CLINICAL REPORTS

Ronald D. Miller, M.D., Editor

Anesthesiology 66:214–216, 1987

Decreased Partial Pressure of Oxygen in Arterial Blood Secondary to Inflation of a Pulmonary Artery Flow-directed Catheter Balloon

M. Kainuma, M.D.,* Y. Shimada, M.D.†

In the presence of a pulmonary artery flow-directed catheter, pulmonary blood flow can change, producing decreases in systemic arterial blood pressure during pulmonary artery wedge pressure (PAWP) measurements in adults and neonates.1-3 However, changes in arterial oxygen tension (PaO₂) during PAWP measurement have not been described, probably due to a lack of adequate continuous oxygen tension monitoring. Recently, an indwelling intraarterial oxygen sensor that continuously monitors arterial oxygen tension became commercially available.4 Using this monitor, we describe a case in which a balloon inflation of a pulmonary artery flow-directed catheter caused a significant decrease in PaO₂ during anesthesia and thoracic surgery.

REPORT OF A CASE

A 57-year-old woman (40 kg, 150 cm) was scheduled for removal of a metastatic lung tumor in the right lower lobe of the lung. She had undergone hysterectomy for sarcoma of the uterus 9 months previously and a left lower lobectomy and wedge resections in both lungs for metastatic lung tumors 3 months before. After a short interval, she was readmitted because of a solitary nodule in the right lower lobe suspected to be a metastatic lung tumor. Preoperative lung function tests showed vital capacity (VC) 70% and forced expiratory volume at 1 s (FEV₁) 95% of predicted values. Preoperative examination by cardiologists revealed no abnormality on chest roentgenogram, ECG, and echocardiogram. Other laboratory data were within normal limits.

Atropine, 0.5 mg im, and hydroxyzine, 50 mg im, were given 30 min prior to entering the operating room. Anesthesia was induced with thiopental, 250 mg iv. Succinylcholine, 60 mg, was used to facilitate endotracheal intubation. Anesthesia was maintained with enfurane (2–2.5%), nitrous oxide (50%) in oxygen, and pancuronium. Ventilation was controlled with a tidal volume of 400 ml at a rate of 12 per min. A 7 Fr pulmonary artery flow-directed catheter (American Edwards Laboratories, Santa Ana, CA) was inserted percutaneously through the right jugular vein. Placement of the tip of the catheter at the right main pulmonary artery and wedged to the lower pulmonary artery was confirmed by palpation by a surgeon during surgery and chest roentgenogram after surgery. A 21-gauge thin-walled arterial catheter was inserted percutaneously in the left radial artery. A continuous intraarterial oxygen tension (PaO₂) monitoring sensor (model 636100, Kontron Instruments, Everett, MA) was attached to, and flushed via a standard arterial blood pressure monitoring system. The sensor assembly was then connected to the indwelling arterial catheter and the electrode was advanced into the radial artery so that its tip protruded 10 cm from the end of the catheter. The PaO₂ was continuously recorded before, during, and after surgery. Systemic arterial pressure (SAP), pulmonary arterial pressure (PAP), and central venous pressure (CVP) also were continuously recorded during surgery.

After the patient was placed in the left lateral decubitus position, PaO₂ decreased from 120 mmHg to 92 mmHg secondary to PAWP measurement with 1.2 ml of balloon inflation. A similar phenomenon appeared repeatedly while CVP, SAP, and heart rate did not change, as shown in figure 1. Blood gas measurements were performed three times during the period using a blood gas analyzer (Corning M-175). PaO₂ values were 120, 90, and 149 mmHg, and completely coincided with PaO₂ values at the respective sampling times for PaO₂ measurements. A decrease in PaO₂ started within 15 s and reached a plateau within 120 s after balloon inflation without exception. After thoracotomy and superior segmentectomy of the right lower lobe, a drop of PaO₂ was similarly observed. After the surgery, we examined the effect of PAWP measurement on PaO₂ in the supine position. A decrease in PaO₂ occurred; however, the decrease was less marked in the supine position when compared with the left lateral decubitus position. The postoperative course was uneventful.

DISCUSSION

The pulmonary vascular bed of normal human lungs has a low resistance and high compliance system. Therefore, the hemodynamic effects of inflation of a pulmonary artery flow-directed catheter balloon are generally considered to be negligible.5 However, Willis et al. reported that the SAP was decreased markedly by inflation of a pulmonary artery flow-directed catheter balloon located at the left main lobe pulmonary artery in a patient after right pneumonectomy. Berry et al.1 also presented two cases with right pneumonectomy in which decreases in SAP and cardiac output and an increase in CVP occurred during a PAWP measurement. These patients had a lim-
FIG. 1. Tracing demonstrates systemic arterial pressure (SAP), pulmonary arterial pressure (PAP), central venous pressure (CVP), and intrarterial oxygen tension (P_{aO_2}). When the balloon of the pulmonary artery flow-directed catheter was inflated with 1.2 ml of air, P_{aO_2} markedly decreased, with little change in SAP and CVP.

ulated lung vascular bed due to right pneumonectomy, so the right ventricle could not adequately pump the blood in the face of increased afterload by occlusion with a small balloon.

When the cardiac output decreases during balloon inflation, P_{aO_2} could decrease in the presence of a fixed intrapulmonary shunt. We could not verify this assumption because we did not measure cardiac output at the time of balloon inflation. If this had been the case, P_{aO_2} could have decreased in a similar degree both in the lateral and supine positions. Moreover, we did not observe decreased SAP as reported by Berry et al.\(^1\) at the time of balloon inflation.

The hydrostatic pressure difference caused by gravity in the lung causes a vertical gradient in the distribution of pulmonary blood flow.\(^6\) Consequently, the ventilation–perfusion inequalities in the lung appear more remarkable in the lateral decubitus than the supine position. In this case, blood flow to the upper (right) lung would be less than to the dependent (left) lung. Balloon inflation in the right lower pulmonary artery could have caused additional redistribution of blood from the right lung to the left, resulting in exaggeration of the ventilation–perfusion inequalities and a decrease in P_{aO_2}. On the other hand, in the supine position, the interrupted blood flow in the right lower pulmonary artery by balloon inflation would not have caused major ventilation–perfusion inequalities. In addition, the total pulmonary vascular bed in our patient was smaller than normal due to wedge resections and left lower lobectomy performed 3 months earlier. Consequently, the balloon occlusion could have caused a markedly exaggerated response in redistribution of blood flow in this patient compared with patients with normal lungs, so that the redistributed blood volume from the right lung to the left would have been large enough to decrease P_{aO_2}.

Another possibility to explain these findings is an increased intrapulmonary shunt flow resulting in exaggeration of ventilation–perfusion inequalities. Jindal et al.\(^7\) recognized an acute increase in bronchial blood flow through bronchopulmonary anastomosis beyond the pulmonary arterial obstruction in isolated left lower lobe of anesthetized dogs, although they did not describe alterations in P_{aO_2} during the experiments. It would, however, be difficult to interpret our observations using this etiology, because even a large systemic arterial–pulmonary vein shunt flow through bronchial circulation would produce modest effects on decreases in P_{aO_2}.

We believe the intravascular oxygen sensors accurately reflect P_{aO_2}. Bratanow et al.\(^4\) reported that mean P_{aO_2} values closely followed mean P_{aO_2}(r = 0.97) for 147 samples over a wide range of values. Sensor drift was less than 10% in 24 h during in vitro bench tests. The response time (90%) was 65.3 ± 9.9 s upward and 56.4 ± 4.6 s downward.\(^8\) As the time required for initiating a decrease and plateau in P_{aO_2} are 15 s and 100 s, respectively, the actual decrease in P_{aO_2} started almost simultaneously with balloon inflation with our patient.

In conclusion, inflation of a pulmonary artery flow-directed catheter balloon may be potentially hazardous in hypoxic patients, particularly when the patients are placed in the lateral decubitus position and/or have impaired lung function due to a limited pulmonary vascular bed.
REFERENCES

Anesthesiology

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

SHUJI DOHI, M.D.,* YUMIKO ISHIZAWA, M.D.,† SHIGEYUKI SAITO, M.D.‡

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 thiopental, 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.

* Associate Professor of Anesthesiology.
† Resident of Anesthesiology.
‡ Assistant Professor.

Received from the Department of Anesthesiology, Institute of Clinical Medicine, University of Tsukuba, Sakuramura, Ibaraki 305, Japan.
Accepted for publication September 3, 1986.
Address reprint requests to Dr. Dohi.
Key words: Carbon dioxide; end-tidal; pulmonary blood flow.
Equipment: balloon-tipped, flow-directed pulmonary artery catheter.
Oxygen: tension.