of history, clinical assessment, blood gas and acid-base profiles and the interaction of the baby to any supportive respiratory devices enhance the bedside application of pulmonary physiologic principles especially for a neonate with respiratory distress. The non-invasive assessment of the three respiratory signals provides objective, valuable on-line data that may be used in an adjunctive manner to monitor, interpret and define the severity of dysfunction. These data do not provide a clinical diagnosis, but can be useful in the following manners: (a) evaluation of alteration and/or limitation in inspiratory/expiratory airflow; (b) evaluation of driving pressure, work and the effort to maintain minute ventilation; (c) evaluation and calculation of the elastic and resistive components of pulmonary dysfunction; (d) calculation of the inspiratory, expiratory and total lung time constants; (e) evaluation of the interaction between spontaneous breathing and conventional mechanical ventilation including CPAP; (f) evaluation of the degree of response to a therapeutic intervention; (g) evaluation of the evolution and resolution of the respiratory disease.

**Optimizing peak inflating pressure**

If a baby is being managed on a pressure-limited ventilatory support, visualizing the concomitant
tidal volume may corroborate the selection of a chosen peak inflating pressure. A suggested goal would be to initially ventilate at the low ‘normal’ value of tidal volume (such as 5 to 6 ml/kg). This provides for a more objective approach than choosing the peak inflating pressure on the basis of auscultation for adequate breath sounds during manual ventilation. Similarly, the tidal volume actually delivered to a neonate can be measured when setting the volume support during volume-controlled ventilation.

Optimizing peak end-expiratory pressure

It is feasible to define an optimal end-distending pressure using pulmonary graphics; however, the process is complex and at present not user-friendly. Using a combination of the effects of driving pressure on tidal volume and visual changes in P-V relationships, one can ascertain whether incremental changes in PEEP lead to pulmonary over- or under-distension or moving to a linear component of the P-V relationship (see Case Study example in Fig. 3 and Tables 5, 6). Because, the clinical goal is to ventilate at the linear portion of the P-V loop, bedside incremental changes in PEEP should only be done by experienced clinicians who can accurately assess the changes in measured data and thereby calculate the impact of PEEP manipulation. As PEEP is increased from 2 to 8 cm HO, the surface would increase in a linear manner unless the lungs are over-distended or under-inflated. The concomitant increase in the expiratory surface area of the lung would improve oxygenation. As one seeks to define a tidal volume that requires the least driving pressure (breathing at the most linear component of the P-V relationship), the optimal PEEP would be the one at which the least oxygen supplement is needed (thus, reduction in PEEP should not lead to an increase in inspired oxygen). It is also important for a clinician to recognize any inadvertent PEEP (Table 7) and realize that the display of PEEP proximal to the endotracheal tube and does not measure inadvertent PEEP.

Optimizing circuit airflow

Usually, the circuit airflow of the ventilator has not been an active decision of the clinician and has been based upon manufacturer’s guidelines. It is well known that excessive circuit airflow can lead to over-distension and inadvertent excessive PEEP. Both of these effects would lead to hypoventilation and subsequent hypercapnia. The pulmonary graphic manifestations would be lower tidal volumes, wider pulmonary hysteresis, pressure over-distension and perhaps turbulence in the airflow signal. These would be immediately corrected by a bedside manoeuvre to reduce the circuit airflow.
Another option to set the circuit airflow is to base the setting on an eight-fold product of desired minute ventilation (tidal volume and respiratory frequency).

Optimizing inspiratory time

Inspiratory time may be increased or decreased (and thereby change the expiratory time) by a clinician as a response to changes in the mean airway pressure and oxygenation. These are usually clinical decisions based on the physiologic understanding of respiratory time constants (product of compliance and resistance). In addition to the impact on oxygenation, the concomitant and often indirect beneficial or deleterious effects of the new inspiratory time can be assessed on the pulmonary graphics. These include the effects on tidal volume, inspiratory and expiratory hysteresis, pressure-volume relationship (such as over distension from excessive mean airway pressure), and flow volume relationships (such as expiratory flow limitation from excessive and inadvertent PEEP) as sequelae of shortened expiratory time.

Optimizing synchrony and rate of ventilatory support

Real-time evaluation of synchronous ventilation on the graphic displays is helpful for nurses, respiratory therapist and physicians to assess non-ventilatory means to correct asynchronous ventilation. The clinical value of the visual display allows for early response to a neonate’s discomfort. Babies who continue to ‘buck’ the ventilator and are not amenable to bedside comforting and nursing measures, may demonstrate their response to ventilatory technologies based synchronized ventilation.

Optimizing inspired oxygen

The process of plotting serial arterial blood gases on the PO2–PCO2 nomogram (Fig. 4) provides the clinician a perspective on the extent of the variation induced either by disease or the operator. Operator-driven swings in oxygenation may be minimized by prospective decisions (such as use of the alveolar gas equation) or by invoking changes in a cautious and incremental manner.

Optimizing ventilatory strategies for permissive hypercapnia

The relationship between alveolar ventilation and arterial carbon dioxide tension is incredibly linear and may be used as an advantage in defining desired goals for ‘permissive’ hypercapnia. Selection of PCO2 value of 50 Torr in lieu of the ‘normal’ 40 Torr is a choice of defining a 25% deviation; this may actually indicate hyperventilation by 25%. This decision could be an elective clinical maneuver but the clinician needs to ensure

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**Table 5. Clinical case**

<table>
<thead>
<tr>
<th>1.15 kg male neonate, at 28 weeks' gestation, with RDS, treated with surfactant and on ventilatory support.</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>Arterial gas: pH = 7.32; PaO2 = 89 torr; PaCO2 = 52 torr</td>
<td></td>
</tr>
<tr>
<td>Settings: 18/5 cm H2O × 35 breaths/min, FiO2 = 0.34</td>
<td></td>
</tr>
<tr>
<td>Driving pressure: 18 = 5 = 13 cm H2O</td>
<td></td>
</tr>
<tr>
<td>Tidal volume (measured): = 7.8 ml (6.8 ml/kg)</td>
<td></td>
</tr>
<tr>
<td>'Effect compliance' = ΔV/ΔP; 7.8/13 = 0.6 ml/cm H2O</td>
<td></td>
</tr>
<tr>
<td>Compliance = Δ volume/ΔP; 7.8 ml H2O or, Δ 1 cm H2O leads to Δ in volume = compliance</td>
<td></td>
</tr>
<tr>
<td>A wean to 17/5, projected tidal volume = 7.8–0.6 = ~ 7.2 ml</td>
<td></td>
</tr>
</tbody>
</table>

**Table 6. Clinical case: alveolar algebra**

<table>
<thead>
<tr>
<th>A dual wean to 17/4, led to a measured Δ in volume of 10.2 ml, 'effective' compliance = ΔV/ΔP; 10.2/13 = 0.78 ml/cm H2O</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td>● Δ 1 cm H2O leads to Δ in volume = 0.78 ml</td>
<td></td>
</tr>
<tr>
<td>● Δ 1 cm H2O to 16/4 will decrease tidal volume to ~ 9.4 ml</td>
<td></td>
</tr>
<tr>
<td>● Δ 1 cm H2O to 15/4 will decrease tidal volume to ~ 8.6 ml</td>
<td></td>
</tr>
<tr>
<td>● Δ 1 cm H2O to 14/4 will decrease tidal volume to ~ 7.8 ml</td>
<td></td>
</tr>
<tr>
<td>With incremental weaning driving pressure could be 10 cm H2O, if successfully weaned to 14/4, and a tidal volume of 7.8 ml (6.8 ml/kg) provided there is no concomitant increase in inspired oxygen</td>
<td></td>
</tr>
</tbody>
</table>

**Table 7. Frequent reasons for inadvertent PEEP**

| ● High ventilatory circuit airflow |  |
| ● Long inspiratory time |  |
| ● Shortened expiratory time |  |
| ● Rapid respiratory rates |  |
| ● Increased airway resistances |  |
| ● Expiratory flow limitation |  |

Another option to set the circuit airflow is to base the setting on an eight-fold product of desired minute ventilation (tidal volume and respiratory frequency).
Figure 4. The arterial PO$_2$-CO$_2$ nomogram plots serial arterial oxygen and carbon dioxide gas tensions. Dashed lines demarcate ‘Normal’ ranges. The dotted lines define the ranges of oxygen saturation. Solid lines connect serial values of blood gases. The mean values, their standard deviation and coefficient of variance for both arterial oxygen and carbon dioxide tensions are shown in the inset. These variations may be due to disease-related or operator-induced effects and may be interpreted by clinical review.

that the decision is not a passive one such that atelectatic lungs are being ventilated. Again, the plotting of serial blood gases on a PO$_2$-PCO$_2$ nomogram (Fig. 4) provides the clinician with a direct visual impact of the recent gas exchange history such that prospective decisions are made consciously and conscientiously.

In conclusion, at present there are no evidence-based studies that would indicate that the optimization of ventilatory support and the reduction in pressure-related barotrauma, volume or airflow over-distension or reduction in alveolar hypoxemia would reduce the severity or incidence of chronic lung disease. Even though this effect may be conjectured on the basis of fundamental principles of pulmonary physiology and clinical acumen, such evidence needs to be gathered. Limitations in designing such studies may be attributed to ability to achieve clinical consensus, define appropriate monitoring endpoints for respiratory and alveolar barotrauma and shift the emphasis from the ventilator (equipment) to the Ventilator (the clinician at the bedside). In the meantime, a clinician needs to be guided a fundamental principle of neonatal ventilation: ‘First, use the least level of support to maintain adequate gas exchange.’

References


Case Study

Use of “effective” compliance measure as a means to predict the change in tidal volume upon adjustment of inflating pressures. Three options are described. When resultant measured tidal volume exceeds or are lower than the predicted tidal volume, these suggest that the baseline settings were at low FRC or high FRC (in conjunction with Figure 3).

Baseline ventilator settings in 28 week preterm neonate who is 4 h age, post surfactant treatment, with arterial blood gases of PO2=89 torr, PCO2=52 torr, pH=7.32 and receiving inspired fractionated oxygen of 0.34

<table>
<thead>
<tr>
<th>PIP/PEEP (cm H2O)</th>
<th>Driving pressure (cm H2O)</th>
<th>Tidal volume (ml)</th>
<th>Effective compliance (ml/cm H2O)</th>
</tr>
</thead>
<tbody>
<tr>
<td>18/5</td>
<td>13</td>
<td>7.8</td>
<td>0.7</td>
</tr>
<tr>
<td>Options to adjust ventilator settings</td>
<td>Option no. 1: Increase PIP by 1 cm H2O</td>
<td>19/5</td>
<td>Option no. 2: Decrease PIP by 1 cm H2O</td>
</tr>
<tr>
<td>New settings</td>
<td>Option no. 3: Dual wean (PIP/PEEP) by 1 cm H2O</td>
<td>17/4</td>
<td></td>
</tr>
<tr>
<td>Expected tidal volume*</td>
<td>8.4 ml (7.8+0.6)</td>
<td>7.2 ml (7.8–0.6)</td>
<td>7.8 ml</td>
</tr>
<tr>
<td>Measured tidal volume</td>
<td>On 19/5</td>
<td>On 17/5</td>
<td>On 17/4</td>
</tr>
<tr>
<td>(a) Baseline settings at sub-optimal FRC</td>
<td>&gt;8.4 ml</td>
<td>&lt;7.2 ml</td>
<td>&gt;7.8 ml</td>
</tr>
<tr>
<td>(b) Baseline settings at optimal FRC</td>
<td>8.4 ml</td>
<td>7.2 ml</td>
<td>7.8 ml</td>
</tr>
<tr>
<td>(c) Baseline settings above optimal FRC</td>
<td>&lt;8.4 ml</td>
<td>&gt;7.2 ml</td>
<td>&lt;7.8 ml</td>
</tr>
</tbody>
</table>

*Expected change in tidal volume is the actual tidal volume plus that due to adjusting the driving pressure. If the driving pressure is increased by 1 cm H2O, then the increase in tidal volume will be equivalent to the ‘effective’ compliance (ml/cm H2O).