Prediction of Arterial Blood pCO₂ by Measuring the Ventilating Gas Exhaust pCO₂ from a Bubble Oxygenator

Jeffrey B. Riley
Emory University Clinic
Atlanta, Georgia

Abstract

Ventilating gas exhaust pCO₂ (PexCO₂) was continuously monitored by a mass spectrometer during hypothermic, total heart lung bypass employing a bubble oxygenator. The hypothesis that the arterial blood pCO₂, temperature corrected to the arterial blood temperature (pCO₂, TBo) is in equilibrium with the PexCO₂ was tested.

While the PexCO₂ trends well with the pCO₂, TBo during rapid cooling, it assumes a lower value compared to the arterial pCO₂ corrected to the nasopharyngeal temperature (pCO₂, TNP) because the blood temperature is much less than nasopharyngeal.

During warming, PexCO₂ reads consistently lower than pCO₂, TBo apparently due to increased blood CO₂ removal (VCO₂) and an inability of the PexCO₂ to come to equilibrium with pCO₂, TBo in the oxygenator. During low VCO₂ and steady state conditions, PexCO₂ predicts pCO₂, TBo well (r = .803, p <.01). The percent disagreement of PexCO₂ and pCO₂, TBo is a linear function of the VCO₂ (r = -.606, p <.01).

Introduction

A continuous, on line monitor of the arterial blood pCO₂ during total heart lung bypass can aid the operator in better controlling the respiratory component of the acid base system. Constant knowledge of the arterial blood pCO₂ allows the perfusionist to monitor the large excursions in the total blood CO₂ content as the patient's metabolic rate and the CO₂ solubility continuously change during total body perfusion with rapid blood stream cooling and warming.

In the human lung with adequate perfusion/ventilation, the end expiratory gas pCO₂ is in equilibrium with the end alveolar capillary blood pCO₂. Therefore, it is reasonable to hypothesize that the blood pCO₂ leaving an artificial oxygenator is in equilibrium with the ventilating gas pCO₂ (PexCO₂) exiting the device following adequate residence time with mixing in the bubble column.

During total heart lung bypass, venous blood enters the bubble oxygenator with a total CO₂ content of approximately 45 to 50 volumes % under normal physiologic conditions. CO₂ dissolved in solution in the plasma and cellular water exerts a pressure (pCO₂). The venous blood pCO₂ becomes the forcing function for CO₂ gaseous exchange with the ventilating gas during tonometry in the oxygenating device bubble column and defoamer area.

The PexCO₂ can be useful in determining the volume exchange per minute of CO₂ (VCO₂, mlCO₂/min) in the transfer device. This value may be quantiated in the gaseous phase by measuring the difference between FiCO₂ (ventilating gas inlet percent CO₂) and FoCO₂ (exhaust ventilating gas percent CO₂) and multiplying by the ventilating gas flow.
Infrared analyzers are available to accurately measure percent CO₂ in the gaseous phase. However, if FoCO₂ is not measured directly, it may be calculated from a PexCO₂ measurement in the device exhaust gas:

\[
\text{FoCO₂} = \frac{\text{PexCO₂}}{(P_{\text{atm}} - P_{\text{H₂O}}) \text{ mmHg CO₂}} \text{ Eq. 2}
\]

where \( P_{\text{atm}} \) is atmospheric pressure adjusted for the pressure of water vapor \( P_{\text{H₂O}} \). \( P_{\text{H₂O}} \) is for 100% humidity and may be adjusted for ventilating gas temperature to increase accuracy. The FiCO₂ may be calculated with calibrated dry gas from meter readings if infrared analysis is not available:

\[
\text{FiCO₂} = \frac{X\% \text{ CO₂} \times \text{CO₂ gas mixture flow}}{(\text{CO₂ gas flow} + \text{O₂ gas flow})} = \frac{\text{ml/minute}}{\text{ml/minute}} \text{ Eq. 3}
\]

where \( X\% \text{ CO₂} = \) the \% CO₂ in the CO₂ gas mixture used in the ventilating gas.

The pressure exerted by the CO₂ in the ventilating gas (\( p_{\text{in CO₂}} \)) at the blood inlet and the venous blood PCO₂ determine the partial pressure gradient for CO₂ gas exchange. The operator partially controls \( V_{\text{CO₂}} \) by altering FiCO₂ and hence the \( p_{\text{in CO₂}} \) for a given venous blood PCO₂ and perfusion circumstance. If adequate mixing time is allowed, the \( p_{\text{in CO₂}} \) should come to equilibrium with the arterial blood PCO₂.

**Method**

To test the hypothesis that the \( p_{\text{in CO₂}} \) is an accurate predictor of PCO₂, \( T_{\text{Bo}} \), the following protocol was executed. The exhaust gas PCO₂ at the gas exit port of the Cobe Laboratories Model 42-221 Optiflo II bubble oxygenator\(^a\) was continuously monitored by an Elmer-Perkin Model 1100 Medical gas mass spectrometer analyzer.\(^b\) The gas exhaust port of the Optiflo II was unobstructed and the exhaust gas sample was drawn to the gas analyzer by a 30 foot small bore vacuum hose at a rate of 225 ml/minute, creating a measurement system response time of about 90 seconds. The continuous sample PCO₂ wave form was presented in the O.R. Suite on a small calibrated scope. The waveform was updated every 90 seconds since the analyzer was on a time sharing system with 14 other O.R. suites. The oxygenator gas temperature was measured by a sterile Yellow Springs Instrument\(^c\) Model 514 hypodermic needle temperature probe introduced into the rapid prime port of the Optiflo II. The gas temperature was employed to estimate the \( P_{\text{H₂O}} \) of the 100% humidity ventilating gas in Equation 2.

Figure 1 presents a typical curve for a measurement system calibration verification performed prior to initiation of bypass. The results are plotted versus time as the FiCO₂ is altered. The 30°C oxygen gas flow was constant at 4 liters/minute. There was no perfusate flow through the oxygenator gas column, therefore the FoCO₂ equaled the FiCO₂. The expected \( p_{\text{in CO₂}} \) calculated from the FoCO₂ and atmospheric pressure \( (P_{\text{atm}}) \) agrees well with the mass spectrometer PexCO₂. The 90-second rise time is consistent at an oxygenator gas flow of 4 liters/minute.

The blood inlet and blood outlet temperatures, blood gas and pH samples were collected from the integral temperature and blood sample ports on the Optiflo II. The inlet and outlet blood gas and pH values analyzed at 37°C were temperature corrected by the analog equations presented by Nunn, Thomas and Severinghaus to the respective blood temperature.\(^2\) The arterial blood PCO₂ was also temperature corrected to the patient temperature (PCO₂, \( T_{\text{NP}} \)) for comparison with \( T_{\text{Bo}} \)-corrected PCO₂ values and \( p_{\text{in CO₂}} \).

Patient blood flow, 100% oxygen and 100% CO₂ dry gas flows were recorded from calibrated twin roller pump and gas flow meters. Simultaneous blood gas and pH determinations and circuit temperatures were collected on 49 consecutive samplings during 15 adult open heart procedures employing the Optiflo II for total cardiopulmonary support. The FiCO₂ from Equation 3, the FoCO₂ from Equation 2, and the \( V_{\text{CO₂}} \) from equation 1 were calculated for each sampling.

The \( p_{\text{in CO₂}} \) was compared to the PCO₂, \( T_{\text{Bo}} \) and the PCO₂, \( T_{\text{NP}} \) in a linear regression model sample pairs. The % disagreement in the \( p_{\text{in CO₂}}, T_{\text{Bo}} - p_{\text{in CO₂}} \) was

\(^a\) Cobe Laboratories, Inc., Lakewood, Colorado, 80215
\(^b\) Elmer-Perkin, Pomona, California, 91767
\(^c\) Yellow Springs Instrument Co., Inc., Yellow Springs, Ohio, 45387
FIGURE 1. Mass spectrometer $p_{ex}CO_2$ measurement system calibration verification. Oxygen gas flow = 4 L/min at 30°C and $P_{ atm}$ = 735 mmHg. The mass spectrometer $p_{ex}CO_2$ is plotted versus time as the FoCO$2$ is varied.

compared to the $V_{CO_2}$ in a linear regression model for the sample pairs.

**Results and Discussion**

Table 1 lists the correlation coefficients for the agreement between the $p_{ex}CO_2$ and $pCO_2$, $T_{Bo}$ during cooling, hypothermia and warming phases of bypass. Overall, the $p_{ex}CO_2$ is a poor predictor of the $pCO_2$, $T_{NP}$ ($r = .389$, $p < .01$). For all samples, the $p_{ex}CO_2$ is a fair predictor of the $pCO_2$, $T_{Bo}$ ($r = .803$, $p < .01$). However, the $pCO_2$ is not routinely temperature corrected to the $T_{Bo}$ in some clinical settings. During rapid cooling, the $T_{Bo}$ is much lower than the $T_{NP}$ and the outlet blood $pCO_2$ is also lower, leading to a large discrepancy between the $p_{ex}CO_2$ and the $pCO_2$, $T_{NP}$ as seen in Figure 2. The lower correlation coefficients during warming and at normothermic equilibrium warranted the comparison of the percent disagreement in $p_{ex}CO_2$ and $pCO_2$, $T_{Bo}$ to the $V_{CO_2}$ (Personnel communication, Marc Vorhees, Cobe Laboratories, Inc.\textsuperscript{a}). The blood $CO_2$ removal is especially large during warming and normothermia after maintaining temperature corrected 37°C, normal $pCO_2$'s during hypothermia and increasing blood $CO_2$ content. Figure 3 presents the relationship between the percent difference in $p_{ex}CO_2$ and $pCO_2$, $T_{Bo}$ to the $V_{CO_2}$ for all sample points. The predicting discrepancy appears to be a function of the blood $CO_2$ removal rate ($r = −.606$, $p < .01$). For most blood flows and gas-to-blood flow ratios, which determine the blood residence time

<table>
<thead>
<tr>
<th>Phase of Bypass</th>
<th>Rapid Cooling</th>
<th>Cold Equilibrium</th>
<th>Rapid Warming</th>
<th>Warm Equilibrium</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slope</td>
<td>.89</td>
<td>.76</td>
<td>.33</td>
<td>.93</td>
</tr>
<tr>
<td>Y-Intercept</td>
<td>4.5</td>
<td>10.8</td>
<td>31.9</td>
<td>6.3</td>
</tr>
<tr>
<td>Degrees of Freedom</td>
<td>10</td>
<td>8</td>
<td>21</td>
<td>2</td>
</tr>
<tr>
<td>Correlation Coefficient</td>
<td>.95</td>
<td>.80</td>
<td>.48</td>
<td>.52</td>
</tr>
<tr>
<td>Significance</td>
<td>$p &lt; .01$</td>
<td>$p &lt; .01$</td>
<td>$&lt; p &lt; .05$</td>
<td>NS</td>
</tr>
</tbody>
</table>
with the gas, the $p_xCO_2$ rarely comes to equilibrium with the $pCO_2$, $T_B0$ in the Optiflo II blood oxygenator. The blood $pCO_2$ predicting power of the $p_xCO_2$ is greatest during hypothermia and situations of low $CO_2$ exchange. The perfusionist can adapt to the discrepancies and use the trend data in the $p_xCO_2$ to control the $pCO_2$, $T_{NP}$.

The availability of the mass spectrometer and the ability to accurately calculate $V_{CO_2}$ with computer assistance allows expanded research capabilities in the clinical setting. Knowledge of the $V_{CO_2}$ allows confirmation of the clinical diagnosis of respiratory acidosis (decreased $V_{CO_2}$, decreased or negative respiratory quotient [R.Q.]) and the converse respiratory alkalosis.

Recently, Abbott, et al., 1980, employed the mass spectrometer to measure ventilating gas $V_{O_2}$, $V_{CO_2}$, and R.Q. in a study of pediatric perfusion cases. Abbott documented CO$_2$ addition to the blood (negative respiratory quotient) during rapid cooling and advocated the use of 10% CO$_2$ in the ventilating gas, and maintenance of an R.Q. = .8 during warming of deep hypothermia patients to optimize perfusion adequacy.

The $V_{CO_2}$ and respiratory quotient are not only a function of the oxygen transfer, venous blood CO$_2$ content, oxygenator gas-to-blood flow ratio, and percent CO$_2$ in the ventilating gas, but the ability of the operator to maintain minimal fluctuations in arterial blood pCO$_2$.

Acknowledgment

The author gratefully acknowledges the valuable assistance of Steven Moffitt, Ph.D., in the statistical analysis as well as the writing of this publication and Norma R. McGraw for technical assistance.

Conclusions

1. The $p_xCO_2$ is a fair predictor of the arterial blood pCO$_2$ temperature corrected to the $T_B0$ and a poor predictor of the pCO$_2$ corrected to the $T_{NP}$ during all phases of hypothermic bypass.

2. The percent disagreement in the $p_xCO_2$ and the pCO$_2$, $T_B0$ is a function of the $V_{CO_2}$. The $p_xCO_2$ is in equilibrium with the $pCO_2$, $T_{NP}$ when $V_{CO_2}$ is minute.

3. The use of the $p_xCO_2$ to calculate $V_{CO_2}$ in the clinical setting provides the operator a tool for diagnosis of respiratory acid base disorders and clinical research in conjunction with on-line monitoring of the $V_{CO_2}$ and respiratory quotient.

Bibliography