Mid-Frequency Ventilation: Unconventional Use of Conventional Mechanical Ventilation as a Lung-Protection Strategy

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BACKGROUND: Studies have found that increasing the respiratory frequency during mechanical ventilation does not always improve alveolar minute ventilation and may cause air-trapping. OBJECTIVE: To investigate the theoretical and practical basis of higher-than-normal ventilation frequencies. METHODS: We used an interactive mathematical model of ventilator output during pressure-control ventilation to predict the frequency at which alveolar ventilation is maximized with the lowest tidal volume ($V_T$) for a given pressure. We then tested our predicted optimum frequencies and $V_T$ values with various lung compliances and higher-than-normal frequencies, with a lung simulator and 5 mechanical ventilators (Dräger Evita XL, Hamilton Galileo, Puritan Bennett 840, Siemens Servo 300 and Servo-i). RESULTS: Compliances between 10 mL/cm H$_2$O and 42 mL/cm H$_2$O yielded $V_T$ between 4.1 mL/kg (optimum frequency 75 cycles/min) and 6.0 mL/kg (optimum frequency 27 cycles/min). The intrinsic positive end-expiratory pressure at the optimum frequency was always less than 2 cm H$_2$O. All the ventilators except the Hamilton Galileo had an optimum frequency near 50 cycles/min, whereas the predicted optimum frequency was 60 cycles/min. CONCLUSIONS: With these ventilators and pressure-control ventilation, alveolar minute ventilation can be optimized with higher-than-normal frequency and lower $V_T$ than is commonly used in patients with acute respiratory distress syndrome. We call this strategy mid-frequency ventilation. Key words: mechanical ventilation, high-frequency ventilation, tidal volume, $V_T$, acute respiratory distress syndrome, acute lung injury. [Respir Care 2008;53(12):1669–1677. © 2008 Daedalus Enterprises]
increasing the frequency in conventional mechanical ventilation is not an effective way to improve gas exchange.\textsuperscript{7,12}

The relationship between frequency and $V_E$ partly depends on the ventilation mode (volume control vs pressure control). This difference is explained by the determinants of $VT$. In volume control the $VT$ is a set parameter. With pressure control the $VT$ (of a passive inspiration) is the result of the interaction between the change in airway pressure (end-inspiratory pressure minus end-expiratory transrespiratory system pressure), the time constant of the respiratory system, and the set inspiratory time.\textsuperscript{13} When the respiratory system characteristics remain constant, the change in pressure difference and inspiratory time determine the $VT$.

While keeping a constant duty cycle, increasing the frequency will have these effects:

1. In volume-control ventilation, the $V_E$ and alveolar $V_E$ increase linearly. The resultant decrease in inspiratory time causes gas-trapping and a linear increase in auto-PEEP and $P_{aw}$.

2. In pressure-control ventilation, $V_T$ decreases due to shortened inspiratory time and auto-PEEP. However, the drop in volume is offset by the increase in frequency, so $V_E$ increases asymptotically.\textsuperscript{14} In contrast, because $V_D$ remains relatively constant, alveolar volume decreases with frequency, and alveolar $V_E$ reaches a maximum value at what we call the optimum frequency. Interestingly, although auto-PEEP develops, $P_{aw}$ doesn’t change.\textsuperscript{14} These characteristics make pressure-control ventilation an attractive option to evaluate higher ventilatory frequencies with conventional mechanical ventilation. However, in 1993 Burke et al found that one of the best intensive-care ventilators at that time could not deliver pressures and volumes consistent with theoretical ideals.\textsuperscript{15} To our knowledge, no further investigations of this sort have been attempted since.

We studied the theoretical and practical basis of using higher-than-normal frequencies with pressure-control ventilation in a simulated paralyzed patient with ARDS. In the first phase of the study we developed an interactive spreadsheet program in which we applied a mathematical model to predict ventilator output with given patient and ventilator variables. We used the mathematical model to predict the frequency that would maximize alveolar ventilation. In the second phase we used a lung model of a patient with ARDS and tested 5 mechanical ventilators at higher frequencies than are commonly used.

Methods

Phase 1: Mathematical Model

We used a validated mathematical model\textsuperscript{14,15} to describe pressure-control continuous mandatory ventilation of a passive, single-compartment, lumped-parameter model. The equations in Table 1 include patient-determined and clinician-set variables, from which the ventilator output is obtained. The original equation for alveolar ventilation\textsuperscript{14} was modified by making $V_D$ a constant volume based on patient weight (rather than a fixed percentage). This modification resulted in an expression for alveolar ventilation that has a maximum value over a range of ventilator frequency from 1 to 150 cycles/min. For convenience, the model was designed to allow input of the $V_D/VT$ ratio rather than absolute values for $V_D$, because $V_D/VT$ can be easily evaluated at the bedside (eg, with a NICO2 monitor, Respironics, Carlsbad, California). $V_D$ is then calculated as the product of $V_D/VT$, body weight, and $VT$ (in mL/kg). For this baseline calculation.

<table>
<thead>
<tr>
<th>Table 1. Modeling Equations for Pressure-Control Ventilation</th>
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<tbody>
<tr>
<td>$V_T = (P_{aw} \times C)(1 - e^{-66D/fRE})(1 - e^{-0.05D/fRE})$</td>
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<tr>
<td>$V_E = f \times V_T$</td>
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<tr>
<td>$V_A = V_T - V_D$</td>
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<tr>
<td>$V_A = f \times V_A$</td>
</tr>
<tr>
<td>auto-PEEP = $P_{aw}(e^{-0.05D/fRE})(1 - e^{-0.05D/fRE})$</td>
</tr>
<tr>
<td>$P_{aw} = P_{aw} + D + $PEEP</td>
</tr>
<tr>
<td>$V_D = \frac{V_D}{VT} \times V_T$</td>
</tr>
<tr>
<td>Required $V_A = \frac{V_D \times 1.35}{0.05}$</td>
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</table>

\begin{align*}
V_T & = \text{tidal volume} \\
P_{aw} & = \text{set pressure limit (cm H}_2\text{O above the set positive end-expiratory pressure)} \\
C & = \text{compliance (L/cm H}_2\text{O)} \\
\varepsilon & = \text{Euler’s constant (the base of natural logarithms; approximately 2.718)} \\
D & = \text{duty cycle (ratio of inspiratory time to total respiratory cycle time)} \\
f & = \text{ventilator frequency (cycles/min)} \\
R_I & = \text{inspiratory resistance (cm H}_2\text{O/L/s)} \\
R_E & = \text{expiratory resistance (cm H}_2\text{O/L/s)} \\
V_D & = \text{minute volume} \\
V_A & = \text{alveolar tidal volume (L)} \\
V_D & = \text{dead space (L)} \\
V_{aw} & = \text{alveolar minute volume (L/min)} \\
\text{auto-PEEP} & = \text{intrinsic positive end-expiratory pressure} \\
P_{aw} & = \text{mean airway pressure}.
\end{align*}
we assumed a $V_T$ of 6 mL/kg, so $V_D$ was assumed to be constant and $V_D/V_T$ increased as the pressure-control $V_T$ decreased.

Once the patient variables were determined, we set the ventilator variables as follows: The duty cycle was adjusted to 45%, to maximize volume delivery (optimal duty cycle). When $R_{1}/R_E$ the optimal duty cycle is 50%, and vice versa if $R_{1} < R_E$. PEEP was set at 9 cm H$_2$O, and the pressure limit above PEEP was adjusted to produce a $V_E$ of approximately 13 L/min at a frequency of 30 cycles/min. These values were selected to match the average values in the ARDS Network study, so that our first hypothesis could be tested with realistic values for alveolar ventilation, $V_T$, and optimum frequency.

Figure 1 shows an example of the model output, based on the lung mechanics and ventilator settings of a simulated patient with ARDS. Alveolar ventilation is maximized at a frequency of 52 cycles/min. The $V_T$ calculated by the model (not shown in Table 1) was 6.4 mL/kg at 30 cycles/min, which was the average frequency used in the ARDS Network study. The optimum frequency of 52 cycles/min yielded an alveolar ventilation of 8.5 L/min, but the required alveolar ventilation for this patient was only 5.3 L/min, so we reduced the inspiratory pressure above PEEP, which reduced the predicted optimum frequency. We considered the $V_T$ calculated by the spreadsheet at the required alveolar $V_E$ and new optimum frequency to be the optimum tidal volume ($V_T$). We repeated this procedure for a range of compliance settings: 0.15–0.63 mL/cm H$_2$O/kg (ie, 10–42 mL/cm H$_2$O for a simulated body weight of 66 kg), to match the average value in the ARDS Network study.

**Phase 2: Ventilator Performance**

We used a high-fidelity servo lung simulator (ASL5000, IngMar Medical, Pittsburgh, Pennsylvania) to evaluate the relationship between frequency and alveolar $V_E$ with 5 common intensive-care ventilators (Evita XL, Dräger, Lübeck, Germany; 840, Puritan Bennett/Tyco, Mansfield, Massachusetts; Galileo, Hamilton, Bonaduz, Switzerland; Servo-i, Siemens Elema, Solna, Sweden; and Servo 300, Siemens Elema, Solna Sweden) and compared the ventilators’ performance to the theoretical results from the mathematical model. We directly measured and/or calculated the ventilator output. The lung simulator uses a computer to control the movement of a piston according to the equa-
tion of motion for the respiratory system. Constant compliance is simulated by moving the piston such that:

\[ \frac{dV}{dt} = \frac{dP}{dH} \times C \]

where \( d \) is the derivative with respect to time (t), \( V \) is volume, \( P \) is pressure, and \( C \) is compliance. The constant resistance is calculated as:

\[ R = \frac{dP}{dV/dt} \]

where \( R \) is resistance. So the piston is moved at a speed of:

\[ \frac{dV}{dt} = \frac{dP}{R} \]

The lung simulator can also model non-constant resistance and compliance and patient ventilatory efforts (simple triggering efforts or full spontaneous breathing).

We set the lung simulator to model a passive respiratory system, composed of a single linear constant resistance and single constant compliance: \( R_1 = R'_E = 10 \text{ cm H}_2\text{O/ L/s} \), and compliance of 0.025 L/cm H\(_2\)O. These parameters were kept constant during all the experiments. Data from the lung simulator were sampled at 500 Hz. Tidal volume was measured as the excursion of the piston inside the lung simulator (measured and displayed by the lung simulator’s software). Alveolar volume was calculated as the \( V_T \) minus an assumed constant \( V_D \) of 150 mL.

Each ventilator was connected to the lung simulator via a conventional circuit (approximately 180 cm) with separate inspiratory and expiratory limbs (Airlife, Cardinal Health, McGaw Park, Illinois). We also attached an adult humidifier (MR250, Fisher & Paykel, Auckland, New Zealand) filled to the level line with unheated water. We used the same circuit for all the experiments. We calibrated and tested the ventilators for leaks prior to the experiments.

**Experiment Protocol**

All experiments were conducted with room air (fraction of inspired oxygen 0.21) and are reported as measured. The ventilator was set to deliver pressure-control continuous mandatory ventilation (ie, all breaths were time-triggered, pressure-limited, and time-cycled).

The mathematical model is based on a perfect square pressure waveform, so we set the ventilators to deliver as-perfect-as-possible square waveform (ie, minimum pressure rise time). The inspiratory pressure above PEEP (driving pressure) was set at 20 cm H\(_2\)O. No PEEP was applied (to avoid any interference from the exhalation manifold). The duty cycle was 50% (inspiratory-expiratory ratio 1:1) or as close as possible to 50% if the ventilator settings would not allow precisely 50%. The pressure rise time (if adjustable) and “plateau %” (explained below) were set to achieve an immediate rise to peak pressure. The ventilator frequency was increased in increments of 10 cycles/min, starting at 10 cycles/min, up to the maximum frequency achieved by the ventilator. Given the stability of the model, we report only one run for each experiment. After each incremental step we allowed 1 min to elapse before obtaining readings. Tidal volume, \( P_{aw} \), and auto-PEEP (after an expiratory pause of 2 s) were recorded from the lung simulator. We report the exhaled \( V_T \) because this was the output common to all the ventilators. Exhaled \( V_T \) and alveolar ventilation were calculated with the lung-simulator data.

Because no ventilator is expected to produce a mathematically perfect square pressure waveform, we defined the difference between the actual ventilator output and the theoretical output for \( V_T \), \( P_{aw} \), and auto-PEEP as performance error (performance error = lung-simulator value minus value predicted by mathematical model). We obtained high-definition recordings of the \( P_{aw} \) tracings for comparative analysis.

**Statistical Analysis**

Continuous variables are reported as mean ± SD and minimum and maximum values, as appropriate. Group comparisons of quantitative variables are descriptive and graphed to represent the mathematical model and ventilator performance.

**Results**

**Phase 1: Mathematical Model**

Decreasing the inspiratory pressure with the lung parameters in Fig. 1 decreased the maximum alveolar ventilation to match the required alveolar ventilation calculated by the model, so the optimum frequency decreased to 45 cycles/min and optimum \( V_T \) was 4.8 mL/kg.

Using the procedure described in the methods section to adjust the spreadsheet model, and varying the compliance between 10 mL/cm H\(_2\)O and 42 mL/cm H\(_2\)O yielded \( V_T \) values between 4.1 mL/kg (optimum frequency 75 cycles/min) and 6.0 mL/kg (optimum frequency 27 cycles/min). At the optimum frequency, auto-PEEP was always < 2 cm H\(_2\)O.

Overall, the model behavior (in response to single changes in variables) can be summarized as follows. Increasing the compliance (by shifting the alveolar ventilation vs frequency curve to the left) increased the alveolar ventilation and reduced the optimum frequency. Decreasing the resistance or increasing the inspiratory pressure increased both the alveolar ventilation and the optimum
frequency. Changing the applied PEEP affected only the $P_{aw}$. Changing the duty cycle in either direction away from 50% (inspiratory-expiratory ratio 1:1) decreased both the alveolar ventilation and the optimum frequency. When $R_I > R_E$, the optimal duty cycle is > 50%, and vice versa: if $R_I < R_E$ the optimal duty cycle is < 50%. Increasing the $V_{TD}$ decreased the optimum frequency. The interactive model is available on request to the authors.

Phase 2: Ventilator Performance

Figure 2 illustrates the pressure waveforms recorded by the lung simulator. The Galileo had the least square waveform (discussed below). The 840 had the squarkest waveform (ie, best compared to the ideal step function of the mathematical model). The waveform from the Evita XL at 100 cycles/min represents a duty cycle of 0.20 because of its limited inspiratory-time settings at high frequency.

In addition to the clinician-set variables (PEEP, inspiratory pressure, frequency, and inspiratory-expiratory ratio), other variables inherent to each ventilator need to be adjusted to deliver the best waveform (Table 2). The Evita XL and the Servo-i have limited inspiratory-time settings, which altered the duty cycle at higher frequencies and account for the $V_T$ differences (Fig. 3).

The “inspiratory rise time percent,” “$P_{ramp}$ (ms),” “plateau %,” and “slope rise time” all refer to the speed to reach the plateau of the square waveform. The shortest rise time delivers the best waveform and the largest $V_T$. The shortest rise time on the Galileo is 50 ms, which causes the pressure waveform to lose its squareness and affects the $V_T$ delivered. Interestingly, the 840 had the best square waveform with high demand settings (100 cycles/min), but was unable to deliver the predicted $V_T$ because it produced less pressure-overshoot than the other ventilators. So the 840’s better pressure control was a disadvantage in this context.

Ventilation Outcome Variables

Figure 3 shows the major outcome variable (alveolar ventilation as a function of frequency). With the exception of the Galileo, all the ventilators had an optimum frequency of about 50 cycles/min, whereas the predicted optimum frequency was 60 cycles/min. The Evita XL and Servo-i were the closest to the alveolar $V_{TE}$ predicted. The Galileo had an optimum frequency of 40 cycles/min because of its consistently lower $V_T$.

Figure 4 shows frequency versus auto-PEEP, $P_{aw}$, and $V_T$. For $V_T$, the Evita XL’s performance curve most closely followed the predicted curve up to 70 cycles/min. All the ventilators produced higher auto-PEEP and $P_{aw}$ than predicted by the mathematical model, because airway pressure failed to drop immediately to baseline on exhalation. The effects of limited ventilator settings are seen in the data from the Evita XL and Servo-i at > 70 cycles/min (inspiratory time limited), and with the Hamilton Galileo throughout (slow rise time).

As expected, none of the ventilators was able to perform overall as the mathematical model predicted (Table 3). However, the $V_T$ delivered was within 10% of predicted up to frequencies of 30 cycles/min. Although the Evita XL and Servo-i continued to perform within 10% of predicted up to 70–90 cycles/min, this may be due to overshooting the set inspiratory pressure (see Fig. 2).
**Discussion**

We have demonstrated, in a theoretical and physical model, that, during pressure-control ventilation at a constant duty cycle, increasing the frequency above commonly used frequencies might improve alveolar ventilation. Furthermore, we showed that we can achieve this with low $V_T$ and a constant $P_{aw}$. Interestingly, for many patients with relatively severe ARDS (ie, resistance $\leq 15$ cm H$_2$O/L/s, compliance $\leq 35$ mL/cm H$_2$O, and $V_D/V_T \approx 0.5$) the model predicts that maximum alveolar ventilation can be obtained with a $V_T < 6$ mL/kg at a frequency of $> 35$ cycles/min.

Most clinicians, we think, find the terms “high-frequency ventilation” and “conventional ventilation” of use in teaching and general communication about mechanical ventilation. The method we are proposing is neither. Thus, by referring to our proposed strategy as mid-frequency ven-
tilation we highlight the fact that conventional ventilators (not specialized high-frequency devices) can and possibly should be used at frequencies higher than the accepted normal range for patient size and also higher than commonly reported in sick ventilated patients. We are also pointing to a direction of inquiry that is diametrically opposed to the most popular ventilation mode today (volume control continuous mandatory ventilation, particularly in patients with ARDS).

The equation for alveolar ventilation described by Marini et al treated $V_D$ as a fixed percentage of the $VT$.

\[ V_A = VT - V_D \]

where $V_D$ is assumed to be a fixed value, \(^21\) the alveolar-ventilation-versus-frequency curve displays a peak rather than approaching an asymptote (see Fig. 1). The peak in alveolar ventilation corresponds to an optimum frequency and optimum $VT$. Importantly, in pressure-control ventilation, as described by the mathematical model and confirmed in our experiment, the $P_{aw}$ remained stable throughout the rise in frequency despite the frequency-dependent air-trapping (manifested as auto-PEEP). Indeed, auto-PEEP is predictable and can thus be quantitated nearly as accurately as externally applied PEEP.

A high ventilatory frequency with a conventional ventilator was initially examined while researching high-frequency positive-pressure ventilation, but was later abandoned for highly specialized ventilators.\(^\text{22,23}\) There have been isolated reports and cases series that used prototypes\(^\text{24}\) and adult\(^\text{25}\) and neonatal ventilators.\(^\text{26}\) Recently, Rich SCOEUR et al\(^\text{10}\) evaluated 6 patients with ARDS treated with a permissive hypercapnia strategy (by reducing $VT$). Mechanical ventilation was optimized by increasing the frequency (from $18 \pm 0$ cycles/min to $30 \pm 4$ cycles/min) until auto-PEEP was detected. A significant reduction in $P_{aCO_2}$ (from $84 \pm 24$ mm Hg to $60 \pm 16$ mm Hg) was observed.

Richard et al\(^\text{11}\) described a $P_{aCO_2}$ reduction from $61 \pm 19$ mm Hg to $43 \pm 15$ mm Hg after increasing the ventilator frequency from $17 \pm 3$ cycles/min to $30 \pm 3$ cycles/min, though this generated auto-PEEP ($3.9 \pm 1.1$ cm H\(_2\)O). In contrast, Vieillard-Baron et al\(^\text{7}\) did not find a difference in $P_{aCO_2}$ ($51 \pm 7$ mm Hg versus $47 \pm 8$ mm Hg) when they increased the frequency from $15$ cycles/min to $30$ cycles/min, and that strategy generated substantial auto-PEEP ($6.4 \pm 2.7$ cm H\(_2\)O) and hemodynamic compromise. The differences in the results can be explained by differences in patient population, ventilator settings, and instrumental $V_D$.\(^\text{27}\) All the trials used volume-control ventilation, which on its own seems unlikely to avoid the adverse effects of higher frequencies (see below). The only study that used pressure-control ventilation, by Paulsson et al,\(^\text{28}\) included 53 pediatric patients (mean age $4$ y) with ARDS. They used a mean frequency of $80$ cycles/min (range $40–120$ cycles/min), low $VT$ ($3–5$ mL/kg), and high PEEP. Unfortunately, Paulsson et al did not measure auto-PEEP, and no control group was reported.

There are several reasons for preferring pressure-control ventilation over volume-control ventilation at higher frequencies. The compliance of the patient circuit becomes a complicating factor in volume-control ventilation. Because the patient circuit compliance is in parallel with respiratory-system compliance, $VT$ is partitioned between the two. Prediction equations for volume control would have to include circuit compliance and would be more complicated. In contrast, with pressure control, compliances in parallel are exposed to the same pressure drop and hence the effect of patient circuit compliance is minimal so long as a square pressure waveform is maintained (which our results indicate is a reasonable assumption with some ventilators). Also, the higher the frequency, the shorter the

### Table 3. Performance Error During Mid-Frequency Ventilation

<table>
<thead>
<tr>
<th></th>
<th>Servo 300</th>
<th>Servo-i</th>
<th>Evita XL*</th>
<th>840</th>
<th>Galileo</th>
</tr>
</thead>
<tbody>
<tr>
<td>$VT$ (mean ± SD mL)</td>
<td>$-28.8 \pm 25.8$</td>
<td>$4.1 \pm 25.4$</td>
<td>$9.5 \pm 15.4$</td>
<td>$-34.5 \pm 21$</td>
<td>$-83 \pm 57$</td>
</tr>
<tr>
<td>$VT$ range (mL)</td>
<td>$-55$ to $14$</td>
<td>$-33$ to $35$</td>
<td>$-14.8$ to $23$</td>
<td>$-56$ to $-1$</td>
<td>$-144$ to $1$</td>
</tr>
<tr>
<td>auto-PEEP (mean ± SD cm H(_2)O)</td>
<td>$1.9 \pm 1.1$</td>
<td>$1.2 \pm 0.4$</td>
<td>$0.83 \pm 0.6$</td>
<td>$2.4 \pm 1$</td>
<td>$1.9 \pm 0.5$</td>
</tr>
<tr>
<td>$P_{aw}$ (mean ± SD cm H(_2)O)</td>
<td>$1.8 \pm 0.4$</td>
<td>$1.1 \pm 0.4$</td>
<td>$1.1 \pm 0.3$</td>
<td>$1.8 \pm 0.5$</td>
<td>$-0.03 \pm 1.4$</td>
</tr>
</tbody>
</table>

* Performance error = lung-simulator value minus value predicted by mathematical model |

$VT$ = tidal volume |

auto-PEEP = intrinsic positive end-expiratory pressure |

$P_{aw}$ = mean airway pressure
inspiratory time, and the higher the inspiratory flow must be for a given VT. Pressure control (in general) produces higher peak flow than does volume control, and the flow is automatically adjusted to meet demand (ie, as respiratory system mechanics change), as opposed to volume control, which requires an arbitrary operator preset value.

Most ventilators can deliver adequate VT at relatively high frequencies. Optimum ventilator performance (compared to the mathematical model) results from the ventilator’s ability to deliver as square a pressure waveform as possible. The 3 determinants are: the ability to immediately rise to peak pressure; lack of oscillations in the plateau of the waveform; and the ability to immediately return to baseline pressure. Alterations in rise time and the plateau affect the VT delivered. Alterations in expiration cause air-trapping and higher-than-predicted Paw and auto-PEEP. Minimal changes to ventilator software (settings) and circuits (low VD and compliance) should improve delivery of pressure-control ventilation at higher frequencies.

There are limitations to our mathematical model. Certain assumptions have to be made to derive the equations. We assumed a passive respiratory system (ie, to simulate a paralyzed patient). We assumed that inertance was negligible; however, as frequency increases, inertance may play a more important role. Inertance would decrease the VT with a given inspiratory pressure limit and thus account for some of the difference between model predictions and ventilator performance.

Another limitation has to do with assumptions about VD. Physiologic VD changes with frequency, VT, lung perfusion, and instrumentation. Furthermore, other factors make mathematical modeling very difficult, including the effects of mixing time in CO2 removal, other mechanism of gas exchange, and pathology-specific VD alterations. Our model simply assumed a constant VD calculated from the VD/VT (easily obtained at the bedside) at a given VT and normalized for predicted body weight.

The simplicity of our mathematical model, which considers a single unit with constant compliance and resistance, may seem an over-simplification of the lung. However, models as simple as this have been used previously to calculate the behavior of the respiratory system, and were successfully implemented as ventilation modes. Indeed, adaptive support ventilation (a pressure-control mode on Hamilton ventilators) uses the same assumptions as our model (ie, predicted VD and VE based on weight, and constant resistance and compliance). The difference is that adaptive support ventilation predicts optimum frequency and VT based on minimizing the mechanical work of breathing, whereas our model for mid-frequency ventilation maximizes the alveolar VE.

The physical model of the respiratory system we used has the same limitations as the mathematical model. The ASL5000 lung simulator maintains resistance and compliance constant, which is a major difference from the study by Burke et al.

Another limitation is the assumption of equal RL and RE in our ventilator experiments. It is more likely that expiratory resistance is greater than RL, so the optimum frequency in terms of alveolar ventilation would be shifted downwards. Nevertheless, our general results regarding these ventilators’ ability to deliver higher frequencies still apply, with the understanding that clinical application would require bedside determination of both RL and RE.

Conclusions

With both a mathematical and a physical model we demonstrated the feasibility of predicting an optimal frequency and VT based on maximizing alveolar VE during pressure-control ventilation. VT values determined in this way are lower and frequencies are higher than those reported in the literature for ventilating patients with ARDS. Our findings suggest that this approach may offer benefits, compared to conventional volume-control modes.

REFERENCES


