Intermittent CPAP
A New Mode of Ventilation during General Anesthesia

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Background: Airway pressure–release ventilation provides ventilation comparable to controlled mechanical ventilation (CMV), but with lower peak airway pressures and less dead-space ventilation. To obtain these advantages for patients administered general anesthesia, the authors (1) designed a mode similar to airway pressure–release ventilation, intermittent continuous positive airway pressure (CPAP), and compared its efficiency with that of CMV, and (2) assessed the accuracy of end-tidal carbon dioxide tension (PETCO2) as a monitor of the partial pressure of carbon dioxide in arterial blood (Paco2) during CPAP compared with during CMV.

Methods: Twenty anesthetized, tracheally intubated patients received baseline CMV that produced a PetCO2 of approximately 35 mmHg and a pulse oximetry value > 90%. Patients were assigned to undergo alternating trials of CMV and CPAP. During CPAP, CMV was applied to the airway, removed for 1 s, and reapplied at a rate equal to the ventilator rate during CMV. The difference between the carbon dioxide tension in arterial blood and end-tidal gas [P(a–ET)CO2] and the calculation of Paco2/minute ventilation quantified the efficiency of ventilation. Data were summarized as mean ± SD and compared using the Student’s t test.

Results: Peak airway pressure (13 ± 2 vs. 23 ± 5 cm H2O; P < 0.001) and minute ventilation (3.5 ± 1 vs. 4.6 ± 1.2 L/min; P < 0.001) were lower during CPAP than during CMV. The value for Paco2/minute ventilation (11.1 ± 2.9 vs. 7.5 ± 2.6 mmHg L–1 min–1; P < 0.0001) was greater during CPAP. Paco2 was always greater during CMV (6.3 ± 1.6 vs. 1.7 ± 0.9 mmHg; P < 0.0001) and was never > 35 mmHg during CPAP.

Conclusions: During CPAP, less ventilation was necessary to produce a Paco2 comparable to that during CMV. This represents a significant reduction in dead-space ventilation, improved efficiency of ventilation, and a lower value for P(a–ET)CO2. Compared with CMV, CPAP also improves the accuracy of PETCO2 as a monitor of Paco2. (Key words: Airway pressure–release ventilation; end-tidal carbon dioxide; mechanical ventilation; monitoring.)

THE technical and physiologic aspects of mechanical ventilation during surgery have not changed for decades. For example, the use of positive-pressure ventilation during general anesthesia causes a maldistribution of inspired gas relative to pulmonary perfusion. As a result, alveolar dead space increases, and supplemental oxygen must be given to prevent hypoxemia resulting from areas of lung with a low ventilation-to-perfusion ratio. More often than not, relative overventilation causes hypoxic ventilatory responses. Ideally, mechanical ventilation instituted during general anesthesia would provide adequate alveolar ventilation with a minimal increase in airway pressure and a minimal displacement of the operative site. The traditional method of producing ventilation for general anesthesia, controlled mechanical ventilation (CMV), consists of intermittent delivery of a selected tidal volume (TV) with an attendant peak airway pressure of 15–30 cm H2O. Based on our experience with alternate ventilation modes in critically ill patients, we designed a means to produce ventilatory support for anesthetized patients.

Airway pressure–release ventilation (APRV) has been used successfully to ventilate anesthetized animals1–3 and unanesthetized humans.4–7 During APRV, continuous positive airway pressure (CPAP) is titrated to an optimal level. Airway pressure is then decreased intermittently. Gas exits the lungs and lung volume decreases, allowing excretion of carbon dioxide. After a selected release time, airway pressure and lung volume are rapidly reestablished. Spontaneous breathing is possible at any time.

Because most reports have indicated that APRV can augment ventilation with lower peak airway pressure...
and improved efficiency compared with traditional modes of positive pressure ventilation, we designed a similar ventilatory pattern for use in anesthetized patients, intermittent continuous positive airway pressure (CPAP). We had two goals: (1) to compare the efficiency of ventilation during CMV with that during CPAP in patients undergoing general anesthesia and intraabdominal operations and (2) to test the hypothesis that the partial pressure of end-tidal carbon dioxide (P\textsubscript{ETCO\textsubscript{2}}) is a more accurate reflection of the partial pressure of arterial carbon dioxide (P\textsubscript{aCO\textsubscript{2}}) during CPAP than during CMV.

**Materials and Methods**

We studied patients classified as physical status 1 and 2 of the American Society of Anesthesiologists who were scheduled for general anesthesia, intraabdominal operation, and insertion of an intraarterial catheter to monitor blood pressure. All patients signed a consent form approved by the Institutional Review Board. Excluded were patients who had unstable cardiovascular function or severe obstructive lung disease. Chest leads II and V5 were attached for electrocardiographic monitoring, and heart rate was determined electronically. A probe placed on a finger tip allowed oxygen saturation to be measured by pulse oximetry.

Anesthesia and neuromuscular blockade were induced with propofol (1 or 2 mg/kg administered intravenously), or thiopental (2–5 mg/kg administered intravenously) and succinylcholine (1.5 mg/kg administered intravenously), respectively. Anesthesia and neuromuscular blockade were maintained with isoflurane, nitrous oxide, and oxygen or with vecuronium, respectively. All patients were orotracheally intubated. Patients initially were ventilated with CMV using a V\textsubscript{T} of 8–10 ml/kg and a respiratory rate sufficient to keep P\textsubscript{ETCO\textsubscript{2}} between 30 and 35 mmHg. The fractional concentration of inspired oxygen (F\textsubscript{I,O\textsubscript{2}}) was adjusted to keep the S\textsubscript{PO\textsubscript{2}} at 90% or higher. A thermistor placed in the esophagus monitored temperature. A catheter inserted into the radial artery permitted determination of blood pressure, pH\textsubscript{a}, P\textsubscript{aCO\textsubscript{2}}, P\textsubscript{aO\textsubscript{2}}, hemoglobin concentration, and oxyhemoglobin saturation. A flow transducer (VarFlex; Allied Healthcare Products, Irvine, CA) attached to the tracheal tube was connected to a pulmonary mechanics computer (BICORE CP-100; Allied Healthcare Products) to determine V\textsubscript{T}, respiratory rate, minute ventilation (V\textsubscript{E}), peak inspiratory gas flow rate, and peak and mean airway pressures. The pulmonary mechanics monitor is factory calibrated with room air (oral communication, Larry Butcher, Allied Healthcare Engineering Department, 1996), so that volumes calculated during general anesthesia necessitated correction for nitrous oxide and isoflurane. Using a calibrated 1-l super syringe (Hamilton Medical Corp., Reno, NV), identical volumes of room air and a gas mixture identical to that breathed by the patient were injected through the pneumotachograph. The resulting ratio (volume air:volume anesthetic gas mixture) was used to correct the respired gas volumes obtained from the computer during the investigation. The sample tubing of a gas and anesthetic vapor monitor (Ultima; Datex-Engström, Helsinki, Finland) was positioned between the pneumotachograph and the anesthesia breathing circuit to determine the fractional concentration of inspired oxygen, P\textsubscript{ETCO\textsubscript{2}}, and the end-tidal concentrations of isoflurane and nitrous oxide. Baseline data were collected after the abdominal incision was made, and heart rate, mean arterial blood pressure, and end-expired concentration of isoflurane remained unchanged for 30 min. We assumed relatively constant P\textsubscript{aCO\textsubscript{2}} and carbon dioxide production, and we quantified the efficiency of ventilation by calculating P\textsubscript{aCO\textsubscript{2}}/V\textsubscript{E} (the normal value is approximately 7.4 mmHg·1·min\textsuperscript{-1} for awake humans).

Patients were assigned randomly to receive alternating 20-min trials of CMV (using the same characteristics as baseline) and CPAP. To provide CPAP, we used a modified ventilator (model Mark 4A; Bird Corp., Palm Springs, CA). Alveolar ventilation is produced by intermittent release of CPAP nearly to atmospheric pressure (fig. 1). After a release period of 1 s, CPAP is restored and lung volume is reestablished. The respiratory rate during CPAP was the same as during baseline CMV. However, because previous investigations revealed that dead-space ventilation was lower during APRV than during CMV, during CPAP the desired tidal volume was obtained by titration of CPAP to produce a P\textsubscript{ETCO\textsubscript{2}} that was 2 or 3 mmHg more than the value observed during baseline CMV; this was performed to try to produce equivalent alveolar ventilation and P\textsubscript{aCO\textsubscript{2}}.

Data are summarized as mean ± SD. To assess the possibility of a carry over of treatment effect (treatment-period interaction), we compared the differences (mean ± SD) for the two treatment sequences. To compare the differences between the two treatment sequences, we applied the Student’s t test for independent observations. There was no significant treatment-period interaction, therefore, data were compared sta.

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tistically using the Student’s t test for paired observations (two tailed). We used regression analysis to assess the mathematical relation between PetCO₂ and PaCO₂. We tested the null hypothesis that the two regressions were coincident (i.e., they have the same slope and intercept) according to the technique described by Glantz. We also quantified the strength of the association between PetCO₂ and PaCO₂ by calculating Pearson’s product-moment correlation (r). We tested the null hypothesis that the two correlations were the same according to a technique described by Zar.

**Results**

Twenty patients (11 women, 9 men) who were 62 ± 15 yr old and who weighed 88 ± 26 kg underwent similar anesthesia care and operative procedures. The end-tidal concentration of isoflurane (1.1 ± 0.3%), body temperature (35.7 ± 0.5°C), and hemoglobin concentration (10.8 ± 1.5 g/dl) were similar throughout the study, and intertrial data were pooled for summary. There were no differences in variables reflecting cardiovascular function throughout the study (table 1).

Peak airway pressure was less during CPAP than during CMV (table 2). During CPAP, peak airway pressure did not exceed 18 cm H₂O in any patient, and in six patients, it was less than one half that observed during CMV. Although mean airway pressure was higher during CPAP, no adverse cardiovascular consequences were apparent. During CMV, peak inspiratory gas flow rate was 0.7 ± 0.2 l/min. After interruption of CPAP during CPAP, a peak flow of 1.3 ± 0.4 l/min restored the CPAP level. The respiratory rate was similar by design, but during CPAP, comparable PaCO₂ was achieved with less tidal volume and minute ventilation. Thus CPAP improved the efficiency of ventilation, as quantified by a higher value for PaCO₂/V̇E (table 3).

There were no differences in the fractional concentration of inspired oxygen, and arterial blood gas tensions, pH, and oxyhemoglobin saturation did not change during the study. The correlation coefficient between

**Table 1. Summary of Data Reflecting Cardiovascular Function during Intermittent CPAP (CPAP) and Controlled Mechanical Ventilation (CMV) in 20 Patients Undergoing General Anesthesia**

<table>
<thead>
<tr>
<th>Trial</th>
<th>HR (min⁻¹)</th>
<th>SAP (mmHg)</th>
<th>DAP (mmHg)</th>
<th>MAP (mmHg)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPAP</td>
<td>70 ± 12</td>
<td>120 ± 23</td>
<td>70 ± 12</td>
<td>83 ± 18</td>
</tr>
<tr>
<td>CMV</td>
<td>72 ± 11</td>
<td>123 ± 23</td>
<td>64 ± 14</td>
<td>85 ± 17</td>
</tr>
</tbody>
</table>

Data are mean ± SD, and intertrial comparisons were performed using Student’s t test for paired observations.

HR = heart rate; SAP = systolic arterial pressure; DAP = diastolic arterial pressure; MAP = mean arterial pressure.
INTERMITTENT CPAP DURING ANESTHESIA

Table 2. Summary of Data Reflecting Lung Mechanics during Intermittent CPAP (CPAPi) and Controlled Mechanical Ventilation (CMV) in 20 Patients Undergoing General Anesthesia

<table>
<thead>
<tr>
<th>Trial</th>
<th>Peak Paw (cmH₂O)</th>
<th>Mean Paw (cmH₂O)</th>
<th>Vₜ (mL)</th>
<th>RR (min⁻¹)</th>
<th>Vₑ (L/min)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPAPi</td>
<td>13 ± 2*</td>
<td>11 ± 3†</td>
<td>536 ± 148*</td>
<td>7 ± 1</td>
<td>3.5 ± 1.0*</td>
</tr>
<tr>
<td>CMV</td>
<td>23 ± 5</td>
<td>8 ± 2</td>
<td>624 ± 162</td>
<td>7 ± 1</td>
<td>4.6 ± 1.2</td>
</tr>
</tbody>
</table>

Data are mean ± SD, and intertrial comparisons were performed with Student’s t test for paired observations.

Peak Paw = peak airway pressure; Mean Paw = mean airway pressure; Vₜ = tidal volume; RR = respiratory rate; Vₑ = minute ventilation.

* P < 0.007 versus CMV.
† P < 0.01 versus CMV.

PₐCO₂ and PₑTₐCO₂ was closer to unity for CPAPi (r = 0.95) than for CMV (r = 0.80; P < 0.10; fig. 2). There was a significant difference in the lines of least squares resulting from the regression of PₑTₐCO₂ on PₐCO₂ during CMV versus CPAPi (P < 0.001). The slope of the regression (β) of PₑTₐCO₂ on PₐCO₂ was closer to 1 during CPAPi (β = 0.90) than during CMV (β = 0.62). The y-axis intercept (α) was 2.45 during CPAPi and 9.07 during CMV. The arterial-to-end-tidal carbon dioxide partial pressure difference [P(a−ET)CO₂] was always less during CPAPi (1.7 ± 0.9 mmHg) than during CMV (6.3 ± 1.6 mmHg; P < 0.0001) and was never more than 3.5 mmHg during CPAPi. During CMV, P(a−ET)CO₂ ranged from 4 to 10 mmHg.

Discussion

We designed and tried to evaluate CPAPi as a ventilatory support technique for patients undergoing general anesthesia and neuromuscular blockade. We observed that the minute ventilation necessary to achieve similar alveolar ventilation, as reflected by PₐCO₂, was less when patients were ventilated with CPAPi versus CMV. Normal awake Vₜ is approximately 6 mL/kg. Our results indicate that such a Vₜ provided by CPAPi at a rate of 7/min will produce a normal PₐCO₂. Because anatomic dead space was presumed to be nearly constant, the ability to achieve comparable PₐCO₂, with a lower P(a−ET)CO₂ and a lesser minute ventilation was evidence of less alveolar dead-space ventilation during CPAPi.

The reduction in alveolar dead space ventilation rendered PₑTₐCO₂ a more accurate reflection of PₐCO₂ during CPAPi than during CMV. The observation that deadspace ventilation is lower also has been commonly noted during APRV. Although mean airway pressure was higher during our trials of CPAPi, no adverse cardiovascular consequences seemed to occur.

The following equation provides an estimate of the amount of change (Δ) in functional residual capacity (FRC) effected by CPAP:

\[ ΔFRC = CPAPi \times CLT \]

where CLT is lung–thorax compliance. Periodic release of CPAP causes lung volume to decrease, and restoration of CPAP causes lung volume to increase, thus

Table 3. Summary of Data Reflecting Gas Exchange during Intermittent CPAP (CPAPi) and Controlled Mechanical Ventilation (CMV) with an FₐCO₂ = 0.33 ± 0.08 in 20 Patients Undergoing General Anesthesia

<table>
<thead>
<tr>
<th>Trial</th>
<th>pHa</th>
<th>PₐCO₂ (mmHg)</th>
<th>PₐO₂ (mmHg)</th>
<th>SₐO₂ (%)</th>
<th>P(a−ET)CO₂ (mmHg)</th>
<th>PₐCO₂/Vₑ (mmHg/L·min⁻¹)</th>
</tr>
</thead>
<tbody>
<tr>
<td>CPAPi</td>
<td>7.40 ± 0.04</td>
<td>38.6 ± 3.0</td>
<td>110 ± 42</td>
<td>95.6 ± 3.7</td>
<td>1.7 ± 0.9*</td>
<td>11.1 ± 2.9*</td>
</tr>
<tr>
<td>CMV</td>
<td>7.42 ± 0.04</td>
<td>37.0 ± 2.2</td>
<td>117 ± 40</td>
<td>96.1 ± 3.0</td>
<td>6.3 ± 1.6</td>
<td>7.9 ± 2.6</td>
</tr>
</tbody>
</table>

Data are mean ± SD, and intertrial comparisons were performed with Student’s t test for paired observations.

pHₐ = arterial blood pH; PₐO₂ = partial pressure of oxygen in arterial blood; SₐO₂ = arterial blood oxyhemoglobin saturation; P(a−ET)CO₂ = partial pressure of carbon dioxide in arterial blood minus end-tidal gas; PₐCO₂/Vₑ = ratio of PₐCO₂ and minute ventilation.

* P < 0.0001 versus CMV.

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Positive-pressure inflation of the lungs can cause adverse cardiovascular effects. We found no differences in cardiovascular function during CPAPi and CMV, which probably was caused by the low mean airway pressure observed with both modes. Previous investigations have reported adverse cardiovascular sequelae that occurred with intermittent positive-pressure ventilation, but not with APRV. Because APRV provides mechanical ventilation by decreasing airway pressure from a level of CPAP titrated to optimize lung mechanics, peak airway pressure never exceeds CPAP, and mean airway pressure is less than the CPAP level. Therefore, the lack of adverse pressure-related effects is not surprising.

The value for $P(a-Fi)CO_2$ during CPAPi was similar to that observed in spontaneously breathing patients. During spontaneous breathing, inspired gas is distributed predominately to relatively well-perfused alveoli in dependent lung regions, and the end-expired gas closely approximates alveolar gas. However, in anesthetized, paralyzed, and mechanically ventilated patients, the inspired gas is distributed preferentially to poorly perfused or nonperfused alveoli in nondependent lung units, and the end-expired gas contains a significant contribution from alveolar dead space. During normal spontaneous breathing, $P(a-Fi)CO_2$ may range from 1 to 3 mmHg. During CMV, $P(a-Fi)CO_2$ may exceed 12 mmHg and is rarely $< 6$ mmHg. When mechanical inspiration occurs from a lung volume less than normal FRC, the maldistribution of inspired air relative to perfusion is exaggerated. Thus, alveolar dead-space ventilation is greater during CMV, particularly when FRC decreases, as occurs immediately after induction of general anesthesia.

Valentine et al. observed a distribution of less ventilation to nonperfused alveoli (dead space) with similar tidal volume and frequency during APRV than during intermittent mandatory ventilation and pressure support ventilation. Although we did not quantify dead space, the improved efficiency of ventilation during CPAPi versus CMV, as evidenced by a higher value for $P_{aCO_2}/V_{E}$, indicates that alveolar dead-space ventilation was less during CPAPi. As noted in figure 2, the slope and intercept of the least-squares regression lines for $P_{TECO_2}$ versus $P_{aCO_2}$ are different for CMV and CPAPi. Furthermore, the slope of the least-squares regression line for $P_{TECO_2}$ versus $P_{aCO_2}$ obtained during CPAPi was 0.90 and the y-axis intercept was 2.43 mmHg, indicating that $P_{TECO_2}$ closely reflects $P_{aCO_2}$ regardless of the $P_{aCO_2}$ value. In contrast, with higher values of $P_{aCO_2}$

Fig. 2. The relation between end-tidal carbon dioxide tension ($P_{TECO_2}$) versus the partial pressure of arterial carbon dioxide ($P_{aCO_2}$) in 20 patients who received alternate trials of intermittent positive pressure ventilation (CPAP; solid circles) and controlled mechanical ventilation (open circles). The line of identity (dotted) and a mathematically determined line of best fit (solid) associated with each data cloud were plotted. Regression equations for the line of best fit during CPAP and CMV were $P_{TECO_2} = 2.4\, mmHg + 0.9\times P_{aCO_2}$ and $P_{TECO_2} = 9.1\, mmHg + 0.6\times P_{aCO_2}$, respectively, $P < 0.001$ using a technique described by Zar.

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providing alveolar ventilation and excretion of carbon dioxide.
observed during CMV, P(a−ET)O₂ increased, secondary to increased alveolar dead space, rendering PETCO₂ a progressively less accurate monitor of PaCO₂. Presumably, lower peak airway pressure during CPAP caused less alveolar dead space in nondependent lung regions.

Intermittent CPAP, pressure-controlled, inverse ratio ventilation; and APRV share several common characteristics, but conceptually they are quite different. Pressure-controlled, inverse ratio ventilation is applied to critically ill patients with severe respiratory dysfunction, usually with an elevated end-expiratory pressure. Spontaneous breathing is not possible and extreme sedation, muscle relaxation, or both, usually are administered. A pressure limit is selected to produce acceptable V̇t and peak positive airway pressures. As the patient’s condition improves, pressure-controlled, inverse ratio ventilation gradually is discontinued by decreasing inspiratory time and mean positive airway pressure.

Airway pressure-release ventilation is applied to spontaneously breathing patients with a restrictive ventilatory defect to optimize FRC with CPAP. If necessary, the CPAP level is decreased briefly (usually 1 s or less) to allow a decrease in lung volume and carbon dioxide excretion. As the patient’s condition improves, and he or she is more able to breathe, the rate of release is decreased and mean airway pressure increases.

During general anesthesia, CPAP is applied to a level that will produce a desirable V̇t when removed. Then the CPAP is reduced intermittently to produce ventilation; there is no intent to “restore” normal FRC, to optimize lung mechanics, or to improve oxygenation. Then, CPAP can be discontinued at the end of the anesthetic. Although the airway pressure patterns may be similar, pressure-controlled, inverse ratio ventilation; APRV; and CPAP are distinctly different in concept, application, and mode of discontinuation.

Because our patients received continuous neuromuscular blockade, CMV and CPAP both provided total ventilatory support. Existing methods do not permit application of partial ventilatory support techniques, such as intermittent mandatory ventilation or APRV, during anesthesia. However, the use of CPAP to provide partial mechanical support of spontaneously breathing patients who cannot maintain an acceptable PaCO₂ during general anesthesia may have several advantages over the use of CMV: (1) lower mean intrathoracic (pleural) pressure; (2) augmented venous return and improved cardiovascular performance; and (3) better distribution of inspired gas, resulting in improved matching of ventilation and perfusion. All of these advantages occur when APRV is used to provide partial ventilatory support in awake humans. The role for CPAP in partial ventilatory support during general anesthesia should be evaluated.

Our results indicate that CPAP provides more efficient ventilation of patients undergoing general anesthesia, with a significantly lower peak airway pressure compared with CMV. An improved efficiency of ventilation decreases the necessary minute ventilation and permits reduction of V̇t, respiratory rate, or both, thus reducing the magnitude or frequency, respectively, of lung inflation. Thus, there is less respiratory movement and possible improvement in technical conditions during intraabdominal operations. During CPAP, P(a−ET)O₂ approximates the value observed during spontaneous breathing, rendering PETCO₂ a more accurate monitor of ventilation during CPAP than during CMV.

Intermittent CPAP is a unique way to provide ventilatory support to anesthetized patients. It may have significant advantages with regard to gas exchange, efficiency of ventilation, and hemodynamic stability. Future investigations will determine the usefulness of CPAP in clinical practice.

References

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