CONTINUOUS POSITIVE PRESSURE VENTILATION AND OXYGEN DELIVERY

D. M. PHILBIN, R. W. PATTERSON, AND R. A. BARATZ

SUMMARY

The effects of intermittent and continuous positive pressure were studied in mechanically ventilated healthy dogs. No statistically significant changes were produced in oxygen tension, carbon dioxide tension, alveolar-arterial gradients or shunts by continuous positive pressure. It did produce a significant reduction in cardiac output and total oxygen delivery which was reversed by the reintroduction of intermittent positive pressure. The decline in cardiac output was not accompanied by hypotension. The application of continuous positive pressure requires careful assessment of systemic effects to determine its usefulness.

The use of continuous positive-pressure breathing (CPPB) is an increasingly common clinical tool. It has been advocated in certain circumstances, such as pulmonary oedema and the adult respiratory distress syndrome, because it may raise arterial oxygen tension (Pao2) to levels higher than those reached during intermittent positive-pressure breathing (IPPB) (Adams et al., 1969; Ashbaugh et al., 1969; Frumin et al., 1959; McIntyre, Laws and Ramachandran, 1969). Some studies have shown, however, that the effect of CPPB on Pao2, intrapulmonary shunting, and alveolar-arterial oxygen tension differences (A-a)Po2 diff is variable and may be related to the effect on the cardiac output (Cheney, Hornbein and Crawford, 1957; Colgan, Barrow and Fanning, 1971; Gold, Han and Helrich, 1966). It would appear then that the effect on total oxygen delivery (Q x CaO2) might be a more meaningful determinant of the results of CPPB and this study was undertaken to determine the changes in this quantity during IPPB and CPPB.

METHOD

Six healthy mongrel dogs weighing between 14 and 26 kg were anaesthetized initially with intravenous sodium pentobarbitone, 50 mg/kg. The trachea was then intubated with a wide bore endotracheal tube and the cuff inflated to a gas tight fit. Halothane 0.4 to 0.6% as measured by an ASC Fluothane monitor, was subsequently administered via a Copper Kettle with air as the carrier. The oesophageal temperature was monitored and the animal's temperature was kept constant at 37° ± 1°C with a heating pad.

The tip of a cardiac catheter was introduced through the jugular vein into the right ventricle or pulmonary artery (as indicated by the pulse pressure contour). An indwelling teflon catheter was placed in the femoral artery. These were connected to Statham PR-23-2D-300 transducers and a direct writing recorder (Electronics for Medicine, Inc.) for continuous monitoring of pressures. Arterial and mixed venous blood samples were obtained from these catheters. Blood gases were analyzed with a modified Clark oxygen electrode (Mod 133 Instrumentation Laboratory, Boston, Mass.) and with the Severinghaus electrode for carbon dioxide tension at 37°C. Blood pH was measured with an Instrumentation Laboratory pH meter. Blood Po2 was corrected for membrane blood gas difference by tonometry at 37°C. The oxygen capacity was estimated from the haemoglobin concentration and the content calculated from the saturation and tension. The fraction of carbon dioxide and oxygen in mixed expired gas was measured with a Scholander apparatus (Scholander, 1947). The alveolar/arterial oxygen tension difference was calculated assuming that Pco2 was equal to Pao2 and that R was 1.

Daniel M. Philbin, M.D.; Richard W. Patterson M.D.; Robert A. Baratz Ph.D., M.D.; Departments of Anesthesia, Harvard Medical School, Boston, Massachusetts and Columbia University College of Physicians and Surgeons, New York, N.Y.

Presented in part at the American Society of Anesthesiologists National Meeting, Atlanta, Georgia, October 1971.

Supported in part by NIGMS grant 5-P01-GM-09069.

Please address all correspondence to Daniel M. Philbin, Department of Anesthesia, Massachusetts General Hospital, Boston, Massachusetts 02114, U.S.A.
The fraction of cardiac output passing through a shunt \( \frac{Q_s}{Q_T} \) was calculated using the formula (Bartels et al., 1963).

\[
\frac{Q_s}{Q_T} = \frac{(C_{eo} - C_{ao_2})}{(C_{co_2} - C_{vo_2})}
\]

Cardiac output was measured using the standard dye dilution technique with indocyanine green and a Beckman recording densitometer. The densitometer was calibrated with samples of each dog's blood containing a known amount of dye. Injection of dye was through the venous catheter and densitometer sampling was from the femoral artery catheter.

After placement of the endotracheal tube, the dog was ventilated with an Ohio volume controlled ventilator, using a one way valve to eliminate re-breathing. Tidal volume and frequency were adjusted so the end expired carbon dioxide concentration was 3% as measured by an infra-red analyzer (Beckman Model LB-4). The volume and frequency of the ventilator remained unchanged for the remainder of the study. End expiratory pressure was zero.

After the animals were judged to be in a reasonably stable state the experiment was started which consisted of three periods, each of 30 min duration, with measurements being made during the second 15 min of each period. First, IPPB with an end expiratory pressure of zero, (2) CPPB of 10 cm H₂O achieved with a spring-loaded valve in the expiratory line and (3) reinstitution of IPPB.

**RESULTS**

Table I lists the mean values, with standard error, for the measurements and calculations made for all six animals. The initial values for \( P_{ao_2} \) of 101 mm Hg and \( (A-a)P_{o_2} \) of 28.7, indicating relatively good lung function, were not significantly affected by the introduction of CPPB. Reintroduction of IPPB again produced little change.

The initial \( P_{ao_2} \) of 19.6 mm Hg was a reflection of the degree of hyperventilation produced by the IPPB. This rose slightly during CPPB and then returned to the previous level. The introduction of CPPB did not produce any statistically significant changes in either \( C_{ao_2} \), \( C_{vo_2} \) or \( \frac{Q_s}{Q_T} \). \( (A-a)P_{o_2} \) demonstrated a progressive decline during the entire study going from 28.7 mm Hg initially to 25.1 mm Hg during CPPB, to 21.9 mm Hg in the final period but again these differences were not significant.

The introduction of CPPB in period 2 did substantially affect the cardiac output, producing a fall from 3.34 l/min to 2.38 l/min. Concomitant with this was a corresponding reduction in oxygen delivery \( Q \times C_{ao_2} \) which fell from 628.8 ml/l. to 429.5 ml/l. with CPPB and rose again to 559.2 ml/l. with reinstitution of IPPB.

**TABLE I. Cardiorespiratory Data**

<table>
<thead>
<tr>
<th></th>
<th>IPPB</th>
<th>CPPB</th>
<th>IPPB</th>
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<tbody>
<tr>
<td>( P_{ao_2} ) (mm Hg)</td>
<td>19.6 ± 1.9</td>
<td>25.1 ± 3.9</td>
<td>19.9 ± 1.8</td>
</tr>
<tr>
<td>( P_{co_2} ) (mm Hg)</td>
<td>101 ± 8</td>
<td>100 ± 7</td>
<td>109 ± 5</td>
</tr>
<tr>
<td>( \frac{Q_s}{Q_T} ) (%)</td>
<td>13.38 ± 3.8</td>
<td>14.48 ± 4.7</td>
<td>10.68 ± 1.94</td>
</tr>
<tr>
<td>( (A-a)P_{o_2} )diff (mm Hg)</td>
<td>28.7 ± 8</td>
<td>25.1 ± 4.6</td>
<td>21.9 ± 4.8</td>
</tr>
<tr>
<td>( C_{ao_2} ) (ml/100 ml)</td>
<td>17.96 ± 1.1</td>
<td>17.52 ± 0.95</td>
<td>18.0 ± 1.1</td>
</tr>
<tr>
<td>( C_{vo_2} ) (ml/100 ml)</td>
<td>14.94 ± 1.3</td>
<td>14.30 ± 1.5</td>
<td>15.10 ± 1.3</td>
</tr>
<tr>
<td>Cardiac Output (l/min)</td>
<td>3.34 ± 0.62</td>
<td>2.38 ± 0.3</td>
<td>2.97 ± 0.48</td>
</tr>
<tr>
<td>Oxygen Delivery ( (Q \times C_{ao_2}) ) (ml/l)</td>
<td>628.8 ± 165</td>
<td>429.5 ± 82</td>
<td>559.2 ± 130</td>
</tr>
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*Mean ± SE
Figure 1 illustrates the changes produced in some of these parameters by the introduction of CPPB.

**DISCUSSION**

The initial blood gas values obtained in period 1 indicated relatively good lung function and pulmonary gas exchange in these animals. This was corroborated by the values obtained for alveolar/arterial oxygen tension difference and shunt. It was therefore anticipated that the institution of CPPB would produce little change or improvement. The values obtained in period 2 substantiated this.

The low $P_{aO_2}$ in period 1 is indicative of the degree of hyperventilation produced which is not an unusual clinical situation. The slight rise produced by CPPB was obviously not enough to increase the cardiac output.

The marked effect that CPPB may have on the cardiac output has been reported by a number of previous investigations (Baratz, Philbin and Patterson, 1971; Cheney, Hornbein and Crawford, 1967; Colgan, Barrow and Fanning, 1971) under varying circumstances. In this study the presence of halothane may have contributed to this effect. Sykes and associates (1970) also reported in normovolemic dogs a decline in cardiac output and alveolar/arterial oxygen tension difference. One explanation offered is that the increase in lung volume produced by end-expiratory pressure improves the distribution of air in the lung thus delivering more air to overperfused alveoli (thus reducing effective alveolar deadspace). In this study, however, reinstitution of IPPB produced a further decrease in alveolar/arterial oxygen tension difference, though not a statistically significant one. Obviously then a number of factors are involved. It is quite possible that the rise in cardiac output in period 3 produced a better distribution in a lung previously exposed to CPPB. Since all of our measurements were made on room air we cannot differentiate between true shunt and ventilation-perfusion abnormalities.

The contribution of the cardiac output to arterial oxygenation has already been demonstrated (Kelman, et al., 1967). The result of this reduction in cardiac output was inevitably then a substantial reduction in total oxygen delivery. The small changes in arterial oxygen tension difference are a reflection of the relatively small shunt present in these healthy animals. In the presence of a large shunt, a significant decline in the arterial oxygen tension could be anticipated (Philbin et al., 1970). The small rise in arterio-venous oxygen content difference from period 1 to period 2 (3.02 to 3.22 vols %) suggests several possibilities. It is unlikely that the introduction of CPPB produced any significant change in oxygen consumption (Sykes at al., 1970). The decreased cardiac output then most likely led to a decrease in perfusion of some areas and organs. Differential studies were not made but previous work (Baratz, Philbin and Patterson, 1971) suggests that there may be a notable decline in kidney perfusion. The effect on other organ systems can only be speculated.

It is apparent then, that at least under these circumstances, measurements of arterial oxygen tension, shunt and alveolar/arterial oxygen tension difference are not an accurate reflection of the overall effect of CPPB on oxygen delivery. The systemic effects, particularly on the cardiac output must be considered. It should be noted that substantial reductions in the cardiac output can be produced without being reflected in concomitant reductions in arterial pressure (Philbin and Sullivan, 1971).

Obviously data obtained in healthy anaesthetized dogs cannot be completely extrapolated to clinical situations. Nevertheless, these data again emphasize that the use of CPPB requires careful monitoring of a number of parameters in order to accurately assess its effect. In patients with large shunts, unless there is sufficient decrease to compensate for a decline in cardiac output arterial oxygen tension may be unimproved or even decline. If the cardiac output falls enough, flow through some organ systems may be seriously impaired at the expense of a high arterial oxygen tension. The use of CPPB then requires assessment of each individual situation with accurate monitoring.

**REFERENCES**


**VENTILATION A PRESSION POSITIVE CONTINUE ET APPORT D'OXYGENE**

**SOMMAIRE**

Les effets de la pression positive intermittente et continue ont été étudiés chez des chiens sains mécaniquement ventilés. Des modifications statistiquement significatives n'ont pas été produites au niveau de la pression d'oxygène et d'anhydride carbonique, des gradients alvéolaires-arteriels ou des shunts par la pression positive continue.

Elle causa una reducción significativa del débito cardíaco y de l'apport total d'oxygène, reversible par la reintroducción de la presión positiva intermittente. La reducción del débito cardíaco ne s'accompagna pas d'hypotension. L'application d'une pression positive continue nécessite une évaluation soignueuse de ses effets systémiques pour déterminer son utilité.

**KONTINUIERLICHE ÜBERDRUCKBEATMUNG UND SAUERSTOFFVERSORGUNG**

**ZUSAMMENFASSUNG**


**VENTILACION CON PRESION POSITIVA CONTINUA Y APORT DE OXIGENO**

**RESUMEN**

Los efectos de la presión positiva intermitente y continua fueron estudiados en perros sanos ventilados mecánicamente. No fueron producidos cambios estadísticamente significativos en la tensión de oxígeno, tensión de anhidrido carbónico, gradientes alveolares-arteriales o shunts por presión positiva continua. Produjo una reducción significativa en el gasto cardíaco y aporte total de oxígeno que fue invertida por la reintroducción de la presión positiva intermitente. La disminución del gasto cardíaco no fue acompañada por hipotensión. La aplicación de presión positiva continua requiere una evaluación cuidadosa de los efectos sistémicos para determinar su utilidad.