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Changes in Pulmonary Oxygenation and Hemodynamic Responses during Pulmonary Arterial Occlusion Pressure Measurements

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Although the use of the balloon-tipped, flow-directed catheter to measure systemic and pulmonary hemodynamics has contributed greatly to the management of both critically ill and anesthetized patients, a few recent case reports indicate that inflation of the catheter's balloon, per se, also alters systemic hemodynamics.^{1,2} These alterations may occur because inflation of the balloon causes blockade of a significant percentage of the pulmonary vasculature with a consequent reduction in cardiac output (CO).^{1,2} Also, occlusion of a major pulmonary artery by inflating the balloon could affect ventilation-perfusion relationships secondary to a sudden decrease in pulmonary perfusion and a concomitant increase in alveolar dead space.³

In previous reports, even unilateral pulmonary artery occlusion has been described to have no effect on heart rate (HR), arterial pressure, CO, and arterial oxygen saturation in spontaneously breathing humans.⁴⁻⁶ However, we frequently observed that end-tidal carbon dioxide concentration decreases transiently during pulmonary arterial occlusion pressure (PAOP) measurements, as described by Perkins and Bedford.⁷ We believe it has not been sufficiently elucidated as to how balloon inflation of

the balloon-tipped, flow-directed catheter affects hemodynamics and pulmonary oxygenation, although the changes in hemodynamics due to pulmonary arterial occlusion or distention have been studied in experimental animals.⁸⁻¹² We therefore undertook the present study to examine how balloon inflation for measurement of PAOP affects routine monitoring variables such as end-tidal CO₂, arterial blood gases, and CO in anesthetized, mechanically ventilated humans.

METHODS

Studies were performed on 31 adult patients (27-78 yr of age) in the supine position who required a Swan-Ganz triple-lumen pulmonary artery catheter (PAC) and general anesthesia for surgery. This study was approved by our local committee. Patients were premedicated with diazepam, 10 mg, po. Anesthesia was induced with thiamylal, 4-5 mg/kg iv, and tracheal intubation was facilitated with succinylcholine, 40-60 mg iv, or pancuronium, 4-6 mg iv. After induction of anesthesia, a cannula was inserted into the left radial artery and a flow-directed, balloon-tipped catheter (7.5 Fr, American Edwards Laboratories, Puerto Rico) 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 roentgenogram taken before surgery. Anesthesia was maintained with oxygen, nitrous oxide (50%), and enflurane (inspired concentration of 1.5-2.0%). All patients were mechanically ventilated with tidal volume of 8-12 ml/kg and respiratory rate of 10-12 breaths/min, without PEEP.

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Hemodynamic variables such as systemic arterial pressure (SAP), heart rate (HR), pulmonary arterial pressure (PAP), and right atrial pressure (RAP) were continuously measured and recorded in all patients in the supine position. ECG and end-tidal carbon dioxide pressure (PETCO₂; Normocap®, Datex, Finland) were also recorded throughout the study. CO was measured by the thermodilution method in triplicate with 10 ml of iced 5% dextrose solution with a CO computer (COM-1™, American Edwards Laboratories). Arterial blood samples were immediately analyzed for oxygen and carbon dioxide tensions (PaO₂, PaCO₂, pH_a; Corning, model 175).

After steady-state condition was established in each patient by controlled mechanical ventilation, the above measurements were taken while the balloon was still deflated. Then, the PAC balloon was inflated for 2 min with 1.5 ml of air and the measurements were repeated. In 19 of the 31 patients, arterial blood samples were taken for blood gas analyses during the inflation of the PAC balloon, and in nine of these patients the measurements were repeated within 1 min after deflating the balloon. Data were analyzed using Student's *t* test for paired data. Statistical significance was accepted when *P* values were less than 0.05.

RESULTS

There were no statistically significant differences in any variables of oxygenation and systemic and pulmonary hemodynamics before or during inflation of the PAC balloon (table 1). However, the inflation of the balloon caused a slight but statistically significant reduction in PETCO₂ (table

TABLE 1. End-tidal CO₂ and Values of Arterial Blood Gases and Hemodynamic Measurements Before, During, and After Inflation of the Balloon

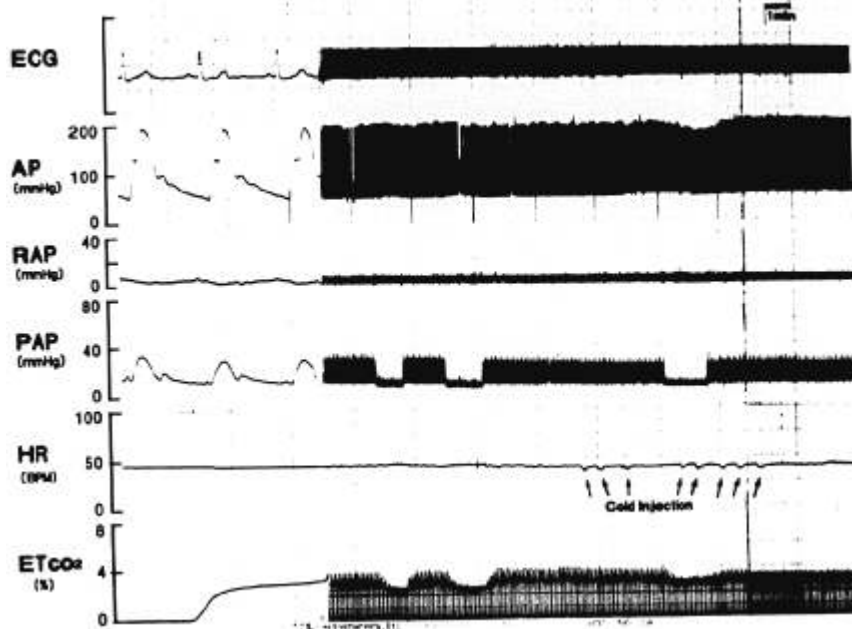
	Inflation of the Balloon		
	Before (n = 31)	During (n = 31)	After (n = 31, or 9†)
PETCO ₂ (mmHg)	33 ± 4	30 ± 5*	34 ± 4
PaCO ₂ (mmHg)	37 ± 6	38 ± 6	36 ± 7 †
PaO ₂ (mmHg)	182 ± 48	171 ± 48	184 ± 42 †
pH _a	7.43 ± 0.06	7.43 ± 0.06	7.43 ± 0.05 †
BE (mEq/l)	1 ± 2	1 ± 2	1 ± 1 †
SAP (mmHg)	123 ± 22	123 ± 22	121 ± 28
DAP (mmHg)	70 ± 12	70 ± 12	70 ± 15
HR (beats/min)	75 ± 13	73 ± 11	72 ± 12
RAP (mmHg)	6 ± 3	5 ± 3	6 ± 3
SPAP (mmHg)	25 ± 6	—	24 ± 6
DPAP (mmHg)	9 ± 5	—	7 ± 6
PAOP (mmHg)	—	9 ± 5	—
CO (l/min)	4.4 ± 1.1	4.3 ± 1.0	4.5 ± 1.2

See text for abbreviations.
Values are mean ± 1 SD.
* *P* < 0.05 vs. before inflation of the balloon.

1, fig. 1); the range of reduction in PETCO₂ was 0–15.2 mmHg. This reduction in PETCO₂ returned immediately (within a few breaths) to preocclusion levels after deflation of the balloon in all patients studied, and PaCO₂ increased slightly (*P* > 0.05, table 1). After deflation of the balloon, all variables returned to the values taken before the inflation (table 1).

Examinations of chest radiographs of the 20 patients by a radiologist revealed that the tip of the PACs was located either in the right main pulmonary artery (ten

FIG. 1. Polygraph tracings of each measured variable. Top to bottom: ECG; arterial pressure (AP); right atrium pressure (RAP); pulmonary arterial pressure (PAP); heart rate (HR); and end-tidal CO₂ concentration (ETCO₂). Notice that inflating the balloon of the pulmonary artery catheter produced a significant decrease in ETCO₂.



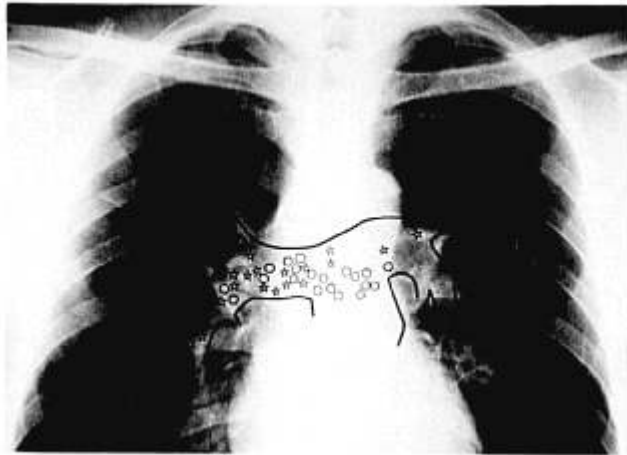


FIG. 2. Positions of both the tip and the thermistor of the PAC were superimposed on a chest radiograph. An asterisk indicates the tip of the PAC and an open circle indicates the position of the thermistor in each of 20 patients.

patients); the right upper lobar artery (one patient); the right interlobar trunk and lower lobar artery (seven patients); or the left main pulmonary artery (two patients) (fig. 2).

Inflation of the balloon of the PAC produced clinically insignificant changes in PaO_2 in most of the patients (slight increase in six patients; increase more than 50 mmHg in two; slight decrease in five; no changes in one, fig. 3). There were three patients, however, who showed a remarkable decrease (more than 50 mmHg) in PaO_2 during inflation of the balloon: one was a patient (PaO_2 decreased from 186 mmHg to 92 mmHg) with the PAC located in the right upper lobar artery; another patient (PaO_2 decreased from 254 mmHg to 166 mmHg) had previously undergone right middle and lower lobectomy; and, in the

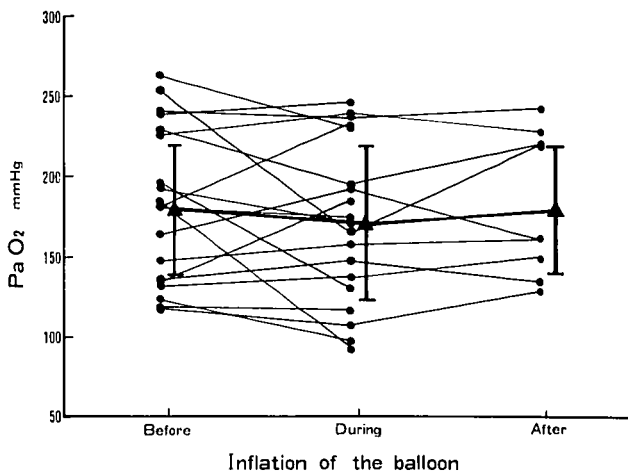


FIG. 3. Effects of inflating the balloon on PaO_2 in 19 patients.

patient with right upper lobe tumor, PaO_2 decreased from 195 mmHg to 132 mmHg. There was no correlation between decreases in PET_{CO_2} and changes in PaO_2 seen during inflation of the PAC balloon and no significant correlation between the changes in PaO_2 due to inflation and control PAP.

The measurements of CO by thermodilution were performed without any difficulty during inflation of the PAC balloon. Bradycardic responses associated with cold injectate¹³ observed in some patients were seen similarly before, during, and after inflation of the PAC balloon (fig. 1).

DISCUSSION

The results of our study indicate that occlusion of a major pulmonary artery by inflation of the PAC balloon led to a small but statistically significant decrease in PET_{CO_2} in anesthetized, mechanically-ventilated subjects without affecting the right ventricular CO as well as systemic hemodynamics. Although it has not been previously described,¹⁴ the decrease in PET_{CO_2} is predictable, because decrease in end-tidal carbon dioxide concentration has been used for proper placement of the PAC catheter⁷ and detection of venous air embolism^{15,16}; in the latter, small air bubbles lodge in peripheral branches of pulmonary circulation and cause mechanical obstruction as well as reflex pulmonary vasoconstriction, resulting in a considerable change in the ventilation-perfusion relationship within the lungs and thus a subsequent increase in alveolar dead space. This could occur similarly during PAOP measurements. Decrease in PET_{CO_2} would reflect changes in pulmonary blood flow or pulmonary perfusion under constant ventilation.^{17,18} Without a significant change of CO during inflation of the PAC balloon, there may not be any change in overall pulmonary blood flow, but some changes in distribution of pulmonary perfusion certainly could occur. Because CO_2 production would not be changed during the balloon inflation, an increase in PaCO_2 should occur because of constant ventilation and the increase in alveolar dead space. However, there were no statistically significant changes in PaCO_2 before, during, and after the balloon inflation. The reason for this is unclear. Because some discrepancies also exist between PaCO_2 and PET_{CO_2} over a period of time in the venous air embolism,¹⁵ decreases in PET_{CO_2} might not be reflected in increases in PaCO_2 within such a short period of time (2 min) for the balloon inflation.

One interesting finding in the present study is a significant decrease in PaO_2 in three patients during inflation of the PAC balloon. With mechanical obstruction of a large pulmonary artery by inflation of the PAC balloon, blood flow to the artery should shift to the other arteries. One patient (60 yr, female, 48 kg) with the PAC located

in the right upper lobar artery showed a remarkable decrease (94 mmHg) in PaO_2 following inflation of the balloon. In an anesthetized, paralyzed, and mechanically ventilated supine subject, the nondependent leaf of the diaphragm has greater excursion during tidal breathing,¹⁹ while regional distribution of pulmonary blood flow remains primarily gravity dependent.²⁰ Therefore, we speculated that the blockade of blood flow to the upper lobe (referred to as zone II²¹) could shift mainly to the lower lobe (referred to as zone III²¹) and result in impaired oxygenation due to a large decrease in the ventilation-perfusion ratio. On the contrary, in most of the other cases in which the PAC located in the lower lobe, it is assumed that a shift of pulmonary blood flow from the lower lobe to the other lobes could have occurred, and thus preserved or improved oxygenation. Even in the cases where the PAC located in the right or left main pulmonary artery, oxygenation was not affected significantly during the inflation. In addition to such conjectured redistribution of pulmonary blood flow, it is also possible that partial obstruction of the pulmonary arterial vasculature during inflation of the PAC balloon could cause local bronchoconstriction due to local hypocapnia,^{5,22} and thus overall ventilation-perfusion relationships could be preserved. However, further studies on the relationship between the changes of PaO_2 and the mechanical effects related to location of the balloon are needed.

Besides the case reported here, we observed another two patients (55 yr, female, 63 kg; 72 yr, male, 51 kg) who also showed a remarkable decrease (88 mmHg, 63 mmHg, respectively) in PaO_2 following inflation of the balloon. One patient previously had had a right middle and lower lobectomy. In such a case with greatly decreased pulmonary vasculature, the shunt effect by the shift of pulmonary blood flow became prominent. In addition, because pulmonary arterial obstruction produces an acute increase in anastomotic bronchial blood flow due to a local increase of prostacyclin and thromboxane in dogs,²³ these humoral agents might contribute to the decrease in PaO_2 during PAOP measurements. Fortunately, in the present series of patients there was no patient in whom PaO_2 decreased lower than 80 mmHg during a 2-min inflation of the PAC balloon. However, there may be some patients with serious pulmonary catastrophe and/or under one-lung ventilation in whom inflating the PAC balloon could cause serious hypoxemia. Because obviously the magnitude of decrease in PET_{CO_2} is not a reliable index for indicating the magnitude of change in PaO_2 during inflation of the PAC balloon, it is crucial to measure PaO_2 in such patients who are needed to inflate the PAC balloon for a little longer time in order to perform examinations such as pulmonary occlusion angiography, which has recently been recommended in acute respiratory failure.^{24,25}

In addition to mechanical obstruction of pulmonary blood flow, inflating a balloon in the pulmonary arterial vasculature might cause an increase in PAP by reflex pulmonary vasoconstriction, as reported in animals,⁸⁻¹¹ and thus a decrease in PaO_2 .^{8,11} This mechanism *via* pulmonary arterial stretch receptors probably could occur even in a case of occlusive distention such as the PAOP measurements. Although we did not measure PAP during inflation of the balloon, increased blood flow to unobstructed pulmonary vasculature could cause pulmonary vascular resistance to decrease due to recruitment or local distention.²¹ Therefore, the occlusive distention could not cause a large increase in PAP as conjectured by the absence of measurable changes in PAP following the deflation and by no changes in HR and SAP during the inflation (table 1). Since potent anesthetic agents suppress the hypoxic pulmonary vasoconstriction,^{26,27} enflurane N_2O anesthesia may have suppressed the reflex pulmonary vasoconstriction. This possible suppressive effect may act beneficially on pulmonary oxygenation. Because, unlike pulmonary air embolism,²⁸ the hypoxic pulmonary vasoconstriction (airway hypoxia) apparently plays no significant role in the pulmonary vascular responses to the balloon inflation, the changes (decrease or increase) in PaO_2 observed are probably dependent on maldistribution or redistribution of pulmonary blood flow through the reflex mechanism along with the mechanical effects and/or the anesthetic effects. In a recent abstract, Spargo²⁹ reported that inflation of the PAC balloon causes a small decrease in PET_{CO_2} , but no change in SAP and HR in intensive care unit patients. Although his results seem to be consistent with ours in anesthetized patients, we cannot exclude the possibility that the anesthetic effects might play an important role in our results.

CO measurement by the thermodilution technique is not possible with the PAC in the wedged position.^{2,29} In our study, the examination of chest radiographs of 20 patients revealed that the thermistor of the PACs was located mainly in the right or left main pulmonary artery (18 patients) and in the proximal part of the right interlobar trunk (two patients) (fig. 2). Although we did not verify the values of CO measured with the thermodilution technique by those with the other methods such as the dye dilution method or the Fick method, as far as the verified location of the thermistors is concerned, interruption of overall pulmonary blood flow seems unlikely to occur during the balloon inflation. In fact, we obtained a smooth thermodilution curve with each measurement, and we did not have any complication related to cold injectate during inflation of the PAC balloon.

We conclude that pulmonary arterial occlusion by the PAC balloon during PAOP measurements causes a significant decrease in PET_{CO_2} but is unlikely to cause impairment in oxygenation and hemodynamics in most su-

pine, mechanically ventilated, anesthetized patients with normal pulmonary vasculature. However, in the patients with significant loss of pulmonary vasculature by disease or surgery and/or those patients with the balloon obstructing the blood flow to the upper lobe, a remarkable decrease in PaO_2 during the balloon inflation can occur. Therefore, the balloon must be deflated as soon as PAOP measurements are taken in such patients to prevent hypoxemia.

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