Original Article

The Temperature-Corrected Versus the Uncorrected PO$_2$ During Hypothermic Cardiopulmonary Bypass—Correlation with Oxyhemoglobin Saturation

Anis S. Baraka, MD, Musa K. Muallem, MD, Maurice A. Baroody, MD, Sania T. Haroun, MD, Abla A. Sibai, MS

Department of Anesthesiology and Department of Biostatistics and Epidemiology, American University of Beirut, Beirut, Lebanon.

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Abstract

In 8 adult patients undergoing cardiopulmonary bypass, the perfusion flow was altered from 2.4 to 1.8 and 1.2 L/min/m$^2$ during hypothermia (26.5 ± 2.3°C). Following rewarming of the patients to 37°C, the perfusion flow was also altered from 2.4 to 1.8 and 1.2 L/min/m$^2$. The resulting mixed venous oxyhemoglobin saturation (S$v_2$) was measured in vivo by in-line oximetry at the actual body temperature, and was correlated with the corresponding temperature-corrected and uncorrected mixed venous oxygen tension (P$v_2$) as measured in the lab at 37°C. Venous blood at the entrance of the oxygenator was used for continuous in-line oximetry, blood gas sampling, and for measuring body temperature.

Correlation of S$v_2$ at the different perfusion flows with the corresponding temperature-corrected P$v_2$ during hypothermia is shifted to the left of the correlation following rewarming of the patients to 37°C. In contrast, correlation of S$v_2$ with the uncorrected P$v_2$ is not significantly different from the normothermic relationship. Also, the uncorrected P$v_2$ and S$v_2$ during hypothermia at a perfusion flow of 1.2 L/min/m$^2$ were not significantly different from that achieved during normothermia at a flow of 2.4 L/min/m$^2$. Thus, maintenance of the temperature-uncorrected P$v_2$ during hypothermia at the normal normothermic value facilitates clinical interpretation and reflects a normal oxyhemoglobin saturation, irrespective of changing the body temperature.

Introduction

Hypothermia is widely practiced during cardiopulmonary bypass (CPB) (1). However, use of temperature-corrected or uncorrected PO$_2$ during hypothermia is controversial (2). Hypothermia shifts the oxyhemoglobin dissociation curve to the left (3) when oxyhemoglobin saturation is correlated with the temperature-corrected PO$_2$. However, correlation between the temperature-uncorrected PO$_2$ and the in vivo oxyhemoglobin saturation has not been previously reported.

The present report investigates during hypothermic CPB, the influence of different perfusion flow rates on the mixed venous oxygen tension (P$v_2$), and correlates the resulting temperature-corrected versus the uncorrected P$v_2$ with the corresponding mixed venous oxyhemoglobin saturation (S$v_2$) as measured in vivo by in-line oximetry. The S$v_2$/P$v_2$ correlations during hypothermia are compared to that achieved following rewarming of the patients to 37°C.

Materials and Methods

The investigation was performed in 8 patients, aged 25 to 65 years and weighing 55 to 75 kg, who underwent coronary artery bypass grafting or valve replacement during CPB. The investigation was approved by the Institution Research

Address correspondence to:
Anis S. Baraka, MD
Professor and Chairman of Anesthesiology
American University of Beirut
Beirut, Lebanon

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Committee, and informed consent was obtained.

The patients were premedicated with 10 mg of morphine sulphate, 25 mg of promethazine hydrochloride, and 0.4 mg of scopolamine hydrobromide, intramuscularly. Anesthesia was induced with 0.1 mg/kg of midazolam hydrochloride, 40 ug/kg of fentanyl citrate, and 0.1 mg of pancuronium bromide. Following orotracheal intubation, ventilation was controlled with 100% oxygen, without any inhalation anesthetic supplementation. Patients were monitored with an electrocardiogram (V5), a radial artery catheter, and a pulmonary artery catheter.

Ringer’s lactate solution, 1,500 ml, was used to prime the bubble oxygenator and circuit. During bypass, the hematocrit level was lowered from a mean preoperative value of 41.2% ± 2.9% to 26.3% ± 3.2%. The patients were perfused by a roller pump at a flow rate of 2.4 L/min/m². During hypothermia, ventilation was controlled with any inhalation anesthetic. The perfusion flow was then decreased to 1.8 L/min/m² and 1.2 L/min/m² The resulting SvO₂ and PVₐO₂ were recorded approximately 5 minutes after each perfusion flow change.

Continuous in-line oximetry of the venous saturation was achieved by the Bentley Oxy-Stat Meter (5,6). The site of body temperature and SvO₂ monitoring, as well as mixed venous gas sampling, was the venous blood at the entrance to the oxygenator (7). PVₐO₂ was measured by ABL300 Radiometer with electrodes kept constant at 37°C. The temperature-uncorrected PVₐO₂ values measured at 37°C were then automatically corrected by the Radiometer according to body temperature.

During CPB, body temperature was decreased, and the heart was arrested after aortic cross-clamping with a cardioplegic solution (K30 mEq/L at 4°C). After approximately 10 to 20 minutes of nonpulsatile CPB, a steady mean arterial pressure was achieved and the mean body temperature was stabilized at 26.5 ± 2.3°C. When surgery was completed, the aortic cross-clamp was released, and the patient was warmed to a body temperature of 37°C.

The study of the influence of different perfusion flows on SvO₂ and PVₐO₂ was completed as described below:

### Table 1: Perfusion Flow

<table>
<thead>
<tr>
<th>Flow (L/min/m²)</th>
<th>Hypothemia (26.5 ± 2.3°C)</th>
<th>Normothermia (37°C)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SvO₂ %</td>
<td>PVₐO₂ Uncorrected</td>
</tr>
<tr>
<td>2.4 L/min/m²</td>
<td>90 ± 4.8</td>
<td>73 ±18 mmHg</td>
</tr>
<tr>
<td>1.8 L/min/m²</td>
<td>83 ± 6.4</td>
<td>57 ± 11 mmHg</td>
</tr>
<tr>
<td>1.2 L/min/m²</td>
<td>73 ± 8.1</td>
<td>44 ± 8 mmHg</td>
</tr>
</tbody>
</table>

1. During hypothermia, SvO₂ and PVₐO₂ were monitored simultaneously when the perfusion flow was maintained at 2.4 L/min/m². The perfusion flow was then decreased to 1.8 L/min/m² and 1.2 L/min/m². The resulting SvO₂ and PVₐO₂ were recorded approximately 5 minutes after each perfusion flow change.

2. The original perfusion flow of 2.4 L/min/m² was then restored and maintained throughout the period of rewarming. When the venous temperature stabilized at 37°C for 10 to 20 minutes, the effect of the different perfusion flows (2.4, 1.8, 1.2 L/min/m²) on SvO₂ and PVₐO₂ were monitored, similar to step 1.

All data are presented as mean ± SD. Analysis of variance (ANOVA) was conducted to check for the effect of different perfusion flows on the mean SvO₂ and PVₐO₂. Since the oxyhemoglobin dissociation curve is sigmoid, correlation between PVₐO₂ and the corresponding SvO₂ was conducted by regression analysis between log PVₐO₂ and the probit of SvO₂. The log-probit transformation straightens the sigmoid curve (8), and facilitates the determination of the P50 values (the partial pressure of oxygen necessary to achieve 50 percent hemoglobin saturation). The Cochran’s method of analysis was used to compare the slopes and elevation of the regression lines. Significant results were identified when the F-ratio deviated significantly from one, the null hypothesis of no different. P<0.05 was considered significant.

### Results

As shown in Table 1, the mean SvO₂, as well as the temperature-corrected and uncorrected PVₐO₂ during hypothermia and after rewarming to 37°C, were significantly decreased when the perfusion flow was decreased from 2.4 L/min/m² to 1.8 L/min/m² and 1.2 L/min/m². Decreasing the perfusion flow...
During hypothermia for corrected $P_VO_2$ which was achieved following rewarming at a flow of 2.4 L/min/m$^2$. Figure 1 shows the individual $SvO_2$ values at the different perfusion flows during hypothermia and after rewarming to 37°C.

Correlation of the log $P_VO_2$ at the different perfusion flows, with the probit of the corresponding $SvO_2$ during hypothermia and following rewarming is depicted in Fig 2. During hypothermia, correlation of $SvO_2$ with the temperature-corrected $P_VO_2$ is shifted parallel and to the left of the correlation following rewarming; the P50 is estimated as 13.5 mmHg which is significantly lower than the P50 of 24 mmHg estimated following rewarming. In contrast, correlation of $SvO_2$ with the uncorrected $P_VO_2$, as well as the estimated P50 of 25 mmHg, are not significantly different from that achieved following rewarming.

**Discussion**

During hypothermia, the use of temperature-uncorrected $P_O_2$ which is measured by electrodes at 37°C, or the $P_O_2$ corrected to the actual body temperature remains a controversial issue (2). This may be particularly important when the blood is desaturated, since $P_O_2$ falls 7.2% for each degree (Centigrade) of temperature drop, while it may only fall at 1.3 percent for each degree (Centigrade) of temperature drop in the fully saturated blood.

Our report shows during cardiopulmonary bypass that correlation of the temperature-corrected $P_VO_2$ achieved during hypothermia at the different perfusion flows with the corresponding $SvO_2$ measured in vivo at the actual body temperature is shifted to the left of the correlation achieved following rewarming of the patient to 37°C. In contrast, correlation of $SvO_2$ with the temperature-uncorrected $P_VO_2$ as well as the estimated P50 are not significantly different from that achieved following rewarming of the patients to 37°C. When blood sampled from a hypothermic patient is analyzed by electrodes at 37°C, the solubility coefficient for oxygen decreases resulting in a proportional increase of $P_O_2$ (10). However, the observed change of $P_O_2$ with temperature is just enough to maintain constant oxyhemoglobin saturation (11). Our results confirm the findings of previous work (12) which indicates that the best correlation to direct measurement of hemoglobin bound oxygen during hypothermia occurs when using uncorrected $P_O_2$ applied to a standard oxygen.
dissociation curve at 37°C.

The present report also shows, during moderate hypothermic CPB (26.5 ± 2.3°C), that decreasing the perfusion flow to 1.2 L/min/m² resulted in SvO₂ and uncorrected PvO₂ which were not significantly different from the values achieved by a flow of 2.4 L/min/m² following rewarming of the patients to 37°C. The whole-body oxygen consumption is decreased by about 50% for every 10°C decrease in body temperature (13,14). Thus, adjusting the perfusion flow during hypothermic CPB according to the body temperature (15-17) will keep the oxygen extraction ratio unchanged, and hence will maintain the SvO₂ and the uncorrected PvO₂ at the normal normothermic values.

The concept of maintaining the uncorrected PvO₂ and SvO₂ during hypothermia at the normal normothermic values appears to be natural and physiologically sound in both man and animals. Following weaning from CPB, it has been shown in moderately hypothermic patients that the mixed venous oxygen saturation and the oxygen extraction rates remain unchanged during the rewarming phase since the changes of cardiac output are associated with similar changes of oxygen consumption (18). Also, observations during hibernation have shown that SvO₂ of animals in hibernation is not essentially different from the active normothermic animals (19). The concept simulates the alpha-stat strategy of carbon dioxide management which shows that maintenance of the uncorrected PCO₂ at the normothermic value will maintain a constant carbon dioxide content at all body temperatures (20).

The use of temperature-uncorrected PO₂ during hypothermia simplifies clinical interpretation because of our familiarity with the normothermic oxyhemoglobin dissociation curve. Thus, maintenance of the temperature-uncorrected PO₂ during hypothermic CPB at the normal normothermic values will be associated with a normal oxyhemoglobin saturation. In contrast, the temperature-corrected PO₂ values and the associated shift of the oxyhemoglobin dissociation curve show wide variations according to the body temperature of the hypothermic patient, if indeed a single "body temperature" exists. When the body temperatures are changing rapidly by perfusion cooling, large thermal differences occur between different organs of the core as well as between the core and shell (21).

In conclusion, our report shows during moderate hypothermic CPB that correlation of the in vivo SvO₂ with the temperature-uncorrected PvO₂ is not significantly different from the correlation achieved following rewarming of the patient to 37°C. Thus, maintenance of the temperature-uncorrected PO₂ during hypothermia at the normal normothermic value facilitates clinical interpretations and ensures a normal oxyhemoglobin saturation, irrespective of changing the body temperature.

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