Role of pulmonary artery catheters in critically ill patients

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The pulmonary artery catheter (PAC), which directly measures pulmonary artery pressure (PAP) pulmonary capillary wedge pressure, left ventricular end diasystolic volume and indirectly measures cardiac output (CO), can help to identify different types of shock and mechanical complications of acute myocardial infarction, including mitral regurgitation, ventricular septal perforation, and tamponade. The PAC also has become a common supplement to clinical assessment for guiding the therapeutic management of hemodynamic parameters.

Swan et al[1] introduced the first PAC in 1970. It has since become a common objective supplement to clinical assessment for guiding the therapeutic management of hemodynamic parameters, offering both direct and indirect measurements of cardiac functions. The minimal requirements for using a PAC should be proficiency in reliably placing the PAC, adequate knowledge of the fundamentals of reading and interpreting a tracing, knowing the pitfalls of interpretation, and applying the data to a reasonable therapeutic intervention. Correct interpretation of PAC tracings has been difficult. Morris et al[2] examined 2711 PAC tracings and found that 31% were inadequate for interpretation.

A 67-year-old male with known case of hypertension, right bundle branch block, ischemic heart disease and a heavy smoker for more than 40 years was presented to the accident and emergency unit of Salmaniya Medical Center, Manama, Bahrain with history of sudden onset shortness of breath and chest discomfort. On arrival to emergency room his examination revealed cachectic man, dyspneic, afebrile, pulse of 67 per minute, blood pressure of 190/109. Chest examination also revealed; a unremarkable cardiovascular and abdomen, a Jugular venous pressure not raised. His arterial blood gas on room air was PH 7.361, PO_2 72, PCO_2 60, HCO_3 33, hemoglobin 12 gm/dl, total white cell count of 6,800 with normal differential, platelets urea and electrolytes. The patient was admitted to the hospital and on the way to ward he had a respiratory arrest and was intubated and within 6 hours he was shifted to the Intensive Care Unit (ICU). Chest radiograph and computerized tomography of the chest carried out showed fibrocystic changes of chronic obstructive pulmonary disease. In the ICU, the patient was hypotensive on ventilator, febrile, tachycardic boluses of fluid was given and was started on antibiotics on which the patient responded. On the next 4 days he settled but repeated attempts of weaning from ventilator failed. The next day the patient developed atrial fibrillation as was evidenced by the wave changes in the infero-lateral leads and became hypotensive. He failed to respond to fluid therapy and inotrops was started but even on maximum doses of dopamine, epinephrine and dobutamine, he did not respond and he was continuously hypotensive. Repeated blood culture was negative. Arterial line central venous catheter was then inserted. Echocardiography carried out showed good left ventricular function with ejection fraction of 60%. His thyroid functions was normal. It was then decided to insert PAC to monitor proper hemodynamics. The tracing on the monitor and tip of the catheter at left atrium (west zone III) was confirmed by chest radiograph. His systemic vascular resistance (SVR) was 655, CO 3.1, pulmonary artery occlusion pressure (PAOP) was 9. The interpretation of the reading indicates that the patient was in septic shock, an intravenous fluid boluses was given, inotropes and hydrocortisone 50 mg was then started every 6 hours. He was also given with cefepime 1 gm every 8 hours and vancomycin 1 gm twice daily. Later, blood and deep tracheal aspirate cultures grown methicillin resistant coagulase negative staphyloccus sensitive to cefipime and vancomycine, patient responded by maintaining good blood pressure without inotrops requiring minimum oxygen to maintain oxygen saturation more than 95%. The patient improved dramatically and PAC was removed after 3 days. On day 23, he had percutaneous tracheostomy. Later, the patient developed a critical care illness neuropathy and myopathy, he deteriorated and died.

The history of pulmonary artery catheter illustrates our enthusiasm for new technology and lead us to incorporate its use into daily practice with little, if any, of the rigorous review required for new pharmacologic therapies. From the time that pulmonary artery catheterization was first performed in the mid-1940s until the early 1970s, pulmonary artery catheters were used almost exclusively in catheterization laboratories to determine whether patients with cardiac disease (primarily congenital and valvular defects) were eligible for surgical intervention.[3] The uses of the pulmonary artery catheter expanded from diagnosis alone to include the direction of therapy when Swan et al[1] introduced the balloon-tipped catheter that could be inserted at bedside. No clinical trials were conducted to determine whether patient outcomes were altered by the data derived from insertion of these catheters or the associated therapeutic interventions. Benefit was simply assumed. Complications include arrhythmias (ventricular

Table 1 - Assessment of shock.

Type of shock	CO	SVR	PAOP
Cardiogenic (myopathies, arrhythmias, valvular disease)	Low	High	High
Hypovolemic (hemorrhage, burn, pancreatitis, dehydration)	Low	Low	Low
Distributive (sepsis, anaphylaxis, neurogenic, endocrine)	High	Low	Low
Early sepsis	High	Low	Low
Late sepsis	Low	Low	High or low
Obstructive (tamponade, pulmonary embolism, tension pneumothorax)	Low	High	High or low

CO - cardiac output, SVR -systemic vascular resistance, PAOP - pulmonary artery occlusion pressure

arrhythmia or right bundle branch block, resulting in complete heart block in patient with preexisting left bundle branch block), pneumothorax, pulmonary infarction (if the balloon is left inflated), knotted PACs, pulmonary artery rupture (due to overinflation and usually heralded by hemoptysis), thrombosis or embolism, and infection (potentially leading to sepsis or endocarditis).

In practice, when the CO is thought to be inadequate, direct measurement allows the effects of inotropic agents or mechanical support (balloon pump or ventricular - assist devices) to be assessed. It can be useful to separate the inotropic and chronotropic effects of therapy by determining the stroke volume. Due to CO = heart rate x stroke volume, stroke volume can serve as an index of contractility. Systemic vascular resistance (SVR) is a measure of vascular tone that is calculated from the CO and pressure gradient across the systemic vascular bed [CO = (mean arterial pressure - right arterial pressure) x SVR x 80]. Since SVR is a calculated average resistance throughout the circulation, often it does not reflect the resistance of individual vascular beds. From a practical standpoint, CO and SVR are used to distinguish among the different types of shock (**Table 1**). Low CO is a characteristic of cardiogenic shock, hypovolemia, obstructive shock, and late distributive or septic shock. Low SVR is found in distributive shock, including that which occurs as a result of sepsis or adrenal insufficiency or which has a neurogenic cause. Understanding the pitfalls of PAC data acquisition and interpretation is essential for a valid interpretation. A PAOP is valid only when there is a static but continuous column of blood between the PAC and the j-point. These conditions are met when the PAC is in physiologic west zone III. West[4] conceptually divided the lungs into 3 zones based on the effects of gravity and the pressure of air in the lungs on blood flow. Under some circumstances, gas pressure (in an alveolus of the lung) can be greater than PAP; there is no blood flow to zone I due to the alveoli in this area cause occlusion of the blood vessels. More commonly, blood flow can be intermittent. This occurs in zone II when alveolar gas pressure is lower than PAP but higher than pulmonary venous pressure. Flow is constant only in zone III, where alveolar pressure is lower than both PAP and pulmonary venous pressure. As the PAC is flow-directed, it should float into zone III. One indication that the PAC may not be in zone III is a measure of PAOP greater than the MPAP.[5]

The use of the pulmonary-artery catheter is currently being studied in patients with other clinical syndromes, including acute lung injury and congestive heart failure. The design and execution of these trials have enhanced our understanding of the complexity of studying a technology that is already so widely used in clinical practice. The determination of which clinical questions are important to ask and the designing of appropriate trials with which to answer those questions have led to debates that would not have been considered a decade ago. These debates represent the progress we have made in research related to critical care and the difficulty posed by the legacy of an over-enthusiastic embracing of technology without adequate assessment. I hope that we are learning from our experience.

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