Effect of Hypothermia on Arterial Versus Venous Blood Gases During Cardiopulmonary Bypass in Man

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Abstract

The effect of moderate hypothermia on arterial versus venous blood gases was investigated in 11 patients during cardiopulmonary bypass. The pump and oxygen flows were maintained at a constant flow of 2.4 L/m²/min throughout bypass. After going on bypass, the central venous blood temperature was dropped to 27.8 ± 0.6°C, and after surgery was completed the patients were rewarmed to 37.0°C. Arterial and venous blood gases were sampled simultaneously from the arterial outlet and the venous inlet lines of the oxygenator every two degrees of temperature change until 37°C was reached. The effect of temperature changes on pCO_2 simulated the alpha-stat strategy of acid-base regulation; the temperature-corrected arterial and venous pCO_2 varied directly and the corrected pH varied inversely with body temperature, while the uncorrected pCO_2 and pH did not show a significant change with changes of body temperature. The arterial pO_2, whether corrected or uncorrected, as well as the venous oxygen saturation and the uncorrected venous pO_2, significantly increased with each increment decrease of body temperature. However, the corrected venous pO_2 did not significantly change with changes of body temperature. The report suggests that maintenance of constant perfusion and oxygen flows at normothermic levels during hypothermic cardiopulmonay bypass will maintain a constant carbon dioxide content and a constant corrected venous pO_2.

Introduction

Hypothermia is widely practiced during cardiopulmonary bypass (CPB) in man. Baraka et al have previously shown that changes of the perfusion[1] and the oxygen flow[2] during CPB can affect oxygenation and carbon dioxide elimination during hypothermia and following rewarming. The present report investigates the effect of changing body temperature on the temperature-corrected and uncorrected arterial versus venous blood gases, while maintaining both the pump flow and the oxygen flow constant throughout CPB.

Method

Investigation was carried out on 11 patients undergoing coronary artery bypass grafting or valve replacement during CPB. Their age ranged between 30-63 yrs, and their weight ranged between 50-80 kg. The investigation was approved by the Institution Research Committee and informed consent was obtained from each patient.

The patients were premedicated with 10 mg morphine, 25 mg promethazine and 0.4 mg scopolamine IM. Anesthesia was induced with 0.1-0.2 mg/kg midazolam, 40 µg/kg fentanyl and a mixture of 0.25 mg/kg alcuronium and 0.1 mg/kg pancuronium. Following tracheal intubation, ventilation was controlled with 100 percent oxygen without any inhalation anesthetic supplementation. All patients were monitored by EKG (V_5), radial artery cannula and PA catheter. A Bentley-10B adult bubble oxygenator[a] was primed by 1500 ml lactated Ringer's solution. The patient was perfused by a roller pump[b] at a flow of 2.4 L/m²/min, and the oxygenator was bubbled by an equal flow of 100 percent oxygen. No carbon dioxide was added to the oxygen flow during cooling or rewarming. Both the pump and oxygen flows were maintained constant throughout the period of investigation. The mean arterial pressure

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a) American Bentley, Irvine, CA 92714
b) Sarns 5000, Ann Arbor, MI 48710

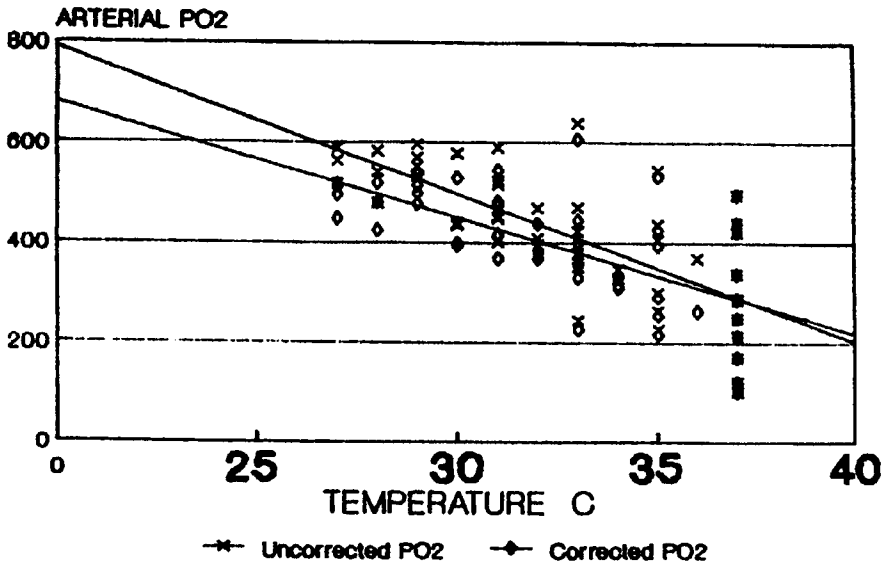


Figure 1

Arterial pO_2 vs. Temperature
Correlation and regression between body temperature (°C) and arterial pO_2 (mmHg) showing a negative linear association between both corrected (r = -0.64, b = -23.1, P < 0.0001) and uncorrected (r = -0.72, b = -29.4, P < 0.0001 pO_2. While the two regression lines meet at higher temperatures, they diverge as the temperature decreases.

during bypass ranged between 50-80 mmHg. The hematocrit was lowered from a prebypass level of 39.6 ± 4.4 percent to 24.6 ± 3.7 percent during CPB. As soon as CPB was instituted, the patients were cooled to a mean venous temperature of 27.8 ± 0.6°C. and the heart was arrested after aortic clamping by cardioplegic solution (K 30mEq/L at 4°C). When the aortic cross clamp was released, the patients were rewarmed to 37°C. Continuous in-line oximetry of the venous oxygen saturation (SVO_2) was achieved by the Bentley Oxy Stat Meter.[a,3] The site for body temperature and SVO_2 measurement was the venous blood at entrance to the pump oxygenator. This is probably the best site during CPB where we can get a truly mixed venous sample at a temperature which most closely mirrors "mean" body temperature.[4,5]

During rewarming, the SVO_2 as well as both the arterial and venous blood gases were monitored every two degrees of temperature change until a central venous blood temperature of 37°C was reached. Arterial and venous samples were taken simultaneously from the arterial outlet and the venous inlet lines of the oxygenator for measuring pO_2, pCO_2, pH and base deficit using an ABL300 Radiometer[c] with electrodes kept constant at 37°C. The temperature-uncorrected values measured at 37°C were then corrected according to body temperature.[6]

Correlation and regression were conducted between body temperature and venous/arterial blood gas parameters. Statistically significant association was identified when the slope of the regression line deviated from zero, the null hypothesis of no linear relationship between the dependent and independent variable. All data are presented as standardized regression coefficient, slope of the regression line and its associated P value. A value of $P < 0.05$ was considered significant.

Results

Arterial Blood

The arterial pO_2, whether temperature-corrected or uncorrected, increased significantly with each increment decrease of body temperature. Correlation between body temperature and pO_2 shows a negative linear association (Figure 1).

The temperature-corrected pCO_2 decreased significantly with each increment decrease of body temperature; correlation between body temperature and corrected pCO_2 shows a positive linear association, while the uncorrected pCO_2 did not show a significant change (Figure 2). In contrast, the corrected arterial pH increased significantly with cooling, while the uncorrected pH did not significantly change (Figure 3). The base deficit was minimal and did not significantly change with changes of body temperature (r = -0.02, b = -0.01, P is NS).

Venous Blood

As shown in Figure 4, the temperature-corrected venous pO_2 (PVO_2) did not show a significant change with changes of body temperature. In contrast, both the uncorrected PVO_2 and the SVO_2 increased with each increment decrease of body temperature.

The effect of temperature changes on venous pCO_2 and pH are similar to its effect on the arterial pCO_2 and pH. The temperature corrected venous pCO_2 varied directly with body temperature, while the uncorrected pCO_2 did not show a significant change (Figure 5). In contrast, the corrected pH increased significantly with

c) Radiometer, Copenhagen, Denmark

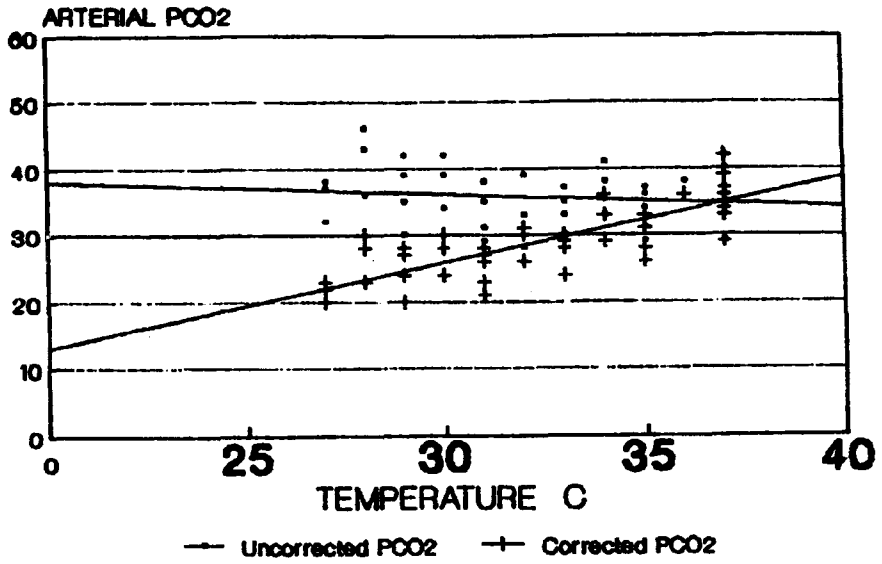


Figure 2

Arterial pCO_2 vs. Temperature
Correlation and regression between body temperature (°C) and uncorrected and corrected arterial pCO_2 (mmHg) showing a positive linear association ($r = .78$, $b = 1.3$, $P < 0.0001$) in the case of corrected pCO_2 and no association ($r = -0.13$, $b = -0.17$, P = NS) in the case of uncorrected pCO_2.

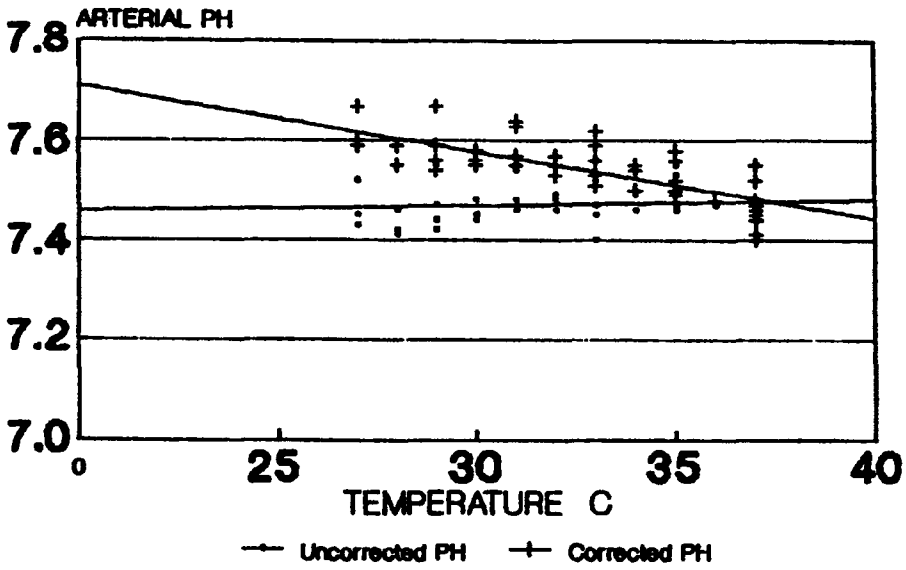


Figure 3

Arterial pH vs. Temperature
Correlation and regression between body temperature (°C) and uncorrected and corrected arterial pH showing a negative linear association ($r = -0.75$, $b = -0.014$, $P < 0.001$) in the case of corrected pH and no association ($r = 0.09$, $b = 0.001$, P = NS) in the case of uncorrected pH.

cooling, while the uncorrected pH did not significantly change (Figure 6). The base deficit was minimal and did not show a significant change with changes of body temperature ($r = 0.19$, $b = 0.07$, P is NS).

Discussion

During CPB, oxygen flow is delivered to the oxygenator to oxygenate the venous return and to provide carbon dioxide elimination. The present report investigates the effect of changing body temperature on the arterial versus venous blood gases during CPB, while maintaining both the pump and oxygen flows constant at 2.4 L/m²/min throughout the period of bypass. No exogenous carbon dioxide was added to the ventilating oxygen flow either during hypothermia or rewarming.

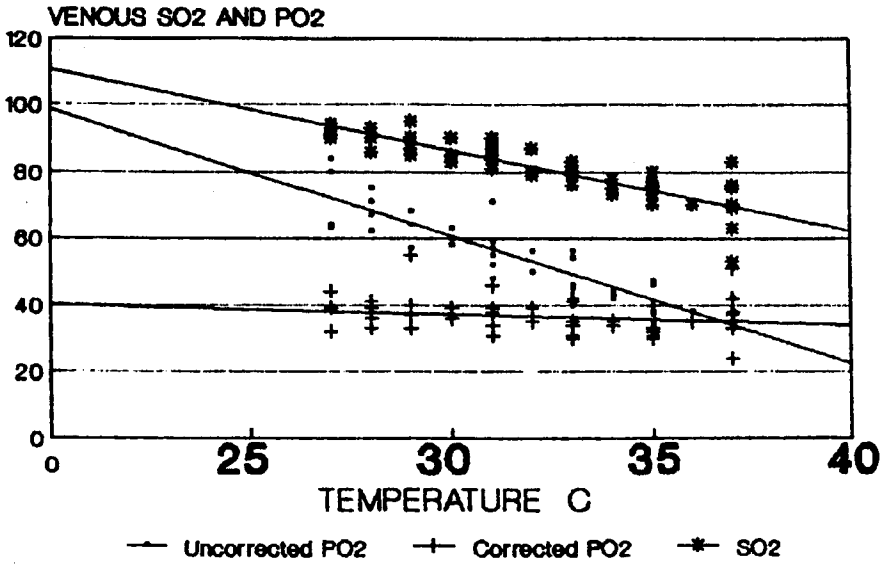
In all our patients, the arterial pO_2, whether temperature-corrected or uncorrected, as sampled from the outlet of the oxygenator, increased significantly with each increment decrease of body temperature. The higher arterial pO_2 during hypothermia may be attributed to the higher SVO_2 of venous return. Also, it may suggest a more efficient oxygenation of the venous return by the bubble oxygenator during hypothermia. Cooling increases the solubility of the bubbled oxygen in blood, and may decrease the size of its bubbles, and hence provides a larger surface area of blood/gas exchange.[7]

Lowering of the core body temperature from 37°C down to 28°C results in an average decrease of oxygen consumption by about 50 percent.[8] Thus, most of the metabolic needs during hypothermia can be met with minimal hemoglobin desaturation. In our patients, the $SV0_2$ significantly increased with each increment de-

Figure 4

Venous SO_2 and pO_2 vs. Temperature

Correlation and regression between body temperature (°C) and venous oxyhemoglobin saturation (SO_2%) and venous pO_2 (mmHg). The correlation with SO2 shows a negative and significant association (r = -0.85, b = -2.4, P < 0.0001). Correlation with uncorrected venous pO_2 shows a similar negative linear association (r = -0.88, b = -3.8, P < 0.0001). However, the corrected venous pO_2 did not significantly change with changes in body temperature (r = -0.15, b = -0.22, P = NS).



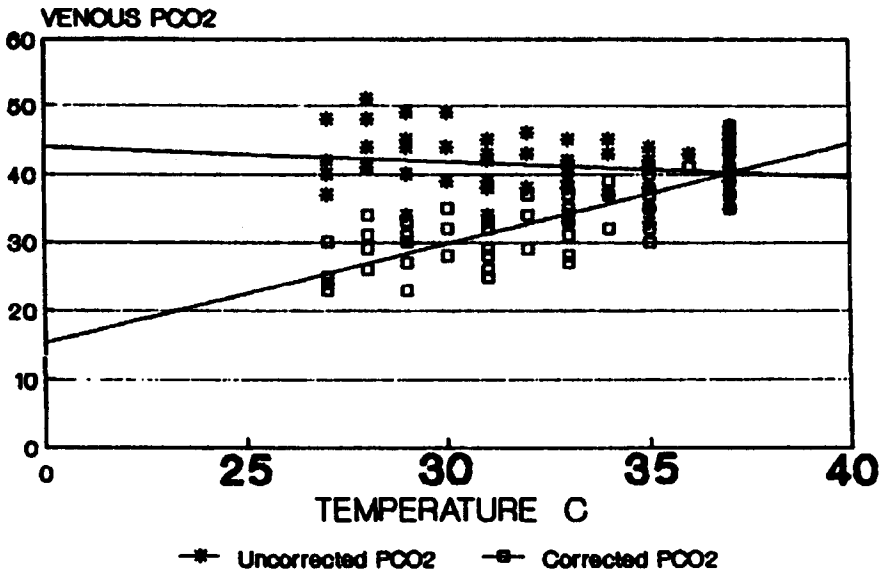


Figure 5

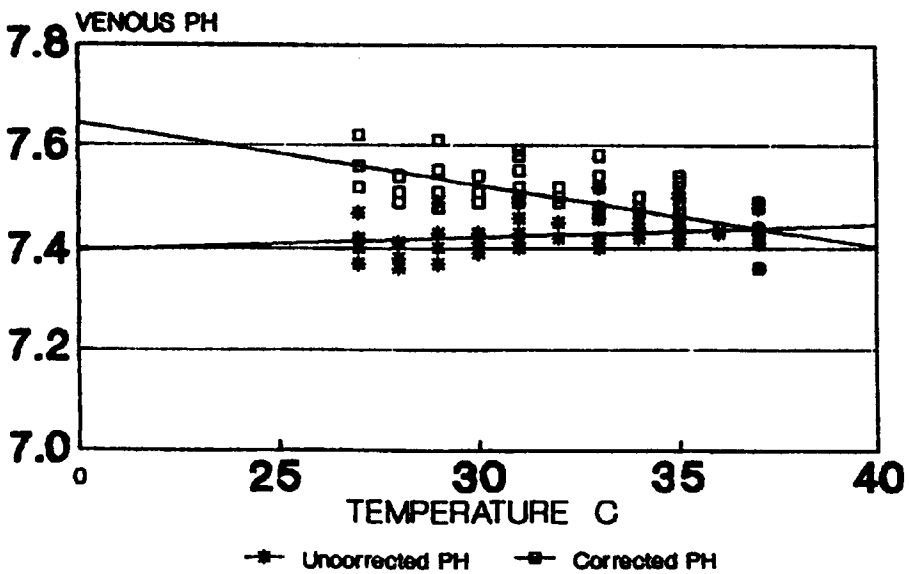
Venous pCO_2 vs. Temperature

Correlation and regression between body temperature (°C) and uncorrected and corrected venous pCO_2 (nnHg) showing a positive linear association (r = 0.79, b = 1.47, P < 0.0001) in the case of corrected pCO_2 and no association (r = -0.16, b = -0.22, P = NS) in the case of uncorrected pCO_2.

Figure 6

Venous pH vs. Temperature

Correlation and regression between body temperature (°C) and uncorrected and corrected venous pH showing a negative linear association (r = -0.71, b = -0.012, P < 0.0001) in the case of corrected pH and no association (r = 0.21, b = 0.003, P = NS) in the case of uncorrected pH.



crease of body temperature. The significant increase of SVO_2 during hypothermia was associated with a corresponding increase of the temperature-uncorrected venous pO_2. However, the temperature-corrected venous pO_2 did not significantly change during cooling, despite the increase of SVO_2 denoting a progressive leftwards shift of the oxyhemoglobin dissociation curve with each increment decrease of body temperature.[9] The cellular pO_2 approximates closely to venous pO_2,[10] and hence the technique may maintain optimal milieu interieur and prevent cellular hyperoxia.

The effect of hypothermia on pCO_2, while maintaining the ventilating oxygen flow constant, simulates the alpha-stat strategy of the poikilotherms which maintain ventilation constant during temperature changes.[11-15] Our report shows that the corrected pCO_2 whether arterial or venous varies directly and the corrected pH varies inversely with body temperature, while the uncorrected pCO_2 and pH remain essentially constant over a wide range of temperature changes. As the body cools, carbon dioxide production will be decreased, and consequently the corrected pCO_2 falls about 4.5 percent per °C, provided ventilation is maintained at the normothermic level.[9] However, cooling increases the carbon dioxide dissolved in the blood by a similar factor[9], and hence the carbon dioxide content as evidenced by the uncorrected pCO_2 does not show a significant change.

In conclusion, our report shows the effect of temperature changes on the arterial and venous blood gases during CPB, while maintaining both the perfusion and oxygen flows at a constant flow of 2.4 $L/m^2/min$ throughout the period of bypass. The report suggests that maintenance of constant perfusion and oxygen flows during hypothermic CPB at normothermic values will maintain a constant carbon dioxide content as evidenced by the temperature-uncorrected pCO_2, and a constant cellular pO_2 as evidenced by the corrected PVO_2. The technique is simple and may ensure optimal milieu interieur at all body temperatures.

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