The Pulmonary Artery Catheter
A Critical Reappraisal

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INTRODUCTION

Balloon floatation pulmonary artery catheters (PACs) have been used for hemodynamic monitoring in cardiac, medical, and surgical intensive care units (ICUs) since the 1970s. In cardiac catheterization laboratories, balloon floatation catheters are routinely used for right heart catheterization. It was estimated in 2000 that approximately 1.5 million balloon floatation catheters were sold annually in the United States.1 Approximately 30% of these catheters were used in cardiac surgery, 30% in coronary care units and cardiac catheterization laboratories, 25% in high-risk surgical and trauma patients, and 15% in medical ICUs. Right heart catheterization is performed primarily to determine hemodynamics and measure cardiac output, typically for the purpose of establishing a diagnosis of underlying pathology and also to guide therapy. With the availability of noninvasive diagnostic modalities, particularly echocardiography, the frequency of diagnostic pulmonary artery (PA) catheterization has declined.

In this review, the evolution of PACs, the results of nonrandomized and randomized studies in various clinical scenarios, the uses and abuses of bedside hemodynamic monitoring, and current indications of PA catheterization in critical care units are discussed.

EVOLUTION OF PA CATHETERS

The history of cardiac catheterization dates back to the early nineteenth century.2 In 1844, Claude

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Bernard performed right ventricular (RV) and left ventricular (LV) catheterization in horses by inserting glass tubes into the jugular veins and carotid arteries. In the 1800s, the performance of cardiac catheterization in horses to measure intracardiac pressures became a common practice among other notable physiologists as well, including Jean-Baptiste Chauveau and Etienne Marey. In the early 1900s, Fritz Bleichroder, Ernest Unger, and W. Loeb performed cardiac catheterization in dogs, testing the effect of drugs injected into the central circulation.

Dr Werner Forssmann is credited with performing the first human right heart catheterization. In 1929, he introduced a urethral catheter into his left antecubital vein and advanced the catheter to his right atrium (RA), documenting this self-catheterization by x-ray. Unfortunately, following publication of this landmark effort, Dr Forssmann lost his clinical practice privileges because of unfavorable press.

In the 1940s, Drs Andre Cournand, Hilbert Ranges, and Dickinson Richards developed catheters that could be advanced into the pulmonary artery and used these catheters in the cardiac catheterization laboratory to study hemodynamics in patients with congenital and acquired heart diseases. Working at Bellevue Hospital in New York, Andre Cournand and Dickinson Richards used PACs not only to measure intracardiac pressures, but also to obtain true mixed venous blood samples from the PA. This permitted measurement of cardiac output by the direct Fick principle for the first time. Cournand and Richards also used PA catheterization for analysis of blood gases, pH, respiratory gas exchange, and blood volume in healthy individuals as well as in patients with cardiopulmonary disease. In 1956, Drs Andre Cournand, Dickinson Richards, and Werner Forssmann were awarded the Nobel Prize in Physiology or Medicine for their pioneering contributions in cardiac catheterization.

In 1949, the concept and technique of measuring pulmonary capillary wedge pressure (PCWP) was developed in the cardiac catheterization laboratory of Dr Lewis Dexter at the Brigham and Women’s Hospital, Harvard Medical School. PCWP was measured by advancing the PAC to the distal branches of the PA. It was assumed that PCWP reflected LV diastolic pressure. In 1953, Lategola and Rahn developed a self-guiding balloon-tipped catheter that could be advanced into the PA and thence into the wedge position.

The development of PACs for a variety of clinical scenarios continued. Dr H.J.C. ‘Jeremy’ Swan, an Irishman and cardiovascular researcher in London moved to the US for a fellowship at the Mayo Clinic in 1951. There he worked with Earl Wood and furthered his interest in cardiac and pulmonary vascular physiology. In 1964, R. D. Bradley, a British intensivist whom Swan had mentored whilst in the UK, developed miniature catheters that could be guided into the PA. In 1965, Drs Fife and Lees constructed self-guiding PACs and used these catheters for a variety of physiologic studies. In 1969, Dr Melvin Scheinman and his colleagues at San Francisco General Hospital, used flow directed catheters to measure right heart pressures.

Meanwhile, Dr Swan at Cedars-Sinai, where he had moved in 1965, was having little success with Bradley’s catheters and wrestled with a design to make it easier to “float” the PAC into the right heart and beyond. In his words: “In the fall of 1969, I was on the beach in Santa Monica, California, with my young children and noted a sailboat with a large spinnaker making good progress in a calm sea. I wondered whether a sail or parachute at the tip of a flexible catheter would solve the problem. I had been a consultant to Edwards Laboratories for several years and brought this proposal for discussion. David Chonette, new product manager, did not favor the solutions suggested, but proposed a small inflatable balloon that would be relatively easy to fabricate. The balloon worked superbly, and sail and parachute were abandoned.” In 1970, Drs Jeremy Swan, William Ganz, and Forrester published details of the first ‘balloon tipped flow directed’ catheters which could be used in patients in the intensive care unit without the use of fluoroscopy. These catheters had two lumens – one to inflate the balloon and the other to record pressure.

The “Swan-Ganz” catheter was further developed by Ganz to measure cardiac output by the thermodilution method. Subsequently introduced balloon flotation catheters with pacing electrodes, which could be used for atrial, ventricular, and atrioventricular sequential pacing. Following this, further modifications included adding additional ports for hemodynamic monitoring and medication infusion, cardiac pacing, and even placing an oximeter at the tip, which allowed for continuous SvO₂ monitoring. Fig. 2 shows the most common device in use today. It is a 7.5-Fr catheter inserted through an 8.5-Fr introducer sheath and has 3 ports for pressure monitoring and infusions, a thermistor probe 3 cm proximal to the tip, and a balloon at the tip inflated via a valve at the hub.
PLACEMENT OF BALLOON FLOATATION CATHETERS AND HEMODYNAMIC MEASUREMENTS

The PAC is typically placed at the bedside without fluoroscopy. Access is established in a central vein as usual and a 10-cm 8.5-Fr introducer inserted. The PAC is then ensheathed in a sterile sleeve (that allows for future sterile manipulation) and passed through the valve in the introducer hub. Between 11 cm and 15 cm, the balloon is inflated and the catheter advanced. The balloon should always be fully inflated during catheter advancement, and fully deflated during catheter withdrawal.

As the catheter is advanced, its position can be identified by characteristic pressure wave forms. When the catheter is in the RA, the RA pressure wave form is noted. The RA pressure wave forms are characterized by two positive waves: an “a,” which occurs during RA systole and a “v,” which occurs during the end of right ventricular (RV) systole. It should be appreciated that in atrial fibrillation, “a” waves are absent. There are also two negative waves: the “x” descent caused by atrial relaxation and the “y” descent, which occurs during RV rapid filling. In atrial fibrillation, the “a” upstroke and the “x” descent are absent. A positive wave called the “c” wave is frequently recognized during the “x” descent. The “c” wave results from doming of the tricuspid valve into RA during RV isovolumic systole.

With further advancement of the catheter, the balloon tip crosses the tricuspid valve and an RV pressure wave form is seen (Fig. 3). The RV pressure waveform is characterized by a sharp upstroke during the isovolumic phase of systole. During the ejection phase, there is much slower rise in pressure. During the isovolumic relaxation phase, there is a sharp downstroke in the RV pressure wave form. Because of the tricuspid valve, the RV systolic pressure is always higher than the RA pressure. During diastole, a rapid filling wave, diastasis, and atrial filling waves are recognized. During the rapid filling phase, RV diastolic pressure rises with inflow of blood from the RA. During the phase of diastasis, there is no further rise in RV pressure, as inflow from the
RA ceases due to the lack of a pressure gradient between the 2 chambers. The atrial filling wave is related to RA contraction at the end of diastole. In atrial fibrillation, the atrial filling wave is absent.

When the catheter with the balloon inflated is advanced from the RV, it crosses the pulmonic valve and floats into the PA (Fig. 3), ultimately arriving at the wedge position recognized by a characteristic pressure wave form (Fig. 3). When the balloon is deflated, the PA pressure waveform is restored (Fig. 3). The PA pressure waveform is characterized by a sharp upstroke and a down stroke interrupted by the dicrotic notch and dicrotic wave. The PCWP wave form is similar to the RA pressure wave form, but at a higher pressure and later in the cardiac cycle (when timed against the QRS complex).

The PCWP is commonly used to assess pulmonary venous and left atrial pressures, which also reflects LV diastolic pressure in the absence of mitral valve obstruction. It should be appreciated that for determination of left atrial pressure by measuring the PCWP, presence of a continuous fluid column between the distal tip of the catheter and the left atrium is necessary (Fig. 4). The continuous fluid column is absent when the alveolar pressure is considerably higher than pulmonary capillary pressure. In these circumstances, the small pulmonary veins and capillaries collapse. This zone of the lung is called West zone 1, and typically correlates anatomically with the upper air fields above the level of the left atrium. The continuous fluid column between the distal tip of the catheter and left atrium can be maintained.

Fig. 3. Hemodynamic waveforms in the RA, RV, PA, and wedge (PCWP) positions of the PAC. (From Disease-a-month. The Swan-Ganz Catheter. Dis Mon 1991;37(8):509–43; with permission.)

Fig. 4. West’s zones of the lung; note that Zone 3 placement of the PAC is critical for the accurate estimation of the left ventricle end diastolic pressure from the PCWP. (From de Beer JM, Gould T. Principles of artificial ventilation. Anaesth Intensive Care Med 2013;14(3):83–93; with permission.)
only when the catheter is placed in an area of the lung where the PCWP is considerably higher than the alveolar pressure. This area is termed West zone 3, located in the most dependent part of the lung below the level of the left atrium.

The wedge position can be verified by withdrawing blood with the balloon inflated for determination of oxygen saturation. If the catheter is in proper wedge position, withdrawn blood will be “arterialized” with an oxygen saturation of 95% or higher. There may be considerable inaccuracy in proper measurement of PCWP by the waveform analysis alone and considerable interobserver variability as well.29

At the time of catheter placement, the difference between PA end-diastolic and mean PCWP should be determined. If the difference does not exceed 5 mm Hg, PA end-diastolic pressure can be used in place of PCWP,30–32 The normal range of RA pressure is 0 to 7 mm Hg. The RA “a” wave pressure is higher than the “v” wave pressure. The normal range of RV systolic pressure is 15 to 25 mm Hg and the normal range of RV end-diastolic pressure is between 0 and 8 mm Hg. The PA systolic pressure is similar to that of RV systolic pressure in the absence of RV outflow obstruction. The normal mean PA pressure (MPAP) is less than 18 mm Hg and the normal mean PCWP is 15 mm Hg or less. The “a” wave pressure is lower than the “v” wave pressure in PCWP.

When the RA and RV are markedly dilated or in patients with severe pulmonary arterial hypertension (not uncommon in the cardiac ICU), it may become necessary to use fluoroscopy for PAC placement because coiling and knotting in the RA or RV can occur.

During hemodynamic monitoring in critical care units, RA, PA, and PCW pressures are typically monitored, in addition to arterial pressure and cardiac output. Arterial and mixed venous oxygen saturations can be used to assess changes in cardiac output.

The RA pressure reflects RV diastolic pressure in the absence of tricuspid valve obstruction. Jugular venous pressure is similar to RA pressure in the absence of superior vena cava or innominate vein obstruction. Thus, bedside measurement of jugular venous pressure provides a reasonable estimate of RA and RV diastolic pressure, meaning that RV filling pressure can be assessed without the use of a PAC. PCWP reflects LV diastolic pressure in the absence of non-Zone III placement and pulmonary venous, left atrial, and mitral valve obstruction. PCWP, however, cannot be estimated without right heart catheterization, even with the use of sophisticated Doppler echocardiography. In the absence of cardiopulmonary disease, RA pressure has only a modest correlation to PCWP.33 In the presence of valvular, myocardial, or pericardial disease, this correlation is even worse.34,35

The RA and PCW pressures are, at best, surrogates for RV and LV filling pressures respectively, which in turn are used to assess RV and LV function. Although it is commonly assumed that right atrial pressure (RAP) and PCWP represent RV and LV preloads, it should be appreciated that true preload refers to ventricular end-diastolic volume. As ventricular volumes are difficult to determine at the bedside in critical care units, RAP and PCWP are used as surrogates for ventricular filling pressures. Ventricular function curves are constructed by relating stroke volume or cardiac output to the ventricular filling pressure (Frank-Starling relation). Stroke volume is calculated by dividing cardiac output by heart rate. Cardiac output is usually determined by the thermodilution method when a PAC is used. Cardiac output can also be determined by dividing total body oxygen consumption by the arterial and mixed venous oxygen content difference (Fick principle).

To construct a ventricular function curve, stroke volume is plotted on the vertical axis and ventricular filling pressure on the horizontal axis (Fig. 5). Obviously, in the absence of mitral or tricuspid valve regurgitation or intracardiac shunts, RV and LV stroke volumes are the same.

Both RV and LV function curves have an initial steep portion and a relatively flat terminal portion. On the steep portion of the function curve, for a given increase in filling pressure, there is a substantial increase in stroke volume. This may occur, for example, during volume expansion by administration of fluids. On the flat portion of the function curve, a similar increase in filling pressure is associated with a smaller increase in stroke volume. In the failing ventricle, the ventricular function curve moves downward and to the right. The steep portion of the depressed ventricular function curve is relatively shorter and the flat portion longer (see Fig. 5). Thus, in failing hearts, for a given increase in filling pressure, the degree of rise in stroke volume is much smaller.

The RV and LV are structurally different: the RV free wall is thinner than that of the LV. The determinants of RV and LV stroke volume are also different. For example, systemic arterial pressure, a component of LV afterload, is higher than PA pressure, a component of RV afterload. The RV is also more compliant than the LV. Thus, RV filling pressure is much lower than that of the LV for a similar end-diastolic volume. As such, the RV function curve sits to the left of the LV function curve.
An understanding of the difference between the RV and LV function curves is clinically important. During volume expansion therapy, for a given increase in stroke volume, RV filling pressure increases much less than LV filling pressure. When stroke volume increases from 15 mL to 40 mL, RA pressure (RV filling pressure) increases from 5 to 10 mm Hg. However, PCWP (LV filling pressure) increases from 15 to 30 mm Hg, and can result in pulmonary edema.

Although in clinical practice, RA and PCWP pressures are used as RV and LV filling pressures, true ventricular filling pressures are transmural. The transmural pressure is the difference between the ventricular distending pressure and the pressure-resisting ventricular distention. Pericardial and mediastinal pressures comprise resisting pressure. Normally, the intrapericardial pressure is 0 and the mediastinal pressure is between $-1$ mm Hg and $-3$ mm Hg. Thus, in normal conditions, RAP and PCWP are assumed to accurately reflect RV and LV filling pressures. However, when intrapericardial and mediastinal pressures change, transmural pressure changes. For example, in pericardial effusion, the intrapericardial pressure is increased. To estimate RV and LV filling (transmural) pressures, pericardial pressure should be subtracted from the RA and PCW pressures.

The cardiac pressure waveforms can vary with the respiratory phase. Typically, the respiratory influence on the waveform is most neutral at end-expiration, where end-expiration depends on whether the patient is breathing spontaneously or is on positive-pressure (assisted) ventilation (Fig. 6).

In various arrhythmias, both PCWP and RAP are altered. In atrial fibrillation, “a” waves are absent and in atrial flutter, flutter waves can be visualized. In the presence of PVCs, cannon waves distort the RAP and PCWP wave forms. Cannon waves are produced when atrial systole occurs while the atrioventricular valves are closed during ventricular systole, such as with complete heart block.

**CARDIAC OUTPUT**

Cardiac output can be determined by the thermodilution technique with a PAC by using the Stewart-Hamilton equation. A fixed volume of
cold fluid (typically 10 mL of normal saline at room temperature) is injected as a bolus into the proximal lumen of the PAC, and the resulting change in the PA blood temperature is recorded by the thermistor at the catheter tip. As the cooler mixed blood flows by the thermistor, the rate of change of temperature and a return back to body temperature is reflected in a temperature concentration curve (Fig. 7). The quicker the rate of change in blood temperature, the greater the flow rate, and, thus, the higher the cardiac output. Conversely, a slower rate of change in blood temperature indicates lower cardiac output.

In certain circumstances, cardiac output cannot be determined accurately by the thermodilution technique. In patients with severe tricuspid regurgitation and intracardiac shunts, prolonged indicator transit time and recycling of the indicator can cause overestimation or underestimation of cardiac output.\textsuperscript{27} Lower limb compression devices, ubiquitous in the ICU, have even been shown to confound thermodilution cardiac output measurement.\textsuperscript{36} Low basal blood temperature, for example during therapeutic hypothermia, may also introduce error in measured cardiac output by the thermodilution technique.

The analysis of arterial pulse pressure wave forms (APWA) method has also been used for measurement of cardiac output. This method estimates intravascular volume status through the use of stroke volume variation (SVV) and, thus, estimates cardiac output.\textsuperscript{37} This technique can be used only in patients on a ventilator. The variables that can be determined by the APWA method are cardiac index (CI) and SVV. A CI of at least 2.8 L/min/m\textsuperscript{2} measured by this technique has been reported to indicate a favorable prognosis.\textsuperscript{37} It has been also reported that SVV can be used to assess volume responsiveness. It should be noted that a poor correlation between the APWA method and thermodilution has been reported.\textsuperscript{38}

A transpulmonary thermodilution technique also can be used to measure cardiac output (CO). In this method, a thermal injectate is delivered into the RA by a central venous catheter and the change in temperature of the blood circulating through the pulmonary and arterial circulation is sensed by a thermistor mounted at the tip of the radial artery. A high correlation exists between CO determined by this technique and CO determined by thermodilution with a PAC.\textsuperscript{39}

The transpulmonary thermodilution technique can be used with global and diastolic volume and lung water.

Transthoracic and transesophageal echocardiography (TTE and TEE, respectively) are being used increasingly in ICUs to assess LV and RV function and filling pressures. Transesophageal echocardiography can be used to assess RV and LV volumes and ejection fraction even in patients

![Fig. 7. The temperature change versus time graph (A) applied to the Stewart Hamilton equation. (B) Notice that the rate of change of temperature is inversely proportional to the cardiac output. (From Marino PL. The ICU book. 2nd edition. Philadelphia: Lippincott Williams and Wilkins; 1997; with permission.)](image-url)
on ventilators. RV systolic pressure can also be determined from the tricuspid regurgitation jet. The measurement of inferior vena cava diameter has been used to assess central venous pressure. Studies on the correlation between Doppler and thermodilution methods of cardiac output determination have varied, some suggesting poor correlation and others reporting reasonable correlation.

**PULMONARY ARTERY DATA**

A plethora of data can be obtained from the PAC (Box 1, Table 1). Once the monitoring system is properly calibrated, zeroed to atmospheric pressure, and the transducer leveled to the phlebotatic axis, the central venous pressure (CVP) and pulmonary artery pressure (PAP) are displayed on the monitor. Typically the blood pressure and heart rate are also displayed. The next step is to aspirate a blood sample from the PA port to obtain the $SvO_2$, inflate the balloon with 1.5 mL of air and measure the wedge pressure at end expiration, and inject 10 mL of normal saline in the RA port of the PAC to obtain cardiac output by thermodilution. With these primary measurements at hand, one can populate various equations as shown in Table 1 and obtain derived data. Fig. 8 illustrates the determinants of $SvO_2$ and the utility of monitoring $SvO_2$, which is a less operator-dependent variable and quite useful when waveform analysis or thermodilution are in question.

**CLINICAL APPLICATIONS**

**Cardiac Catheterization Laboratory**

Balloon floatation catheters are routinely used in cardiac catheterization laboratories for right heart catheterization to record intracardiac pressures and cardiac output. Cardiac output is usually determined by thermodilution, as described earlier. Cardiac output also should be determined by measuring oxygen consumption and arteriovenous oxygen difference (Fick equation), especially in patients with severe tricuspid regurgitation and intracardiac shunts.

In the presence of a large left-to-right shunt, pulmonary blood flow is higher than systemic flow and cardiac output determined by thermodilution will overestimate the true cardiac output. In the presence of a right-to-left shunt, pulmonary blood flow is lower than systemic flow and cardiac output determined by thermodilution will underestimate the true systemic cardiac output.

**Acute Coronary Syndromes**

When the PAC was first introduced, hemodynamic monitoring by right heart catheterization was routinely performed in patients with acute coronary syndromes (ACS) in myocardial infarction research units, and in coronary care units. Numerous studies were performed seeking to identify such features as the optimal left heart filling pressure, the best medical therapy for ACS by application of hemodynamic subsets, or the proper use and effects of diuretics in patients developing heart failure during ACS. The hemodynamic effects of various vasoactive agents in acute myocardial infarction (MI) were also studied by right heart catheterization with the use of PACs. These studies provided useful knowledge in understanding of the hemodynamic alterations, pathophysiologic changes, and rationale for management of patients with acute MI.

Hemodynamic subsets and their clinical correlates in patients with acute MI were useful for assessing severity of cardiac and hemodynamic compromise (Table 2). In subset I, cardiac output and PCWP are normal and there are no signs of hypoperfusion or congestion. In subset II, cardiac output is normal but PCWP is elevated. Clinically, there are signs of pulmonary congestion without signs of hypoperfusion. In subset III, cardiac output is reduced and PCWP is elevated. Clinically, there are signs of hypoperfusion without signs of pulmonary congestion. Patients in cardiogenic shock fall under subset IV and are

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**Box 1**

**Organization of PAC data**

1. **Observe**
   - a. MAP: Blood pressure/mean arterial pressure
   - b. RAP: Central venous pressure/right atrial pressure
   - c. PAP/MPAP: Pulmonary artery pressure and mean pulmonary artery pressure

2. **Do**
   - a. PCWP: Inflate the balloon to obtain pulmonary capillary wedge pressure
   - b. $SvO_2$: Aspirate pulmonary artery blood to obtain $SvO_2$
   - c. CO: Obtain cardiac output by thermodilution via PAC

3. **Derive**
   - a. Cardiovascular dynamics
   - b. Oxygen dynamics
Table 1
Derived parameters for cardiovascular and oxygen dynamics

<table>
<thead>
<tr>
<th></th>
<th>Cardiovascular Dynamics</th>
<th>Oxygen Dynamics</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cardiac index (L/min/m²)</td>
<td>( \frac{CO}{BSA (m^2)} )</td>
<td>( \frac{CO}{BSA (m^2)} )</td>
</tr>
<tr>
<td>Stroke volume (L/beat)</td>
<td>( \frac{CO}{HR (beats/min)} )</td>
<td>( \frac{CO}{HR (beats/min)} )</td>
</tr>
<tr>
<td>Stroke volume index (L/beat/m²)</td>
<td>( \frac{SV}{BSA (m^2)} )</td>
<td>( \frac{SV}{BSA (m^2)} )</td>
</tr>
<tr>
<td>MAP (mm Hg)</td>
<td>( \frac{2\text{Diastolic} + \text{Systolic}}{3} )</td>
<td>( \frac{2\text{Diastolic} + \text{Systolic}}{3} )</td>
</tr>
<tr>
<td>SVR (dyne-sec-cm⁻⁵)</td>
<td>( \frac{\text{MAP} (\text{mm Hg}) - \text{MRAP} (\text{mm Hg})}{\text{CO} (\text{ml/min})} \times 80 )</td>
<td>( \frac{\text{MAP} (\text{mm Hg}) - \text{MRAP} (\text{mm Hg})}{\text{CO} (\text{ml/min})} \times 80 )</td>
</tr>
<tr>
<td>PVRI (L/dyne/sec/cm⁻⁵/m²)</td>
<td>( \frac{\text{MAP} (\text{mm Hg}) - \text{PCWP} (\text{mm Hg})}{\text{CI} (\text{L/min})} \times 80 )</td>
<td>( \frac{\text{MAP} (\text{mm Hg}) - \text{PCWP} (\text{mm Hg})}{\text{CI} (\text{L/min})} \times 80 )</td>
</tr>
<tr>
<td>LVSWI (g·m⁻²·beat)</td>
<td>( 0.0136 [SVI 	imes (\text{MAP} - \text{PCWP})] )</td>
<td>( 0.0136 [SVI 	imes (\text{MAP} - \text{PCWP})] )</td>
</tr>
<tr>
<td>RVSWI (g·m⁻²·beat)</td>
<td>( 0.0136 [SVI 	imes (\text{mPAP} - \text{RAP})] )</td>
<td>( 0.0136 [SVI 	imes (\text{mPAP} - \text{RAP})] )</td>
</tr>
</tbody>
</table>

Abbreviations: BSA, body surface area; CaO₂, arterial O₂ content; CI, cardiac index; CO, cardiac output; DO₂, oxygen delivery; Hb, hemoglobin; HR, heart rate; LVSVI, left ventricular stroke work index; MAP, mean arterial pressure; O₂ ER, oxygen extraction ratio; mPAP, mean pulmonary artery pressure; PCWP, pulmonary capillary wedge pressure; PVR, pulmonary vascular resistance; PVRI, pulmonary vascular resistance index; RAP, right atrial pressure; RSVWI, right ventricular stroke work index; SV, stroke volume; SVI, stroke volume index; SvO₂, mixed venous saturation; SVR, systemic vascular resistance; SVRI, systemic vascular resistance index; DO₂, oxygen consumption.

Additionally hypotensive, with an arterial pressure of 90 mm Hg or less.

It should be noted that these hemodynamic studies were performed before the era of echocardiography and immediate revascularization. With the advent of echocardiography, the need for invasive hemodynamic monitoring has declined markedly. For practical purposes, RV and LV filling pressures and cardiac output can be only approximated by echo-Doppler studies. An analysis of data from the multinational Global Registry of Acute Coronary Events (GRACE) revealed that the rate of PA catheterization was 3.0% in 2007 compared with 5.4% in 2000. In the United States, this number dropped from 10.4% in 2000 to 1.5% in 2007. Furthermore, it was observed that PA catheterization is associated with increased mortality and longer length of hospital stay. Mortality at 30 days was substantially higher among patients with PAC for both unadjusted (odds ratio [OR] 8.7; 95% confidence interval [CI] 7.3–10.2) and adjusted analyses (OR 6.4; 95% CI 5.4–7.6) in all groups except in patients with cardiogenic shock (OR 0.99; 95% CI 0.80–1.23).

Gore and colleagues showed that in patients with heart failure secondary to acute MI, inhospital mortality was 44.8% in those receiving PA catheterization versus 25.3% in those who did not. In patients with hypotension, the mortality was 48.3% in patients who received PA catheterization versus 32.2% in patients who did not. In patients with cardiogenic shock complicating ACS, however, there was no difference in mortality between patients who received PA catheterization (74.4%) and those who did not (79.1%). In the
GUSTO trial, the hazard ratio of 30-day mortality in patients without cardiogenic shock who received PA catheterization was 4.80 (95% CI 3.56–6.47) compared with those who did not. Yet, in patients with cardiogenic shock, there was no difference in the risk of mortality; the hazard ratio was 0.99 (95% CI 0.80–1.23). In a broad retrospective registry analysis of 5841 hospitalized patients with ACS, mortality was higher in patients who received PA catheterization. The results of these studies suggest that there is no indication for routine PA catheterization in patients with ACS, unless there is concomitant cardiogenic shock or other complications. In this setting, hemodynamic monitoring with PACs may be necessary to guide appropriate therapy, particularly when vasoactive drugs are being used.

**RV-MI**

Diagnosis of Right Ventricular Myocardial Infarction (RV-MI) can be established by characteristic hemodynamic findings. RA pressure is elevated and is frequently equal to or higher than PCWP.
In patients with severe RV dysfunction, RV systolic pressure is not as elevated as in patients with pre-capillary pulmonary hypertension due to the RV’s inability to generate high pressure. However, the most distinctive feature of acute RV infarction is a distorted PA pressure waveform (Fig. 9).

Although a PAC is not needed for diagnosis of acute RV infarct, hemodynamic monitoring may be helpful in patients with cardiogenic shock complicating RV infarction.

### Table 2

<table>
<thead>
<tr>
<th>Subset</th>
<th>Clinical Signs: PC (Wet/Dry)</th>
<th>Cardiac Index (L/min/m²)</th>
<th>PAWP (mm Hg)</th>
<th>Hospital Mortality (%)</th>
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</thead>
<tbody>
<tr>
<td>I</td>
<td>PC No (dry)</td>
<td>&gt;2.2</td>
<td>&lt;18</td>
<td>3</td>
</tr>
<tr>
<td>II</td>
<td>PC Yes (wet)</td>
<td>&gt;2.2</td>
<td>&gt;18</td>
<td>9</td>
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<tr>
<td>III</td>
<td>PC No (dry)</td>
<td>&lt;2.2</td>
<td>&lt;18</td>
<td>23</td>
</tr>
<tr>
<td>IV</td>
<td>PC Yes (wet)</td>
<td>&lt;2.2</td>
<td>&gt;18</td>
<td>51</td>
</tr>
</tbody>
</table>

**Abbreviations:** HYP, peripheral hypoperfusion; PAWP, pulmonary artery wedge pressure; PC, pulmonary congestion.


### Mechanical Complications of ACS

Acute mitral regurgitation and septal rupture are the 2 most striking mechanical complications of ACS. Acute mitral regurgitation due to papillary muscle infarction or rupture is associated with a giant “V” wave in the PCWP tracing or a reflected “V” wave in the PAP tracing (Fig. 10). Giant “V” waves are not diagnostic of acute severe mitral regurgitation as they can be observed in ventricular septal rupture and even in aortic and mitral...
stenosis. In these conditions, the magnitude of a normal “V” wave is accentuated if there is increased volume return to the left atrium. However, the reflected “V” wave in the PAP tracing is diagnostic of acute or subacute severe mitral regurgitation. Of course, papillary muscle infarction or rupture can be easily diagnosed by echocardiography.

Ventricular septal rupture is usually associated with a large left-to-right shunt. The characteristic hemodynamic feature is a large step up in $O_2$ saturation in pulmonary arterial and RV blood samples compared with that obtained from the RA (Fig. 11). Echocardiography, however, can be used not only for the diagnosis of the ventricular septal rupture but also to assess the magnitude of shunt. As such, PA catheterization is not required, nor recommended for diagnosis of mechanical complications of ACS.

**Non Acute Coronary Syndromes**

Hemodynamic monitoring with PACs is frequently used in intensive coronary units for management of patients with hypotension and shock not due to ACS or valvular heart disease. Determination of PCWP can distinguish between hemodynamic (cardiogenic) and permeability (noncardiogenic) pulmonary edema. Hemodynamic pulmonary edema is characterized by a PCWP of 25 mm Hg or greater. In patients with permeability pulmonary edema, the PCWP is normal.

Septic and hypovolemic shock are two common conditions for which hemodynamic monitoring was frequently used in ICUs. Determination of RAP, PCWP, cardiac output, and systemic vascular resistance allows differentiation between cardiogenic, hypovolemic, and septic shock.

In cardiogenic shock, RAP and PCWP are usually elevated, but PCWP is higher than RAP. Cardiac output is reduced, systemic vascular resistance is usually elevated, and systemic systolic blood pressure is low. In hypovolemic shock, both RAP and PCWP are lower than normal, cardiac output and arterial pressure are reduced, and systemic vascular resistance is normal or elevated depending on the magnitude of hypotension and reduction of cardiac output. In septic shock, RAP and PCWP are normal before fluid therapy, cardiac output is normal or higher than normal, and systemic vascular resistance is abnormally low.
However, in critically ill patients with septic or hypovolemic shock, PA catheterization has been associated with an increased risk of mortality (OR of death was 1.24, 95% CI 1.03–1.049).\(^5^8\)

In a randomized clinical trial performed in the United Kingdom, there was no difference in mortality, organ dysfunction, or length of hospital stay between patients receiving PA catheterization and patients who were not catheterized.\(^5^9\) In a multicenter study, 676 patients with noncardiogenic shock or acute lung injury or both were randomized to receive PA catheterization or no catheterization. There was no difference in 30-day mortality between the 2 groups.\(^6^0\)

In a randomized trial sponsored by National Heart, Lung, and Blood Institute, 1001 patients were randomized to receive PA catheterization or central venous catheter placement to assess the relative efficacy of PACs in decreasing mortality and morbidity of patients with acute respiratory distress syndrome.\(^6^1\) In the PAC group, a slightly higher percentage of patients were in shock and received vasopressors. However, there was no difference in hospital mortality, ICU length of stay, or total hospital days. There was a higher incidence of arrhythmias in the PAC group. Also, although hospital costs were similar in both groups, the long-term cost was higher in the PAC group and there was a mean loss of 0.3 quality-adjusted life years in the PAC group.\(^6^2\) Thus, PA catheterization did not provide any advantage over the use of central venous catheters in the management of patients with acute respiratory syndrome and routine use in this setting is not recommended.

PA catheterization has also been used for the management of high-risk surgical patients. In the perioperative setting, optimizing oxygen delivery by volume expansion therapy or hemodynamic manipulation with vasoactive drugs guided by PAC monitoring was associated with reduced mortality and morbidity and improved prognosis in high-risk surgical patients.\(^6^3,6^4\) However, a randomized clinical trial of 1994 patients (after screening 3803) reported that there was no advantage of PA catheterization compared with standard care for the management of high-risk surgical patients in the perioperative period.\(^6^5\) In-hospital mortality between the 2 groups was 7.7% and 7.8% in the standard care and catheterized groups, respectively. One-year survival was also similar in both groups. Although morbidity, including complications such as MI, heart failure, and arrhythmias, was similar in the 2 groups, the incidence of pulmonary embolism was higher in patients who received PA catheterization.

The need for perioperative and intraoperative hemodynamic monitoring with PACs in cardiac surgical patients remains controversial. In a prospective observational multicenter study, the effects of PA catheterization were assessed in 5065 patients undergoing coronary artery bypass surgery.\(^6^6\) Some patients were monitored by transesophageal echocardiography only, whereas other patients were monitored by PA catheterization only. A third group received both TEE and PA catheterization and a fourth group received neither. Propensity score matched-pair analysis was used to statistically generate comparisons.
and yielded 1273 matched pairs receiving PA catheterization versus no hemodynamic monitoring. The composite primary end point included death from any cause, cerebral dysfunction (stroke or encephalopathy), renal dysfunction, cardiac dysfunction (MI or congestive heart failure) or pulmonary dysfunction (acute respiratory distress syndrome). Secondary end points included the use of inotropic agents, duration of intubation, and length of stay in ICUs. The primary end point occurred in 21.3% of the 271 patients receiving a PAC and in 15.4% of the 196 patients who did not receive PAC monitoring. The adjusted OR was 1.68 (95% CI 1.24–2.26; P<.001). In patients receiving a PAC, all-cause mortality was 3.5% compared with 1.7% in patients who did not receive PA catheterization (adjusted OR 2.08; CI 1.11%–3.88%, P = .02). The incidence of cardiac, renal, and cerebral dysfunction, as well as the use of inotropic drugs, duration of intubation, and length of stay in the ICU, was also higher in patients receiving PA catheterization. The results of this study thus suggest that PA catheterization can produce deleterious effects in patients undergoing coronary artery bypass surgery.

In another randomized trial, hemodynamic monitoring with PACs was compared with transpulmonary thermodilution (TTD) in patients undergoing combined valve repair surgery.67 Patients were randomized in two equal groups of 40. In the PAC group, cardiac index, mean arterial pressure, and PCWP were monitored during goal-directed therapy. In the TTD group, global end-diastolic volume index, extravascular lung water index, and oxygen delivery index were additionally monitored. Hemodynamic improvement was greater in the TTD group and duration of mechanical ventilation was longer in the PAC group. The results of this study suggest that TTD is better than PAC during goal-directed therapy in patients undergoing combined valve repair surgery.

It should be recognized that in these studies, PA catheterization was performed not for the diagnosis of hemodynamic abnormality but for therapeutic guidance. This required prolonged catheterization, which may be associated with undesirable complications.

### Chronic Systolic Heart Failure

In patients with severe chronic heart failure, PA catheterization has been performed to determine the severity of hemodynamic abnormalities to guide therapy and assess prognosis. In this context, clinical subsets based on hemodynamic abnormalities have been established and therapies specific to these subsets have also been developed.68,69 With regard to prognosis, it has been reported that PCWP greater than 25 mm Hg, cardiac index less than 2.2 L/min/m², LV stroke work index less than 45 g/m², and systemic vascular resistance greater than 1800 dyn.s.cm⁻⁵ indicated poor prognosis.70,71 It should be appreciated that prognosis can also be assessed by history and physical examination and evaluation of response to therapy.

To determine whether PA catheterization is helpful in improving clinical outcomes in patients hospitalized with severe chronic systolic heart failure, the Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) trial was performed.72 In this study, 433 patients were randomized to assess effectiveness of therapy guided by PA catheterization or by clinical assessment. The primary end point was days-alive out of hospital within the first 6 months. The secondary end points were changes in quality of life, exercise tolerance, and biochemical and echocardiographic parameters. PAC use did not improve the primary outcome. The number of days-alive out of hospital in first 6 months was 133 days in the PAC group and 135 days in the clinical assessment group (hazard ratio 1.00, 95% CI 0.82–1.21; P = .99). Mortality in the PAC group was 10% and 9% in the clinical assessment group. In-hospital complications were 21.9% in the PAC group and 11.5% in the clinical assessment group (P = .04). These data suggest that routine use of PACs does not improve prognosis of patients with severe chronic heart failure and may be associated with a higher complication rate.

### Pulmonary Hypertension

The etiology of pulmonary hypertension (PH) can be suspected by physical examination, heralded by the presence of a loud pulmonic component of the second heart sound. The presence or absence of mitral and aortic valve diseases and LV myocardial disease provides clues regarding whether PH is postcapillary, precapillary, or mixed. PH resulting from intracardiac shunts can also be suspected by bedside physical examination. Echo-Doppler studies should be routinely performed in all patients suspected of PH. However, right heart catheterization is necessary not only for diagnostic confirmation, but also to establish the etiology and guide therapeutic approach.73,74

A mean PA pressure at rest of greater than 25 mm Hg establishes the diagnosis of PH. Postcapillary pulmonary hypertension is defined when elevated PCWP is the principal mechanism for PH. In patients with postcapillary pulmonary
hypertension, pulmonary vascular resistance is normal or only slightly elevated and the difference between PA end diastolic pressure and mean PCWP is 5 mm Hg or less.

In patients with precapillary pulmonary hypertension, PCWP is normal, pulmonary vascular resistance is markedly elevated, and the difference between the PA end diastolic pressure and mean PCWP is significantly greater than 5 mm Hg.

In patients with mixed-type pulmonary hypertension, PCWP is elevated and there is also increased pulmonary vascular resistance. The difference between PA end diastolic pressure and mean PCWP is greater than 5 mm Hg.

During hemodynamic evaluation of patients with pulmonary hypertension, oxygen saturations in the intracardiac chambers should be determined to exclude significant intracardiac shunts as well. The need and importance of PA catheterization in the diagnosis and management of patients with PH is apparent.

**Transplantation**

PA catheterization is routinely performed for evaluation of potential heart transplant recipients. In addition to determining PA pressure, pulmonary vascular resistance and the severity of right and left heart failure, the reversibility of pulmonary vascular resistance in response to pulmonary vasodilators is tested to determine whether heart, or combined heart and lung transplantation is indicated. Patients on continuous infusion of a single high-dose intravenous inotrope or multiple intravenous inotropes, in addition to continuous hemodynamic monitoring of LV filling pressures, are classified as Status 1A priority for purposes of heart transplantation.

Following transplantation, hemodynamics are monitored by PAC placement as well. Transient RV failure following cardiac or pulmonary transplantation therapy is common and therapies are frequently guided by hemodynamic abnormalities determined by PA catheterization.

**COMPLICATIONS**

Although the incidence of serious complications during hemodynamic monitoring with PACs is low, complications related to the placement and prolonged use do occur and can be significant. Complications during insertion of PACs include inadvertent puncture of the arteries, production of pseudoaneurysms, and hemothorax or pneumothorax. The incidence of inadvertent arterial puncture is approximately 3% to 9% and bleeding from the arterial puncture site can usually be stopped by compression of the artery. Injury of the thoracic duct may occur during attempts to cannulate the left internal jugular or left subclavian vein and can lead to formation of chylothorax. The risk of catheter-related venous thrombosis is approximately 2% when subclavian veins are used, 8% when internal jugular veins are used, and 22% when femoral veins are used. These complications, however, are not unique to the use of PACs. The use of any central venous catheter can be associated with these complications. Air embolism, a potentially fatal complication in the presence of intracardiac shunts, occurs rarely.

Although cardiac arrhythmias occur frequently during placement of PACs, these arrhythmias are usually transient. Atrial and ventricular premature beats, nonsustained atrial and ventricular tachycardia, and conduction anomalies can occur during placement. The incidence of arrhythmias ranges from 12.5% to 70.0% during placement of PACs. The incidence of ventricular premature beats is between 52% and 68% and that of nonsustained ventricular tachycardia is 1% to 53%. Sustained ventricular tachycardia or ventricular fibrillation requiring direct current shocks occurs in fewer than 1% of patients. In patients with pre-existing left bundle branch block, complete heart block may be precipitated during insertion of PACs and pacing may be required. The incidence of transient right bundle branch block is less than 5% and is benign.

Traumatic injury of the tricuspid valve apparatus can also occur. Catheters can become entangled within the tricuspid valve chordate and severe tricuspid valve regurgitation can occur during removal of the entangled catheter.

As with any other central venous catheter, bacteremia, sepsis, and catheter site infection are potential complications as well. The reported incidence of sepsis and bacteremia is between 1.3% and 2.3%. Because the tricuspid valve can be traumatized during PAC placement, tricuspid valve endocarditis is potentially a serious complication. The reported incidence of right-sided endocarditis is between 2.2% and 7.1%.

Pulmonary artery rupture presents as sudden profuse hemoptysis and is almost always a fatal complication, but is rare, with a reported incidence between 0.03% and 0.20%. The reported mortality is 70% and the risk factors are PA hypertension and older age. The forceful overinflation of the balloon and keeping the balloon inflated for prolonged periods of time are the most common causes of PA rupture. Rarely, PA dissection can be caused by the tip of the catheter, and can progress to complete rupture.
Knotting of pulmonary catheters is an uncommon complication, and the estimated incidence is 0.03%. When the PA pressure wave form cannot be obtained despite advancing the PAC beyond 60 to 70 cm, use of fluoroscopy can reduce the risk of knotting. Catheter techniques for unknotting exist, although occasionally, extraction by venotomy is required.

**CONTROVERSY SURROUNDING THE PAC CATHETER**

There was tremendous enthusiasm following the introduction of the PAC, a technology that seemed to make it easy to estimate right and left heart-filling pressures and rapidly measure response to therapeutic interventions. Suddenly, it seemed, cardiopulmonary physiology could be studied without cumbersome technology and many physician scientists spent the good part of a decade exploring their favorite ideas. Cohn, an early surgical intensivist, for instance used the PAC to develop an “automated physiologic profile” to monitor hemodynamic, oxygen consumption and tissue utilization data in perioperative patients. Louis Del Guercio, a surgeon and one of the founders of the Society for Critical Care Medicine (SCCM) and its sixth president, used the “automated physiologic profile” to reduce operative mortality in elderly patients, using a variety of interventions. Shoemaker, the third president of the SCCM, and others investigated whether “supernormal” oxygen delivery improved outcomes in high-risk surgical patients. As discussed in the previous section, Forrester and colleagues used the PAC in acute MI for prognostic and therapeutic purposes. The ability to easily generate reams of seemingly meaningful physiologic data was all too alluring for many intensivists to resist. Before long, the use of the PAC had become “routine” in all kinds of ICUs: cardiac, surgical, and medical. The rapidity of adoption of this technology was like few seen before. Indeed, Paul Marino remarked “The PAC is not just important for the specialty of critical care, it is responsible for the specialty of critical care.” Soon, there was an explosion of anecdotal data reporting improved outcomes, no difference at all, as well as worse outcomes in a variety of patient populations in uncontrolled settings. In 1996, Connors and colleagues reported that the use of a PAC in 5735 critically ill patients was associated with an apparent lack of benefit and led to increased mortality and increased utilization of resources. The inevitable backlash followed, and at times it was shrill. Editorials with titles such as “Death by pulmonary artery flow-directed catheter,” “Swan song for the Swan-Ganz catheter?” and “Is it time to pull the pulmonary artery catheter?” appeared in leading journals. They were prompted largely by uncontrolled studies that suggested that mortality, the length of intensive care, and hospital costs were greater when a pulmonary catheter was used in a variety of clinical settings in various heterogeneous patient populations. In 1997, the SCCM tried to address this uncertainty by issuing a Consensus Statement which did little to address the confusion, as there were no randomized controlled trials (RCTs) to base those recommendations on. The first RCTs started appearing in 2002. In carefully controlled studies, the “routine” use of PACs in various populations, high-risk surgical patients, patients with shock and acute respiratory distress, critically ill patients, and patients with congestive heart failure, there was no improvement in outcomes. This led to a rapid and precipitous decline in the use of PACs across all types of ICUs in the United States. Of course, this raises questions about maintaining ongoing competence in procedural skills and implications for clinical care and training of newer generations of cardiologists and intensivists.

As the use of the PAC declined and its use presumably more selective, a 2013 Cochrane review of 13 studies with a total of 5686 ICU patients showed that use of a PAC did not alter the mortality, general ICU or hospital LOS, or cost for adult patients in intensive care. The quality of evidence was high for mortality and LOS but low for cost analysis. The investigators made another important point: that newer, less-invasive hemodynamic monitoring tools need to be validated against the PAC before clinical use in critically ill patients.

The fact is that the PAC, which is essentially a diagnostic tool, has been judged by clinical outcomes, standards that are meant to determine the efficacy of therapeutics. Indeed, few other diagnostic tools have been subject to such rigorous and perhaps misdirected scrutiny. Although PACs are invasive, rendering them more prone to complications, both observed and unseen, they are diagnostic tools at best, and when considered in that context, provide data not easily obtained by other means. As early as 1990, Iberti and his group at Mount Sinai had shown that there was a significant deficit in physicians’ knowledge and understanding of PAC data. For example, only 59.5% of attending physicians could correctly identify the PCWP waveform. One cannot condemn chest radiographs or electrocardiograms as deficient if expert interpretation of these tests is lacking. In the scenario of clinical ambiguity for a pathophysiologically complex and
critically ill patient, the clinician must first use the necessary tools to garner information, and then use this information to make an informed decision. To attribute clinical outcomes to a diagnostic decision in this setting is not warranted. Certainly, indiscriminate use of any invasive test in the wrong setting is undesirable, and may pose unnecessary risk. Clearly, “routine PA catheterization” lacks fundamental grounding in that no diagnostic test should be performed “routinely” without an appropriate context, even a simple venous blood sample.

With respect to risk, even when used in the appropriate population, clinicians should ensure that catheters are placed, used, and maintained optimally and that known pitfalls are avoided to minimize the risk. Avoidance of pitfalls includes not only PAC specific factors, but also increasing the reliance on operator independent data points such as mixed venous oxygen saturation as well as correlation with other laboratory markers of organ perfusion. Prompt removal of PACs after procurement of data also limits risk by minimizing the duration of invasive exposure. Additionally, expertise in obtaining and interpreting the waveforms and a firm fundamental understanding of the data derived will allow clinicians to make the most of data garnered following PAC placement. Even an uncomplicated catheter placement is rendered useless if data gathering or interpretation is faulty.

With respect to a multitude of less invasive hemodynamic tools, the PAC has been used in many more patients and clinical scenarios and subjected to more RCTs than any of these tools. The plethora of existing data suggesting that “routine” PA catheterization in ICUs is not beneficial is likely multifactorial: poor patient selection, inability to identify and interpret data, untoward invasive risk, and the lack of effective therapies for the condition studied. As Richard and colleagues correctly observed “Even if the purpose of monitoring with PAC is ultimately to save lives, it would be unrealistic to believe that the prognosis of patients could be improved by its presence alone.”

A reasonable approach to the selective use of the PA catheter would be to

1. Understand the pitfalls and optimize procedural and interpretive handling, thus minimizing risk
2. Deploy only with appropriate patient selection and clinical scenario, thus maximizing potential benefit
3. Use only as long as necessary, remove as soon as possible
4. Correlate with nonoperator-dependent, highly sensitive variables such as SvO$_2$
5. Follow trends rather than focus solely on absolute values
6. Limit insertion and use by teams that have special training or expertise in performing and interpreting hemodynamic monitoring, just as we do with echocardiography or ventilators, for example
7. Continue research to better understand patient selection and define appropriate indications

**CURRENT INDICATIONS FOR PA CATHETERIZATION**

In light of the preceding, it becomes clear that in the hands of an operator who is fully aware of the many pitfalls and complications of the PAC and highly experienced in the synthesis and interpretation of data obtained from the PAC, it can be a valuable diagnostic tool. In the hands of such an operator, there is no diagnostic intervention that has been applied as widely, performed as easily, and subject to such intense scrutiny as the PAC. There is no demonstrated mortality benefit in applying this diagnostic tool “routinely” to large

<table>
<thead>
<tr>
<th>Box 2</th>
<th>Indications for PA catheterization</th>
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<tr>
<td>Not indicated as routine in high-risk cardiac and noncardiac patients</td>
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<tr>
<td>Indicated in patients with cardiogenic shock during supportive therapy</td>
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<tr>
<td>Indicated in patients with discordant right and left ventricular failure</td>
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<tr>
<td>Indicated in patients with severe chronic heart failure requiring inotropic, vasopressor, and vasodilator therapy</td>
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<tr>
<td>Indicated in patients with suspected “pseudosepsis” (high cardiac output, low systemic vascular resistance, elevated right atrial and pulmonary capillary wedge pressures)</td>
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<tr>
<td>Indicated in some patients with potentially reversible systolic heart failure, such as fulminant myocarditis and peripartum cardiomyopathy</td>
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<tr>
<td>Indicated for the hemodynamic differential diagnosis of pulmonary hypertension</td>
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<tr>
<td>Indicated to assess response to therapy in patients with precapillary and mixed types of pulmonary hypertension</td>
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<tr>
<td>Indicated for the transplantation workup</td>
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heterogeneous populations of critically ill patients, nor is there increased mortality or length of stay. It is also abundantly clear that in current clinical practice, no assessment of pulmonary hypertension or management of advanced heart failure or evaluation for heart transplant proceeds without PA catheterization. In most other scenarios, despite all that has been written, patient selection and tailored therapy is still an art and is highly operator dependant. Indications that are relevant to the use of the PAC in cardiac critical care are listed in Box 2.

SUMMARY

The PAC has come a full circle. Devised by cardiologists to probe the inner workings of the heart, it has inspired much research and clinical use. Over time, evidence has developed that harm could result if it is not used judiciously. Nevertheless, it remains a simple diagnostic intervention, done at the bedside with proven utility in cardiac critical care. Many technologies seek to supplant the PAC, but none has been subjected to as much clinical use and scrutiny. It is paramount that the user be intimately familiar with the pitfalls, the dangers of misinterpretation, and the potential complications of this device. More importantly, thoughtful patient selection and judicious use of this device can mitigate any possible harm and maximize utility.

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