Alteration of Pulmonary Oxygenation by Pulmonary Artery Occluded Pressure Measurements in Mechanically Ventilated Patients*

Yumiko Ishizawa, M.D.; and Shuji Dohi, M.D.

A significant decrease in \( \text{PaO}_2 \) occurs in some patients during PAOP measurements. To examine the incidence and the types of patients whose \( \text{PaO}_2 \) decreases during PAOP measurements, we studied the changes of \( \text{PaO}_2 \) in 101 anesthetized, mechanically ventilated adult patients. During a 2-min inflation of a PAC balloon, seven patients (6.9 percent) developed marked decrease in \( \text{PaO}_2/\text{FiO}_2 \) (more than 100 mm Hg). Neither age nor size of patients, decrease in end-tidal \( \text{CO}_2 \), nor baseline value of PAP correlated with the decreases in \( \text{PaO}_2 \). A decrease in \( \text{PaO}_2/\text{FiO}_2 \) (more than 50 mm Hg) was observed more frequently in female patients and in patients with cardiac disease, especially those whose PAP values were above normal. Although a significant decrease in \( \text{PaO}_2 \) during PAOP measurements does not appear to occur often, our results suggest that the changes in \( \text{PaO}_2 \) should be evaluated in patients with severely compromised pulmonary oxygenation.

\[ \text{PAOP} = \text{pulmonary artery occluded pressure}; \text{PAC} = \text{pulmonary artery catheter}; \text{SAP} = \text{systemic arterial pressure}; \text{DAP} = \text{diastolic arterial pressure}; \text{RAP} = \text{right atrial pressure} \]

The measurements of PAOP using a flow-directed, balloon-tipped PAC may produce rapid onset of significant hypoxemia either by temporary cessation of mechanical ventilation\(^{1,2} \) or by occlusion of a major pulmonary arterial vasculature. The former has been well recognized and thus a simple electronic circuit was constructed for the measurements without separation of the patient from the ventilator.\(^{1,2} \) The latter decrease in \( \text{PaO}_2 \) due to inflating the balloon of the PAC per se has recently been reported by us\(^3 \) and others.\(^{4,5} \)

Although the information obtained with inflating the PAC balloon is invaluable and contributes greatly to the management of both critically ill and anesthetized patients, the decrease in \( \text{PaO}_2 \) could cause deterioration of the patient's condition in some situations. Therefore, we expanded our study to know its incidence and to examine the types of patients who tend to develop a remarkable decrease in \( \text{PaO}_2 \) during PAOP measurements and whether the changes in \( \text{PaO}_2 \) are related to the existing level of pulmonary vascular tone.

**Methods**

Studies were performed on 101 anesthetized adult patients (27 to 82 years of age) who were in the supine position and who required a Swan–Ganz triple-lumen PAC and general anesthesia for surgery. This study was approved by our local review committee. Primary disorders in these patients included pulmonary carcinoma (\( n = 21 \)), mitral or aortic valve disease (\( n = 18 \)), coronary artery disease (\( n = 12 \)), other cardiac disease (\( n = 7 \)), hepatic carcinoma (\( n = 10 \)), abdominal aortic aneurysm (\( n = 7 \)) and other disorders (\( n = 26 \)). Anesthesia technique maintained was selected from the following: enflurane, 1.5 to 2.0 percent, inspired, \( \text{N}_2\text{O} \)/\( \text{O}_2 \) moderate-dose Fentanyl, 2 to 10 \( \mu \text{g/kg} \), \( \text{N}_2\text{O} \)/\( \text{O}_2 \); lumbar epidural anesthesia plus \( \text{N}_2\text{O} \)/\( \text{O}_2 \)/\( \text{F} \) high-dose Fentanyl, 70 to 100 \( \mu \text{g/kg} \), \( \text{N}_2\text{O} \)/\( \text{O}_2 \). After induction of anesthesia, a catheter was inserted into the left radial artery and a flow-directed, balloon-tipped catheter (PAC, 7.5 Fr, American Edwards Laboratories) was directed into the pulmonary artery through the right internal jugular vein. The PAC was placed in the most proximal position of the pulmonary artery, which gave a satisfactory PAOP by inflating the balloon with 1.5 ml of air. The location of the tip of the PAC was documented from a chest radiograph taken before surgery. All patients were mechanically ventilated with a \( \text{FiO}_2 \) of 0.33 or 0.5, tidal volume of 10 to 12 ml/kg and respiratory rate of 10 to 12 breaths per minute, without PEEP.

After steady-state condition was established, hemodynamic values of each patient such as SAP, HR, PAP, RAP, and \( \text{Paco}_2 \) (Normocap Days) were measured and recorded continuously. An arterial blood sample was also taken for arterial blood gas analysis (\( \text{PaCO}_2 \), \( \text{pH} \), base excess; Corning, model 175) while the PAC balloon was still deflated in each patient. Then the PAC balloon was inflated for 2 min with 1.5-ml of air and the previously noted measurements were repeated. Cardiac output was measured by the thermodilution method in triplicate with 15 ml of 5 percent dextrose solution with a CO computer (COM-1 TM, American Edwards Laboratories) before inflation of the balloon. All measurements were performed before surgery. Accuracy of the PAOP reading was assured by demonstrating a and v atrial waveforms in its tracing and when the mean PAOP was lower or equal to the DPA.*

Data for paired samples were analyzed using Student's \( t \) test. Chi-square analysis with Yates correction and linear regression analysis with least squares method were utilized to provide type of patients and a relationship between changes in \( \text{PaO}_2 \) and the other variables as appropriate. Statistical significance was accepted when \( p \) values were less than 0.05.

*From Department of Anesthesiology, Institute of Clinical Medicine, University of Tsukuba, Tsukuba City, Ibaraki, Japan. Manuscript received September 6; revision accepted December 19.

Reprint requests: Dr. Dohi, Institute of Clinical Medicine, University of Tsukuba, Tsukuba City, Ibaraki 305, Japan
RESULTS

Inflation of the PAC balloon caused a slight but statistically significant reduction in PETCO₂ (0 to 15 mm Hg, Table 1), but no significant differences in any variable of pulmonary and systemic hemodynamics (Table 2). However, there was a significant decrease (more than 50 mm Hg) in PaO₂/FIO₂ in 15 patients (14.9 percent); seven of these patients (6.9 percent) showed a more remarkable decrease (more than 100 mm Hg) in PaO₂/FIO₂ during PAOP measurement (Fig 1). On the other hand, 11 out of 101 patients (10.9 percent) showed a significant increase (more than 50 mm Hg) in PaO₂/FIO₂. There was no significant correlation between changes in PaO₂/FIO₂ and those in PETCO₂ (r = 0.26), nor between changes in PaO₂/FIO₂ and control PAP (r = 0.09, Fig 2).

Although patients' ages or sizes (body surface area, body mass index and height) did not correlate with the changes in PaO₂/FIO₂, there was a significant difference in the changes in PaO₂/FIO₂ between male and female patients (Fig 1); 11 of 15 patients who showed remarkable decreases in PaO₂/FIO₂ (more than 50 mm Hg) were female.

The patients with coronary artery disease tended to have decreases in PaO₂/FIO₂ (Table 3) and in those with either coronary artery disease or valvular heart disease, there were significant correlations between changes in PaO₂/FIO₂ and control SPAP (Fig 3).

Twenty-nine patients (28.7 percent) were cigarette smokers and they were all male patients. No significant change in PaO₂/FIO₂ was found in both smoking and nonsmoking male patients.

Examinations of the chest radiographs of 60 patients revealed that the tip of the PAC was located either in the right main pulmonary artery (33 patients), the right side of lobar arteries (23 patients), or the left main pulmonary artery (four patients). In the patients with the PAC located in the right main pulmonary artery, PaO₂/FIO₂ decreased significantly during the balloon inflation (p < 0.05). Among five different anesthesia techniques used, the patients with high-dose Fentanyl anesthesia only had a statistically significant decrease in PaO₂/FIO₂ during the PAC balloon inflation (Fig 4).

DISCUSSION

In the present study, we found that only seven of 101 patients (6.9 percent) developed more than a 100 mm Hg decrease in PaO₂/FIO₂ during the inflation of PAC balloon; the majority of patients showed a relatively small decrease or increase in PaO₂. Neither the decrease in PETCO₂ nor baseline PAP was correlated

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Table 1 — Values of Arterial Blood Gases and End-Tidal CO₂ before and during Inflation of the Balloon

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>During</th>
<th>Δ(Range)</th>
</tr>
</thead>
<tbody>
<tr>
<td>PaO₂/FIO₂ (mm Hg)</td>
<td>449 ± 87</td>
<td>439 ± 94</td>
<td>-9 ± 58</td>
</tr>
<tr>
<td>Male (n = 61)</td>
<td>433 ± 89</td>
<td>437 ± 95</td>
<td>3 ± 41</td>
</tr>
<tr>
<td>Female (n = 40)</td>
<td>468 ± 83</td>
<td>440 ± 96†</td>
<td>-28 ± 73</td>
</tr>
<tr>
<td>PaCO₂ (mm Hg)</td>
<td>35 ± 5</td>
<td>35 ± 6</td>
<td>0 ± 3</td>
</tr>
<tr>
<td>pH</td>
<td>7.44 ± 0.05</td>
<td>7.44 ± 0.05</td>
<td>-0 ± 0.02</td>
</tr>
<tr>
<td>Base excess (mEq/L)</td>
<td>0.8 ± 2.4</td>
<td>1.0 ± 2.6</td>
<td>0.2 ± 1.4</td>
</tr>
<tr>
<td>PETCO₂ (mm Hg)</td>
<td>31 ± 5</td>
<td>28 ± 5†</td>
<td>-2 ± 4</td>
</tr>
</tbody>
</table>

*Values are mean ± 1 SD.
†p < 0.05 vs before inflation of the balloon.

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Table 2 — Hemodynamic Variables before and during Inflation of the Balloon

<table>
<thead>
<tr>
<th></th>
<th>Before</th>
<th>During</th>
</tr>
</thead>
<tbody>
<tr>
<td>SAP (mm Hg)</td>
<td>121 ± 23</td>
<td>120 ± 23</td>
</tr>
<tr>
<td>DAP (mm Hg)</td>
<td>60 ± 17</td>
<td>66 ± 15</td>
</tr>
<tr>
<td>HR (breaths per minute)</td>
<td>70 ± 16</td>
<td>70 ± 17</td>
</tr>
<tr>
<td>SPAP (mm Hg)</td>
<td>24 ± 17</td>
<td></td>
</tr>
<tr>
<td>DPAP (mm Hg)</td>
<td>9 ± 4</td>
<td></td>
</tr>
<tr>
<td>PAOP (mm Hg)</td>
<td>...</td>
<td>7 ± 4</td>
</tr>
<tr>
<td>RAP (mm Hg)</td>
<td>6 ± 3</td>
<td>6 ± 3</td>
</tr>
<tr>
<td>CO (L/min)</td>
<td>4.4 ± 1.4</td>
<td></td>
</tr>
</tbody>
</table>

*Values are mean ± 1 SD.

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Figure 1. Changes of PaO₂/FIO₂ during PAOP measurements with respect to gender difference.
with the decrease in PaO₂ during PAOP measurements. The maximum decrease in PaO₂/FIO₂ induced by the balloon inflation was 186 mm Hg in the present series of patients. Such a degree of decrease in PaO₂ must be crucial and large enough to deteriorate oxygen delivery to the tissues if it occurs in patients with cardiopulmonary instability or acute respiratory failure, even though CO remained unaffected during PAOP measurements.

Although the phenomenon caused by the balloon inflation is not the same as that of pulmonary embolism, both the balloon and embolus have a tendency to flow to oxygenated, well perfused lung regions and produce mechanical obstruction of pulmonary blood flow to those regions with a concomitant increase in the dead space. Hypoxemia in acute pulmonary embolism is well known as a consistent and important clinical feature, and its severity seems to be dependent on the degree of occlusion of pulmonary vasculature and probably bronchoconstriction due to released chemical mediators. Although the degree of occlusion can be predicted by the magnitude of Pao₂-PetCO₂ difference in pulmonary embolism, our results indicate that the magnitude of the decrease in PetCO₂ could not be a predictable value for the decrease in PaO₂ during PAOP measurements.

Inflation of the PAC balloon could affect pulmonary oxygenation in at least four ways: by mechanical obstruction of pulmonary blood flow, by neuronal response of pulmonary vasculature, by local changes of existing humoral mediators and thus, probably, by changes in bronchial motor tone. All of these could cause redistribution of pulmonary blood flow within the lungs. The mechanical obstruction of the major pulmonary artery due to the balloon has been considered a main cause for a remarkable decrease in PaO₂ in patients with reduced pulmonary vasculature. Lung size as assessed by total lung capacity and airway area are significantly smaller in female compared with male adults, even in the subjects matched for height. It is thus possible that smaller lung capacity in females could be responsible for enhancing the ventilation-to-perfusion mismatching during the balloon obstruction and thus for the decrease in PaO₂. The balloon inflation might also induce neuronal responses such as reflex pulmonary vasoconstriction and hypoxic bronchial constriction, and pulmonary vascular responses to hypoxia; vasoactive substances are different between male and female animals. Therefore, although no report on gender difference exists in human pulmonary vasoreactivity, we cannot exclude the possibility that neuronal as well as humoral factors might be responsible, to some extent, for the present results. Further, other factors affecting pulmonary vasculature and vasoreactivity, such as cigarette smoking as well as a patient's age, must be taken into account for the present results.

Patients with coronary artery disease or with valvular heart disease seem to be candidates for the PaO₂ decrease when their resting PAP was elevated, as demonstrated in Figure 3. Both inhalational and opiate anesthetic agents also affect pulmonary oxygenation and vasoreactivity, which seem to be dependent on the existing level of vascular tone. On the other hand, sympathectomy of the pulmonary vasculatures, in this case induced by cervical epidural anesthesia, is unlikely to affect the vasoreactivity to the balloon manipulation. Thus, it remains unclear whether the PaO₂ decrease during the balloon inflation was caused by purely mechanical obstruction, and/or by changes in reflex responses due to either anesthetics used or the disease per se.

Table 3—Changes in PaO₂/FIO₂ following the Inflation of the Balloon in Patients with Cardiac Disease, Pulmonary Carcinoma and Other Disorders

<table>
<thead>
<tr>
<th>Primary Disorders</th>
<th>n</th>
<th>Before</th>
<th>During</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valvular heart disease</td>
<td>19</td>
<td>456 ± 68</td>
<td>456 ± 99 (mm Hg)</td>
</tr>
<tr>
<td>Coronary artery disease</td>
<td>15</td>
<td>450 ± 95</td>
<td>418 ± 112 (mm Hg)</td>
</tr>
<tr>
<td>Other cardiac disease</td>
<td>7</td>
<td>483 ± 87</td>
<td>450 ± 71 (mm Hg)</td>
</tr>
<tr>
<td>Pulmonary carcinoma</td>
<td>21</td>
<td>433 ± 104</td>
<td>427 ± 105 (mm Hg)</td>
</tr>
<tr>
<td>Others</td>
<td>39</td>
<td>431 ± 81</td>
<td>440 ± 83 (mm Hg)</td>
</tr>
</tbody>
</table>

*Values are mean ± 1 SD.
†p<0.05 vs before inflation of the balloon.
In addition to significant changes in blood distribution within the lungs, the mechanical obstruction of pulmonary blood flow might increase anastomotic blood flow. The importance of these factors for the changes, both a decrease or an increase in \( \text{PaO}_2 \), could also be dependent on the degree of obstructed area of the pulmonary vasculature and the position of the balloon as indicated in the present results. However, a recent study has verified that the distal tip of the PAC moves synchronously with the balloon inflation and deflation. Since we took the chest radiographs during the deflation, the importance of the influence of the obstructed area due to the presence of the PAC balloon on the changes in \( \text{PaO}_2 \) may need to be reexamined.

We conclude that although pulmonary arterial occlusion by the PAC balloon could not cause severe hypoxemia in the majority of anesthetized, mechanically ventilated adult patients, in some female patients and patients with cardiac and coronary artery disease, a significant decrease in \( \text{PaO}_2 \) could occur during the PAOP measurements probably due to redistribution and/or maldistribution of pulmonary blood flow. Since CO is maintained during the balloon inflation, significant impairment in the oxygen delivery to the tissues is unlikely to occur. However, our results suggest that PAOP measurements may cause impairment in oxygenation in patients with reduced pulmonary vasculature and/or with compromised pulmonary oxygenation such as acute pulmonary failure.

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