Laboratory Evaluation of the Vortran Automatic Resuscitator Model RTM

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BACKGROUND: One device that has been proposed to address the need for emergency ventilation is the Vortran Automatic Resuscitator. OBJECTIVE: To test the hypothesis that increasing load (ie, increasing resistance or decreasing compliance) significantly affects minute alveolar ventilation. METHODS: A Vortran Automatic Resuscitator was connected to a passive lung model and we measured load with 8 combinations of 4 compliances (14, 28, 46, and 63 mL/cm H2O) and 2 resistances (20 and 42 cm H2O/L/s). Source gas flow was either 20 or 40 L/min. We measured tidal volume (VT), frequency, inspiratory time, expiratory time, peak inspiratory pressure, and intrinsic positive end-expiratory pressure. We calculated the ratio of inspiratory time to total cycle time (TI/Ttot), minute ventilation, minute alveolar ventilation, and estimated PaCO2. Raw data were summarized with descriptive statistics. A subset of the experimental data (outcome measures for conditions with high and low values for resistance, compliance, and source gas flow) was analyzed with a 2-level factorial design, with standard “design of experiments” procedure, including analysis of variance. Differences associated with p values ≤ 0.05 were considered significant. RESULTS: Assuming the model lung represented a 68-kg adult, the measured VT ranged from a low of 1.7 mL/kg to a high of 16.7 mL/kg. TI/Ttot was greatly affected by the input flow. At 40 L/min the average TI/Ttot was 30%, and at 20 L/min TI/Ttot was 52%. As the load increased, VT decreased and frequency increased. However, neither the minute ventilation nor the minute alveolar ventilation stayed constant. Minute ventilation ranged from 5.2 L/min to 11.3 L/min at 40 L/min source flow. More importantly, minute alveolar ventilation ranged from zero to 9.8 L/min, resulting in a calculated PaCO2 range of over 100 mm Hg to 16 mm Hg, respectively. Indeed, calculated PaCO2 was never in the normal range (35–45 mm Hg). “Design of experiments” analysis showed that VT was affected by compliance and resistance (p < 0.001 and p < 0.05, respectively). Frequency was affected only by compliance (p < 0.001). Minute alveolar ventilation was affected by compliance and resistance (p < 0.001 and p < 0.01, respectively). Minute alveolar ventilation increases as compliance increases and/or resistance decreases, but these variables were essentially independent. CONCLUSIONS: The Vortran Automatic Resuscitator showed an automatic increase in frequency and decrease in VT that resulted in inappropriate levels of minute alveolar ventilation over a range of compliance and resistance values expected in paralyzed patients ventilated for respiratory failure. The variable performance under changing load, along with the lack of alarms, should prompt caution in using the Vortran Automatic Resuscitator for emergency ventilatory support in situations where the patient cannot be constantly monitored by trained and experienced operators. Key words: disaster, ventilator, design of experiments, ventilator load, lung model, disaster preparedness, alveolar minute ventilation, continuous spontaneous ventilation. [Respir Care 2007;52(12):1718–1727. © 2007 Daedalus Enterprises]
and delivers a stable minute ventilation ($V_\dot{E}$) when com-
tilators available.1– 6 The challenge has been highlighted
influenza epidemic], hospitals are increasingly develop-
whether from terrorist actions or natural disaster [eg, avian
large numbers of victims who need mechanical ventilation
by the fact that current estimates suggest that the number
the Vortran Automatic Resuscitator model RTM (VAR),
study evaluated the bench performance of one such device,
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Fig. 1. Schematic of the Vortran Automatic Resuscitator model
RTM. $F_{I\text{O}_2}$ = fraction of inspired oxygen.

Introduction

In the context of disaster preparedness for managing
large numbers of victims who need mechanical ventilation
(whether from terrorist actions or natural disaster [eg, avian
influenza epidemic]), hospitals are increasingly develop-
strategies to make large numbers of mechanical ven-

The manufacturer states: “The VAR self-adjusts by in-
creasing respiratory rate and decreasing tidal volume ($V_T$)
and delivers a stable minute ventilation ($V_E$) when com-
pliance decreases.”8 However, even if $V_E$ remains con-
stant, a decreasing $V_T$ will decrease minute alveolar ven-
tilation and hence may adversely affect gas exchange. In a
previous study, we found that the VAR output is highly
unstable with changes in patient load.9 The present study
was designed specifically to test the hypothesis that in-
creasing load (ie, increasing resistance or decreasing com-
pliance) will have a significant effect on minute alveolar ven-
tilation. The device is powered by connecting it to a 50 psi
gas source (which will provide 40 L/min inspiratory flow
through the ventilator’s internal resistance) or to a medical
gas flow meter, which can provide 15– 40 L/min. Every
breath is patient-triggered, flow-limited, and patient-cy-
cled. The control variable is pressure, because the device
is designed to maintain a fairly constant inspiratory pres-
sure as the load (ie, due to respiratory system mechanics)
changes. However, the data indicate that the pressure con-
trol is not very precise. Inspiration is pressure-triggered
and pressure-cycled. Both of these phase variables (trigger
and cycle) are affected by the patient’s ventilatory muscle
activity and/or respiratory system mechanics. If the patient
makes an active inspiratory effort large enough to drop
airway pressure to or below the positive end-expiratory
pressure (PEEP), inspiration is pressure-triggered. If the
patient makes an active expiratory effort large enough to
drive airway pressure to the value set with the peak inspir-
atory pressure (PIP) dial (see Fig. 1), inspiration is
pressure-cycled. If there is no ventilatory muscle activity,
the VAR can be set to auto-trigger, in which case the
inspiratory and expiratory times are affected by the load
imposed by the mechanics of the respiratory system. For
example, during inspiration, airway pressure rises linearly
at a rate determined by the flow and the respiratory system
elastance, as described by the equation of motion for the
respiratory system (eg, as flow or elastance increases, the
pressure cycle threshold is reached more quickly and the
inspiratory time decreases). Once inspiration cycles off,
airway pressure decays exponentially, as determined by
the time constant of the total system (ie, the respiratory
system elastance, the airways resistance, and the resistance
provided by the rate dial). When the pressure decays to the
intrinsic PEEP (auto-PEEP) level, inspiration is triggered.
Thus, the patient either actively or passively controls both
the timing and size of every breath, which makes the breaths
spontaneous by definition.10 It follows logically that the
breathing pattern is pressure-controlled continuous spon-
taneous ventilation. Note that this particular example of
continuous spontaneous ventilation is different from other
forms of continuous spontaneous ventilation in that a
backup rate can be set even though the patient is para-
lyzed. However, the backup rate will increase as the load
increases, and vice versa.

The rate dial can be set so that the ventilator no longer
triggers automatically. According to the VAR’s user guide,
“Under these circumstances, the VAR is delivering pres-
sure-supported ventilatory support and the patient must
trigger the VAR to begin subsequent full inhalations.”11
However, the form of pressure-controlled continuous spon-
taneous ventilation provided by the VAR should not be
confused with the pressure-support mode common on most
ventilators, where each breath is flow-triggered or pres-
sure-triggered, pressure-limited, and flow-cycled.

Methods

Device Description

The VAR (Fig. 1) is a device that provides a single
mode: pressure-controlled continuous spontaneous venti-
With the adult VAR model we tested in the present study, PIP can be adjusted from 20 cm H$_2$O to 50 cm H$_2$O and frequency from 8 breaths/min to 20 breaths/min. The PEEP is intrinsic to the device; that is, it generates auto-PEEP, which ranges from 2 cm H$_2$O to 5 cm H$_2$O and, according to the manufacturer, is approximately one tenth of the set PIP.\textsuperscript{11}

**Equipment Calibration**

We evaluated the effect of load on the performance of 3 new VARs, using a lung model (Adult/Pediatric Lung Model, IngMar Medical, Pittsburgh, Pennsylvania). The lung model had flow and pressure sensors that output to data-acquisition-and-analysis software (Analysis Plus, Novametrix Medical Systems, Wallingford, Connecticut).

The flow sensor’s calibration was verified with a 3-L calibration syringe (Cybermedic, Pulmonary Data Service, Louisville, Colorado). Air was passed through the lung model, and the resulting volume (ie, integrated flow) data were compared to the syringe markings. Syringe volumes ranged from 100 mL to 800 mL (in increments of 100 mL). Error was defined as the difference between measured and true (syringe) values, expressed as a percent of the true value. The maximum error of the volume measurements was 6%.

Though the lung model has integral pressure sensors, we used an external digital manometer (Pneumogard, Novametrix Medical Systems, Wallingford, Connecticut), because resistances were added external to the lung model. The calibration of the Pneumogard manometer was verified with a water manometer (Dwyer Instruments, Michigan City, Indiana). The average error of the Pneumogard manometer was 1%.

The lung model consisted of 2 bellows connected by tubing and pneumatic switches. The switches allowed adjustment of series resistance for each lung. They also allowed the ventilator to be connected to either one lung or both lungs in parallel. The compliance of each lung is adjustable by engaging 0, 1, or 2 springs. The available range of compliance for this model, using different combinations of springs and bellows, was validated by ventilating it with a rectangular pressure waveform and recording pressure and volume data measured by the lung model’s sensors. The compliance of each combination of springs and bellows was calculated as the slope of the linear regression of volume versus pressure. The pressure-volume relationship was linear (minimum $r^2 = 0.99$). The actual compliances used in subsequent experiments are shown in Table 1.

The lung model has a minimum nominal airway resistance built in. The model resistance plus the resistance of an 8.0-mm inner-diameter endotracheal tube (ETT) attached to the model airway opening was the low value for resistive load in subsequent experiments. However, the simulator’s airway resistances are nonlinear, so we wished to avoid using them for the value of high resistive load. Instead, we built a linear resistor from specifications in United States patent number 4,691,187 (variable linear resistor). The linear resistor was constructed by compressing a standard 9.0-mm inner-diameter ETT between 2 blocks of wood secured together with one screw on each corner. The resistance of the device was adjusted by the degree of compression of the tube. The resistance of the device was evaluated as the slope of the linear regression of its flow-pressure curve. Pressure was measured with the water manometer and flow with a mass flow meter (model 4000, TSI, Shoreview, Minnesota). The linear resistor was adjusted to give a resistance of 35 cm H$_2$O/L/s over the flow range 15–45 L/min. The device was highly linear ($r^2 = 0.99$). Connecting this resistor to the ETT and the lung model (at the lowest resistance setting) represented the high resistive load used in subsequent experiments.

The total (dynamic) resistance of the low and high configurations was measured with a ventilator’s lung mechanics software (iVent201,VersaMed, Pearl River, New York). The ventilator was set at an inspiratory flow of 30 L/min (halfway between the flows of 20 L/min and 40 L/min used in subsequent experiments). The low resistance (8.0-mm inner-diameter ETT plus lung model resistance setting of one) was 20 cm H$_2$O/L/s. The high resistance (8.0-mm inner-diameter ETT plus linear resistor plus lung model resistance setting of one) was 42 cm H$_2$O/L/s. The values of low compliance and high compliance and low resistance are within the range found in patients ventilated for respiratory failure.\textsuperscript{12–14}

The high resistance represented a value that might be found in a patient with airway secretion problems or bronchial constriction.\textsuperscript{15}

The VAR was powered through a standard medical airflow meter (Timeter Instrument, St Louis, Missouri). At settings of 20 L/min and 40 L/min the flows (as measured by the mass flow meter) were 18.0 L/min and 37.9 L/min, respectively.

### Table 1. Compliance and Resistance Values and Number of Lungs and Springs Used in the Experiments

<table>
<thead>
<tr>
<th>Compliance (mL/cm H$_2$O)</th>
<th>A</th>
<th>B</th>
<th>C</th>
<th>D</th>
</tr>
</thead>
<tbody>
<tr>
<td>Lungs used in experiment (n)</td>
<td>1</td>
<td>2</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Springs engaged in experiment (each)</td>
<td>2</td>
<td>2</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Resistance (cm H$_2$O/L/s)</td>
<td>High 42</td>
<td>Low 20</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

ABCD
Experimental Procedures

This study was designed to test the hypothesis that increasing the load (ie, increasing resistance or decreasing compliance) will have a significant effect on minute alveolar ventilation. The scenario of increasing load with progressive lung disease is one that would be expected when using the VAR in a disaster situation. Therefore, we selected initial settings to be appropriate for an adult with normal lungs but not breathing spontaneously (ie, the lung model was used to simulate passive inspiration and expiration). Thereafter, the VAR settings were not changed, so that V˙E was a function of changes in lung model settings.

The experimental set-up is shown in Figure 2. Normal lung condition was simulated with the lowest resistance value and the highest compliance value (see Table 1). The gas supply to the VAR was set at either 20 L/min or 40 L/min, which represented the low and high limits of the range of operation. The gas supply was air from a compressor. The VAR’s green adapter (for delivering 100% oxygen) was in place. The PIP dial was set to the lowest indicated value (20 cm H2O). The VAR’s user guide suggests that the V T may be set by “observ(ing) the rise and fall of the chest.” The PIP of 20 cm H 2O produced a large excursion of the lung model bellows.

Measurements of PIP at the airway opening (ie, the connection between the VAR and the ETT) were made with the Pneumogard manometer. Frequency, V T, inspiratory time, expiratory time, and auto-PEEP measurements were made with the fixed-orifice pneumotachometer and analyzed with the data-acquisition-and-analysis software.

After measuring the initial values with the normal lung conditions, the load was changed, in random order, with various combinations of high and low resistance and compliances ranging from low to high (A through D, as shown in Table 2) for a total of 8 combinations (including the initial normal load).

Data Analyses

The mean and standard deviation was calculated for the data from the repeated measurements. The ratio of inspiratory time to expiratory time (T I/T tot) was calculated as:

\[
\frac{T_I}{T_{tot}} = \frac{T_I}{T_I + T_E} \times 100\% \quad (1)
\]

V˙E was calculated as the product of V T and frequency. Minute alveolar ventilation was estimated as:

\[
alveolar V_E = (V_T - V_D) \times f \quad (2)
\]

where alveolar V E is minute alveolar ventilation, V D is dead space volume, assumed to be 150 mL in a normal 68-kg adult, and f is ventilatory frequency.

The estimated P eCO2 resulting from the estimated minute alveolar ventilation was calculated as:

\[
P_{eCO2} = \frac{\dot{V}_{CO2} \times (P_B - P_{H2O})}{0.9286 \times \text{alveolar } V_T} \quad (3)
\]

where \(\dot{V}_{CO2}\) is the carbon dioxide production (assumed normal value 200 mL/min at standard temperature and pressure, dry), P B is the barometric pressure (assumed to be 760 mm Hg), P H2O is the partial pressure of water in alveolar gas (assumed to be 47 mm Hg), 0.9286 is the factor used to convert alveolar V E from atmospheric temperature and pressure dry to standard temperature and pressure dry (atmospheric pressure was assumed to be 760 mm Hg, and room temperature was assumed to be 21°C), and alveolar V E was assumed to be measured at atmospheric temperature and pressure dry.

The change in functional residual capacity (∆FRC) was calculated from the compliance of the model and the associated auto-PEEP:

\[
\Delta FRC = C \times \text{auto-PEEP} \quad (4)
\]

A subset of the experimental data (ie, outcome measures for conditions with high and low values for resistance, compliance, and source gas flow) was analyzed as a 2-level factorial design with standard “design of experiments” procedures. The specific steps were as follows:

1. Construct a table showing the standard template for organizing high and low values of the experimental vari-
ables and interaction effects (represented with plus symbols and minus symbols, respectively) and mean values of the responses. The pattern of pluses and minuses for interaction effects was calculated by multiplying the parent (main effect) terms. For example, in the first row, the interaction effect CR (ie, compliance and resistance) is + because C is – and R is – also, and the product of 2 negatives is a positive. To this table was added calculated values for main effects and interaction effects. Effects were calculated as:

\[
\text{Effect} = \frac{\sum Y^+}{n^+} - \frac{\sum Y^-}{n^-}
\]

(5)

where \(Y^+\) is the response values for high levels of the effect, \(Y^-\) is the response values for low levels of the effect, \(n^+\) is the number of responses for high levels of the main effect, and \(n^-\) is the number of response for low levels of the main effect. For example,

2. Sort the absolute values of the effects in ascending order.

3. Calculate the cumulative probabilities of the data from step 2. In this study design there were 3 main effects and 4 interaction effects, so the 0 to 100% probability scale was divided into 7 equal segments.

4. The absolute values of the effects are plotted on the horizontal axis versus the midpoints of the probability segments on the vertical axis, using a probability scale. The

<table>
<thead>
<tr>
<th>Table 2. Experimental Results*</th>
</tr>
</thead>
<tbody>
<tr>
<td>At Flow of 40 L/min</td>
</tr>
<tr>
<td>Set</td>
</tr>
<tr>
<td>C R</td>
</tr>
<tr>
<td>D L 1,134 (5)</td>
</tr>
<tr>
<td>C L 734 (5)</td>
</tr>
<tr>
<td>B L 456 (9)</td>
</tr>
<tr>
<td>D H 746 (10)</td>
</tr>
<tr>
<td>C H 448 (13)</td>
</tr>
<tr>
<td>B H 277 (15)</td>
</tr>
<tr>
<td>A L 198 (12)</td>
</tr>
<tr>
<td>A H 120 (33)</td>
</tr>
</tbody>
</table>

*Summary values for 3 measurement runs, sorted by minute alveolar ventilation. The values for compliance (C) and resistance (R) are shown in Table 1. The initial setting was frequency 10 breaths/min and PIP = 20 cm H_2O (according to the scale on the Vortran Automatic Resuscitator), shown in the first row of data. All subsequent settings (changes in C and R only) were in random order.

\(V_T\) = tidal volume
\(f\) = frequency
\(T_I\) = inspiratory time
\(T_E\) = expiratory time
\(PIP\) = peak inspiratory pressure
auto-PEEP = intrinsic positive end-expiratory pressure
\(T_I/T_{tot}\) = ratio of inspiratory time to total respiratory cycle time
\(V_{Ei}\) = minute ventilation
alv \(V_{EI}\) = minute alveolar ventilation (tidal volume minus assumed normal adult dead space [150 mL] times frequency)
\(P_{a\text{CO}_2}\) = estimated arterial carbon dioxide tension assuming normal carbon dioxide production (200 mL/min)

\(\Delta FRC\) = change in functional residual capacity associated with the PEEP (ie, trapped gas).
result is a normal plot of effects for each response. The purpose of the normal plot is to judge whether the data are normally distributed. Normally distributed data lie approximately on a straight line on this type of plot.

5. Data from 2-level factorial designs typically show a pattern of mostly linear points at low values of cumulative probability, with a few outliers. The data on the straight line are considered nonsignificant random variations, whereas the outliers are considered to be possibly from other populations (i.e., significantly different). The hypothesis that outliers represent significantly different mean values of the responses is tested with analysis of variance (ANOVA) as follows:

6. The outliers on the normal plot are labeled and included as effects in the ANOVA model.

7. The sum of squares (SS) for the effects is calculated as:

\[ SS = \frac{8}{4} (\text{effect})^2 \]  

8. The sum of squares for the model is the sum of the SS values for the effects in the model.

9. The sum of squares for the residual is the sum of the SS values for the remaining effects that were considered insignificant (i.e., the effects that fell approximately on the line in the normal plot).

10. The mean square (MS) is calculated as the ratio of the SS to the degrees of freedom (DF):

\[ MS = \frac{SS}{DF} \]  

Each effect is based on 2 averages, high versus low, so it has 1 degree of freedom. The model and the residual have as many degrees of freedom as the number of effects they contain.

11. The F statistic is calculated as the mean square for the model, or each effect divided by the mean square of the residual:

\[ F = \frac{MS}{MS_{\text{residual}}} \]  

12. The p values are derived from a table of F statistic values. Effects associated with p values ≤ 0.05 were considered significant.

Results

General Observations

The experimental data are shown in Table 2, sorted from highest to lowest \( V_T \). One general observation is that the output of the device was relatively unstable (i.e., highly variable \( V_T \) and frequency). The coefficient of variation in the measured and calculated values was relatively large, reaching a high of 33% for measured \( V_T \) and 79% for calculated \( P_{\text{aCO}} \). Instability was generally greater when the device was operated at 40 L/min, compared to 20 L/min. Even though the VAR is designed to cycle inspiration off at the preset pressure (which was unchanged during the experiments), the actual PIP range was 23.7–31.7 cm H\(_2\)O at 40 L/min. Again, the variation in PIP was less at 20 L/min than at 40 L/min. PIP increased as resistance increased or compliance decreased. Average auto-PEEP was 5 cm H\(_2\)O at 40 L/min and 6 cm H\(_2\)O at 20 L/min. These auto-PEEP levels corresponded to 18% of PIP at 40 L/min and 23% of PIP at 20 L/min.

\( V_T \) decreased as resistance increased or compliance decreased (Fig. 3). Also, at every combination of resistance and compliance, the \( V_T \) at 20 L/min was greater than at 40 L/min, due to a longer inspiratory time in each case (Fig. 4). In general, the \( V_T \) values were less than predicted from the product of set flow and measured inspiratory times. This was due to the error in the flow meter powering the VAR and also the error due to gas compressed in the delivery circuit and lung model between the VAR and the model flow sensor. Assuming the model lung represented a 68-kg adult, the measured \( V_T \) values ranged from a low of 1.7 mL/kg to a high of 16.7 mL/kg.

The \( T_I/T_{\text{tot}} \) in Table 2 (defined as inspiratory time divided by the sum of inspiratory time and expiratory time) was greatly affected by the input flow. At 40 L/min the average \( T_I/T_{\text{tot}} \) was 30%, and at 20 L/min \( T_I/T_{\text{tot}} \) was 52%, which represents an inverse inspiratory-expiratory ratio.

As the load increased, the \( V_T \) decreased and the frequency increased (Fig. 5). However, neither the \( V_E \) nor the minute alveolar ventilation stayed constant. \( V_E \) ranged from 5.2 L/min to 11.3 L/min at 40 L/min source flow. More importantly, minute alveolar ventilation ranged from zero to 9.8 L/min, resulting in calculated \( P_{\text{aCO}} \) values of over 100 mm Hg and 16 mm Hg, respectively. Indeed, calculated \( P_{\text{aCO}} \) was never in the normal range (35–45 mm Hg).

As described in the methods section, the initial setting of the VAR was for a normal patient, with the lowest PIP marked on the device (20 cm H\(_2\)O) and a low frequency (10 breaths/min). However, this setting resulted in an inappropriately large \( V_T \) and hyperventilation (i.e., \( P_{\text{aCO}} = 24 \) mm Hg at both source gas flows). This initial setting also resulted in a high auto-PEEP (7–9 cm H\(_2\)O), which produced a volume of trapped gas larger (633–823 mL) than the expected normal adult \( V_T \) (500 mL).

Statistical Analyses

Table 3 shows the data for the 2-level factorial “design of experiments” analysis. Figures 6, 7, and 8 show
the normal plots for absolute values of the effects from Table 3. Figure 6 suggests that $V_T$ is markedly affected by compliance ($C$) and to a lesser extent by resistance ($R$) and the interaction of compliance and resistance ($CR$). However, ANOVA results showed that only $C$ and $R$ had significant effects ($p < 0.001$ and $p < 0.05$, respectively).
Figure 7 shows that the frequency seems to be affected only by compliance. ANOVA confirmed this ($p < 0.001$).

The main outcome variable, minute alveolar ventilation, seems to be affected by compliance most strongly, followed by resistance, compliance-resistance interaction, and source gas flow (see Fig. 8). Table 4 shows the ANOVA results. As with $V_T$, only resistance and compliance had significant effects. Figure 9 shows the interaction plot for minute alveolar ventilation. Minute alveolar ventilation increases as compliance increases and/or resistance decreases. The fact that the lines are virtually parallel indicates that the effects of compliance are essentially independent of the effects of resistance.

**Discussion**

The literature that accompanies the VAR states: “The VAR automatically delivers a lower $V_T$ and a higher re-
spiratory rate and is ideal for patients with ARDS [acute respiratory distress syndrome] with decreasing compliance," and that the VAR "delivers a stable $V_E$ when compliance decreases from a healthy 0.07 L/cm H$_2$O to a stiff 0.02 L/cm H$_2$O." The data from our study indicate that $V_E$ is affected by both compliance and resistance, as well as source gas flow. Under conditions of changing load (from changes in lung mechanics), the $V_E$ varies widely. Of similar impact is the fact that minute alveolar ventilation under our experimental conditions was never in the normal range, as indicated by the calculated PaCO$_2$. Furthermore, the $T_I/T_{tot}$ may go above 50% at low source gas flow, which will increase mean airway pressure and may result in inadvertent hemodynamic consequences.

Our findings indicate that auto-PEEP was a higher percentage of PIP (ie, 18–23%) than indicated by the manufacturer (10%). High auto-PEEP may have adverse physiologic consequences.

Data from our study support data from other researchers who concluded that changes in lung conditions result in unpredictable changes in rate and $V_T$. Our simulated 68-kg patient would require $V_T$ in the range 270–410 mL (4–6 mL/kg), according to the ARDS Network guidelines. The $V_T$ values in our study were in that range 13% of the time (2 of 16 experimental conditions). Other studies have found that variation in VAR performance is also unpredictable with positional changes. With an apneic patient it may be difficult if not impossible to adjust PIP and rate to give an appropriate $V_E$ for a given set of lung mechanics, using only chest rise as a guide. For example, with a high (normal) compliance and a low resistance, a low-normal rate setting (10 breaths/min) resulted in substantial hyperventilation.

In one experimental condition (lowest compliance, highest resistance), one of the VAR devices we tested stopped triggering. When tapped, the VAR would trigger for a few breaths and then stop again.

A number of scenarios that would result in mass casualty respiratory failure describe patients with substantial...
pulmonary dysfunction. Blast lung injury, inhaled anthrax, plague, and avian flu can all end in the physiologic condition termed ARDS. The principles of ARDS management dictate accurate delivery of VT, control of the fraction of inspired oxygen and PEEP, and limiting the airway pressures. The VAR devices tested in the present study did not satisfy any of those criteria. Additionally, in a scenario that results in a surge of patients who require ventilation and a reduction in the ratio of caregivers to casualties, alarms will be essential. The VAR lacks alarms in the event of low VT, apnea, low pressure, or disconnect.

The major limitation of this study was that we simulated a passive patient. Presumably, a patient who is able to trigger the VAR could maintain a more consistent minute alveolar ventilation and hence gas exchange. Furthermore, we made no effort to increase the VT by adjusting PIP (and hence decreasing frequency) as the load increased. Therefore, our results should be generalized only to patients unable to trigger inspiration and unattended by trained operators.

Conclusions

The VAR showed an automatic increase in frequency and decrease in VT that resulted in inappropriate levels of minute alveolar ventilation over a range of compliance and resistance values expected in paralyzed patients ventilated for respiratory failure. The variable performance under changing load, along with the lack of alarms, prompts concern regarding the VAR for emergency ventilatory support in situations where patients cannot be constantly monitored by trained and experienced operators.

REFERENCES