Lung (2005) 183:209–219
DOI: 10.1007/s00408-004-2537-9

Lung

© Springer Science+Business Media, Inc. 2005

Principles of Pulmonary Artery Catheterization in the Critically Ill

Eleanor M. Summerhill,[1,2] and Michael Baram[1,3]

[1]Brown Medical School, Providence, Rhode Island, USA; [2]Internal Medicine Residency Program, Memorial Hospital of Rhode Island, 111 Brewster St., Pawtucket, Rhode Island, 02860, USA; [3]Rhode Island Hospital, 593 Eddy St., Providence, Rhode Island, 02903, USA

Abstract. The pulmonary artery catheter (PAC) may be helpful in determining the etiology of shock, lactic acidosis, pulmonary edema, oliguric renal failure, pulmonary hypertension, and a number of cardiac abnormalities. In addition, it may also be useful in guiding fluid and vasoactive therapy. However, although hemodynamic data from the pulmonary artery catheter (PAC) is widely used diagnostically and therapeutically in the care of critically ill patients, the use of the catheter has not been shown to provide outcomes benefit. In fact, there is some evidence to suggest that placement of the PAC may actually be detrimental. The reasons for this are unclear, but it has been shown that both physicians and nurses frequently misinterpret waveforms and other data obtained from the PAC. Presently, there are a number of ongoing randomized, controlled trials investigating the use of the PAC in specific clinical situations and/or patient populations as well as using specific treatment strategies. In the meantime, if any benefit is to be achieved, it is imperative that clinicians have a thorough understanding of the indications, contraindications, complications, and pitfalls of data interpretation prior to using the catheter. These are reviewed in this article.

Key words: Pulmonary artery catheter—Shock—Hemodynamic monitoring—Pulmonary artery wedge pressure.

Introduction

Hemodynamic data obtained from the balloon-tipped pulmonary artery catheter (PAC) has been widely used in critically ill patients, both diagnostically and to

Correspondence to: Eleanor M. Summerhill(401) 729-2522(401) 729-2202; email: Eleanor_Summerhill_MD@Brown.edu

guide therapy, since the development of the catheter more than thirty years ago [17]. Nevertheless, the outcomes benefit of pulmonary artery catheterization has never been proven. In a landmark observational study published in 1996, PAC use was associated with an increased risk of mortality in a group of 5735 mixed medical and surgical ICU patients [3]. This finding has lead to widespread controversy regarding the use of PACs. A 1997 consensus panel urged further investigation in the form of randomized, prospective controlled trials in specific patient populations [12]. These investigations are ongoing today. In the interim, predominant expert opinion supports continued use of PACs in a judicious manner, following careful evaluation of the potential risks and benefits to each individual patient [4, 12].

Given the above considerations, a PAC should be placed only if an accurate assessment of hemodynamic status cannot be determined from a detailed clinical history, careful physical exam, and noninvasive measurements alone. When interpreted and applied correctly, information obtained from the PAC may provide the clinician with more accurate data regarding intravascular volume, cardiac function, and tissue oxygen delivery and utilization in critically ill patients. However, in order for any benefit to be achieved, the clinician must have a thorough understanding of the interpretation and use of hemodynamic data obtained from the PAC. It is clear from multiple studies [1, 6, 7] that both physicians and nurses frequently make erroneous determinations of the pulmonary artery wedge pressure (PAWP), which may lead to inappropriate clinical decision-making. Given all of the above, it is essential that clinicians have an extensive understanding of the indications, contraindications, complications, and pitfalls of data interpretation prior to using the PAC.

Indications

The PAC may be helpful in determining the etiology of shock, lactic acidosis, pulmonary edema (cardiogenic vs. noncardiogenic), oliguric renal failure, pulmonary hypertension, and multiple cardiac abnormalities such as mitral regurgitation, atrial and ventricular septal defects, cardiac tamponade, restrictive cardiomyopathy, and tachyarrythmias. It may also be useful in guiding fluid and vasoactive therapy over time [9]. Traditionally, the PAC has also been used during and after cardiac and major noncardiac surgery in high-risk patients. However, in a large recently published multicenter, randomized controlled trial, no survival benefit was found in a group of elderly high-risk surgical patients assigned to therapy guided by PAC [15]. Another recent multicenter, randomized, controlled study in which 676 adult patients with acute respiratory distress syndrome (ARDS) and/or shock were enrolled also found no significant difference in morbidity or mortality in those patients managed with a PAC versus those who were not [13].

Insertion Technique

The standard PAC is a meter long. It is equipped with proximal and distal ports facilitating measurement of intravascular pressures, infusion of vasoactive agents

and fluids, and blood sampling. At the tip is a thermistor used to calculate cardiac output and a balloon that may be inflated and deflated as necessary. Some catheters are also equipped with an additional right ventricular port for temporary pacemaker insertion.

Before placement, the catheter and tubing must be flushed and filled with fluid through which intravascular pressures are transmitted to a transducer. The equipment is then zeroed to atmospheric pressure at the level of the patient's left atrium (midaxillary line, fourth intercostal space) and calibrated. The electronic signal from the transducer is subsequently amplified and displayed on a monitor. Failure to remove all air bubbles from the tubing may result in "damping" of the waveform tracing, making the characteristic contour of the waveforms less distinct and systolic pressure readings erroneously low. The waveform may also be dampened if a clot obstructs the catheter tip.

The PAC is inserted percutaneously through an 8.5 French introducer, which also serves as an additional venous access port. Because of anatomic ease of insertion, the preferred sites are the right internal jugular, left subclavian, right subclavian, and left internal jugular veins, in that order. Additional sites include the femoral, external jugular, and antecubital veins. Although placement of the catheter is usually guided by pressure waveform monitoring, fluoroscopic visualization may be necessary in some cases, particularly from a femoral site. After the catheter is inserted to the 15–20-cm mark, ensuring that the tip has entered the venous circulation, the balloon is inflated with 1.5 cc of air. From this point forward, the catheter must be advanced with the balloon fully inflated to prevent damage to the myocardium, cardiac valves, or pulmonary artery branches. When the catheter is withdrawn, the balloon must first be deflated to avoid valvular injury.

As shown in Figure 1, the catheter advances first into the right atrium (RA), then across the tricuspid valve into the right ventricle (RV), and across the pulmonic valve into the pulmonary artery (PA). As the catheter continues to advance with the balloon inflated, it will eventually wedge in a branch of the PA, occluding blood flow. The pulmonary artery occlusion, or wedge pressure (PAWP), will then be recorded from the distal lumen. With balloon deflation, a PA pressure tracing will be recorded. From subclavian or internal jugular sites, the RV is generally reached at approximately 30 cm, the PA at 40 cm, and the PAWP at 50 cm. If a PAWP tracing is not obtained after the catheter has been advanced more than 15 cm beyond the RV, the balloon should be deflated and the catheter withdrawn to the RA before attempting placement again. This will prevent excess catheter from coiling and possibly knotting in the RV. After insertion, a chest X-ray is obtained to ensure proper catheter placement and rule out pneumothorax. The catheter tip should be visualized within the proximal third of the hemithorax. Prior to securing the catheter, the balloon is reinflated. If less than 1.0 cc of air is required to obtain a PCWP, the catheter must be pulled back.

Interpretation of Waveform Tracings and PAWP

The characteristic waveforms obtained from the PAC are depicted in Figure 2. RA tracings show relatively small positive deflections known as a and v waves,

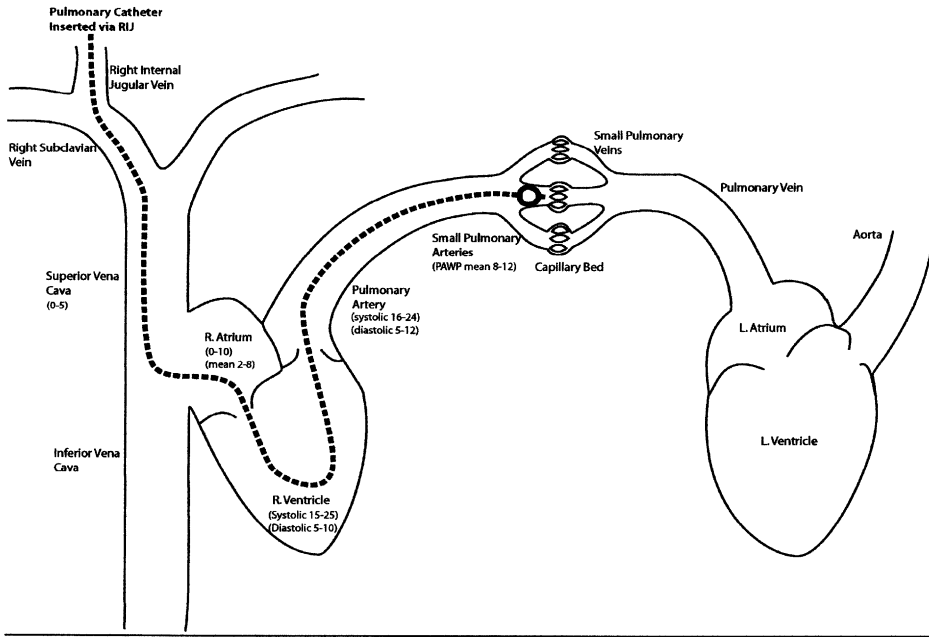

Fig. 1. Diagram depicting the path of the PAC through the right side of the heart and PA, along with the relationship of the PCWP to LA pressure. Normal pressures are given in mmHg. RIJ = right internal jugular vein.

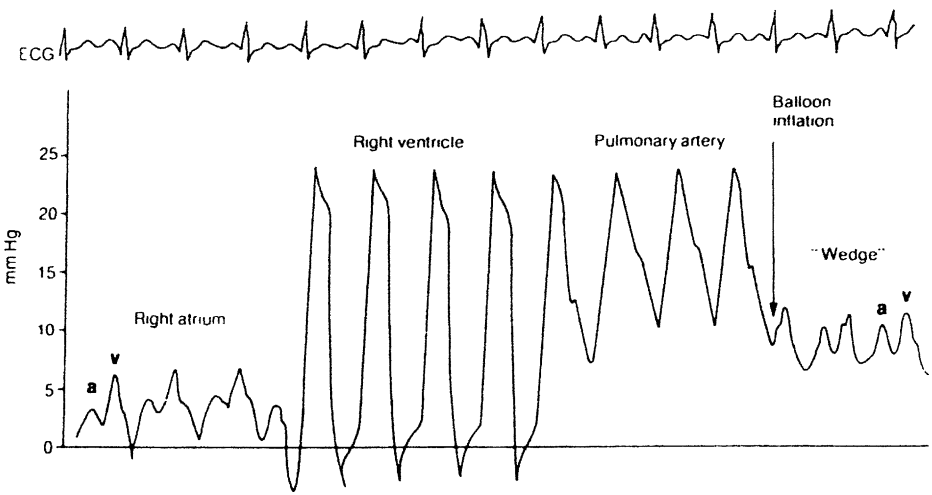

Fig. 2. Characteristic pressure waveforms recorded during PAC insertion. During diastole, when the tricuspid valve is open, pressures in the RA and RV are equal to central venous pressures. After passing through the pulmonic valve, there is a step-up in diastolic pressure as a result of pulmonic valve closure. The high systolic pressures in the RV and PA are generated by RV contraction, with a dicrotic notch representing pulmonic valve closure. The a and v waves seen on the PAWP tracing are transmitted from the LA. [Reprinted from Matthay MA (1983) Invasive hemodynamic monitoring in critically ill patients. Clin Chest Med 4(2):233–249, with permission from Elsevier].

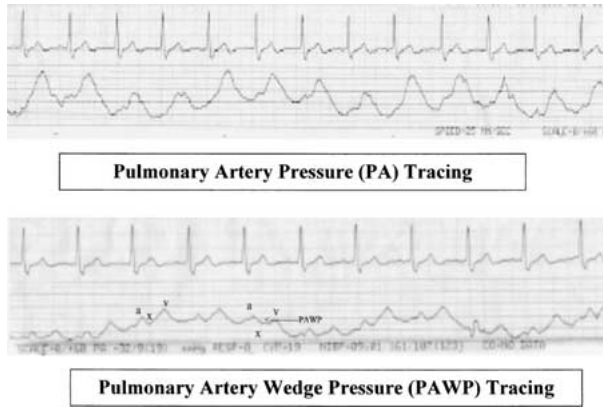

Fig. 3. PA and PAWP tracings from a spontaneously breathing patient with normal respiratory variation. (An easy way to remember the direction of deflection during expiration is the mnenomic: v is for valley in ventilated patients, and a is for apex in the alive and well). The a wave is the first upward deflection seen on the wedge tracing after the p wave on the ECG and represents atrial systole. It is followed by the x descent, the v wave representing ventricular systole, and the y descent. The mean PAWP is measured at end-expiration at the midpoint between the top of the a wave and the nadir of the x descent.

Table 1. Conditions under which PAWP is not equivalent to LVEDP

- Mitral stenosis
- Mitral insufficiency
- Severe aortic regurgitation
- LA myxoma
- Decreased ventricular compliance (myocardial ischemia, pericardial disease)
- Increased ventricular compliance (dilated cardiomyopathy)
- Pulmonary venous obstruction
- Increased end-expiratory pleural pressure (intrinsic or extrinsic PEEP)
- Non-zone 3 placement of catheter

representing atrial and ventricular systole, followed by the x and y descents, respectively. The c wave, representing tricuspid valve closure, and x^1 descent are not always visible. Because of greatly increased systolic pressures, the waveform changes dramatically upon entry into the RV. The PA tracing is notable for a step-up in diastolic pressure along with a distinctive dicrotic notch, representing pulmonic valve closure. Systolic pressures in the RV and PA are equivalent.

The PAWP is recognized by a drop in the mean pressure along with the presence of atrial waveforms (Fig. 3). As depicted in Figure 1, these are transmitted from the left atrium (LA) by a continuous static column of fluid created from the catheter tip to the LA, as long as there is no intervening venous obstruction. This fluid column is also continuous with the left ventricle (LV) when the mitral valve is open. Thus, during diastole, the PAWP represents the left ventricular end diastolic pressure (LVEDP). In general, there is good correlation between PAWP, LA pressure, and LVEDP [10]. Situations in which PAWP *does not* reflect LVEDP are listed in Table 1.

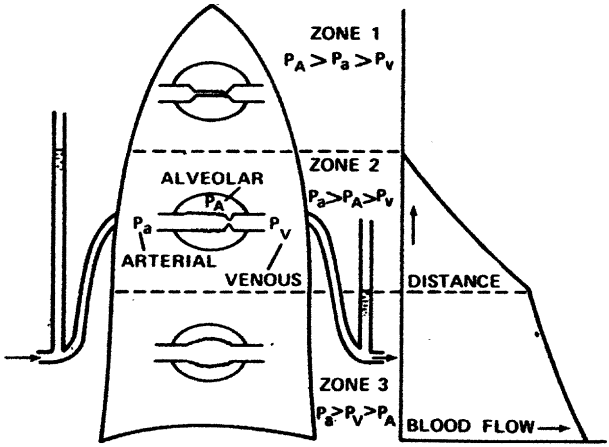

Fig. 4. Regional blood flow distribution zones in the lung [from West JB, Dollery CT, Naimark A (1964) Distribution of blood flow in isolated lung; relation to vascular and alveolar pressures. J Appl Physiol 19:713–724, used with permission].

Pitfalls in PAWP Interpretation

The most common sources of error in PAWP interpretation include: (l) failure to correctly identify end-expiration when determining the mean PAWP, (2) failure to adjust for the effects of positive end-expiratory pressure (PEEP), and (3) placement of the catheter in non-zone 3 lung [10].

The PAWP must be measured at end-expiration, when pleural pressures are close to zero. It is important to remember that in spontaneously breathing patients, intrathoracic pressures are *negative* during inspiration and *positive* during exhalation. Thus, the negative deflection in the PAWP tracing will occur during inspiration and the positive deflection during exhalation. If the patient is on positive-pressure ventilation, the situation is usually reversed. However, if the patient is struggling against the ventilator, there may be large negative deflections with attempted spontaneous inspiration. In these cases, observation of the duration of positive and negative deflections may be helpful in determining end expiration. Normally, inspiratory time is greater than expiratory time, with inspiratory to expiratory (i:e) ratios on the order of 1:2 or 1:3. Remember this will not be the case if the patient is ventilated in an *inverse* ratio mode.

Identification of end-expiration may be particularly difficult in the patient with tachypnea or large respirophasic intrathoracic pressure swings. Therefore, it is important to manually interpret the strip recording rather than relying on automated computations. Brief paralysis with neuromuscular blocking agents may be helpful in determining PAWP on these tracings. When a and v waveforms are prominent on the PAWP tracing, the mean pressure should be measured at the halfway point between the peak of the a wave and the nadir of the x descent [16].

Both extrinsic PEEP (applied through the ventilator) and intrinsic PEEP (as a result of expiratory flow limitation and dynamic hyperinflation) may result in an overestimate of PAWP, and therefore LVEDP, due to increased intrathoracic

Table 2. Methods to confirm lung zone 3 catheter placement

• Tip of the catheter at or below the LA on lateral supine chest radiograph
• Clearly delineated atrial waveforms
• Respiratory variation of PAWP is ≤ 50% static airway pressure (peak − plateau)
• No more than 50% of an increase in extrinsic PEEP should be transmitted to the PAWP

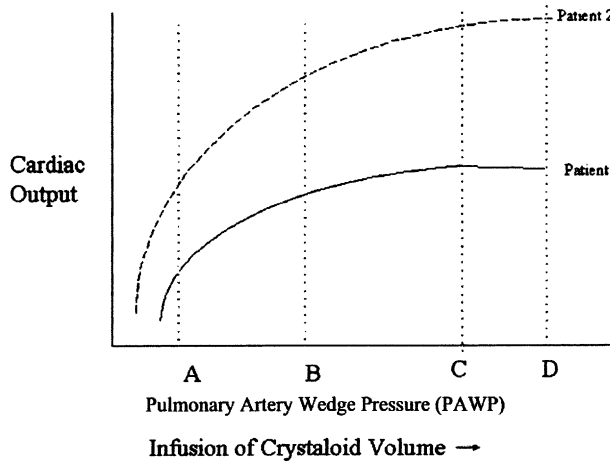

Fig. 5. The Frank–Starling principle states that the strength of cardiac contraction depends on muscle fiber length at end-diastole (preload). By infusing volume, a Starling curve of the heart may be generated. In patient 1, fluid administration increases PAWP/LVEDP, with resultant increases in CO until point C, after which there is no further improvement (point D). Thus, the ideal preload is achieved at point C. In contrast, because of differences in ventricular compliance and/or afterload, with further increases in PAWP/LVEDP, further improvement in CO is seen up to point D.

pressures. To determine the true transmural pressure, an esophageal balloon may be placed, or PEEP may be withdrawn for a very brief period of time (1–2 cardiac cycles) [9]. The first method requires equipment that is not readily available, and the second may cause instability in the patient's respiratory and/or cardiac status. A more practical approach may be to make a rough estimate of the effects of PEEP. In two clinical studies, levels of PEEP $<$ 10 cm H_2O in patients with ARDS did not significantly alter PAWP measurements. At levels of PEEP $>$ 10 cm H_2O, the measured PAWP rose by 2–3 cm H_2O with every 5 cm H_2O increase in PEEP [5, 8].

Measurement of PAWP under non-zone 3 conditions is the third major source of error. Non-zone 3 regions are areas of the lung where mean pulmonary vein (PV) pressure is less than alveolar pressure (Fig. 4). If the catheter is in a region where alveolar pressure is greater than PV pressure, then the measured PAWP will reflect alveolar pressure rather than the LVEDP. Methods to determine zone 3 catheter placement are listed on Table 2 [9, 10].

Because myocardial contractility, compliance, and afterload affect the relationship between PAWP/LVEDP and cardiac output (CO), the optimal PAWP is highly invidualized. Therefore, it is important to follow the Frank–Starling curve

Table 3. Calculation of cardiac output via the Fick method

$CO = \frac{VO_2}{10(CaO_2 - CvO_2)} = \frac{VO_2}{10[(SaO_2 - SvO_2)(1.34 \text{ ml } O_2/\text{g Hgb})(\text{Hgb g/dl})]}$

CO = cardiac output (L/min)

VO_2 = oxygen consumption (ml/min); VO_2 may be roughly estimated as [125 cc/min/m^2 × BSA (m^2)]

CaO_2–CvO_2 = ateriovenous oxygen content difference (ml/L)

SaO_2 = arterial O_2 saturation; measured from arterial blood

SvO_2 = mixed venous O_2 saturation; obtained from the PA in the absence of an intracardiac shunt

Hgb = serum hemoglobin

Table 4. Calculation of systemic and pulmonary vascular resistances

$SVR = \frac{MAP - Pra}{CO}$

SVR = systemic vascular resistance (dyns/cm^5)

MAP = Mean arterial pressure

Pra = right atrial pressure

$PVR = \frac{Ppa - Ppw}{CO}$

PVR = pulmonary vascular resistance (dyns/cm^5)

Ppa = mean pulmonary artery pressure

Ppw = mean pulmonary artery wedge pressure

over time in each patient, measuring PAWP, systemic blood pressure, and CO following serial fluid challenges. Confounding variables such as vasoactive drugs and PEEP must be kept constant during this period of time (Fig. 5). Hydrostatic (cardiogenic) pulmonary edema becomes a consideration at PAWP over 22–25 mmHg [9].

Cardiac Output Measurement

CO is usually obtained by the thermodilution method, whereby 10 cc of cold saline is injected through the proximal (RA) port. This mixes with blood traveling through the RV and into the PA. There the temperature drop is sensed by the thermistor. The area under the temperature–time curve is integrated and is inversely proportional to the flow rate, or CO. As this measurement is highly dependent on the rate at which the saline is injected, it may be quite variable. Therefore, five measurements are obtained, and the three most consistent averaged. CO measurements may be inconsistent between operators and also subject to equipment error after the catheter has been in place for several days. Both left-to-right and right-to-left intracardiac shunts may lead to an overestimation of the CO. In the setting of tricuspid regurgitation, the thermodilution method may either underestimate or overestimate the CO [9, 11, 16]. If questions regarding accuracy arise, calculating the CO by the Fick equation may be helpful (Table 3).

Systemic and Pulmonary Vascular Resistance

Systemic vascular resistance (SVR) and pulmonary vascular resistance (PVR) are not directly measured but may be calculated from data obtained from the PAC.

Table 5.. Complications of pulmonary artery catheterization

Complications of central venous access	Bleeding Hematoma Arterial puncture or cannulation Infection Pneumothorax Hemothorax
Arrythmias	Atrial tachyarrythmias Ventricular tachyarrythmias Right bundle branch block Complete heart block
Catheter-induced injury	Myocardial perforation Valvular injury Pulmonary artery rupture Pulmonary infarction Catheter entrapment on intravascular devices Catheter knotting Thrombosis and embolism Air embolism
Thrombocytopenia	(Catheters are heparin-impregnated)
Misinterpretation of data	

These are derived from Ohm's law: Voltage (V) = Current (I) × Resistance (R). As applied to human physiology, these equations are stated in Table 4. SVR and PVR represent the afterload on the left and right sides of the heart, respectively. It is important to remember that an error in any one of the input variables may result in a large error in the calculation of vascular resistance.

Contraindications

Most contraindications to the insertion of a PAC are relative. If the patient has significant coagulopathy, prudence dictates correction prior to catheter insertion if at all possible. If significant uncorrected coagulopathy is present, the preferred insertion sites are the femoral or internal jugular vein, as these veins are externally compressible. Human recombinant activated protein C therapy must be discontinued for at least 2 hours prior to catheter placement and withheld until adequate hemostasis is achieved following insertion [2]. Catheterization must also be withheld within 24 hours of thrombolytic therapy.

Complications

The complications associated with PAC use are listed in Table 5. A few of the more clinically important complications are reviewed here. Ventricular arrythmias

during passage of the catheter are fairly common, ranging from an estimated incidence of 11%–68% [9]. Critically ill patients in the ICU may be more prone to developing arrythmias because of electrolyte disturbances, acidosis, ongoing myocardial ischemia, hypoxemia, and elevated endogenous and exogenous catecholamine levels. Metabolic disturbances should therefore be corrected prior to placement if possible, and the catheter should be advanced rapidly through the RV. In some instances, the catheter may need to be withdrawn to the RA and antiarrythmics given prior to further catheterization attempts.

Transient right bundle branch block (RBBB) occurs in 0.05%–5% of catheterizations. In a series of 82 patients with left bundle branch block (LBBB), only two episodes of complete heart block (CHB) occurred [9]. Patients at greatest risk of developing CHB are those with new LBBB, as in acute myocardial infarction [10]. In the setting of longstanding LBBB, as a precaution external pacing should be set up prior to catheter insertion. Higher-risk patients with new LBBB and a very compelling indication for PAC placement should first receive prophylactic transvenous pacemaker placement.

Migration of the catheter tip or failure to deflate the balloon may result in pulmonary infarction distal to the occluded arterial branch. The most serious complication of PA catheterization is pulmonary artery rupture, which is heralded by hemoptysis and is usually fatal. Risk factors include pulmonary hypertension, balloon migration, and hypothermia [18].

Conclusion

The PAC has been extensively used in the ICU setting, without evidence of proven outcomes benefit. Rather, in 1996, the results of a study by Connors et al. [3] raised concerns regarding the safety of continued PAC use. These findings may have been due to methodology, directly related to known complications of PAC use, or related to the inability of health professionals to accurately interpret and/or make appropriate clinical decisions based on data obtained from the catheter. Thus, it is very important that clinicians develop an extensive familiarity with the indications, complications, and principles of PAC use. In addition, further studies are needed to define patient populations most likely to benefit from the use of the catheter and to determine whether specific treatment strategies such as early goal-directed therapy [14] based on data obtained from the PAC might improve outcomes.

Acknowledgments. The authors would like to thank Randy Sid, M.D., for his artwork in Figure 1.

References

1. Al-Kharrat T, Zarich S, Amoateng–Adjepongy, Mantous CA (1999) Analysis of observer variability in measurement of pulmonary artery occlusion pressures. Am J Respir Crit Care Med 160:415–420

2. Bernard GR, Vincent JL, Laterre PF, LaRosa SP, Dhainaut JF, Lopez-Rodriguez A, et al. for the Recombinant Human Protein C Worldwide Evaluation in Severe Sepsis (PROWESS) Study Group (2001) Efficacy and safety of recombinant human activated protein C for severe sepsis. N Engl J Med 344:699–709
3. Connors AF Jr, Speroff T, Dawson NV, Thomas C, Harrell FE Jr, Wagner D, et al.(1996) The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 276:889–897
4. Dalen JE, Bone RC (1996) Is it time to pull the pulmonary artery catheter? JAMA 276:916–918
5. Dhainaut JF, Devaux JY, Monsallier JF, Brunet F, Villemant D, Huyghebaert MF. (1986) Mechanisms of decreased left ventricular preload during continuous positive pressure ventilation in ARDS. Chest 90:74–80
6. Iberti TJ, Fischer EP, Leibowitz AB, Panacek EA, Silverstein JH, Albertson TE and the Pulmonary Artery Catheter Study Group (1990) A multicenter study of physicians' knowledge of the pulmonary artery catheter. JAMA 264:2928–2932
7. Iberti TJ, Daily EK, Leibowitz AB, Schecter CB, Fischer EP, Silverstein JH and the Pulmonary Artery Catheter Study Group (1994) Assessment of critical care nurses' knowledge of the pulmonary artery catheter. Crit Care Med 22:1674–1678
8. Jardin F, Farcot JC, Boisante L, Curien N, Margairaz A, Bourdarias JP (1981) Influence of positive end-expiratory pressure on left ventricular performance. N Engl J Med 304:387–392
9. Leatherman JW, Marini JJ (1998) Clinical use of the pulmonary artery catheter In: Hall JB, Schmidt GA, Wood LDH (eds) Principles of Critical Care 2nd ed. McGraw-Hill, New York, pp 155–176
10. Matthay MA, Dorinsky PM (2000) Managing the patient with hemodynamic insufficiency, shock, and multiple organ failure In: George RB, Light RW, Matthay MA, Matthay RA (eds) Chest Medicine: Essentials of Pulmonary and Critical Care Medicine 3rd ed. Williams and Wilkins, Baltimore, pp 609–618
11. Perret C, Tagan D, Feihl F, Marini JJ (1996) The Pulmonary Artery Catheter in Critical Care Blackwell Science, Ltd., Cambridge, UK
12. Pulmonary Artery Catheter Consensus Conference (1997) Consensus statement. Crit Care Med 25:910–925
13. Richard C, Warszawski J, Anguel N, Deye N, Combes A, Barnoud D, et al. for the French Pulmonary Artery Catheter Study Group (2003) Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome. JAMA 290:2713–2720
14. Rivers E, Nguyen B et al Havstad S, Ressler J, Muzzin A, Knobtich B, et al. for the Early Goal-Directed Therapy Collaborative Group (2001) Early goal-directed therapy in the treatment of severe sepsis and septic shock. N Engl J Med 345:1368–1377
15. Sandham JD, Hull RD, Brant RF, Knox L, Pineo GF, Doig CJ, et al. (2003) A randomized, controlled trial of the use of pulmonary-artery catheters in high-risk surgical patients. N Engl J Med 348:5–14
16. Sharkey SW (1997) A Guide to Interpretation of Hemodynamic Data in the Coronary Care Unit Lippincott, Williams and Wilkins, Philadelphia
17. Swan HJC, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D (1970) Catheterization of the heart in man with the use of a flow-directed balloon-tipped catheter. N Engl J Med 283:447–451
18. Weidemann HP, Matthay MA, Matthay RA (1984) Cardiovascular–pulmonary monitoring in the intensive care unit (Part 2)? Chest 85:656–668

Accepted for publication: January 12, 2005