Constant Positive-pressure Breathing and Cardiorespiratory Function

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The cardiopulmonary effects of intermittent (IPPB) and constant positive-pressure breathing (CPPB), with and without expiratory resistance, were studied in ten healthy anesthetized dogs. Tidal volume, flow rate and changes in functional residual capacity (FRC) were measured by capacitance respirometry, a new remote monitoring technique which measures intrapulmonary volume changes as a function of changes in surface area. CPPB with and without expiratory resistance significantly reduced intrapulmonary shunting and increased FRC, but produced no change in A-aDO₂. Reductions in cardiac output which occurred with CPPB were directly related to mean airway pressure. CPPB without expiratory resistance but with an end-expiratory plateau of positive pressure minimized increases in mean airway pressure and seemed to be a safer and better method of CPPB. (Key words: Constant positive-pressure breathing; Expiratory resistance; Capacitance respirometry.)

LAboratory and clinical evidence indicates that constant positive-pressure breathing (CPPB) may raise the arterial oxygen tension to levels higher than those reached during intermittent positive-pressure breathing (IPPB).¹⁻⁶ Two methods currently used to achieve constant positive-pressure breathing are: 1) airflow resistance during expiration adjusted to produce an end-expiratory positive pressure; 2) positive-pressure breathing without expiratory resistance but with an end-expiratory plateau of positive pressure. The present study evaluates

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the cardiovascular responses of ten dogs to periods of spontaneous respiration (SR), IPPB, and the two methods of CPPB.

Methods

Anesthesia was induced with thiopental (25 mg/kg) given intravenously and, following tracheal intubation, maintained with 1 percent inspired halothane. Cannulae were placed in the femoral artery and in the right ventricle via the external jugular vein. At least 30 minutes following induction and during spontaneous respiration, control measurements of functional residual capacity (FRC) were made using the closed-circuit helium-dilution technique, and cardiac output (CO) was measured by the dye-dilution technique, employing a Beckman cardiodensitometer. Intrapulmonary shunting was determined during inhalation of air (total shunt) and oxygen (true shunt) by analysis of end-expired air, arterial blood, and mixed venous blood, employing the formula (Qo/Qc = Cc - Ca/Cc - Cr). Oxygen content values were calculated using the Severinghaus blood gas calculator.⁶

Airway pressure was recorded using a Sanborn 266B transducer. Mean airway pressure was determined by planimetric integration of the pressure curves. Simultaneous recordings of airflow, tidal volume and minute volume were made, employing capacitance respirometry and integrating preamplifiers.

The capacitance respirometer measured intrapulmonary volume changes as a function of changes in surface area.⁵ By incorporating the anesthetized dog in a capacitor bridge circuit (fig. 1), surface area changes of the thoracic cage and abdomen were measured during respiration. The capacitance respirometer is analogous to a two-plate capacitor, with air as the dielectric material. The dog served as the variable plate, and a wire mesh screen placed
anterior to the supine dog served as the constant plate. As the surface area of the breathing animal varied in respect to the constant plate, a change in capacitance resulted. This capacitance change, measured by a sensitive capacitor bridge circuit (Fig. 1), was recorded as a change in voltage. Tracings of the changes in voltage comprised the “capacitance spirogram.” Simultaneous spiromgrams from the capacitance respirometer and a Wedge respirometer were recorded during spontaneous respiration with each dog to calibrate the capacitance measurements (Fig. 2). The capacitance respirometer was also checked with a Wright spirometer and by introducing gas volumes of 100–400 ml through the endotracheal tube with a one-liter calibrated syringe. Volume corrections for BTPS during spontaneous breathing and an additional volume correction for pressure were necessary during positive-pressure breathing. The calibrated capacitance volumes were found to be nearly linear over a range of several tidal volumes.

Measurements of cardiac output and shunting during inhalation of air and oxygen were repeated following 15 minutes of: intermittent positive-pressure breathing (IPPB); constant positive-pressure breathing with end-expired pressures of 5 and 10 cm H2O (CPPB-5, CPPB-10); CPPB-5 with expiratory resistance (CPPB-ER5).

Spontaneous respiration and various types of controlled respiration were possible with the circuit illustrated in figure 3. During IPPB, inspiration was accomplished by ventilator closure of the mushroom valve and direction of a preset volume of gas to the subject. Expiration occurred through the expiratory resistance valve, adjusted to the full-open position. CPPB-ER5 was accomplished by adjusting the expiratory resistance valve so that a pressure of 5 cm H2O existed at the end of expiration (Fig. 4). CPPB-5 and CPPB-10 were achieved by turning the three-way valve to deliver expired air to the spirometer. Weights were added to the spirometer bell to give either 5 or 10 cm H2O of sustained positive pressure following expiration (Fig. 5). A notched metallic skirt was added to the spirometer rim so that expired air was disseminated smoothly, minimizing artifacts in the pressure tracings.

Changes in FRC induced by positive-pressure respiration were determined from changes in the tidal volume baseline as recorded by the capacitance respirometer (Figs. 4 and 5). Respiratory rate and tidal volume were maintained nearly constant during all patterns of ventilation. The pressure pattern sequence...
was randomized and included a second control period in which measurements were made during spontaneous respiration.

Results

No significant changes in cardiac output, FRC or true shunting occurred following the change from spontaneous respiration to IPPB during inhalation of oxygen (table 1). With CPPB-5, the cardiac index fell significantly ($P < 0.001$) and FRC increased 18 per cent. Increasing the end-expired pressure to 10 cm H$_2$O raised mean FRC to 38 per cent above control levels and further reduced the cardiac index ($P < 0.05$). Cardiac index during CPPB-ER5 was nearly the same as that with CPPB-10 (fig. 6).

Although the end-expired pressures with CPPB-5 and CPPB-ER5 were similar, the cardiac index was significantly ($P < 0.05$) lower when expiratory resistance was used to achieve an end-expired pressure of 5 cm H$_2$O. Because of the markedly reduced airflow associated with expiratory resistance (fig. 4), deflation of the lung was prolonged, causing an increase in mean airway pressure and reducing cardiac output.

The mean total shunt during spontaneous breathing of air was significantly reduced during CPPB-5 ($P < 0.05$), yet no further decrease was effected by changing to CPPB-10 or CPPB-ER5 (fig. 7). The true shunt measured during oxygen inhalation, however, de-

![Simultaneous wedge and capacitance spiromograms for calibration of capacitance respirometer.](Fig. 2.

![Schematic of breathing circuit for producing constant positive-pressure breathing with and without expiratory resistance.](Fig. 3.

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creased significantly with progressive increases in mean airway pressure, falling from 14.5 per cent during spontaneous respiration to a low of 8.8 per cent during CPPB-10 ($P < 0.01$).

In spite of marked changes in cardiac output, FRC, and shunting induced by various degrees of positive-pressure breathing, no significant changes in A-a$\Delta$O$_2$ occurred (table 1). Pulmonary ($C_\text{O}_2 - C_\text{O}_2$) and systemic ($C_\text{O}_2 - C_\text{O}_2$) oxygen content differences during spontaneous and positive-pressure breathing are represented in figure 8. Pulmonary oxygen content differences, like the A-a$\Delta$O$_2$, were unchanged during the various forms of positive-pressure respiration. The systemic oxygen content differences, however, rose or fell in direct accord with changes in mean airway pressure.

**Discussion**

**Positive-Pressure Breathing and V/Q**

Bergman had demonstrated that the distributions of inspired gas in healthy humans during artificial and spontaneous respiration were...
Fig. 5. Tidal volume, airway pressure and airflow changes with constant end-expiratory positive-pressure breathing (5 cm H₂O).

the same and were not affected by varying respiratory waveforms during artificial ventilation.⁵,¹⁰ Similarly, it was shown in dogs that the distribution of ventilation was not altered by atelectasis.¹¹ In the present study, positive-pressure respiration resulted in significant reductions in both total and true shunt. The ratio of true to total shunt increased with IPPB but then decreased significantly with higher mean airway pressures, suggesting a reduction in atelectasis or vascular shunting rather than a more equitable distribution of ventilation relative to perfusion.

Positive-Pressure Breathing and Cardiac Output

It has long been known that positive-pressure breathing, by increasing the mean intrathoracic pressure, affects the filling pressure of the right heart, causing a reduction in cardiac output.¹² Constant positive-pressure ventilation achieved by imposing resistance to expiratory airflow (CPPB-ER3) produced the greatest depression of cardiac output in our study, and appears to be the least desirable method of achieving reductions in intrapulmonary shunting. An increase in rate or tidal volume in the presence of expiratory resist-
Table 1. Cardiorespiratory Effects of Intermittent (IPPB) and Constant Positive-Pressure Breathing, with (CPPB-ERS) and without (CPPB-5, CPPB-10) Expiratory Resistance

<table>
<thead>
<tr>
<th></th>
<th>Spontaneous</th>
<th>IPPB</th>
<th>CPPB-5</th>
<th>CPPB-10</th>
<th>CPPB-ERS</th>
<th>Spontaneous</th>
</tr>
</thead>
<tbody>
<tr>
<td>$V_i$ (ml)</td>
<td>273 ± 21</td>
<td>250 ± 28</td>
<td>284 ± 21</td>
<td>284 ± 30</td>
<td>273 ± 27</td>
<td>330 ± 35</td>
</tr>
<tr>
<td>$f$ (min)</td>
<td>15 ± 1.0</td>
<td>13 ± 0.8</td>
<td>13 ± 0.7</td>
<td>13 ± 0.5</td>
<td>13 ± 0.8</td>
<td>12 ± 1.1</td>
</tr>
<tr>
<td>Peak airway pressure (cm H$_2$O)</td>
<td>—</td>
<td>11.5 ± 1.03</td>
<td>16.5 ± 1.23</td>
<td>20.4 ± 1.26</td>
<td>17.1 ± 0.87</td>
<td>—</td>
</tr>
<tr>
<td>Mean airway pressure (cm H$_2$O)</td>
<td>—</td>
<td>3.2 ± 0.35</td>
<td>8.0 ± 0.28</td>
<td>12.9 ± 0.73</td>
<td>10.5 ± 0.42</td>
<td>—</td>
</tr>
<tr>
<td>∆ FRC (Per Cent)</td>
<td>—</td>
<td>15 ± 1.5</td>
<td>15 ± 3.9</td>
<td>15 ± 2.3</td>
<td>15 ± 2.7</td>
<td>—</td>
</tr>
<tr>
<td>A-aDO$_2$ (torr)</td>
<td>169 ± 15</td>
<td>172 ± 15</td>
<td>164 ± 11</td>
<td>150 ± 16</td>
<td>150 ± 20</td>
<td>141 ± 13</td>
</tr>
<tr>
<td>$Q_e/Q_r \times 100$</td>
<td>14.7 ± 0.5</td>
<td>14.1 ± 1.8</td>
<td>12.1 ± 0.5</td>
<td>12.5 ± 2.4</td>
<td>12.4 ± 1.6</td>
<td>12.3 ± 1.1</td>
</tr>
<tr>
<td>$Q_a/Q_r \times 100$</td>
<td>14.5 ± 0.9</td>
<td>13.2 ± 1.2</td>
<td>11.5 ± 1.2</td>
<td>8.8 ± 1.1</td>
<td>9.0 ± 1.4</td>
<td>12.3 ± 1.1</td>
</tr>
<tr>
<td>True shunt/total shunt (O$_2$)</td>
<td>0.78</td>
<td>0.94</td>
<td>0.85</td>
<td>0.70</td>
<td>0.73</td>
<td>0.68</td>
</tr>
<tr>
<td>Cardiac index (l/min/m$^2$)</td>
<td>4.18 ± 0.25</td>
<td>3.81 ± 0.26</td>
<td>3.09 ± 0.25</td>
<td>2.63 ± 0.18</td>
<td>2.32 ± 0.33</td>
<td>4.11 ± 0.31</td>
</tr>
</tbody>
</table>

Fig. 6. Effects of spontaneous and positive-pressure breathing on cardiac index.

Fig. 7. Intrapulmonary shunting during spontaneous and positive-pressure breathing.

Fig. 8. Pulmonary and systemic oxygen content gradients during spontaneous and positive-pressure breathing.

ance could also cause escalations of both mean and end-expiratory pressures, with the likelihood of overdistention of the lungs. Expiratory resistance valves have been used clinically in the treatment of respiratory problems, and controls capable of producing expiratory resistance have appeared on postoperative ventilators. Because of the potential for considerable depression of cardiac output which expiratory resistance may cause, use of these valves for all patients should be accompanied by careful monitoring of cardiac output.

Since mean airway pressure is inversely related to cardiac output, the waveform selected for constant positive-pressure ventilation, ideally, should be that which accomplishes tidal exchange and increases FRC.
while minimizing mean airway pressure. The present study suggests that these goals are achieved with waveforms featuring passive expiration, unimpeded expiratory airflow, and a sustained plateau of positive pressure. There is evidence that end-expiratory pressures as low as 0.5–1 cm H2O are sufficient to stabilize lung function and shunting during prolonged constant-volume respiration. A plateau pressure of 5 cm H2O, however, can evoke significant reductions in shunt, and has proven effective in seriously ill patients.14–16

MONITORING DURING POSITIVE-PRESSURE BREATHING

Caution must be exercised in interpreting changes or lack of changes in A–aDO2 during positive-pressure breathing should mean airway pressure be altered. We found, as Bergman found in humans, that varying the respiratory waveform and mean airway pressures had no significant effect on A–aDO2. In our dogs, however, shunting was reduced. Reductions in cardiac output related to increase in mean airway pressure masked the favorable effect of positive-pressure breathing on shunting, and accounted for the lack of change in A–aDO2. Similar reductions in cardiac output may have been responsible for the lack of improvement or decrease in Pao2 during positive-pressure breathing reported by others.17,15

Assessment of the effects of CPPB on intrapulmonary shunting thus requires knowledge of the systemic effects of CPPB. In lieu of measurement of cardiac output, this can be most easily done by simultaneous measurement of arterial and mixed venous blood.

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References