Oxygenator Exhaust Capnography for Prediction of Arterial Carbon Dioxide Tension During Hypothemic Cardiopulmonary Bypass

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Abstract: Continuous monitoring and control of arterial carbon dioxide tension (P\textsubscript{a}CO\textsubscript{2}) during cardiopulmonary bypass (CPB) is essential. A reliable, accurate, and inexpensive system is not currently available. This study was undertaken to assess whether the continuous monitoring of oxygenator exhaust carbon dioxide tension (PexCO\textsubscript{2}) can be used to reflect P\textsubscript{a}CO\textsubscript{2} during CPB. A total of 33 patients undergoing CPB for cardiac surgery were included in the study. During normothermia (37°C) and stable hypothermia (31°C), the values of PexCO\textsubscript{2} from the oxygenator exhaust outlet were monitored and compared simultaneously with the PaCO\textsubscript{2} values. Regression and agreement analysis were performed between PexCO\textsubscript{2} and temperature corrected-P\textsubscript{a}CO\textsubscript{2} and temperature uncorrected-P\textsubscript{a}CO\textsubscript{2}. At normothermia, a significant correlation was obtained between PexCO\textsubscript{2} and PaCO\textsubscript{2} (r = 0.79; p < 0.05); there was also a strong agreement between PexCO\textsubscript{2} and PaCO\textsubscript{2} with a gradient of 3.4 ± 1.9 mmHg. During stable hypothermia, a significant correlation was obtained between PexCO\textsubscript{2} and the temperature corrected-P\textsubscript{a}CO\textsubscript{2} (r = 0.78; p < 0.05); also, there was a strong agreement between PexCO\textsubscript{2} and temperature corrected-P\textsubscript{a}CO\textsubscript{2} with a gradient of 2.8 ± 2.0 mmHg. During stable hypothermia, a significant correlation was obtained between PexCO\textsubscript{2} and the temperature uncorrected-P\textsubscript{a}CO\textsubscript{2} (r = 0.61; p < 0.05); however, there was a poor agreement between PexCO\textsubscript{2} and the temperature uncorrected-P\textsubscript{a}CO\textsubscript{2} with a gradient of 13.2 ± 3.8 mmHg. Oxygenator exhaust capnography could be used as a mean for continuously monitoring P\textsubscript{a}CO\textsubscript{2} during normothermic phase of cardiopulmonary bypass as well as the temperature-corrected P\textsubscript{a}CO\textsubscript{2} during the stable hypothermic phase of CPB. Keywords: exhaust capnography, hypothermia, cardiopulmonary bypass.

The continuous monitoring of the membrane oxygenator exhaust CO\textsubscript{2} (PexCO\textsubscript{2}) has been suggested as a substitute for continuous monitoring of P\textsubscript{a}CO\textsubscript{2}. However, conflicting results have been reported (1,2). These conflicting results could be attributed to the induction of hypothermia and the rewarming phase of cardiopulmonary bypass (CPB) (3,4), the type of oxygenator (2), the technique and site for CO\textsubscript{2} monitoring (5,6), or the site of temperature monitoring (2). This study was undertaken to determine the validity of exhaust CO\textsubscript{2} for the continuous prediction of temperature-corrected versus uncorrected P\textsubscript{a}CO\textsubscript{2} during CPB using a membrane oxygenator.

MATERIALS AND METHODS

The study was approved by the Institutional Review Board at the American University of Beirut. Because the study involved no alterations in routine patient management, the Board waived the need for a prior informed consent.

Patients of both genders (18 men/15 women, mean age: 64.2 ± 7.1 years, length of CPB: 50 ± 5 min) and who were undergoing elective cardiac surgery for coronary artery bypass grafting with hemodilution hypothermic CPB using the alpha-stat strategy for carbon dioxide homeostasis and using a membrane oxygenator were included in the study. Anesthesia was induced with midazolam (2 mg), thiopentone (3 mg/kg), xylazine (2 mg/kg), sufentanil (25 μg), and vecuronium (0.6 mg/kg), to be followed by tracheal intubation and positive pressure ventilation. Anesthesia was maintained with isoflurane 1–2% in oxygen supplemented with cisatracurium (0.15 mg/kg/h), sufentanil (1 μg/kg/h), and midazolam (0.1 μg/kg/min). After heparinization (heparin of 4 mg/kg to achieve an ACT > 450 s), CPB was started using a roller pump (Sarns 8000, 3M Health Group, Ann Arbor, MI) and a Trillium membrane oxygenator (Medtronic, Minneapolis, MN).

Nonpulsatile pump flow at 2.4 L/m\textsuperscript{2}/min and a similar oxygen flow were maintained throughout bypass. On bypass, hypothermia was induced by actively cooling pa-
tients down to 31°C and cold crystalloid cardioplegia was used for myocardial protection. The blood temperature was continuously monitored with an in-line temperature probe in the CPB circuit. Hypothermia or normothermia was defined as stable after 5–15 min of cooling or rewarming with no more than ±1°C fluctuation in temperature. The oxygenator exhaust outlet of the CPB circuit was connected to a capnograph sensor (Novametrix, Willinford, CT) for continuous monitoring of exhaust CO₂ (PexCO₂). A 100-cm length of anesthetic tubing was attached behind the capnograph adaptor to act as a reservoir for the exhaust gases and thus prevent entrainment of room air. The same capnograph was used on all patients throughout the whole study. Before each use, it was calibrated and checked using a gas containing a known concentration of carbon dioxide (5% balanced with nitrogen) according to the manufacturer’s instructions and was allowed to warm up for at least 1–2 h before use. All measurements were made during full flow bypass without ejecting heart and mechanical ventilation (i.e., during aortic cross clamp time) and no measurements were made during the cooling and rewarming phases of the CPB. The readings of the capnograph (PexCO₂) were noted simultaneously with and rewarming phases of the CPB. The time was defined as stable after 5 min. The same capnograph was used on all patients throughout the whole study. Before each use, it was calibrated and checked using a gas containing a known concentration of carbon dioxide (5% balanced with nitrogen) according to the manufacturer’s instructions and was allowed to warm up for at least 1–2 h before use. All measurements were made during full flow bypass without ejecting heart and mechanical ventilation (i.e., during aortic cross clamp time) and no measurements were made during the cooling and rewarming phases of the CPB. The readings of the capnograph (PexCO₂) were noted simultaneously with sampling of the arterial blood samples and monitoring of the blood temperature. Arterial blood gas samples collected from the oxygenator outlet and obtained on all patients at temperatures of 31°C and 37°C were immediately subjected to duplicate measurement by a bench blood gas analyzer (ABL700, Radiometer, Copenhagen, Denmark) and both temperature-corrected PₐCO₂ and temperature-uncorrected PₐCO₂ values were determined. The blood gas analyzer was calibrated to two-point calibration immediately before starting CPB and to one-point calibration every 30 min during CPB.

**Statistical Analysis**

The means and standard deviations of exhaust PCO₂ (PexCO₂), temperature-corrected PₐCO₂, and temperature-uncorrected PₐCO₂ were obtained at 31°C and 37°C. Also, the means and standard deviations of the gradients between PexCO₂ and PₐCO₂ at 37°C and between PexCO₂ and temperature-corrected and temperature-uncorrected PₐCO₂ at 31°C were obtained. These mean values were compared with the Student’s t test and the analysis of variance. The degrees of correlation between PexCO₂ and the temperature-corrected PₐCO₂ and temperature-uncorrected PₐCO₂ were determined by regression analysis. Also, the degree of agreement between PexCO₂ and the temperature-corrected PₐCO₂ and temperature-uncorrected PₐCO₂ were determined with the Bland-Altman analysis (7). Statistical significance was considered at p < 0.05.

A power analysis using a Type I error of 5% and Type II error of 80% indicated that for a non-significant bias between PₐCO₂ and PexCO₂ a sample size of at least 30 patients to be included in the study.

**RESULTS**

The mean ± standard deviation of PexCO₂ and PₐCO₂ during normothermia (37°C) as well as the temperature-corrected and temperature-uncorrected PₐCO₂ during stable hypothermia (31°C) are presented in Table 1.

<table>
<thead>
<tr>
<th>Temperature</th>
<th>PexCO₂</th>
<th>PₐCO₂</th>
<th>Temperature-Corrected PₐCO₂</th>
<th>Temperature-Uncorrected PₐCO₂</th>
</tr>
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<tbody>
<tr>
<td>37°C</td>
<td>30</td>
<td>33.9 ± 3.1</td>
<td>37.1 ± 2.9*</td>
<td>N/A</td>
</tr>
<tr>
<td>31°C</td>
<td>33</td>
<td>26.3 ± 2.9</td>
<td>N/A</td>
<td>29.0 ± 3.0*</td>
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<td></td>
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<td>39.4 ± 4.8*</td>
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*p < 0.05 vs. PexCO₂.

N/A, not applicable.

**DISCUSSION**

Close monitoring and control of PₐCO₂ are important during all phases of CPB as carbon dioxide is the main determinant of acid-base balance and the cerebral blood flow (3.8). This supports the use of continuous monitoring of arterial PₐCO₂ during normothermic and hypothermic CPB. However, in-line continuous monitoring of PₐCO₂ is expensive and prone to errors particularly when used for long duration (5.6).

Our results showed that during CPB using a membrane oxygenator, there is a strong agreement between arterial and exhaust carbon dioxide tensions at normothermia. During hypothermia a similar strong agreement was observed between temperature-corrected arterial carbon dioxide and exhaust carbon dioxide tensions; however, a
Poor agreement was observed between temperature-uncorrected arterial carbon dioxide and exhaust carbon dioxide tensions. Our results are in agreement with those reported by Potger et al. (9), which suggested that oxygenator exhaust capnography might be a simple and inexpensive adjunct to the bench blood gas analyzer in continuously estimating PaCO2 during CPB. The current findings together with the independent correlation by Potger et al. (9) suggest that continuous monitoring of PexCO2 can be used as a surrogate marker of the temperature-corrected PaCO2 during CPB in addition to the intermittent correlative blood gas analysis.

Our data showed that exhaust capnography can parallel closely the PaCO2 during normothermia and the temperature-corrected PaCO2 during stable hypothermia. During normothermia, the average gradients between exhaust carbon dioxide tension and the arterial carbon dioxide tension were 3.4 ± 1.9 mmHg. During stable hypothermia, a similar gradient of 2.8 ± 2.0 mmHg was observed between the exhaust carbon dioxide tension and the temperature-corrected PaCO2. These gradients are significantly smaller than the high gradient of 13.2 ± 3.8 mmHg obtained between exhaust carbon dioxide tension and temperature-uncorrected arterial carbon dioxide tension during stable hypothermia.

Zia et al. (1) reported that the oxygenator exhaust capnography during CPB in addition to the intermittent correlative blood gas analysis.

Figure 1. Correlation between the partial pressure of arterial carbon dioxide (PaCO2) and partial pressure of exhaust carbon dioxide (PexCO2) at normothermia. r = 0.79, p < 0.05.

Figure 2. Difference in the partial pressure of arterial carbon dioxide (PaCO2) and partial pressure of exhaust carbon dioxide (PexCO2) against the mean of the two measurements at normothermia.

Table 2. Mean and SD of the difference (mmHg) between PexCO2 and PaCO2 at normothermia and during hypothermia when corrected and not corrected to blood temperature.

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<tr>
<th></th>
<th>n</th>
<th>Mean</th>
<th>SD</th>
</tr>
</thead>
<tbody>
<tr>
<td>Normothermia (37°C)</td>
<td>30</td>
<td>3.8</td>
<td>3.7</td>
</tr>
<tr>
<td>Stable hypothermia (31°C)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Temperature-corrected</td>
<td>33</td>
<td>2.3</td>
<td>2.6</td>
</tr>
<tr>
<td>Temperature-uncorrected</td>
<td>33</td>
<td>13.2*</td>
<td>3.8</td>
</tr>
</tbody>
</table>

*p < 0.05 vs. Normothermia.

Figure 3. Correlation between the partial pressure of temperature-corrected arterial carbon dioxide (Temperature corrected-PaCO2) and partial pressure of exhaust carbon dioxide (PexCO2) during stable hypothermia. r = 0.78, p < 0.05.

Figure 4. Difference in the partial pressure of temperature-corrected arterial carbon dioxide (Temperature corrected-PaCO2) and partial pressure of exhaust carbon dioxide (PexCO2) against the mean of the two measurements during stable hypothermia.
nography could be used as an inexpensive means of continuously monitoring $P_aCO_2$ during cooling and stable hypothermic phases of CPB; however, they reported a gradient of $11.8 \pm 2.7$ mmHg between the exhaust $CO_2$ tension and the temperature-corrected $P_aCO_2$ during stable hypothermia. Their results are different from our current study, which showed a smaller gradient of $2.8 \pm 2.0$ mmHg during stable hypothermia. This could be attributed to the fact that Zia et al. (1) used a bubble oxygenator while in the current study a membrane oxygenator was used. There are important differences between bubble and membrane oxygenators with respect to transfer of carbon dioxide (10,11). It has been previously reported that bubble oxygenators are relatively inefficient at removing carbon dioxide (10).

O’Leary et al. (2) reported that measurement of carbon dioxide partial pressure in exhaust gases from a membrane oxygenator during CPB was not a useful method for estimating $P_aCO_2$. In their study, O’Leary et al. used the nasopharyngeal temperature during hypothermia to correct the $P_aCO_2$ values; this may not be the most appropriate site for correcting the $P_aCO_2$. Using the temperature of the blood at the point that carbon dioxide is sampled rather than the nasopharyngeal temperature represents a more logical option for temperature correction of $P_aCO_2$. In the current study the blood temperature was used to obtain the temperature-corrected $P_aCO_2$ during hypothermia.

Our findings suggest that during CPB using the membrane oxygenator, exhaust capnography may reflect continuously the $P_aCO_2$ during normothermia, as well as the temperature-corrected $P_aCO_2$ during stable hypothermia. However, most centers nowadays are using during hypothermic CPB the alpha-stat and not the pH-stat strategy for acid-base management, and hence depend on the temperature-uncorrected rather than the temperature-corrected $P_aCO_2$ values (12–14).

REFERENCES


