Membrane Oxygenator Exhaust Capnography for Continuously Estimating Arterial Carbon Dioxide Tension During Cardiopulmonary Bypass

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Abstract: Typically, the standard practice for measuring the arterial blood carbon dioxide tension (PaCO2) during cardiopulmonary bypass (CPB) is to take intermittent blood samples for analysis by a bench blood gas analyzer. Continuous inline blood gas monitors are available but are expensive. A potential solution is the capnograph, which was evaluated by determining how accurately the carbon dioxide tension in the oxygenator exhaust gases (PECO2) predicts PaCO2. A standard capnograph monitoring line was attached to the exhaust port of the membrane oxygenator. During CPB, the capnograph reading and arterial blood temperature were recorded at the same time as routine arterial blood gases were taken. One hundred fifty-seven blood samples were collected from 78 patients. A good correlation was found between the PECO2 and the temperature corrected PaCO2 (r² = 0.833, P < .001). There was also a reasonable degree of agreement between the PECO2 and the temperature corrected PaCO2 during all phases of CPB: accuracy (bias or mean difference between PaCO2 and PECO2) of −1.2 mmHg; precision (95% limits of agreement) of ± 4.7 mmHg. These results suggest that oxygenator exhaust capnography may be a simple and inexpensive adjunct to the bench blood gas analyzer in continuously estimating PaCO2 of a clinically useful degree of accuracy during CPB.

Keywords: oxygenator exhaust capnography, carbon dioxide tension, cardiopulmonary bypass.

During cardiopulmonary bypass (CPB), intermittent blood sampling using a bench blood gas analyzer is the standard procedure for determining arterial blood carbon dioxide partial pressures (PaCO₂). Typically, only several samples are taken during CPB. Therefore, prolonged periods of CPB can occur without blood carbon dioxide monitoring despite changes in patient status. Ideally, continuous monitoring and control of blood gas CO₂ is warranted as an abnormal PaCO₂ during CPB is implicated in patient morbidity (1).

Continuous inline blood gas monitors are available but are perceived to be expensive to use (2). A potentially economical alternative is the capnograph, which estimates the PaCO₂ in the ventilated patient but remains unused during CPB.

Previous studies have investigated the use of the capnograph in analyzing the carbon dioxide tension in the exhaust gases emitted by the oxygenator (PeCO₂). A reasonable relationship was seen between the PeCO₂ and the PaCO₂ when measured on membrane oxygenators and corrected for arterial blood temperature (3–6).

This study aims to validate further the use of the capnograph in patients on CPB by determining how accurately the PeCO₂ measured from the gas outlet port of a membrane oxygenator (Affinity NT, Medtronic, Australia) predicts PaCO₂.

MATERIALS AND METHOD

During cardiopulmonary bypass (CPB), intermittent blood sampling using a bench blood gas analyzer is the standard procedure for determining arterial blood carbon dioxide partial pressures (PaCO₂). Typically, only several samples are taken during CPB. Therefore, prolonged periods of CPB can occur without blood carbon dioxide monitoring despite changes in patient status. Ideally, continuous monitoring and control of blood gas CO₂ is warranted as an abnormal PaCO₂ during CPB is implicated in patient morbidity (1).

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MATERIALS AND METHOD

In a single blinded, prospective observational study, 78 nonsequential adult patients undergoing cardiac surgery (coronary artery bypass, valve, and aortic surgery) using CPB were studied. All surgeons and perfusionists were included. With the approval of the hospital ethics committee, informed consent was not required as no change was made in the patients’ clinical management.

After premedication with lorazepam, morphine, and
metoclopramide, the patient was induced with fentanyl, pancuronium, and midazolam. Ventilation with 100% oxygen was adjusted to an end tidal carbon dioxide (ETCO2) of 30–35 mmHg. Isoflurane or sevoflurane was used for anesthetic maintenance. After heparinization to achieve a target activated clotting time of at least 400 seconds, CPB commenced.

Nonpulsatile CPB was performed using a membrane oxygenator (Affinity NT, Medtronic, Australasia) and either a roller or centrifugal pump with a 25 μm filter. The circuit was primed with 2000 mL of Hartman’s solution and 10,000 units heparin. One percent isoflurane and midazolam were used for anesthetic maintenance during CPB. The pump flow rate commenced at a rate of 2.4 L/min/m2 and was subsequently adjusted to yield a mixed venous saturation of at least 70%. Arterial pressures were kept between 50 to 70 mmHg, and the minimum systemic temperatures ranged from 28 to 34°C. The PaCO2 and acid base were controlled by using the alpha-stat strategy. The fraction of inspired oxygen entering the oxygenator ranged from 0.6 to 1.0.

A 10-cm piece of ¼ inch PVC tubing (incorporating a Y-piece to prevent accidental overpressurization of the oxygenator and a luer lock connector for capnograph attachment) connected the exhaust port of the oxygenator with a 6-m length of corrugated gas scavenging tubing that was attached to the waste gas scavenging system of the anesthetic machine. A standard 3-m capnograph monitoring tubing connected the capnograph to the luer lock.

The two sidestream infrared capnograph instruments (Datex AS/3 Airway Module G-AiO, Datex Instrumentation Oy, Helsinki, Finland) sampled at a rate of 200 mL/min resulting in a gas sampling delay of less than 3 seconds. Both were calibrated according to the manufacturer instructions during the regular theater quality control schedule. The bench top blood gas analyzer (Ciba 288, Ciba Corning Diagnostic Corp, Medfield, MA) used a Severinghaus electrode and was automatically calibrated to two points every 2 hours and one point every half hour. Blood gas quality controls were checked daily.

Upon initiation of CPB, the capnograph was switched from monitoring the ETCO2 in the patient ventilator circuit to monitoring the oxygenator exhaust gases. The timing of the blood gas sampling was dictated by the perfusionist’s routine practice with most cases generating two to three specimens. Samples were taken only after at least 60 seconds had elapsed after any changes in ventilation, pump flow, or fluid administration and could be confirmed by a horizontal capnograph trace. Any room air contamination of the oxygenator exhaust gases was detected by a rapidly fluctuating capnographic baseline; these values being rejected. Immediately upon aspirating a blood specimen for blood gas analysis, the perfusionist would record the arterial blood temperature, pump and gas flows. Because the perfusionist was blinded to the digital capnogram reading, the person analyzing the blood gas would note this number when the specimen was drawn. The sample was immediately analyzed at 37°C and was subsequently corrected to the arterial blood temperature as measured from the oxygenator blood outlet using the integrated thermister (see Appendix).

The phase of “active cooling” was induced by setting the heater–cooler unit (HCU) water temperature to at least 5°C lower than the patient’s nasopharangeal (NP) temperature. Arterial blood temperatures were kept between 50 to 70 mmHg, and the minimum systemic temperatures ranged from 28 to 34°C. The phase of “stable hypothermia” was seen when the HCU temperature approximated the NP temperature during hypothermia. “Active rewarming” of the hypothermic patient was undertaken by setting the HCU water temperature to between 38 to 40°C with reductions in this setting as the patient’s NP temperature approached normothermia (37°C). Once the patient was normothermic, and the HCU was set to about 37°C, “stable normothermia” ensued.

The strength of the relationship between the P ECO2 and the PaCO2 values was examined by linear regression. The agreement between the two variables was analyzed using Bland and Altman’s method, whereby the relationship between the two variables is presented in terms of accuracy or bias (mean difference between the two variables) and precision or 95% limits of agreement (1.96 times the standard deviation of the difference between the two variables) (7).

RESULTS

One hundred and fifty-seven blood samples were analyzed. Figure 1 displays the relationship between the temperature corrected PaCO2 and the oxygenator P ECO2. The P ECO2 slightly overestimated the PaCO2, but showed a strong positive linear correlation ($r^2 = 0.83, P < .001$).

There was a reasonable degree of agreement between the temperature corrected PaCO2 and the oxygenator P ECO2 during all phases of CPB (Table 1, Figure 2). The accuracy and precision were better during active cooling and stable hypothermia than during rewarming where there was a tendency for P ECO2 to over-read (Figures 3–5). The phase of stable normothermia was not addressed because of inclusion of only nine samples.

The P ECO2 tended to over-read with higher arterial temperatures ($r = -0.48, P < .001$), showed a tendency to over-read with lower oxygenator gas flows ($r = 0.49, P < .001$), while pump flows had no discernable effect on the P ECO2 readings ($r = 0.04, P = ns$) (Figures 6–8).
DISCUSSION

This study further validates the use of the capnograph for predicting the PaCO₂ in patients on CPB by measuring the PECO₂ from the gas outlet port of a membrane oxygenator. Despite the tendency of the capnograph to over-read the PaCO₂ during all stages of CPB by an average of 1.2 mmHg, because the precision was reasonably tight with 95% of samples 4.7 mmHg above or below, it remained clinically useful. Furthermore, because the differences within the levels of agreement are clinically acceptable, the bias should not affect the usability of capnography because we simply need to subtract it from the PECO₂.

Our results with the Affinity NT membrane oxygenator concurs with other studies investigating the use of membrane oxygenator exhaust capnography to estimate temperature corrected PaCO₂, yielding similar degrees of accuracy, precision, and predictability. Carmerlengo and Dearing determined a mean difference of only 0.6 mmHg and a high correlation of 0.97 between blood and exhaust gas pCO₂ for a SciMed oxygenator (3). Aittomäki determined high correlations between blood and exhaust gas pCO₂ ranging from 0.94 to 0.96 for the Medtronic Maxima, Cobe CML, and Dideco Compactflow oxygenators (4), while Lockwood and colleagues calculated a correlation of 0.91 for the Bard HF-570 (5). Weightman and Sheminant presented clinically acceptable mean differences and 95% limits of agreement between blood and capnograph pCO₂ for the Terumo Capiox SX oxygenator (bias 0.9; precision ± 5.0 mmHg) (6). Conversely, O’Leary and colleagues did not find oxygenator exhaust capnography to be useful for estimating PaCO₂ in the Cobe CML (bias 1.4; precision ± 11.6 mmHg); however, they corrected the PaCO₂ to the nasopharageal temperature and not the arterial blood temperature (8).

Of some significance is that these PaCO₂ results are similar in magnitude to those obtained by the continuous in-line CDI 500 blood gas monitoring system (Terumo-Sarns, Ann Arbor, MI) (bias −1.1; precision ± 7.0 mmHg) (9). Furthermore, our study, together with others reporting bias, falls within the target values of ± 5 mmHg established by the US Clinical Laboratory Improvement act of 1988 (CLIA ’88) for blood gas analyzers (10).

Of interest is the reduction in accuracy and precision upon active rewarming. Studies examining the relationship between PECO₂ and PaCO₂ in bubble oxygenators have also yielded a more variable relationship during rewarming. These investigations showed a PaCO₂ exceeding PECO₂, probably because of an incomplete equilibration of the blood and gas phase CO₂ in the less efficient bubble oxygenators during the higher CO₂ production rate associated with rewarming (11,12). However, our study showed a higher PECO₂ than PaCO₂. One explanation is that the in-line arterial temperature probe was under-reading the actual arterial blood temperature during active rewarming; if the correct (higher) blood temperature was available it would have yielded a higher temperature corrected PaCO₂ (13). A higher predicted PaCO₂ than measured may also indicate a closer correspondence between the PECO₂ and the mixed venous PCO₂ (PvCO₂). This phenomenon was noted by previous researchers in the countercurrent designed Medtronic Maxima, Dideco Compactflow, and the Terumo Capiox E; the venous blood entering these membrane oxygenators equilibrating with the gas exiting the devices (4,14). Hypothermia, by decreasing the metabolic production of CO₂, would tend to reduce the PCO₂ gradient between arterial and venous blood thereby reducing the bias with the opposite occurring during rewarming. Lower oxygenator gas flows also increased the bias of PECO₂ exceeding PaCO₂. Higher oxygenator gas flows may dilute the PvCO₂ influenced...
PECO₂ and facilitate the mixing and equilibration of any gas phase gradients resulting in an “improvement” in the bias. Further investigations would require measurements of PvCO₂ to investigate this phenomenon in the “cross-current” designed Medtronic Affinity NT.

To use capnography while on CPB, the perfusionist extrapolates from the PECO₂ to estimate PaCO₂. No further arithmetic correction of the extrapolated PaCO₂ is required if using the “pH-stat” management technique. If using the “alpha-stat” approach, a PCO₂ variation with blood temperature normogram or algorithm is needed to calculate the PaCO₂ at 37°C. Another approach to aid in “alpha-stat” acid base regime is the creation of regression signatures whereby at any given arterial blood temperature, the PECO₂ can be identified to yield normocapnia (15). However, a regression signature should be devel-
oped by the institution for each brand of membrane oxygenator used not only due to the differences in oxygenator design but also in monitoring equipment and techniques. In practice, adjusting oxygenator gas flow to yield a \( \text{PeCO}_2 \) that equals arterial temperature returns alpha stat normocapnia.

A limitation of this study was that both measurement systems were calibrated using different standards, but this is normal in clinical practice and should not deter from its application. The issue of room air contamination of the oxygenator exhaust gases was not quantified; however, it occurred more frequently with the low oxygenator fresh gas flow rates associated with small and hypothermic patients. Room air contamination of the exhaust gases could be further addressed by the manufacturers in developing a modified oxygenator that rationalizes the extensive openings in the housing and incorporates an optimally positioned capnography monitoring port. Such modifications

![Figure 4](image_url)  
**Figure 4.** Agreement between capnograph \( \text{PeCO}_2 \) and blood \( \text{PaCO}_2 \) during stable hypothermia.

![Figure 5](image_url)  
**Figure 5.** Agreement between capnograph \( \text{PeCO}_2 \) and blood \( \text{PaCO}_2 \) during active rewarming.
could provide a real and economical alternative to in-line blood gas monitoring. In addition, monitoring volatile anesthetics and PO2 would broaden the applications of oxygenator exhaust gas monitoring (5).

In summary, our results suggest that oxygenator exhaust capnography may be a simple and inexpensive adjunct to the bench blood gas analyzer in continuously estimating PaCO2 of a clinically useful degree of accuracy during CPB.

REFERENCES


APPENDIX

Formula for temperature correction: PCO2 (art temp) = PCO2 (37°C) × 10 \((0.019(“Art-temp”-37))\)