After fifty years of cardiopulmonary bypass (CPB), we are still debating the optimal value for arterial blood pCO2 during CPB for different patient groups. The availability of improved cerebral function and cerebral artery velocity and emboli monitors has given perfusionists and physicians renewed motivation to be even more precise in our control of pCO2 (1).

After reading Editor Stammers’ J Extra Corpor Technol volume 36 number 2 letter and Bill Horgan’s guest editorial in the same issue, it set me to thinking about my perfusion education and my mentors (2,3). Jim Dearing wrote both about perfusion education and about perfusion techniques. Jim’s writing about perfusion education was often controversial, especially in the late 1970s and early 80s (4). Jim’s published works on perfusion techniques were usually novel and educational.

Leon Camerlengo, after becoming a perfusionist, went on to medical school and is now a practicing cardiovascular anesthesiologist. As a student, he teamed up with Dearing to present this issue’s classic article at the 1980 Philadelphia AmSECT conference. Camerlengo and Dearing’s publication contributes two perfusion technology scientific building blocks:

1) If you know the patient’s oxygen consumption rate, you can predict an oxygenator “optimal ventilation rate” to use at the beginning of CPB; and

2) by continuously monitoring the oxygenator outlet fraction CO2 or the pCO2, you can control the ventilation rate to precisely control the arterial blood pCO2.

They probably got the idea to analyze the gas exiting the oxygenator from Snider, et al. (5). Snider’s work influenced the AAMI standards for manufacturer oxygenator development and testing.

Many perfusionists and anesthesiologists have employed end-tidal CO2 analyzers (infra-red and mass spectrometer) to continuously monitor oxygenator outlet gas pCO2, which is almost in equilibrium with the arterial blood pCO2 (6,7).

Oxygenator capnography may still be a strong clinical research or learning tool for perfusionists who have a renewed interest to study the physiologic benefits of more precise control of pCO2 during CPB. Camerlengo’s J Extra Corpor Technol classic is the starting place for clinicians and students.

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REFERENCES


Write to riley.267@osu.edu to nominate a J Extra Corpor Technol article as a classic.
Precise Control of PCO₂ During Cardiopulmonary Bypass

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Introduction

Maintenance of normal arterial PCO₂ (PaCO₂) during cardiopulmonary bypass (CPB) is essential to both normal acid-base balance and proper blood flow distribution. Respiratory acidosis and more frequently alkalosis are commonly reported conditions concurrent with CPB. Furthermore, decreased PaCO₂ causes a shift in the oxyhemoglobin dissociation curve to the left resulting in less oxygen being available to the tissues at a given PO₂. This couple these events with the fact that decreased PaCO₂ decreases cerebral perfusion and the importance of maintaining normal values becomes readily apparent.

PaCO₂ is primarily a function of CO₂ production, minute ventilation, and inspired CO₂ concentration (FiCO₂). In the past, due to the relative oxygenating inefficiency of our gas exchange devices, we have been forced to hyperventilate in order to oxygenate. The only means left to prevent respiratory alkalosis was to add CO₂ to the ventilating gas, thus reducing the CO₂ driving gradient. With the advent of efficient membrane oxygenators (MO) and their independent control of the two respiratory blood gases, precise regulation of PaCO₂ has become possible by manipulating the ventilatory flow rate (QG). This study was designed to demonstrate the effectiveness of this method for PaCO₂ control during CPB.

Methods and Materials I

A mathematical method was developed (Equation 1) to estimate the optimal ventilation (EOV) required for normothermic perfusion.

Equation 1: Estimation of Optimal Normothermic Minute Volume

\[
EOV \text{ (ml/min)} = \frac{\text{BSA (m²)} \times 80 \text{ (ml/m²/min)}}{.053}
\]

This method was utilized during 50 consecutive CPB procedures utilizing either a SciMed* or Travenol** Membrane Oxygenator. Blood samples were drawn from a well flushed arterial sampling port during normothermic perfusion. The samples were subjected to blood gas analysis (BGA) by an Instrumentation Laboratory (IL) Micro 13 blood gas analyzer,*** operating at 37°C. Ninety-five percent confidence limits were constructed for the measured PaCO₂'s.

Methods and Materials II

A simple proportionality formula (Equation 2) was developed for correcting abnormal PaCO₂ during stable periods of CPB. This method was utilized during 100 consecutive CPB procedures using either MO device. Once stable CPB was attained, a blood sample was taken from a well flushed arterial sampling port of the MO, and analyzed by the IL Micro 13 blood gas analyzer operating at 37°C. The raw data were temperature corrected, if necessary, to the patient's blood temperature measured by an arterial temperature

* Sci-Med Life Systems, Inc., Minneapolis, MN 55441
** Travenol Laboratories, Inc., Deerfield, IL 60015
*** Instrumentation Laboratory, Inc., Model 326.10, Lexington, MA 02173
probe. Once the temperature corrected PaCO₂ (measured PaCO₂) was determined, the new QG required to normalize the PaCO₂ was derived from Equation 2.

Equation 2: Simple Proportionality Formula Used For Estimating Optimal Minute Volume

$$\text{New Minute volume (mL/min) = Old minute volume (mL/min)} \times \frac{\text{Measured PaCO}_2 \text{ (mm Hg)}}{\text{Desired PaCO}_2 \text{ (mm Hg)}}$$

After the newly adjusted QG was maintained for at least five minutes, a second arterial blood sample was analyzed. Ninety-five percent confidence limits were constructed for the measured PaCO₂'s.

Methods and Materials

PaCO₂ control was attempted using an in-line infra-red CO₂ concentration analyzer**** (Figure 1). The sampling probe from the analyzer was strategically placed so that the CO₂ concentration in the exhaust gas from either a membrane or bubble oxygenator was continuously monitored. The QG was altered until the desired expired CO₂ concentration (FeCO₂) was attained. Results were displayed in percent concentration and exhaust gas PCO₂ (PexCO₂) were determined with Equation 3.

Equation 3: Formula used for Calculation of PexCO₂

$$\text{PexCO}_2 \text{ (mm Hg) = Percent Concentration} \times \text{PCO}_2 \text{ (mm Hg)}$$

These data were compared to measured PaCO₂ determined by an IL Micro 13 blood gas analyzer. One hundred and twenty samples were drawn from an arterial sampling port located next to an in-line temperature probe. Samples were analyzed during all stages of CPB and temperature corrected accordingly. Correlation between the measured PaCO₂ with expired CO₂ concentration as well as PexCO₂ was determined. Statistical comparisons were made using the student's t-test. Results were expressed as mean ± 1 SD.

Results

Sixty-three blood samples were analyzed after estimation of the optimal ventilatory flow rate during normothermic perfusion. With the use of Equation 1, the measured PaCO₂ averaged 38 ± 3.1 mm Hg (mean

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**** Sarns, Inc., Ann Arbor, MI 48103
***** Cavitron Neonatal Monitor, Model No. PM-20N, Anaheim, CA 92802
****** Sci-Med Life Systems Inc., Minneapolis, MN 55441
Travenol Laboratories Inc., Deerfield, IL 60015
******* Shiley Scientific Inc., Irvine, CA 92714 S-070 and S-100
William Harvey, Division of C.R. Bard Inc., Santa Ana, CA 92705 H-400 and H-1000


table 1

comparison of measured PaCO₂ with CO₂ concentration
in exhaust gas from blood oxygenators

<table>
<thead>
<tr>
<th>Device</th>
<th>Temperature range (°C)</th>
<th>No. of samples</th>
<th>Percent concentration CO₂ in exhaust gas</th>
<th>Calculated PEO₂ (mm Hg)</th>
<th>Measured PaCO₂ (mm Hg)</th>
<th>Percent divergence from measured PaCO₂</th>
</tr>
</thead>
<tbody>
<tr>
<td>MO</td>
<td>15 - 38</td>
<td>60</td>
<td>4.8±1.0</td>
<td>36.5±9.3</td>
<td>37.1±8.6</td>
<td>4.0±3.3</td>
</tr>
<tr>
<td>BO</td>
<td>15 - 38</td>
<td>60</td>
<td>4.7±1.0</td>
<td>34.4±7.2</td>
<td>35.1±6.9</td>
<td>4.8±4.2</td>
</tr>
</tbody>
</table>

±1 SD). Ninety-five percent of the measured PaCO₂ fell within the range of 32–44 mm Hg.


Results II

One hundred and seventy-two consecutive blood samples were analyzed after changing QG based upon Equation 2 during periods of stable CPB. With the use of Equation 2, the measured PaCO₂ averaged 39±1.27 mm Hg (mean ±1 SD). Samples were analyzed during any stable period of CPB with a temperature range of 17°C–38°C. Ninety-five percent of the measured PaCO₂ fell within the range of 34–44 mm Hg.

Results III

Table I summarizes the data for the 120 consecutive blood samples analyzed with either a MO or BO. Figure 2 vividly demonstrates the very high correlation which existed between the measured PaCO₂ and the CO₂ concentration in the exhaust gas from either device (r = 0.97, p < .001).

The difference between measured PaCO₂ and PEO₂ was expressed as percent divergence. The PEO₂ deviated from the measured PaCO₂ by an average of 4.0 ± 3.3% (MO) and 4.8 ± 4.2% (BO).

Discussion

In the past, QG control has predominantly been by empirical estimation and no consistently good technique has been reported. This becomes obvious when examining operational instructions for the various gas exchange devices. Hyperventilating gas to blood flow rates are recommended and frequently it is suggested that CO₂ should be added to the ventilating gas. Using pure oxygen, the gas flow in blood oxygenators can be adapted to match the metabolic production of CO₂.

The mathematical formula used in Method I is based on normal oxygen consumption and CO₂ production data. The generally accepted value for O₂ consumption is approximately 150 ml/m²/min while the value for CO₂ production is approximately 120 ml/m²/min, a respiratory quotient (RQ) of 0.8. Oxygen consumption and CO₂ production are reduced by approximately one-third due to anesthesia, skeletal muscular paralysis, and artificial ventilation. This means that the average patient undergoing open-heart surgery will have a normothermic O₂ demand of approximately 100 ml/m²/min and a CO₂ production of approximately 80 ml/m²/min.

Once the CO₂ production rate has been estimated, the ventilation rate required to achieve a desired PaCO₂ can be calculated. Since the PaCO₂ and FeCO₂ correlate so closely in the artificial lung, the estimated optimal ventilation rate should be that minute volume

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FIGURE 3. Comparison of Measured PaCO₂ with Calculated PEO₂

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in which the produced CO₂ will be diluted to achieve the desired FeCO₂. For example, if the barometric pressure is 760 mm Hg and the desired PaCO₂ is 40 mm Hg, the desired FeCO₂ must be 5.3% (40/760 × 100). So, the desired QG is that flow that will dilute the CO₂ produced to a concentration of 5.3% or CO₂ production/5.3%.

During CPB variation in patient’s CO₂ production occurs due to changes in metabolic activity. These changes are caused by the induction of hypothermia, varying levels of anesthesia, and the use of muscle relaxants. The variations in CO₂ production necessitate changes in ventilation rate if the PaCO₂ is to be kept within the desirable range. Changes are made using a simple proportionality formula once the CO₂ production is estimated from a measured PaCO₂ and ventilation rate (QG):

\[
\text{CO}_2\text{ production} = \frac{\text{measured PaCO}_2}{P_{\text{Barometric}}} \times \text{QG}
\]

The formula is derived as follows:

\[
\text{New QG} = \frac{\text{CO}_2\text{ production}}{\text{Desired FeCO}_2} = \frac{\text{Measured PaCO}_2}{P_{\text{Barometric}}} \times \text{Old QG}
\]

inverting the denominator and multiplying:

\[
\text{New QG} = \frac{\text{Measured PaCO}_2}{P_{\text{Barometric}}} \times \frac{P_{\text{Barometric}}}{\text{Desired PaCO}_2} \times \text{Old QG}
\]

cancelling the \(P_{\text{Barometric}}\):

\[
\text{New QG} = \frac{\text{Measured PaCO}_2}{\text{Desired PaCO}_2} \times \text{Old QG}
\]

Equations 1 and 2 can be used for all oxygenators, but do not appear to be useful for bubble oxygenators due to the BO’s relative oxygenating inefficiency. At gas to blood flow ratios required to achieve normal PaCO₂, the PaO₂ usually became less than 80 mm Hg. As increasing oxygenating efficiency is achieved in bubble oxygenators, these formulae may be applicable to these devices.

Use of the in-line exhaust gas infra-red CO₂ concentration analyzer produced excellent results in both bubbler and membrane oxygenators. The PaCO₂ could essentially be dialed in by fine tuning QG in MO’s or the FiCO₂ in BO’s to produce a favorable exhaust gas CO₂ concentration. Not only was the device consistent during periods of stable CPB, but dynamic changes in the patient’s CO₂ production could be continuously monitored. Such rapid assessment of the patient’s respiratory status can only improve the adequacy of perfusion.

Conclusions

1. Optimal ventilation can be estimated by using Equation 1 at the onset of by-pass and by modifying QG during bypass with Equation 2.

2. Expired CO₂ concentration can be monitored by an infra-red CO₂ analyzer from either a MO or BO and be used to control PaCO₂. This technique obviates the necessity of measuring PaCO₂.

References


