Pulmonary Vascular Resistance before and after Cardiopulmonary Bypass*  

The Effect of PaCO₂

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To examine whether CPB influences pulmonary vascular sensitivity to CO₂, we compared the effect of slight induced hypocarbia and hypercarbia on pulmonary circulation before and after CPB in ten mechanically ventilated patients undergoing CABG. Hypocarbia was produced by increasing tidal volume slightly and hypercarbia was then induced by adding CO₂ to the inspired gas mixture. In another ten patients, hypercarbia was produced after CPB by decreasing ventilator rate and the cardiopulmonary responses to hypercarbia, produced by the two methods of CO₂ elevation, were compared. Slight respiratory acidosis induced by CO₂ inhalation did not change PVR before CPB but effected a 50 percent increase after CPB. Hypercarbia induced by alveolar hypoventilation after CPB increased PVR by 40 percent. During the increased CO₂ production after hypothermic CPB, pulmonary vasoconstriction would be expected to occur and impair right ventricular performance. Therefore, tight control of PaCO₂ with appropriate adjustment of ventilatory support is mandatory.

(Chest 1989; 95:773-78)

Reports published so far suggest that the pulmonary circulatory response to a given stimulus is more dependent on the initial state of the pulmonary vasculature than on the stimulus itself.⁴ We demonstrated recently that moderately induced respiratory acidosis produced an exaggerated pulmonary vasconstriction reaction after CPB in patients undergoing CABG.³ Pulmonary hypertension was completely reversible by restoration of normal PaCO₂.

No study has previously assessed the CO₂ sensitivity of the pulmonary circulation before and after CPB. The present study was designed to find out whether CPB renders pulmonary vasculature particularly sensitive to CO₂. We compared the effect of slightly induced hypocarbia and hypercarbia on pulmonary circulation both before and after CPB in mechanically ventilated CABG patients with normal preoperative PVR. The cardiopulmonary responses to PaCO₂ elevation produced by addition of CO₂ to the inspired gas mixture and those produced by alveolar hypoventilation were compared as well.

**Materials and Methods**

**Patients**

We studied 20 consenting adult patients scheduled to undergo CABG according to a protocol approved by the Human Subjects Review Committee of our institute. We excluded patients with impaired left ventricular function (ejection fraction <0.4) or elevated mean pulmonary artery pressure;¹ those with a previous history of any other cardiac abnormality or major pulmonary dysfunction, as well as those having evidence of coronary artery disease or diffuse or significant stenosis (>50 percent) of the left main coronary artery. Hemodynamic instability due to intraoperative myocardial ischemia or bleeding after surgery and the need for vasoactive medication at any time of the study necessitated that we excluded 13 additional patients who initially were accepted for the study.

The patients were randomly divided into two groups of ten patients each. In group 1 patients, slight hypercarbia was achieved both before and after CPB by adding CO₂ to the gas mixture delivered by the ventilator, while minute ventilation and FIO₂ were maintained unchanged. In group 2 patients, who were only studied after CPB, slight hypercarbia was induced by decreasing the ventilator frequency with unchanged tidal volume.

**Anesthesia and CPB**

The patients' antianginal medications (nitrates, beta-adrenergic blockers, calcium-entry blockers) were continued until the morning of surgery. Approximately one hour after premedication with morphine 0.2 mgkg⁻¹ and scopolamine, 0.006 mgkg⁻¹, with the patient under local anesthesia, vascular catheters were introduced into a peripheral vein, radial artery and pulmonary artery. Anesthesia was induced with diazepam, 2.5 to 10 mg, and fentanyl, 30 μgkg⁻¹, and maintained with a constant infusion of fentanyl at a rate of 0.3 μgkg⁻¹ · min⁻¹ until the end of surgery. Muscle relaxation was provided using pancuronium. After endotracheal intubation, the patients were connected to a Servo 900B ventilator to receive intermittent positive pressure ventilation with a mixture of oxygen and air (FIO₂ 0.5) and zero end-expiratory pressure. Tidal volume was adjusted to achieve normocarbia using a respiratory rate of 12 min⁻¹.

Nonpulsatile CPB was maintained with bubble oxygenation, moderate hypothermia to a nasopharyngeal temperature of 26 to 28°C, and mild hemodilution. Heparinization and reversal of heparinization using a slow protamine chloride infusion were accomplished with the activated coagulation time method. Diazepam in

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Supported by a grant from the Paulo Foundation.

Manuscript received May 25; revision accepted September 6.

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doses of 10 to 20 mg was given to ensure sleep during CPB. Caval cannulation, cold potassium cardioplegia, and external cooling of the heart were used for myocardial protection. During aortic closure, the patients were disconnected from the ventilator with their airways kept open to ambient air. An FIO2 of 1.0 was used temporarily after weaning from CPB.

Correct positioning of the pulmonary artery catheter tip in the right main pulmonary artery was verified by chest roentgenogram taken soon after surgery. During the study interventions, balanced Ringer’s solution was infused at a rate of 1.5 ml kg⁻¹ h⁻¹.

**Study Design and Measurements**

In group 1 patients, the first study period started shortly after induction of anesthesia. After a period of ten minutes of stabilized normocarbia (PaCO₂, 39.0 ± 2.6 mm Hg [SD]), tidal volume was slightly increased, and hemodynamic and blood gas values were obtained after ten minutes of stabilized hypocarbia (PaCO₂, 34.1 ± 2.0 mm Hg). Thereafter, we induced slight hyperventilation by adding CO₂ (2.6 ± 0.4 percent) to the inspired gas while ventilatory volume and FIO₂ were kept unchanged. The measurements were repeated, when at least ten minutes had elapsed after reaching stabilized hypocarbia (PaCO₂, 46.7 ± 2.0 mm Hg). The second study period in group 1 patients began about 2½ hours after CPB, when the patients were still asleep, paralyzed, and mechanically ventilated. The study sequence just described was followed and the measurements were obtained after a period of ten minutes of stabilized hypocarbia (PaCO₂, 34.0 ± 2.1 mm Hg) and hypocarbia (PaCO₂, 47.1 ± 2.6 mm Hg).

In group 2 patients, hypocarbia (PaCO₂, 34.6 ± 1.2 mm Hg) was produced in a corresponding manner about 2½ hours after CPB. Following this, hypocarbia (PaCO₂, 46.9 ± 1.6 mm Hg) was achieved by decreasing the ventilator rate by about 60 percent from the initial value of 12 min⁻¹, while tidal volume was kept unchanged. Pulmonary and systemic hemodynamics and blood gases were again assessed after a stabilization period of ten minutes at each stage. The time required to attain a steady level of hyperventilation was about ten minutes in group 1 and 20 to 30 minutes in group 2.

Fifty percent inspired oxygen was secured throughout the study period by continuous monitoring with a paramagnetic oxygen analyzer. Continuous monitoring of the end-tidal CO₂ concentration (Multigas capnometer, Datex Instrumentarium Corp) and frequent blood gas analyses were used to guide appropriate ventilatory or inspiratory CO₂ adjustments. Leads 2 and V₅ of the ECG were recorded throughout the study. Intravascular pressures were obtained using AE 840 (AME) transducers zeroed to the mid-thoracic level and recorded on a multichannel recorder (Nihon Kohden Corp). Cardiac output was determined using a thermodilution technique with 10 ml of 0.9 percent saline solution at room temperature injected at end-expiration, and expressed as the mean of three values calculated from well-formed curves. The end-expiratory pressure waveforms were used for the determination of DAP-PCWP and for the calculation of MAP and MPAP. The PVR and SVR were calculated using standard formulae.¹

Arterial and mixed venous blood samples were drawn anaerobically into 5-ml glass syringes which were kept in ice until they were analyzed immediately after each study period for blood gases with an automatic blood gas analyzer (ABL 4, Radiometer) and for blood oxygen saturation and hemoglobin content with a CO-oximeter (IL 282, Instrumentation Laboratories). The values obtained were used for the calculation of QO₂.²

The Wilcoxon signed rank test was used to compare hemodynamic responses caused by CO₂ challenge in group 1 patients before and after CPB. The Mann-Whitney rank sum test was used to compare hemodynamic responses to PaCO₂ changes after CPB between the two groups. Simple linear regression analysis was used for correlation between variables. A p<0.05 was considered statistically significant. Data are presented as means ± SD.

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**Table 1—Characteristics of the 20 CABG Patients Randomized to Two Study Groups**

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=10)</th>
<th>Group 2 (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (yr)</td>
<td>49.7 ± 8.9</td>
<td>50.6 ± 6.5</td>
</tr>
<tr>
<td>Body surface area (m²)</td>
<td>1.90 ± 0.20</td>
<td>1.99 ± 0.22</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>0.60 ± 0.10</td>
<td>0.60 ± 0.11</td>
</tr>
<tr>
<td>FVC (% of predicted value)</td>
<td>100.6 ± 9.3</td>
<td>93.1 ± 8.7</td>
</tr>
<tr>
<td>FEV₁ (% of predicted value)</td>
<td>99.1 ± 12.3</td>
<td>93.0 ± 12.8</td>
</tr>
</tbody>
</table>

*Values are means ± SD or number of patients.

**RESULTS**

The two study groups were comparable with respect to patient characteristics (Table 1), and baseline hemodynamic and blood gas values obtained before induction of anesthesia (Table 2). The values of arterial and mixed venous pH, PaO₂, and PaCO₂ in the two groups are given in Table 3.

**Pulmonary Hemodynamics**

After CPB, MPAP (p = 0.005), PVR (p = 0.005) and DAP-PCWP gradient (p = 0.021) increased significantly from pre-CBP values. Before CPB no significant changes were observed in pulmonary hemodynamics when PaCO₂ was increased in group 1 patients (Fig 1 to 3). After CPB, however, a significant increase in MPAP, PVR and DAP-PCWP gradient at unchanged CO₂ was observed, when PaCO₂ was increased from slight hypocarbia to slight hypercarbia (Fig 1 to 3). The baseline level of PVR after CPB significantly predicted the magnitude of the hypercarbic response in PVR only in group 1 patients (r = 0.72; p<0.05). The method by which hypercarbia was produced after

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**Table 2—Results of Baseline Hemodynamic and Blood Gas Measurements**

<table>
<thead>
<tr>
<th></th>
<th>Group 1 (n=10)</th>
<th>Group 2 (n=10)</th>
</tr>
</thead>
<tbody>
<tr>
<td>HR (beats min⁻¹)</td>
<td>58 ± 4</td>
<td>52 ± 5</td>
</tr>
<tr>
<td>CI (1 min⁻¹ m⁻²)</td>
<td>2.98 ± 0.83</td>
<td>2.68 ± 0.24</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>88 ± 6</td>
<td>79 ± 14</td>
</tr>
<tr>
<td>MPAP (mm Hg)</td>
<td>17.0 ± 4.2</td>
<td>18.5 ± 3.1</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>10.6 ± 3.3</td>
<td>11.3 ± 2.3</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>6.6 ± 3.0</td>
<td>6.9 ± 1.6</td>
</tr>
<tr>
<td>pHa</td>
<td>7.40 ± 0.02</td>
<td>7.39 ± 0.02</td>
</tr>
<tr>
<td>PaO₂ (mm Hg)</td>
<td>80.9 ± 17.8</td>
<td>82.5 ± 23.7</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>42.5 ± 3.0</td>
<td>40.8 ± 3.5</td>
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</table>

*Values are means ± SD.
CPB did not significantly affect the magnitude of the observed pulmonary vasoconstrictory reaction (Fig 1 to 3). The change in DPAP-PCWP gradient induced by hypercarbia correlated rather well with the change in PVR after CPB in group 1 patients (r = 0.79; p = 0.005). In group 2 patients, however, this correlation was poor (r = 0.52; p = 0.128).

No consistent changes in Qs/Qt were found during CO2 challenge. In group 1, Qs/Qt decreased slightly before CPB (from 8.3 ± 7.4 percent to 5.2 ± 6.8 percent) and remained unchanged after CPB (7.3 ± 5.7 percent during hypocarbia and 7.6 ± 3.7 percent during hypercarbia). In group 2, the Qs/Qt values were 9.5 ± 4.5 percent during hypocarbia and 12.5 ± 5.1 percent during hypercarbia. The difference in Qs/Qt changes between groups 1 and 2 was not significant.

**Systemic Hemodynamics**

In group 1 patients HR, CVP, PCWP and the ratio of CVP to PCWP increased significantly after CPB (Table 4). During the hypercarbic challenge in group 1, a significant difference between the pre-CPB and post-CPB changes was noticed in PCWP (a small increase before and a small decrease after CPB).

After CPB, neither the systemic hemodynamic values obtained during hypocarbia nor the hemodynamic responses to hypercarbia differed significantly between groups 1 and 2.

**Discussion**

A few hours after CPB, slight hypercarbia resulted in pulmonary vasoconstriction reaction at unchanged blood flow in our mechanically ventilated patients recovering from CABG. In contrast, before CPB we were unable to demonstrate any significant change in pulmonary circulatory pressure variables during induced hypercarbia. The pulmonary vasoconstriction reaction observed after CPB seemed to be independent of the method by which the changes in PaCO2 were accomplished.

Respiratory acidosis has been shown to induce pulmonary vasoconstriction in several animal species and in humans with diseases ultimately leading to pulmonary hypertension. Only a few studies concerning humans with healthy lungs and normal pulmonary vasculature exist so far and they have given conflicting results. The greater response in animals as compared with humans may be partly explained by the abundant muscularity in the muscular part of the pulmonary arteries and relatively high resting pulmonary artery pressure which have been documented in many animals. The mechanism by which hypercarbia induces pulmonary vasoconstriction is currently not known. Recent findings suggest, however, that the initial state of the pulmonary artery smooth muscle might be important for the response. Thus, acute hypercarbia induces pronounced pulmonary vasoconstriction.

**Table 3—pH, Po2, and PCO2 in Arterial and Mixed Venous Blood**

<table>
<thead>
<tr>
<th>Group 1</th>
<th></th>
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<th>Group 2</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Before CPB</td>
<td>After CPB</td>
<td></td>
<td>Before CPB</td>
<td>After CPB</td>
</tr>
<tr>
<td>pHa</td>
<td>7.46 ± 0.01</td>
<td>7.35 ± 0.02</td>
<td></td>
<td>7.42 ± 0.02</td>
<td>7.32 ± 0.04</td>
</tr>
<tr>
<td>pHc</td>
<td>7.42 ± 0.02</td>
<td>7.33 ± 0.02</td>
<td></td>
<td>7.38 ± 0.03</td>
<td>7.29 ± 0.01</td>
</tr>
<tr>
<td>PaO2 (mm Hg)</td>
<td>202.0 ± 77.0</td>
<td>224.5 ± 69.7</td>
<td></td>
<td>199.5 ± 53.9</td>
<td>181.2 ± 46.2</td>
</tr>
<tr>
<td>PVo2 (mm Hg)</td>
<td>41.3 ± 6.7</td>
<td>45.1 ± 7.1</td>
<td></td>
<td>36.4 ± 5.5</td>
<td>40.5 ± 7.1</td>
</tr>
<tr>
<td>PaCO2 (mm Hg)</td>
<td>34.1 ± 2.0</td>
<td>46.7 ± 2.0</td>
<td></td>
<td>34.0 ± 2.1</td>
<td>47.1 ± 2.6</td>
</tr>
<tr>
<td>PCO2 (mm Hg)</td>
<td>40.6 ± 2.4</td>
<td>51.0 ± 0.9</td>
<td></td>
<td>40.7 ± 3.3</td>
<td>52.0 ± 3.0</td>
</tr>
</tbody>
</table>

*Values are means ± SD.

*p < 0.01; compared with change from hypocarbia to hypercarbia before CPB.
striction in adults and children with diseases affecting pulmonary circulation,\textsuperscript{9,10} while the corresponding response is absent or weak in healthy individuals.\textsuperscript{11} Moreover, nitrous oxide has been shown to induce pulmonary vasoconstriction only in those cardiac patients who have an elevated PVR.\textsuperscript{14} Our results seem to be in agreement with these findings: pulmonary pressure response to hypercarbia was observed only after CPB when the baseline PVR was elevated. Yet, the level of PVR after CPB significantly predicted the magnitude of the change in PVR only in group 1 patients. So, we are not certain whether the structure-function interaction in the pulmonary vascular bed mentioned earlier explains the mechanism of CO\textsubscript{2} response in our patients, who had acute changes in their pulmonary vasculature rather than chronic changes due to persistent pressure or volume overload. It is possible that pure mechanical obstruction of pulmonary vessels by aggregated cells contributed to the difference between pre- and post-CBP pulmonary CO\textsubscript{2} response.\textsuperscript{15,16}

Most of the indirect systemic circulatory effects of hypercarbia are mediated by the release of endogenous catecholamines.\textsuperscript{17} Adrenergic mechanisms also might be involved in pulmonary vasoconstrictor response to hypercarbia. In an animal experiment, pulmonary vasoconstriction produced by hypercarbia was potentiated by beta-blockade, suggesting unmasking of alpha-receptor mediated vasoconstriction.\textsuperscript{18} Another pulmonary vasoconstrictor, nitrous oxide, recently has been shown to liberate catecholamines from isolated strips of pulmonary artery.\textsuperscript{19} If catecholamines are important mediators of pulmonary circulatory effects of hypercarbia, the differences in the level of anesthesia before and after CPB might partially account for our results. High-dose fentanyl anesthesia effectively blocks the increase of plasma catecholamines associated with surgical stimulation, whereas during recovery from anesthesia this inhibitory effect wears off as shown by conspicuous increases in circulating catecholamine levels. In addition, diazepam has been shown to decrease plasma catecholamine levels when it is combined with fentanyl induction.\textsuperscript{20} Consequently, the deeper level of anesthesia before CPB might have blocked the catecholamine-liberating effect of hypercarbia, thereby attenuating its hemodynamic effects.

In a recent study of ASA II-III patients with normal

\textbf{FIGURE 2.} Changes in PVR corresponding to alterations in PaCO\textsubscript{2} in ten CABG patients before and after CPB (group 1) and in ten CABG patients after CPB (group 2). In group 1 hypercarbia was induced by adding CO\textsubscript{2} to the inspiratory gas mixture and in group 2 by decreasing ventilator rate. Values are means \pm SD. \textit{p}\textsubscript{1} = comparison of PVR changes before and after CPB in group 1. \textit{p}\textsubscript{2} = comparison of PVR changes after CPB between groups 1 and 2.

\textbf{FIGURE 3.} Changes in DPAP-PCWP corresponding to alterations in PaCO\textsubscript{2} in ten CABG patients before and after CPB (group 1) and in ten CABG patients after CPB (group 2). In group 1 hypercarbia was induced by adding CO\textsubscript{2} to the inspiratory gas mixture and in group 2 by decreasing ventilator rate. Values are means \pm SD. \textit{p}\textsubscript{1} = comparison of DPAP-PCWP changes before and after CPB in group 1. \textit{p}\textsubscript{2} = comparison of DPAP-PCWP changes after CPB between groups 1 and 2.
Table 4—Systemic Hemodynamics*

<table>
<thead>
<tr>
<th></th>
<th>Before CPB</th>
<th>After CPB</th>
<th></th>
<th>Before CPB</th>
<th>After CPB</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>34.1±2.0</td>
<td>46.7±2.0</td>
<td>34.0±2.1</td>
<td>47.1±2.6</td>
<td>34.6±2.1</td>
</tr>
<tr>
<td>HR (beats min⁻¹)</td>
<td>60±6</td>
<td>58±5</td>
<td>79±9</td>
<td>79±8</td>
<td>71±6</td>
</tr>
<tr>
<td>CI (L min⁻¹ m⁻²)</td>
<td>2.51±0.51</td>
<td>2.59±0.45</td>
<td>2.47±0.44</td>
<td>2.54±0.50</td>
<td>2.37±0.33</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>80±12</td>
<td>78±10</td>
<td>81±14</td>
<td>73±17</td>
<td>81±11</td>
</tr>
<tr>
<td>PCWP (mm Hg)</td>
<td>5.7±2.1</td>
<td>6.4±2.4</td>
<td>7.2±2.2</td>
<td>6.8±2.4</td>
<td>7.5±2.5</td>
</tr>
<tr>
<td>CVP (mm Hg)</td>
<td>4.0±2.3</td>
<td>5.1±3.0</td>
<td>7.6±2.9</td>
<td>8.6±3.7</td>
<td>7.3±2.3</td>
</tr>
<tr>
<td>CVP/PCWP</td>
<td>0.70±0.24</td>
<td>0.76±0.21</td>
<td>1.03±0.15</td>
<td>1.30±0.45</td>
<td>1.01±0.34</td>
</tr>
<tr>
<td>SVR (dyne·cm⁻²·s⁻¹)</td>
<td>1331±317</td>
<td>1222±193</td>
<td>1325±346</td>
<td>1104±348</td>
<td>1288±302</td>
</tr>
</tbody>
</table>

*Values are means ± SD. In group 1, hypercapnia was induced by adding CO₂ to the inspiratory gas mixture, whereas in group 2 hypercapnia was achieved by diminishing the respiratory rate.

\[ \text{Group 1} \]

\[ \text{Group 2} \]

PVR by Wattwil and Olsson, the pulmonary and systemic arterial pressures as well as PVR and CO increased significantly during hypercapnia induced before surgery. By allowing the patients to inspire a mixture containing CO₂, 50 percent oxygen and 50 percent nitrous oxide, a considerably higher level of PaCO₂ than in our study was attained (PaCO₂ 8.6±0.9 kPa). It is possible that the differences in the patients' clinical condition, in the background anesthesia, and in the degree of hypercapnia are responsible for the differences observed between the results of Wattwil and Olsson and our pre-CBP findings.

Hypoxic pulmonary vasoconstriction affects pulmonary perfusion more extensively after than before CPB and administration of 100 percent oxygen decreases PVR after CPB. Hypercapnia and hypoxia additively augment pulmonary vasoconstriction, reducing intrapulmonary right-to-left shunting of blood. It has been suggested that augmentation of HPV in respiratory acidosis primarily is mediated by blood PaCO₂ and not blood pH, while in contrast attenuation of HPV in respiratory alkalosis is due to blood pH. In our present study, changes in blood PaCO₂ were accompanied by changes in blood pH; therefore, their separate effects on pulmonary circulation are difficult to define. We have previously demonstrated a small decrease in shunt fraction after CPB when PaCO₂ was brought from hypocapnia to hypercapnia with CO₂ inhalation. In the present study, no decrease in the intrapulmonary shunt fraction was observed with CO₂ exposure. Even a small increase in shunt, accompanied by a decrease in arterial blood oxygenation was seen, when slight hypercapnia was produced by decreasing ventilator rate. In the present study, however, changes in PaCO₂ were smaller and the calculation of shunt fraction was based on direct measurement of blood oxygen saturation rather than on the calculation from blood gas values as in our earlier study. Whatever the direction of the change in shunt, the magnitude of the change seems to be relatively small. Therefore, potentiation of HPV is not likely to be the sole mechanism behind the increase in PVR with hypercapnia after CPB. Finally, the decrease in ventilator rate, which was used to produce hypercapnia in group 2 patients, may have contributed to the rise in PVR as a consequence of changed intrathoracic pressure-volume conditions.

Right ventricular function is reversibly compromised during the immediate recovery phase after CABG. There is experimental evidence that the increased afterload stress brought about by hypercarbic pulmonary vasoconstriction is potentially deleterious to right ventricular performance. The decrease in systemic arterial pressure observed in our patients during hypercapnia might further impair right coronary artery blood flow. No episode of frank acute right heart failure was observed in this series in which we allowed only modest variation in PaCO₂. Yet, the increase in the ratio of CVP to PCWP after CPB and a tendency to a further increase during CO₂ challenge might indicate impending deterioration of right ventricular function. How well the changes in the ratio of CVP to PCWP reflect changes in right and left ventricular filling is, however, obscure without the knowledge of myocardial compliance.

In conclusion, CPB seems to markedly modify pulmonary vascular sensitivity to CO₂ resulting in elevated MPAP, PVR and DPAP-PCWP gradient at unchanged flow during slight hypercapnia after CPB. Increased CO₂ production caused by increased metabolism is likely to occur in CABG patients during recovery from CPB. Increased right ventricular workload caused by elevated pulmonary artery pressure might impair right ventricular function for several hours after CPB, when right ventricular reserves are limited. We recommend tight control of PaCO₂ by continuous monitoring of end-tidal CO₂ concentration with appropriate adjustment of ventilatory support.
REFERENCES

1. Bagshaw RJ, Cox RH. Pulmonary vascular response is dependent upon initial state of the vasculature. Anesthesiology 1983; 59:205-06