Continuous Cardiac Output Measurement Despite Right Ventricular Rupture and a Malpositioned Pulmonary Artery Catheter

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ABSTRACT

The pulmonary artery catheter is recommended to be placed in the exact location to obtain correct values. We present a case of a pulmonary artery catheter recording continuous cardiac output, even after its tip ended up outside the ruptured right ventricle. An 83-year-old female was scheduled for an emergency operation due to aortic dissection. Preoperative echocardiography showed normal heart with tamponade. In the operating room, insertion of an introducer was uneventful. A pulmonary artery catheter was smoothly inserted, showing typical changes in the pressure waveform. But the pulmonary arterial pressure increased and its waveform dampened soon after. Hypotension and tachycardia occurred, and continuous cardiac output was similar to initial values. After cardiotomy, the surgeon found a laceration of posterior part of the right ventricular wall and the protruding distal tip of pulmonary artery catheter. Repair of ruptured wall and dissected aortic arch was done under hypothermic cardiopulmonary bypass. Weaning from bypass and postoperative recovery were uneventful. In conclusion, the shape of the pulmonary arterial pressure waveform and the ability to measure continuous cardiac output are not indicative of the exact location of the pulmonary artery catheter. We suggest the routine confirmation of the pulmonary artery catheter in correct location by transesophageal echocardiography.

Keywords: Peroperative complications, pulmonary artery catheterization, entricular free wall rupture

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INTRODUCTION

Pulmonary artery catheters (PACs) are commonly used to obtain hemodynamic information, including continuous cardiac output (CCO). Correct hemodynamic values are obtained by placing the tip and thermal filament of the PAC in the pulmonary artery (PA) and right ventricle (RV), respectively (1). We present a case in which a PAC recorded CCO even after its tip ended up outside the ruptured RV.

CASE REPORT

An 83-year-old female (height, 148 cm; weight, 35 kg) patient had an acute aortic dissection (type A), and emergency surgery was planned. Preoperative echocardiography showed normal ventricular wall thickness and contractility (ejection fraction of 65%) with large amount of tamponade.

Initial blood pressure and heart rate were 90/63 mmHg and 75 beats/min, respectively. After induction of anesthesia, the right internal jugular vein was cannulated to insert a 9-Fr introducer in the Trendelenburg position. No resistance or arrhythmia occurred during the guidewire introduction. An 8-Fr PAC was inserted through the introducer sheath after the integrity of its balloon was verified. The balloon was inflated with 1.5ml of air when the PAC recorded a right atrium (RA) pressure waveform. The PAC was passed smoothly into the PA on the first attempt, and typical changes in the pressure waveform from the RA to the RV to the PA were observed. Transesophageal echocardiography (TEE) showed that the tip of the PAC was in the main PA. The PAC balloon was deflated and the catheter was fixed at 38 cm. The central venous pressure (CVP) and pulmonary arterial pressure (PAP) were 8 and 32/13 mmHg, respectively. After calibration of CCO monitoring, a cardiac output (CO) of 2.3 L/min and a systemic vascular resistance (SVR) of 1,230 dynes·s/cm$^5$ were measured. The patient’s
head was rotated to the right for cannulation of the left external jugular vein, at which time the PAP increased to 70–80 mmHg and its waveform dampened. The patient’s systolic blood pressure and heart rate were 60–70 mmHg and 110–120 beats/min, respectively. There were no changes in either the value or the waveform of the CVP, CO, and SVR. Electrocardiography showed sinus tachycardia. TEE confirmed that the ventricle was compressed by an increased amount of tamponade. In addition, the tip of the PAC was not found in the main PA or RV, although the thermal filament of the PAC was shown in the RV on the TEE. Hypotension (80–90 mmHg) and tachycardia (100–110 beats/min) persisted. The CO and SVR were measured as 2.0–2.3 L/min and 1,200–1,250 dynes⋅s/cm$^5$, respectively, although the waveform of PAP was still dampened. We suspected PA rupture by PAC migration. The patient was administered fluid and dopamine, and prompt axillary arterial cannulation for cardiopulmonary bypass (CPB) was performed.

After cardiotomy, the patient’s systolic blood pressure decreased to 50–60 mmHg and fresh blood gushed continuously from the posterior part of the heart. Upon careful examination of the patient’s heart, the surgeon identified a laceration approximately 3 cm in length in the posterior wall of the RV. The distal tip of the PAC protruded through the ruptured wall (Fig. 1). Surgical repair of the ruptured RV and dissected aortic arch was performed under prompt CPB. The patient was weaned from the CPB without incident and no adverse events occurred during the postoperative period.

**DISCUSSION**

RV rupture can occur during catheter insertion or surgical manipulation of the heart (2-6). The former was the more likely cause in this case because, after cardiotomy, the heart was not manipulated except for only simple evacuation of preexisting tamponade. A postoperative
chest X-ray revealed that the introducer tip was located above the RA, and there were no problems during guidewire introduction. Therefore, the cause of the rupture was probably the PAC rather than the introducer-dilator system or the guidewire.

Based on a review of the hemodynamic changes, the most probable sequence of events leading to the rupture was as follows. After insertion of the PAC, TEE confirmed that its tip was in the main PA. In the PAC protocol followed at our hospital, but in contrast to general practice, the PAC is not wedged by advancing it several centimetres beyond the main PA due to the risk of PA injury. In this patient, the fixed depth of the PAC was slightly less than average, although the patient was relatively short. Thus, migration of the PAC into the RV may have occurred when the patient’s head was rotated to the right, during which time the RV wall may have been damaged by the stiff catheter with the deflated balloon, causing it to rupture. Rupture of the RV would have allowed escape of the PAC into the tamponade, leading to a sudden change in the value and waveform indicated by the PAC. The waveform of the PAP may have been dampened because, during the slight movement of the patient during rotation of her head, the tip of the PAC likely made contact with either the external wall of the RV or the pericardium.

Initially, we did not consider the RV rupture as the cause of hemodynamic instability because the thickness of the RV wall was normal. Risk factors for ventricular perforation during catheterization include a small chamber size, a rigid catheter due to hypothermia, outflow tract obstruction, and myocardial infarction (2). The RV wall had not been thinned by a myocardial infarction in this case, unlike previous reports of ventricular perforation during catheterization. However, the PAC may have readily come into contact with the RV wall because of the diminished size of the RV due to the tamponade in this patient. The Trendelenburg position and the hyperdynamic systolic activity may have raised the position of the heart during contraction and contributed to RV rupture.
The PAC used for CCO measurement contains a thermal filament and a thermistor. The thermal filament is 10 cm in length and located 15–25 cm from the distal tip of the catheter, and the thermistor is positioned at the catheter surface 4 cm from the distal tip of the catheter. Commercially available CCO monitoring systems are based on the pulsed warm thermodilution technique, in which a small energy impulse is used as the indicator. The thermal filament emits a pulsed low heat energy signal every 30 to 60 s, allowing blood in the RV to be heated. The thermistor measures minute temperature changes in the PA blood and generates a thermal curve. CO is calculated from the area beneath the thermal curve. The average value of CO measured over the previous 3–6 minutes was displayed on the monitor, which updates CO every 30–60 s (1).

In this case, there was little change in CCO after the PAP wave changed. This suggested that there was no change in the position of the thermal filament and thermistor of the PAC after the RV ruptured. TEE confirmed that the thermal filament of the PAC remained in the RV after the PAP wave changed. The thermistor of PAC was found right outside of the collapsed RV during CPB (Figure). Therefore, the thermistor of PAC may remain in the RV that was filled with blood before CPB, and it may sense all forward flow above the ruptured site. However, the sensed flow may partially escape into the tamponade, and all sensed flow may not be real CO. Therefore, the CCO displayed after the PAP wave dampened may not have been reliable, even though the thermal filament and thermistor of the PAC were in the same position.

In practice, PAC location is speculated with the values and waveforms measured by PAC without additional examination, such as fluoroscopy. PAC migration is also suspected only based on changes in the shape of the PAP waveform and the ability to measure CCO. However, we did not attempt to adjust the location of the PAC on the assumption of PAC migration when the wave of PAP dampened. Instead, we performed a prompt TEE
examination to confirm the location of the PAC. This would increase the likelihood of the early detection of PAC migration and suspicion of an injury by PAC. Recently, routine use of intraoperative TEE was recommended for cardiac and major vascular surgery, in which PAC is inserted. We recommend a confirmatory TEE examination for the location of PAC whenever PAC insertion is completed or PAC migration is suspected.

In conclusion, RV rupture during catheterization may occur even if the RV wall is of normal thickness. Careful catheterization is required, especially in patients with a decrease in the chamber size of the RV. In addition, the ability to measure CCO is not indicative of the exact location of the PAC. A change in the shape of the waveform, indicated by PAC changes, may be suggestive of PAC migration, and the location of the catheter should be confirmed by an additional TEE.
REFERENCES


Malposition of Pulmonary Artery Catheter

Figure: The pulmonary artery catheter protruding from ruptured right ventricle. The arrow indicates the thermistor of the pulmonary artery catheter.