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

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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 C02 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. The 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.
Irradiated analyzers are available to accurately measure percent CO\textsubscript{2} in the gaseous phase. However, if FoCO\textsubscript{2} is not measured directly, it may be calculated from a PexCO\textsubscript{2} measurement in the device exhaust gas:

\[
FoCO_2 = \frac{P_{ex}CO_2}{(P_{atm} - P_{H_2O})} \text{ mmHg CO}_2 \text{ mmHg Eq. 2}
\]

where Patm is atmospheric pressure adjusted for the pressure of water vapor P\textsubscript{H\textsubscript{2}O}. P\textsubscript{H\textsubscript{2}O} is for 100\% humidity and may be adjusted for ventilating gas temperature to increase accuracy. The FiCO\textsubscript{2} may be calculated with calibrated dry gas from meter readings if infrared analysis is not available:

\[
FiCO_2 = \frac{X\% CO_2 \times CO_2 \text{ gas mixture flow}}{(CO_2 \text{ gas flow} + O_2 \text{ gas flow})} \text{ ml/minute} \text{ Eq. 3}
\]

where X\% CO\textsubscript{2} = the \% CO\textsubscript{2} in the CO\textsubscript{2} gas mixture used in the ventilating gas.

The pressure exerted by the CO\textsubscript{2} in the ventilating gas (p\textsubscript{in}CO\textsubscript{2}) at the blood inlet and the venous blood pCO\textsubscript{2} determine the partial pressure gradient for CO\textsubscript{2} gas exchange. The operator partially controls V\textsubscript{CO\textsubscript{2}} by altering FiCO\textsubscript{2} and hence the p\textsubscript{in}CO\textsubscript{2} for a given venous blood pCO\textsubscript{2} and perfusion circumstance. If adequate mixing time is allowed, the p\textsubscript{ex}CO\textsubscript{2} should come to equilibrium with the arterial blood pCO\textsubscript{2}.

Method

To test the hypothesis that the p\textsubscript{ex}CO\textsubscript{2} is an accurate predictor of pCO\textsubscript{2}, T\textsubscript{Bo}, the following protocol was executed. The exhaust gas pCO\textsubscript{2} at the gas exit port of the Cobe Laboratories Model 42-221 Optiflo II bubble oxygenator\textsuperscript{a} was continuously monitored by an Elmer-Perkin Model 1100 Medical gas mass spectrometer analyzer.\textsuperscript{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 pCO\textsubscript{2} 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\textsuperscript{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\textsubscript{H\textsubscript{2}O} 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 FiCO\textsubscript{2} 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 FoCO\textsubscript{2} equaled the FiCO\textsubscript{2}. The expected p\textsubscript{ex}CO\textsubscript{2} calculated from the FiCO\textsubscript{2} and atmospheric pressure (P\textsubscript{atm}) agrees well with the mass spectrometer PexCO\textsubscript{2}. 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.\textsuperscript{2-4} The arterial blood pCO\textsubscript{2} was also temperature corrected to the patient temperature (pCO\textsubscript{2}, T\textsubscript{NP}) for comparison with T\textsubscript{Bo}-corrected pCO\textsubscript{2} values and p\textsubscript{ex}CO\textsubscript{2}.

Patient blood flow, 100\% oxygen and 100\% CO\textsubscript{2} 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 FiCO\textsubscript{2} from Equation 3, the FoCO\textsubscript{2} from Equation 2, and the V\textsubscript{CO\textsubscript{2}} from equation 1 were calculated for each sampling.

The p\textsubscript{ex}CO\textsubscript{2} was compared to the pCO\textsubscript{2}, T\textsubscript{Bo} and the pCO\textsubscript{2}, T\textsubscript{NP} in a linear regression model sample pairs. The % disagreement in the p\textsubscript{a}CO\textsubscript{2}, T\textsubscript{Bo} - p\textsubscript{ex}CO\textsubscript{2} was

\textsuperscript{a} Cobe Laboratories, Inc., Lakewood, Colorado, 80215
\textsuperscript{b} Elmer-Perkin, Pomona, California, 91767
\textsuperscript{c} Yellow Springs Instrument Co., Inc., Yellow Springs, Ohio, 45387
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 $p_{CO_2}$, $T_{Bo}$ during cooling, hypothermia and warming phases of bypass. Overall, the $p_{ex}CO_2$ is a poor predictor of the $p_{CO_2}$, $T_{NP}$ ($r = .389, p < .01$). For all samples, the $p_{ex}CO_2$ is a fair predictor of the $p_{CO_2}$, $T_{Bo}$ ($r = .803, p < .01$). However, the $p_{CO_2}$ 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 $p_{CO_2}$ is also lower, leading to a large discrepancy between the $p_{ex}CO_2$ and the $p_{CO_2}$, $T_{NP}$ 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 $p_{CO_2}$, $T_{Bo}$ to the $V_{CO_2}$ (Personnel communication, Marc Vorhees, Cobe Laboratories, Inc.). The blood $CO_2$ removal is especially large during warming and normothermia after maintaining temperature corrected $37^\circ C$, normal $p_{CO_2}$'s during hypothermia and increasing blood $CO_2$ content. Figure 3 presents the relationship between the percent difference in $p_{ex}CO_2$ and $p_{CO_2}$, $T_{Bo}$ 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.
FIGURE 3. The percent disagreement in the mass spectrometer pCO₂, T_Bo = p_aCO₂ versus the V̇_CO₂ for 49 blood gas samplings during all phases of hypothermic total heart lung bypass employing the Optiflo II blood oxygenator.

with the gas, the p_aCO₂ rarely comes to equilibrium with the pCO₂, T_Bo in the Optiflo II blood oxygenator. The blood pCO₂ predicting power of the p_aCO₂ is greatest during hypothermia and situations of low CO₂ exchange. The perfusionist can adapt to the discrepancies and use the trend data in the p_aCO₂ to control the pCO₂, T_NP.

The availability of the mass spectrometer and the ability to accurately calculate V̇_CO₂ with computer assistance allows expanded research capabilities in the clinical setting. Knowledge of the V̇_CO₂ allows confirmation of the clinical diagnosis of respiratory acidosis (decreased V̇_CO₂, 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 V̇_O₂, V̇_CO₂, and R.Q. in a study of pediatric perfusion cases. Abbott documented CO₂ addition to the blood (negative respiratory quotient) during rapid cooling and advocated the use of 10% CO₂ in the ventilating gas, and maintenance of an R.Q. \(= \cdot 8\) during warming of deep hypothermia patients to optimize perfusion adequacy.

The V̇_CO₂ and respiratory quotient are not only a function of the oxygen transfer, venous blood CO₂ content, oxygenator gas-to-blood flow ratio, and percent CO₂ in the ventilating gas, but the ability of the operator to maintain minimal fluctuations in arterial blood pCO₂.

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Conclusions

1. The p_aCO₂ is a fair predictor of the arterial blood pCO₂ temperature corrected to the T_Bo and a poor predictor of the pCO₂ corrected to the T_NP during all phases of hypothermic bypass.

2. The percent disagreement in the p_aCO₂ and the pCO₂, T_Bo is a function of the V̇_CO₂. The p_aCO₂ is in equilibrium with the pCO₂, T_NP when V̇_CO₂ is minute.

3. The use of the p_aCO₂ to calculate V̇_CO₂ 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 V̇_CO₂ and respiratory quotient.

Bibliography