Haemodynamic monitoring: pulmonary artery catheterization

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Of all the monitors at our disposal, the use of the pulmonary artery catheter (PAC) arouses the greatest debate amongst anaesthetists. Incontrovertably, it provides information concerning the cardiovascular and metabolic state of the patient that is not available by more conventional monitoring¹ but it does so at a price. The potential complications described with its use are far from innocuous and the dollar cost of its insertion and treatment of complications can be considerable. Add to this the paucity of literature in which benefits have been demonstrated in terms of mortality and morbidity and it is easy to understand the reluctance with which the PAC has been received into many hospitals. This review will summarize much of the controversy in a discussion of the methodology, complications, interpretations and indications of the use of the PAC.

Methodology^{2,3}

The pulmonary circulation can be catheterized through any large peripheral vein but the most common in anaesthesia practice are the internal jugular and subclavian veins. The internal jugular vein is more accessible and has the advantage of being the shortest and most direct route into the pulmonary circulation. Its most serious disadvantage is the proximity of the carotid artery which, in a recent series, was inadvertently punctured in 1.9 per cent of attempts at internal jugular catheterization. Other disadvantages include post-operative patient discomfort and the difficulty in maintaining sterility with a suitable dressing. The subclavian approach is superior in this respect but the incidence of pneumothorax can be unacceptably high. Catheterization of the pulmonary circulation is easier from the left subclavian vein than from the right. The basilic vein is preferable to either the internal jugular or subclavian veins in those patients

who are anticoagulated because of the ease with which pressure can be applied should it be traumatized.

The insertion of a PAC introducer sheath is facilitated by the availability of disposable kits which invariably employ the Seldinger technique.⁵ In addition most kits include an adjustable, flexible catheter sheath for attachment proximal to the haemostasis valve in order to preserve sterility during manipulations of the catheter.

Prior to insertion, the PAC is connected to suitably balanced, calibrated transducers and the lumens flushed. The transducers should be zero referenced to the antero-posterior midline of the chest and calibrated to a low pressure range. It is important that the balloon is inflated with air before insertion to confirm the integrity of the balloon and determine its maximum volume. Upon insertion to 20 cm the balloon is inflated and the pressure tracings and ECG observed during passage of the catheter into the pulmonary circulation. (See Figure).

From the point of insertion in the internal jugular vein, the tip of the catheter should reach the right atrium after 20–25 cm have been inserted, the right ventricle after 30 cm and pulmonary artery after 40 to 45 cm. Excessive catheter length in the right ventricle carries with it the risk of endocardial damage and knotting of the catheter. The balloon should be maximally inflated so that the catheter tip does not project beyond the balloon. Despite this precaution, PVC's are very common during passage of the PAC through the right ventricle, but the use of prophylactic lidocaine is unwarranted. Progress

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FIGURE Tracing obtained from pulmonary artery catheter during its introduction into pulmonary artery. Note: (a) abrupt increase in pressure upon entering right ventricle; (b) increase in end diastolic pressure – RVEDP; (c) increase in diastolic pressure upon entering pulmonary artery – PADP; (d) dicrotic notch in pulmonary artery pressure waveform; (e) 'a' and 'v' waves in pulmonary wedge pressure.

into the pulmonary artery is more difficult in patients with atrial fibrillation, low cardiac output or right ventricular dilatation and passage can sometimes be facilitated during deep inspiration and "rushing" the catheter.

Verification of correct placement of the catheter can only be made from accurate interpretation of the pulmonary wedge tracing which in turn is dependent on faithful transduction. The natural resonant frequency of the system which facilitates this transduction should be at least ten times the fundamental frequency being measured and should be optimally damped in order to faithfully reproduce the waveform. This can be achieved using short lengths of low compliance pressure tubing with the minimum number of stopcocks from which all air bubbles have been purged. Blood clots should be discouraged from forming by using a continuous flush device with heparin and saline. In the wedged position, the tracing should have the characteristic pressure waveform of the left atrium with discernable 'a' and 'v' waves. The mean pulmonary wedge pressure should be equal to or less than the pulmonary diastolic pressure and the waveform promptly disappear upon deflation of the balloon. It should return promptly upon reinflation to the maximum balloon volume of 1.5 ml for a 7 Fr gauge PAC. Should the wedge tracing appear at a balloon volume of less than 1.5 ml, the PAC should be withdrawn until maximum inflation is possible. The danger of distal migration of the catheter is constant and should always be considered. Maximum balloon inflation in this situation carries with it the risk of pulmonary artery rupture, a potentially catastrophic event. In order to avoid inadvertent

injection of fluid, the syringe used to inflate the balloon should be kept attached at all times between wedge readings and in the deflated position.

Complications^{6,7}

The complications of this procedure can be associated with either the insertion of the PAC or its prolonged presence in the pulmonary artery. A recent retrospective review of over 6000 PAC insertions has put the incidence of complications for this procedure into perspective.⁴ By far the most common complication was the occurrence of dysrhythmias in 72 per cent of patients whereas the total incidence of serious complications was approximately 0.2 per cent. Uncontrollable pulmonary haemorrhage accounted for the one death in the series which was a consequence of PAC insertion.

Reference has already been made to carotid artery puncture and care should be taken to determine the presence of the needle in the artery before the 8 Fr gauge PAC introducer sheath has been inserted. Should the artery be catheterized with the larger sheath prior to cardiac surgery, some authorities have advocated surgical repair before anticoagulation and the institution of cardiopulmonary bypass.⁸ This has been deemed unnecessary so long as firm external compression is applied for five minutes and the area surgically prepared should subsequent haemorrhage be evident.⁴

Other complications of catheter insertion include pneumothorax, air embolism, nerve damage and dysrhythmias. Pneumothorax should be relatively rare from the internal jugular approach although pleural placement of the catheter sheath and subsequent infusion of fluid into the pleural cavity have been reported. Air embolism can be avoided by performing the technique in the head-down position until such time as the introducer sheath has been inserted and the haemostasis valve shown to be competent. It is particularly important that the PAC introducer sheath and haemostasis valve be firmly assembled before use in order to avoid disconnection and possible air embolism during subsequent manoeuvres.

Whereas the vast majority of dysrhythmias spontaneously resolve with the passage of the PAC into the pulmonary circulation, persistent dysrhythmias requiring antiarrhythmic drugs or counter-shock do occur. Their incidence is particularly high in patients with hypoxemia ($\text{PaO}_2 < 60$ torr) and

acidosis ($\text{pH} < 7.0$).⁹ Pre-existing left bundle branch block is not a contraindication to PAC insertion, although a transvenous pacing capability must be available should complete heartblock ensue.⁴

With the placement of the PAC in the pulmonary circulation, complications that can occur are more serious although fortunately less frequent. They include balloon rupture, sepsis, pulmonary infarction and pulmonary artery rupture. Rupture of the balloon should be suspected if full inflation does not meet with the customary resistance and no wedge tracing is observed. Up to 35 per cent of catheters become contaminated but only 2 per cent of insertions are associated with septicemia. Catheter-related infections are more likely if catheters are left in place for more than three to four days in patients with bacteremia or inflammation occurs around the insertion site.

The incidence of pulmonary infarction has decreased substantially over the years from 7.2 per cent in 1974 to 0.64 per cent in the recent report by Shah.⁴ Factors associated with pulmonary infarction are thought to be distal migration of the catheter into the continuous wedge position and thromboembolism. The PAC should never be left in the continuous wedge position and if a persistent wedge trace is observed, the catheter should be withdrawn and repositioned. It is possible that the occurrence of fewer pulmonary infarcts has been related to the use of continuous flush devices and heparin-bonded catheters which may have reduced the incidence of catheter-related thrombogenicity. Pulmonary infarction is often asymptomatic and while the PAC is in place, a chest x-ray should be taken daily to verify the absence of a peripheral opacity.

Pulmonary artery rupture is indeed a dramatic event carrying with it a mortality of 25 to 40 per cent. Risk factors include pulmonary hypertension and anticoagulation. Its highest incidence is during cardiopulmonary bypass when the heart is manipulated causing distal migration of the catheter into an empty, narrowed pulmonary artery tree. In addition, the temperature-sensitive material becomes more rigid during hypothermia thus increasing the chance of perforation by the catheter tip. In situations other than during cardiopulmonary bypass, the likelihood of perforation can be decreased by identifying the pulmonary artery pressure tracing

before slowly inflating the balloon with no more than 1.5 ml of air. Should flushing by hand be required to rectify a damped trace, it is prudent to withdraw the catheter slightly to avoid flushing in the wedged position.

Interpretation^{2,3,10}

Right and left ventricular preload can be estimated from the information derived from the PAC. Starling's Law states that fibre stretch governs the force of contraction of the myocardium but since volume is more difficult to measure, it is inferred clinically from a knowledge of pressure. However, because of the curvilinear relationship between pressure and volume this inference can be quite misleading if ventricular compliance alters. The central venous pressure (CVP) can be measured from the proximal lumen which is located 30 cm from the catheter tip and in the right atrium. With the balloon inflated in the wedge position, flow distal to the PAC ceases and the pressure waveform in the left atrium is transmitted to the distal lumen of the catheter.

There have been numerous papers which have demonstrated the inadequacy of the central venous pressure in predicting the pulmonary wedge pressure (PWP).^{11,12} Correlation often exists between the two but the slope of that relationship is unpredictable and in some patients with poor left ventricular function a negative correlation exists.¹³ What this means clinically is that the PWP cannot be accurately inferred from the CVP in any given patient but must be measured directly.

Pulmonary artery diastolic pressure however can be used as a safer substitute for PWP in the absence of pulmonary hypertension or tachycardia. Indeed the balloon should not be inflated at a pulmonary artery diastolic pressure of less than 10 mmHg because of the risk of pulmonary artery rupture and the failure of the PWP to provide additional information.

The PWP will faithfully reflect LAP only if there is an unbroken column of blood between the catheter tip and the left atrium. This will occur, in the absence of clots, in zone III areas of the lung in which the pulmonary venous pressure is greater than the alveolar pressure.¹⁴ Zone III is not an anatomically distinct area but is a physiological zone dependent upon airway characteristics, position of the patient and intravascular volume status.

Fortunately, it is the area of greatest pulmonary blood flow which facilitates the correct placement of the flow-directed PAC. It is unusual for airway pressure to be transmitted to pulmonary wedge pressure when the PAC catheter tip is below the level of the left atrium. The correct position of the PAC can be verified on lateral chest x-ray. A non-zone III position can be assumed if the PAC tracing reflects airway pressure and loses the characteristic wave-form of the left atrium. Furthermore, if greater than half of the applied PEEP is transmitted to the PWP, then it is probable that the catheter tip is situated in either zones I or II.

The interpretation of the effects of PEEP on the pulmonary circulation can often be confusing. PEEP is transmitted to the pleural cavity and juxtacardiac structures in a degree inversely proportional to the compliance of the lung. It is transmural pressure (intracavity pressure – pressure of juxtacardiac structures) rather than PWP which is a true estimation of filling pressure but the two are often assumed to be equal because the pleural pressure is close to that of the atmosphere. If PEEP is transmitted to the pleural cavity, transmural pressure might be substantially less than measured PWP. The question can be resolved by measuring pleural pressure directly. Alternatively, there is a good correlation between esophageal pressure and pleural pressure when measured in the lateral decubitus position but both manoeuvres have limited clinical application. The removal of PEEP has been advocated while the PWP is measured, but the measurement so obtained clearly no longer reflects the haemodynamic characteristics that formerly prevailed. In addition, the ensuring hypoxia may jeopardize the patient. We recommend that ventilation with PEEP be continued and that the PWP be measured at end expiration. The presence of the catheter tip in zone III should be verified and PEEP confined to less than 10 cm H₂O. In these circumstances, transmural pressure is approximately equal to measured PWP. Should greater levels of PEEP be required, transmural pressure can be estimated by subtracting half the additional PEEP from the measured PWP.

In certain rare circumstances the PWP and pulmonary venous pressure may be considerably greater than LAP. This will occur in situations of pulmonary venous obstruction by mediastinal

fibrosis or tumour, lung tumours, vasculitis or atrial myxomas.

Of fundamental importance in the interpretation of PWP, is the relationship between LAP and left ventricular end diastolic pressure (LVEDP). The clearest waveform of PWP reflects only the LAP but it is the relationship of the latter to the filling of the left ventricle which truly reflects preload. The LAP is greater than LVEDP when the mitral valve is stenosed and similarly, during mitral regurgitation, the contribution of left ventricular systole to LAP can cause LVEDP to be overestimated. It is often possible to detect the reflux of mitral regurgitation by the presence of large V waves on the pulmonary wedge tracing. In contrast, aortic regurgitation can cause the LVEDP to be significantly greater than LAP because the mitral valve closes prematurely as a consequence of retrograde filling of the ventricle from the aorta.

When left ventricular compliance is decreased, LAP no longer reflects LVEDP because of the added boost that atrial contraction imparts to left ventricular preload and systolic volume. In this situation, LVEDP is best determined from the height of the A wave on the wedge tracing.

Finally, it is important to realize the variable relationship between LVEDP and left ventricular end diastolic volume. In order to optimize stroke volume, normal wedge pressures may have to be considerably exceeded in situations of low myocardial compliance due to ischemia, hypertrophy or infiltration. Indeed it is the relationship of PWP to cardiac output in which the use of the PAC is invaluable.

The thermodilution technique of cardiac output determination correlates well with Fick and dye dilution methods and enables periodic haemodynamic profiles of the patient to be performed. In such a way it is possible to determine the ideal PWP that is compatible with maximum cardiac output, and changes in haemodynamic function can readily be detected. The technique relies on the rapid injection of a fixed volume of ice cold saline and assumes a constant blood flow for its passage over the distally placed thermistor. Naturally, the technique is affected by normal stroke volume and respiration, but these effects can be minimized by obtaining the mean of three determinations rejecting those that vary by more than 10 per cent. The

technique is invalid in the presence of intracardiac shunts and tricuspid regurgitation.

The presence of the catheter in the pulmonary artery enables the mixed venous oxygen saturation (SvO_2) to be determined. We have already discussed the information that can be derived from a knowledge of SvO_2 , but briefly it enables the clinician to detect changes in cardiac output and tissue oxygenation. Determination of SvO_2 can be performed in two ways. Blood can be aspirated from the distal lumen and analysed by laboratory oximetry or alternatively, using fibreoptic technology, an online monitor of SvO_2 is currently available. (Oximetrix ShawTM catheter oximeter system, Oximetrix Inc., Mountain View, California.)

Indications

There is a growing consensus of opinion on the indications for the pulmonary artery catheterization in the environment of the Intensive Care Unit.^{6,10} Forrester *et al.* have demonstrated that uncomplicated acute myocardial infarction is not an indication for PAC insertion because of the accuracy with which clinical criteria can predict haemodynamic status.¹⁵ However, should complications supervene which necessitate the administration of inotropic or vasodilator drugs, then it is more accurate to use information derived from the PAC to assess the effects of pharmacological management.

In the absence of acute myocardial infarction, Connors *et al.* have demonstrated that routine clinical evaluation of critically ill patients does not provide enough information to assess haemodynamic status accurately.¹⁶ Indeed clinical criteria correctly predicted cardiac index in only 44 per cent of cases and pulmonary wedge pressure in only 42 per cent of cases, leading them to conclude that pulmonary artery catheterization is indicated in those patients who have not responded to therapy deemed appropriate after careful clinical evaluation.

In the management of adult respiratory distress syndrome, the optimization of PEEP in terms of its effects on oxygen transport, is greatly facilitated if a PAC is in place. The diagnosis of noncardiogenic pulmonary oedema can only be made if the pulmonary wedge pressure is known. Patients

requiring mechanical ventilation for exacerbation of chronic obstructive pulmonary disease often display right ventricular failure but a knowledge of pulmonary wedge pressure may be required to accurately diagnose left ventricular failure.

The indications for pulmonary artery catheterization during anaesthesia have not been carefully studied. It has been suggested that patients with good ventricular function undergoing coronary artery surgery do not benefit from pulmonary artery catheterization¹³ but Mangano's study did not address the issue of variability in regression¹⁷ nor did it explore the benefits of cardiac output determination in the perioperative period. There have been attempts to reproduce Mangano's conclusions for patients undergoing aortic surgery, but determination of preoperative ejection fraction by multigated cardiac analysis scan has not been able to identify subgroups of patients in whom the CVP faithfully reflects the PWP.¹¹

As an index of ischemia, Kaplan and Wells have demonstrated that an increase in PWP antedates changes in the electrocardiogram (ECG), at least in individual patients.¹⁸ Their mean data however were unable to support the superiority of the PAC since ECG evidence of ischemia was accompanied by increases in CVP as well as increases in PWP. Furthermore, Lieberman *et al.* found that neither the CVP nor the PWP were as good a predictor of ischemia as was systemic hypotension.¹⁹ A systemic blood pressure of less than 90 mmHg predicted ECG changes of ischemia 79 per cent of the time whereas increases in CVP above 10 mmHg or PWP above 15 mmHg predicted ischemia just 24 per cent of the time.

There has been a trend towards the use of the PAC in major vascular surgery such as aortic reconstruction stemming from Attia's observation that the application of the aortic cross-clamp can be associated with changes suggestive of altered ventricular compliance in patients with severe coronary artery disease.²⁰ It is well known that at least 50 per cent of these patients have some degree of coronary artery disease and one study has gone so far as to suggest that prophylactic coronary artery surgery be carried out before aortic reconstruction is embarked upon.²¹ Attia observed that the normal response to aortic cross-clamp is a significant decrease in CVP but application of aortic cross-

clamp involved significant increases in PWP but no change in CVP. The authors concluded that PAC facilitates the management of patients undergoing aortic surgery, but it would appear that diligent observation of the CVP will be just as instructive and less invasive.

No study has yet demonstrated the superiority of the PAC over a simple monitor of CVP in terms of its effect on patient outcome. This is remarkable considering the morbidity, effort and expense associated with pulmonary artery catheterization and anaesthetists are justified in being cautious when presented with a list of indications for its use. We believe that all patients undergoing prolonged major surgery should have some monitor of filling pressure. A CVP is sufficient in the majority of patients but with information derived from a thermodilution pulmonary artery catheter, the haemodynamic status of patients with heart disease can be more accurately managed, particularly in the immediate post-operative period.

It is upon arrival in the Recovery Room that the PAC is especially useful. Alterations in systemic vascular resistance can result from a combination of pain, hypothermia, medications and an unstable intravascular volume. Systemic vascular resistance can only be accurately quantitated from a knowledge of cardiac output, and the resultant information provides a rational basis for subsequent management.

In summary, the thermodilution pulmonary artery catheter enables the physician to monitor the filling pressures of right and left ventricles, cardiac output and mixed venous oxygen saturation. It provides the means whereby the haemodynamic status of the patient can be accurately determined and enables the physician to monitor the effects of pharmacological intervention. It behooves the physician to become familiar with the technique, complications, interpretations and indications of pulmonary artery catheterization.

References

- 1 Swan HJC, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. *N Engl J Med* 1970; 284: 447-51.
- 2 Wiedemann HP, Matthay MA, Matthay RA. Cardiovascular-pulmonary monitoring in the Intensive Care Unit (Part 1). *Chest* 1984; 85: 537-49.
- 3 O'Quinn R, Marini JJ. Pulmonary occlusion pressure: clinical physiology, measurement, and interpretation. *Am Rev Respir Dis* 1983; 128: 319-26.
- 4 Shah KB, Rao TLK, Laughlin S, El-Etr AA. A review of pulmonary artery catheterization in 6245 patients. *Anesthesiology* 1984; 61: 271-5.
- 5 Seldinger SI. Catheter replacement of its needle in percutaneous arteriography. *Acta Radiol* 1953; 39: 368-76.
- 6 Wiedemann HP, Matthay MA, Matthay RA. Cardiovascular-pulmonary monitoring in the Intensive Care Unit (Part 2). *Chest* 1984; 85: 656-68.
- 7 Pace NL. A critique of flow-directed pulmonary arterial catheterization. *Anesthesiology* 1977; 47: 455-65.
- 8 Schwartz AJ, Jobes DR, Greenhow E, Stephenson LW, Ellison N. Carotid artery puncture with internal jugular cannulation. *Anesthesiology* 1979; 51: S160.
- 9 Sprung CL, Pozen RG, Rozanski JJ, Pinero JR, Ersler BR, Castellanos A. Advanced ventricular arrhythmias during bedside pulmonary artery catheterization. *Am J Med* 1982; 72: 203-8.
- 10 Goldenheim RD, Kazemi H. Cardiopulmonary monitoring of critically ill patients. *N Engl J Med* 1984; 311: 776-80.
- 11 Martin DE, Nicholas GG, Osbakken MD. Does preoperative ejection fraction predict CVP/PCWP correlation during aortic surgery? *Anesthesiology* 1982; 57: A20.
- 12 Gelman S, McDowell HA, Proctor JE. Do left and right performance curves predict CVP/PAOP correlation during aortic surgery? *Anesth Analg* 1984; 63: 175-284.
- 13 Mangano DT. Monitoring pulmonary arterial pressure in coronary artery disease. *Anesthesiology* 1980; 53: 364-70.
- 14 West JB, Dollery CT, Naimark A. Distribution of blood flow in isolated lung: relation to vascular and alveolar pressures. *J Appl Physiol* 1964; 19: 713-24.

- 15 *Forrester JS, Diamond GA, Swan HJC.* Correlative classification of clinical and hemodynamic function after acute myocardial infarction. *Am J Cardiol* 1977; 39: 137–45.
- 16 *Connors AF, McCaffree DR, Gray BA.* Evaluation of right-heart catheterization in the critically ill patient without acute myocardial infarction. *N Engl J Med* 1983; 308: 263–7.
- 17 *Lowenstein E, Teplick R.* To (PA) catheterize or not to (PA) catheterize – that is the question. Editorial. *Anesthesiology* 1980; 53: 361–3.
- 18 *Kaplan JA, Wells PH.* Early diagnosis of myocardial ischemia using the pulmonary artery catheter. *Anesthesiology* 1981; 60: 789–93.
- 19 *Lieberman RW, Orkin FK, Jobes DR, Schwartz AJ.* Hemodynamic predictors of myocardial ischemia during halothane anesthesia for coronary artery revascularization. *Anesthesiology* 1983; 59: 36–41.
- 20 *Attia RR, Murphy JD, Snider M, Lappas DG, Darling C, Lowenstein E.* Myocardial ischemia due to infrarenal aortic cross-clamping during aortic surgery in patients with severe coronary artery disease. *Circulation* 1976; 53: 961–5.
- 21 *Hertzer NR.* Myocardial ischemia. *Surgery* 1983; 93: 97–101.