

---

# TECHNIQUE

---

## Effect of Oxygen Flow on pCO<sub>2</sub> and pO<sub>2</sub> During Cardiopulmonary Bypass in Man

---

Anis Baraka, MD, Sania Haroun MD, Maurice Baroody, MD, Maud Nawfal, MD, Aliya Dabbous, MD, Abla Sibaii, BS, Salim Jamal, BS and Sana Shamli, BS

Departments of Anesthesiology, Epidemology and Biostatistics  
American University of Beirut  
Beirut, Lebanon

**Key words:** Cardiopulmonary bypass, bubble oxygenator, acid-base balance, alpha-stat, pCO<sub>2</sub>, pH, oxygenation, pO<sub>2</sub>

---

### Abstract

The effect of oxygen flow on arterial pO<sub>2</sub> and pCO<sub>2</sub> was investigated during cardiopulmonary bypass in three groups of patients undergoing open heart surgery. A Bentley-10 adult bubble oxygenator was used in all patients. The perfusion was maintained in the three groups at a constant flow of 2.3 l/m<sup>2</sup>/min throughout bypass. The oxygenator was bubbled in Group 1 by oxygen at an oxygen:perfusion ratio of 0.75:1.0 while 1.0:1.0 ratio was used in Group 2 and 1.5:1.0 ratio was used in Group 3. After going on bypass, moderate hypothermia was achieved, and the patients were rewarmed when surgery was completed.

The report shows that the arterial pCO<sub>2</sub>, and pO<sub>2</sub> may be determined during cardiopulmonary bypass by the bubbled oxygen flow. Increasing the oxygen:perfusion ratio from 0.5:1.0 to 1.0:1.0 is associated with a significant decrease of pCO<sub>2</sub> and increase of pO<sub>2</sub>. Further increase of the oxygen:perfusion ratio to 1.5:1.0 resulted in further decrease of pCO<sub>2</sub> without a significant increase of pO<sub>2</sub>. The results suggest that optimal oxygenation and carbon dioxide elimination can be achieved by using an oxygen:perfusion ratio 1.0:1.0 during hypothermia and following rewarming.

### Introduction

During cardiopulmonary bypass (CPB) using a bubble oxygenator, oxygen flow is delivered to the oxygenator to oxygenate the venous blood and to eliminate carbon dioxide (1, 2). This report investigates patients undergoing open heart surgery, the effect of different oxygen flows on the arterial pCO<sub>2</sub> and pO<sub>2</sub> during the hypothermic phase of CPB as well as following rewarming.

### Method

Investigation was carried out on 32 patients undergoing coronary artery bypass grafting or valve replacement during

*Address correspondence to: Anis Baraka, MD, Professor & Chairman, Dept. of Anesthesiology, American University of Beirut, 850 Third Ave., 18th Floor, New York, NY 10022.*

CPB. Their ages ranged between 35-60 years of age, and their weight ranged between 43-80 kg. The investigation was approved by the Institution Research Committee and informed consent was obtained from patients.

The patients were premedicated with 10 mg. morphine, 25 mg. promethazine and 0.4 mg. scopolamine. Anesthesia was induced with 0.1-0.2 mg/kg midazolam, 20-40 ug/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% oxygen without inhalation anesthetic supplementation. All patients were monitored by EKG (V<sub>5</sub>), radial arterial catheter and Swan Ganz PA catheterization.

A Bentley-10 adult bubble oxygenator (a) was only primed by 1500 ml lactated Ringer's solution. The patient was perfused by a roller pump (b) at a flow of 2.3 l/m<sup>2</sup>/min. The patients were randomly divided into three groups. In Group 1 (10 patients), the oxygenator was bubbled by 100% oxygen at an oxygen:perfusion ratio of 0.75:1.0, by 1.0:1.0 ratio in Group 2 (12 patients), and by 1.5:1.0 ratio in Group 3 (10 patients). In each group, both the pump and oxygen flows were maintained constantly throughout CPB. No carbon dioxide was added to the oxygen flow during cooling or rewarming. In the three groups, the mean arterial pressure during bypass ranged between 50-80 mmHg. The hematocrit was lowered during CPB to 23.8 ± 3.3% in Group 1, to 25.1 ± 2.9% in Group 2 and to 23.1 ± 2.3% in Group 3.

As soon as CPB was initiated, the patients were rapidly cooled to a rectal temperature of 28.7 ± 1.2°C in Group 1, to 28.4 ± 2.8°C in Group 2 and to 28.7 ± 1.6°C in Group 3. The heart was then arrested after aortic cross clamping by about 20 ml/kg of cardioplegic solution (K<sup>+</sup> 30 mEq/l at 4°C). When surgery was completed, the aortic cross clamp was released and the patient was rewarmed to a mean rectal temperature of 33.8 ± 1.2°C in Group 1, to 34.8 ± 0.9°C in Group 2 and to 34.0 ± 0.9°C in Group 3. After 20-30 minutes at both the low and high temperatures, an arterial sample was taken from the arterial

---

a. American Bentley, Irvine, CA 92714

b. Sarns 5000, Sarns/3M, Ann Arbor, MI, 48103

Table 1: The mean arterial pCO<sub>2</sub> and pH, corrected and uncorrected, which were achieved during hypothermia and following rewarming by using the different oxygen:perfusion flow ratios:

Group I: oxygen:perfusion ratio 0.75:1.0  
 Group II: oxygen:perfusion ratio 1.0:1.0  
 Group III: oxygen:perfusion ratio 1.5:1.0

		<u>HYPOTHERMIA</u>		<u>NORMOTHERMIA</u>	
		Corrected	Uncorrected	Corrected	Uncorrected
GROUP I	Pco <sub>2</sub> mmHg	30.0 ± 6.0	43.3 ± 6.7	34.1 ± 3.2	39.8 ± 3.4
	pH	7.48 ± 0.07	7.31 ± 0.05	7.44 ± 0.04	7.40 ± 0.03
GROUP II	Pco <sub>2</sub> mmHg	23.7 ± 4.1	37.8 ± 6.4	27.3 ± 2.7	31.0 ± 2.5
	pH	7.58 ± 0.06	7.44 ± 0.65	7.53 ± 0.03	7.50 ± 0.03
GROUP III	Pco <sub>2</sub> mmHg	20.4 ± 5.3	30 ± 5.9	25.3 ± 3.5	27.9 ± 3.8
	pH	7.6 ± 0.06	7.50 ± 0.05	7.53 ± 0.06	7.49 ± 0.06

Table 2: The mean arterial pCO<sub>2</sub> corrected and uncorrected, which were achieved during hypothermia and following rewarming by using the different oxygen:perfusion flow ratios:

Group I: oxygen:perfusion ratio 0.75:1.0  
 Group II: oxygen:perfusion ratio 1.0:1.0  
 Group III: oxygen:perfusion ratio 1.5:1.0

		<u>HYPOTHERMIA</u>		<u>NORMOTHERMIA</u>	
		Corrected	Uncorrected	Corrected	Uncorrected
GROUP I	Po <sub>2</sub> mmHg	473.1 ± 76.5	536.4 ± 85.6	222 ± 144	237.7 ± 143
GROUP II	Po <sub>2</sub> mmHg	526.2 ± 71.0	599.5 ± 90.9	379.8 ± 98.4	392.8 ± 100.4
GROUP III	Po <sub>2</sub> mmHg	522.7 ± 37.2	563.2 ± 62.1	306 ± 126	318.8 ± 125.3

Figure 1

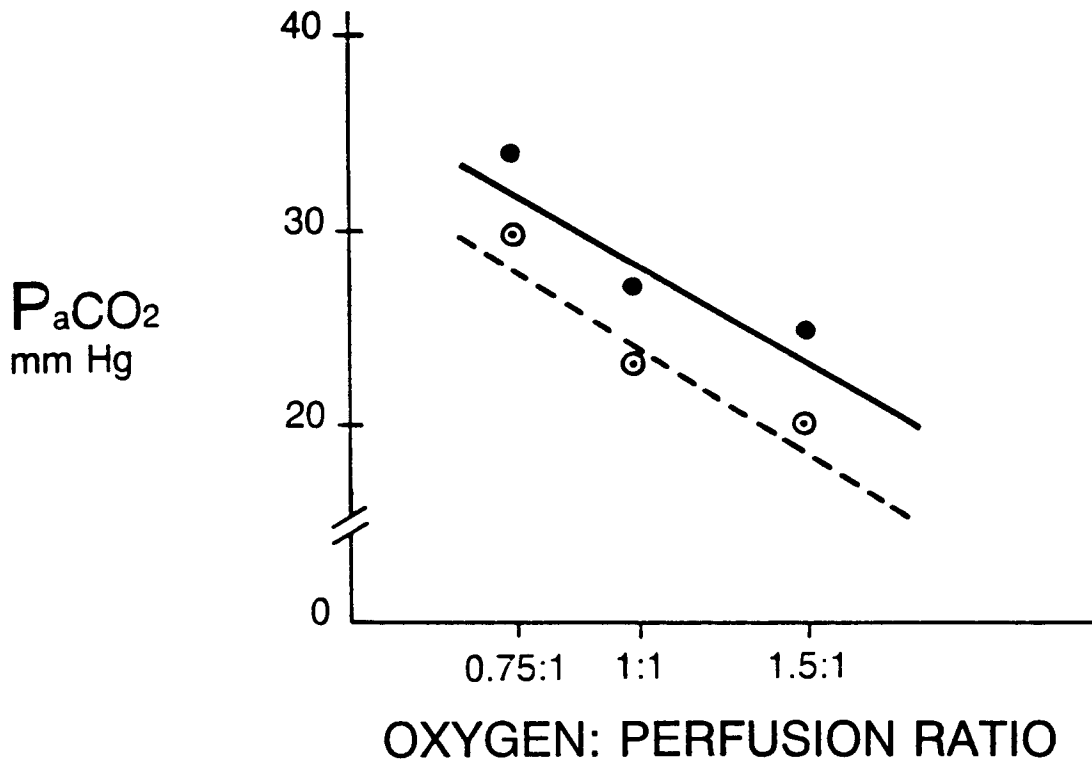
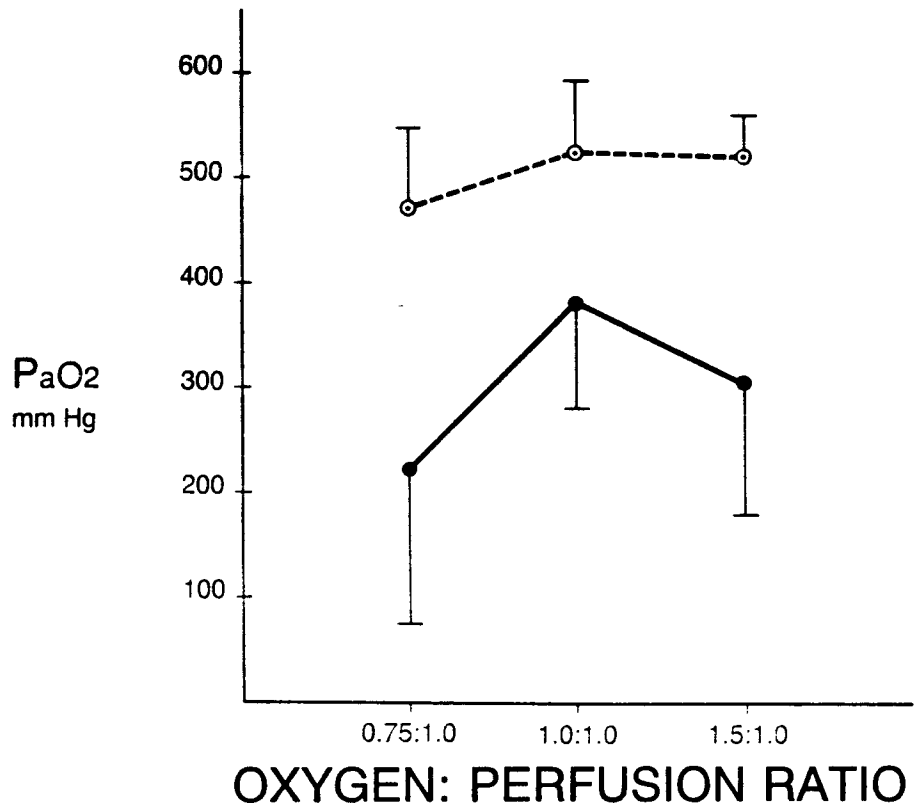


Figure 2



outlet line of the oxygenator, for measuring pCO<sub>2</sub>, pH and pO<sub>2</sub> using ABL300 Radiometer with electrodes kept constant at 37°C. The uncorrected values measured at 37°C were then corrected according to body temperature.

All data are presented as mean ± SD. The paired t-test was used to compare values within the same group, while the unpaired t-test was used to compare data among the different groups. P<0.05 was considered significant.

## Results

As shown in Table 1, the arterial pCO<sub>2</sub> was inversely related to the oxygen flow. Increasing the oxygen:perfusion flow ratio from 0.75:1.0 in Group 1 to 1.0:1.0 in Group 2 was associated with a significant decrease of pCO<sub>2</sub> and increase of pH during both hypothermia and following rewarming. Further increase of the oxygen:perfusion flow ratio to 1.5:1.0 in Group 3 was associated with a further decrease of pCO<sub>2</sub> and increase of pH.

The effect of temperature changes on pCO<sub>2</sub> and pH was qualitatively the same in the three groups. The corrected pCO<sub>2</sub> was significantly lower and the pH was significantly higher during hypothermia than following rewarming. In contrast, the uncorrected pCO<sub>2</sub> was significantly higher and the pH was significantly lower during hypothermia. Figure 1 depicts the relationship between the different oxygen flows and the corrected pCO<sub>2</sub> during both hypothermia and following rewarming.

As shown in Table 2, increasing the oxygen:perfusion flow ratio from 0.75:1.0 in Group 1 to 1.0:1.0 in Group 2 was associated with a significant increase of pO<sub>2</sub> during both hypothermia and following rewarming. Further increase of the ratio to 1.5:1.0 in Group 3 did not produce further increase of pO<sub>2</sub>.

The effect of temperature changes on pO<sub>2</sub> was qualitatively the same in the three groups. Both the corrected and uncorrected pO<sub>2</sub> values were significantly higher during hypothermia than following rewarming. Figure 2 depicts the relationship between the different oxygen flows and the corrected pO<sub>2</sub> during both hypothermia and following rewarming.

## Discussion

During CPB and using a bubble oxygenator, oxygen is bubbled through the oxygenator to provide oxygenation of the venous return and to ensure carbon dioxide elimination. The amount of oxygen that can be transferred into the blood depends primarily on the total surface or exchange achieved by the bubbling process (1, 2).

Our report shows that the oxygen flow affects oxygenation. Using an oxygen:perfusion flow ratio of 1.0:1.0 is associated with maximal pO<sub>2</sub> values during both hypothermia and after rewarming. Increasing the oxygen flow to 1.5:1.0 does not result in a further improvement of oxygenation, while decreasing the oxygen flow to 0.75:1.0 can compromise oxygenation.

The effect of temperature changes on arterial pO<sub>2</sub>, while keeping the perfusion and oxygen flows constant, was qualitatively the same in all patients. In the three groups,

hypothermia was associated with a higher arterial pO<sub>2</sub> than that achieved after rewarming. This can be attributed to the decreased oxygen consumption during hypothermia; VO<sub>2</sub> can drop by about 50% at 28°C (4). Following rewarming, the metabolic rate and oxygen consumption increase and hence, rewarming is associated with increased oxygen extraction as evidenced by the significant decrease of SvO<sub>2</sub> (5).

The present report also shows during CPB that the arterial pCO<sub>2</sub> is inversely proportional to the oxygen flow used. Increasing the oxygen:perfusion flow ratio from 0.75:1.0 to 1.0:1.0 is associated with a significant decrease of pCO<sub>2</sub> and an increase of pH. Further increase of oxygen:perfusion flow ratio to 1.5:1.0 is associated with a further decrease of pCO<sub>2</sub> and an increase of pH.

During CPB, the oxygen flow delivered to the oxygenator provides carbon dioxide elimination and hence PaCO<sub>2</sub> is determined by the following formula:

$$PaCO_2 = (P_B - 47) \frac{V_{CO_2}}{\text{Gas Flow}}$$

Normocarbica can be therefore achieved during normothermia by using an oxygen flow which is approximately equal to the alveolar ventilation volume (6). During hypothermia, the carbon dioxide production is decreased and hence using the same oxygen flow will result in low corrected pCO<sub>2</sub> values. However, because of the increased carbon dioxide solubility by cooling, the carbon dioxide content as evidenced by the "uncorrected" pCO<sub>2</sub> remains unchanged or is even increased. Thus, keeping the oxygen flow constant during both hypothermia and following rewarming will maintain the carbon dioxide content despite the change of corrected pCO<sub>2</sub>. This technique simulates the alpha-stat strategy of acid-base regulation in the poikilothermic animals which maintain ventilation constantly during temperature changes (7-11).

In conclusion, during CPB using a Bentley-10™ bubble oxygenator, the arterial pO<sub>2</sub> and pCO<sub>2</sub> can be controlled by the oxygen flow delivered to the oxygenator. Optimal oxygenation and carbon dioxide elimination can be achieved by using an oxygen:perfusion ratio 1.0:1.0 during both hypothermia and following rewarming.

## References

1. Clark LC Jr: Optimal flow rate in perfusion. In: *Extracorporeal Circulation*. JG Allen (ed), Springfield, Ill, Thomas, 1958, pp 150-163.
2. Montgomery V: Respiratory characteristics of extracorporeal pump-oxygenators. *J Phar Cardiovasc Surg* 39 288-304 1960.
3. Severinghaus JW, Stupfel N, Bradley AF: Variation of serum carbonic acid pK with pH temperature. *J. Appl Physiol* 9: 197-200, 1956.
4. Harris EA, Seelye ER, Squire AW: Oxygen consumption during cardiopulmonary bypass with moderate hypothermia in man. *Br J. Anaesth* 43:1113-1120, 1971.
5. Baraka A, Baroody M, Haroun S, Nawfal M, Dabbous A, Sibai A, Jamal S, Shamli S: Continuous venous oximetry during cardiopulmonary bypass: Influence of temperature changes, perfusion flow, and hematocrit levels. *J Cardiothoracic Anesthesia*, 4:35-38,

1990.

6. Baraka A: pCO<sub>2</sub> control by fresh gas flow during controlled ventilation with a semi-open circuit. *Br J Anaesth* 41: 527-530, 1969.
7. Rhan H: Body temperature and acid-base regulation. *Pneumologie* 151:87-94, 1974.
8. Reeves RB: An imidazole alpha-stat hypothesis for vertebrate acid-base regulation: Tissue carbon dioxide content and body temperature in bullfrogs. *Respir Physiol* 14: 219-236, 1972.
9. White FN, Weinstein Y: Carbon dioxide transport and acid-base balance during hypothermia. In Utley, JR (ed): Pathophysiology and Techniques of Cardiopulmonary Bypass (vol 2). Baltimore, Williams and Wilkins, 1983, pp. 40-48.
10. Blayo MC, Lecompte Y, Pocidalo JJ: Control of acid-base status during hypothermia in man. *Respir Physiol* 42: 287-298, 1980.
11. Swan H. Metabolic rate, temperature and acid-base control: The best strategy and our needs to achieve it. *J Extra Corp Tech* 17:65-73, 1985.