Pitfalls in Interpretation of Pulmonary Artery Catheter Data

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INVASIVE MONITORING using the Swan-Ganz-type pulmonary artery catheter (PAC) has become a standard clinical technique for providing a quantitative assessment of cardiopulmonary function. In principle, the quantitative measurements made possible by this technology can establish an objective basis for both early detection of trends and evaluation of therapy. Because of the general absence of alternative technology, it is understandable that the PAC has had widespread clinical application. Unfortunately, however, measurement and calculation of hemodynamic parameters obtained from it are frequently undertaken without sufficiently serious consideration of the technical limitations and principles involved. This review emphasizes the conditions necessary for correct use of the PAC and informs the unwary practitioner of the many potential pitfalls that complicate and sometimes invalidate the interpretation of PAC data. Table 1 lists the abbreviations used in this review.

Misinterpretation of Pressure Measurements From the PAC

The PAC was designed to estimate left-sided vascular pressures via catheterization of the right side of the heart. It is well known that RAP may not reflect left ventricular filling pressures reliably and that there are often great disparities between the RAP and PAP distal to an occluding balloon, or PAOP, not only regarding the absolute magnitude of these pressures, but also their relative direction of change with acute hemodynamic interventions.1-4

In contrast, the LAP, PAOP, and LVEDP usually are in close agreement, especially at the end of diastole.5,6 However, that relation requires a static uninterrupted column of blood to be present from the tip of the PAC to the aortic valve (Fig 1). At the end of diastole, when there is no net flow in this common chamber, if the pulmonary vascular bed distal to the catheter tip (ie, the pulmonary capillaries and veins) is patent, no pressure gradients should exist. When the tip of the PAC is isolated from the upstream arterial pressure by inflating its balloon, the following are approximately equal: PAEDP, PAOP, PVP, LAP, and LVEDP. However, a variety of conditions may invalidate this pressure relationship.

Effects of Intrathoracic Pressure on Measurement of PAOP

As previously noted, accurate reflection of pressures in the left side of the heart by the PAC depends on the presence of a patent column of blood at the pulmonary capillary level. West et al7 showed that there is a marked variation of blood flow within the lung as a result of interrelationships between alveolar and vascular pressures (Fig 2). West described three physiologic zones of the lung. In zone I, Pa exceeds both Pa and Pv, and no blood flow occurs. In zone II, flow is primarily determined by the balance between Pa and Pa because in this zone Pa > Pa > Pv. Balloon inflation and PAC wedging may convert zone II status to zone I status by preventing blood flow. Only in zone III, where Pa > Pa > Pa, will pulmonary capillaries remain open and allow a patent conduit between the tip of the pulmonary artery catheter and the left atrium. Therefore, if the tip of the pulmonary artery catheter is in either zone I or zone II, PAOP will reflect alveolar pressure rather than mean LAP. PAOP
Table 1. Abbreviations

<table>
<thead>
<tr>
<th>Abbreviation</th>
<th>Description</th>
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<tr>
<td>APRV</td>
<td>airway pressure release ventilation</td>
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<tr>
<td>$C_p$</td>
<td>pulmonary compliance</td>
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<tr>
<td>CO</td>
<td>cardiac output</td>
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<tr>
<td>CPAP</td>
<td>continuous positive airway pressure</td>
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<tr>
<td>$C_w$</td>
<td>chest wall compliance</td>
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<td>LAP</td>
<td>left atrial pressure</td>
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<td>LVFP</td>
<td>left ventricular end-diastolic pressure</td>
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<td>LVEDV</td>
<td>left ventricular end-diastolic volume</td>
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<td>LVSWI</td>
<td>left ventricular stroke work index</td>
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<tr>
<td>MAP</td>
<td>mean arterial pressure</td>
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<td>PA</td>
<td>pulmonary arterial pressure</td>
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<td>PAC</td>
<td>pulmonary artery catheter</td>
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<td>PAEDP</td>
<td>pulmonary artery end-diastolic pressure</td>
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<td>PAOP</td>
<td>pulmonary artery occlusion pressure</td>
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<tr>
<td>$P_w$</td>
<td>airway pressure</td>
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<tr>
<td>$P_c$</td>
<td>pulmonary capillary pressure</td>
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<tr>
<td>PEEP</td>
<td>positive end-expiratory pressure</td>
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<tr>
<td>$P_{pl}$</td>
<td>pleural pressure</td>
</tr>
<tr>
<td>$P_{vo_2}$</td>
<td>pulmonary mixed venous oxygen pressure</td>
</tr>
<tr>
<td>PVP</td>
<td>pulmonary venous pressure</td>
</tr>
<tr>
<td>RAP</td>
<td>right atrial pressure</td>
</tr>
<tr>
<td>RVSWI</td>
<td>right ventricular stroke work index</td>
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<tr>
<td>Sao2</td>
<td>arterial oxyhemoglobin saturation</td>
</tr>
<tr>
<td>SvO2</td>
<td>mixed venous oxyhemoglobin saturation</td>
</tr>
<tr>
<td>SVRI</td>
<td>systemic vascular resistance index</td>
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<td>Vo2</td>
<td>oxygen consumption</td>
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will reflect LAP accurately only when the PAC tip is in zone III. Fortunately, most of the lung is in zone III when the patient is supine, and flow-directed pulmonary artery catheters usually enter zone III because most blood is flowing to that area.\(^6\)\(^9\) Although a flow-directed pulmonary artery catheter will rarely flow into zone I, zone III can be effectively converted to zone I or zone II status by loss of intravascular volume, change in position, airway obstruction, or application of positive-pressure ventilation (especially PEEP). Thus, the administration of fluid to a hypovolemic patient receiving PEEP may in rare instances result in a decrease in PAOP, while left ventricular end-diastolic volume actually increases, and a zone II PA catheter tip position is converted to a zone III catheter tip position.\(^1\)\(^0\)

Occlusion of the pulmonary artery in a segment of lung where the pulmonary venous pressure does not exceed the alveolar pressure (West's zones I and II) may be difficult to appreciate clinically, but should be suspected in the presence of any of the predisposing conditions mentioned previously. It is not uncommon for these conditions to be combined in critically ill patients with acute lung injury—patients in whom increased pulmonary capillary permeability is managed by reducing LAP and oxygenation problems are corrected with application of high levels of PEEP. A non–zone III catheter position may be recognized by observing the effects of sudden increases or decreases of PEEP on the measured PAOP. With normal $C_L$ and $C_W$, the $\Delta P_{pl}$ at the end of passive exhalation should be approximately one half of the $\Delta P_{aw}$. This is necessary because in normal lungs with ventilation at normal tidal volumes, $C_L$ approximates $C_W$ and the following relation exists:

$$\Delta P_{pl} \approx \Delta P_{aw} \cdot C_L/(C_L + C_W).$$

From this equation, it can be deduced that if the PAOP increases by greater than 50% of the change in the applied PEEP, a non–zone III catheter tip position is likely.\(^6\)

If the tip of the pulmonary artery catheter is vertically below or at the level of the left atrium, zone III conditions usually exist, unless extreme hypovolemia or high levels of PEEP are
Fig 2. Effect of PAC position on measurement of PAOP in three zones of the lung.

I: PA > Pa > Pv
II: Pa > PA > Pv
III: Pa > PV > PA

Applied.* Anteroposterior chest radiographs may not reliably locate the position of the tip of the PAC in relation to the left atrium, and lateral chest radiographs taken with the catheter in the wedge position or with its balloon inflated may be required to ascertain that the catheter is positioned correctly below the left atrium.* Other criteria to confirm zone III placement of the pulmonary artery catheter tip follow. First, correct placement is confirmed by a phasic PAOP tracing with A and V waveforms. Those waveforms promptly convert to a pulmonary arterial trace with balloon deflation and reappear with balloon inflation.* Second, blood should be easy to aspirate from the distal port of the PAC to exclude lodgement in a vessel without adequate blood flow. Third, the mean PAOP should be less than or equal to the PAEDP and less than the mean pulmonary artery pressure (unless large V waves are present). Finally, in the wedged position, arterialized blood can be aspirated, or an increase in mixed venous hemoglobin saturation to systemic arterial levels or above them can be shown if using an oximetric PAC. The first three criteria are more important because incomplete arterialization of a wedge position sample may occur if the tip of the pulmonary artery catheter is in a low ventilation-perfusion segment of the lung, despite being in zone III.*

Patients breathing spontaneously without continuous positive airway pressures (CPAP) may change status from zone III to zone II during the expiratory phase of ventilation as alveolar pressures become positive (especially if airway obstruction or bronchospasm occurs), while patients who are mechanically ventilated without PEEP may change zone III during exhalation to zone II during positive-pressure inspiration. Because intrathoracic (and alveolar) pressure is closest to atmospheric pressure at the end of exhalation, regardless of whether spontaneous or positive-pressure ventilation is occurring, intravascular pressures should be measured at this point to minimize the influence of intrathoracic pressure swings. Most time-based electronic sampling of pressure data is nonselective. Therefore, respiratory artifact affects the PAOP on a digital readout.* Most digital readouts may simply detect the highs and lows during some standard interval and produce data that are entirely artifactual. Electronic determinations of pressures by monitoring systems may be counterproductive in such instances. Sophisticated electronic algorithms are available to eliminate respiratory artifact, but even these systems may not work well in patients with tachypnea who are receiving intermittent mandatory ventilation.* In most patients with normal respiratory rates, direct observation of the respiratory pattern at the bedside will allow end-expiration to be defined. When patients are tachypneic, measurement of airway pressure may be required to define end-expiration.* Simultaneous paper recording of the airway pressure and pulmonary vascular pressure tracings will usually allow PAOP to be determined at end-expiration. However, when patients are weak or have high pulmonary or airway circuit compliance, end-expiration may be determined with more sensitivity and reliability by using airway thermistors.*

Although measurement of pulmonary vascular pressures at end-expiration minimizes the effects of pleural pressure changes on pulmonary vascular pressures, holding one's breath at end-expiration with an open glottis may be difficult for even the most alert and cooperative critically ill patient in respiratory distress. Use of hard-copy graphic displays or electronic algorithms to detect end-expiration are better alternatives in this setting. If an electronic-type averaged digital printout is used to determine PAOP, the systolic readout value will more closely reflect the correct end-expiratory PAOP than the mean value in a nontachypneic patient breathing spontaneously without CPAP. This occurs because the negative pressure of inspiration will be associated with a progressively more negative PAOP, rising back to baseline at end-expiration. Similarly, in the patient receiving positive-pressure ventilation without PEEP, the diastolic digital readout value will approximate the end-expiratory PAOP.

The practice of altering ventilatory patterns to measure vascular pressures in the pres-
ence of high levels of PEEP should be condemned. In any patient, errors in measurement of PAOP may be caused by the increased alveolar pressure at end-expiration that results in fewer zone III lung units. In addition, PEEP alters intrapleural pressure to varying degrees, depending on pulmonary compliance. Reduction in PEEP level or disconnection from the ventilator to measure vascular pressures may result in a completely different hemodynamic setting, because venous return may suddenly increase, producing a rebound hypervolemia in central vessels. In addition, many critically ill patients requiring high levels of PEEP may become severely hypoxemic and develop increased intrapulmonary shunt caused by alveolar collapse if PEEP is discontinued. This alveolar hypoxia is not only dangerous to patients, but may itself increase pulmonary vascular pressures.

Effective transmural pressure is the pressure inside the vessel minus the pressure around the vessel. PEEP may decrease the effective transmural pressure because the increase in vascular intraluminal pressure may be less than the increase in surrounding pleural pressure. When pulmonary and thoracic compliances are low, PEEP does not markedly increase intrapleural pressure, and intraluminal pressure generally will reflect transmural distending pressure. When pulmonary compliance is normal or increased, intrapleural and juxtacardiac pressures increase with PEEP, and intraluminal pressure no longer reflects true filling pressures. In addition, as noted above, PEEP may increase the sizes of zones I and II, especially in the hypovolemic patient. Because intraluminal vascular pressures are commonly measured, it is not uncommon to see an increased central venous pressure and PAOP during PEEP therapy when venous return to the heart is diminished. A discrepancy between transmural PAOP and intravascular PAOP may also be observed in patients with chronic obstructive lung disease who have gas trapping in lung units with elevated airway resistance and compliance, prolonged gas time constants, and short expiratory times. Gas trapping is associated with positive pleural pressures and increased end-expiratory lung volumes, providing an "auto-PEEP" effect. The effect of increased alveolar pressure caused by auto-PEEP on pleural pressure is variable and depends primarily on pulmonary compliance. The discrepancies between intravascular PAOP and transmural distending PAOP produced by auto-PEEP are essentially the same as when therapeutic PEEP is applied. Both can be measured in patients whose muscles are completely relaxed by occluding the expiratory circuit of the ventilator at end-exhalation and using the end-expiratory airway pressure as a reflection of the alveolar pressure.

Measurement of transmural distending pressure, though difficult, can be done. Extravascular pressure is approximated by intrapericardial or intrapleural pressure, which can be measured directly by inserting catheters in the pleural space or by an intraesophageal balloon. Neither method is widely applied in clinical practice. Insertion of pleural catheters is an invasive procedure with potentially serious complications. Esophageal balloons are not well tolerated by alert patients and require the lateral decubitus position for best accuracy. Many formulas have been developed to estimate the effect of PEEP on PAOP measurement. However, because pulmonary compliance and the effects of PEEP have wide interpatient variability, the usefulness of these formulas is questionable. Because clinicians do not usually quantitate transmural PAOP, clinical evaluation is more important than intravascular PAOP in assessing preload in patients when PEEP is applied.

Other modes of ventilatory support in critically ill patients may also produce discrepancies in measurement of the true distending transmural PAOP. Inverse-ratio ventilation is a form of positive-pressure ventilation that increases end-expiratory lung volume and produces a PEEP-like effect by dramatically shortening expiratory time. Intravascular PAOP will overestimate true PAOP when inverse-ratio ventilation is used. Conversely, airway pressure release ventilation (APRV) is a rediscovered form of "CPAP with release" that involves the intermittent release of CPAP to ambient pressure for 1 to 1.5 seconds. Ventilation to the same PaCO₂ with APRV in both normal and noncompliant lungs is reported to occur with lower mean airway and pleural pressures than conventional positive-pressure ventilation. Although these lower pressures should lessen the disparity between transmural and intravascular PAOP compared with conventional positive-pressure ventilation, when large
transpulmonary pressure gradients ($P_{AW} - P_{PL}$) are necessary for adequate APRV, PAOP should be measured at the end of the positive-pressure plateau to minimize the effects of a large swing of intrathoracic pressure created by APRV.

**Other Conditions in Which PAOP Overestimates LVEDP**

There are a variety of other conditions that result in discrepancies between PAOP and LVEDP (Table 2). Tachycardia produces shorter diastolic times and diminishes ventricular filling. When the heart rate is increased sufficiently, the left atrium contracts against a partially closed mitral valve, and an end-diastolic gradient exists between the PAEDP and the PAOP or LVEDP. In humans, when heart rates are greater than 115 per minute, PAEDP is greater than PAOP. PAOP may also exceed and not reflect LVEDP if increases of pulmonary vascular resistance occur even in the absence of tachycardia, because there may be a functional discontinuity in the common chamber between the PAC tip and the left atrium at the level of the pulmonary veins. Such increases in resistance of the pulmonary venous system to blood flow may occur as a result of sepsis, pulmonary venous occlusive disease, and other entities, and cause the PAOP and PAEDP to exceed the LAP.

Obstruction to pulmonary venous drainage may cause PAOP (and PVP) to exceed LVEDP. If the mitral valve orifice is obstructed by mitral stenosis, an artificial valve, or a left atrial mass (clot, myxoma), LAP and PAOP may both exceed LVEDP. If the pulmonary veins are compressed by a mediastinal neoplastic or fibrotic process, or pulmonary venous thrombosis occurs, PVP and PAOP will exceed LAP, although LAP will reflect LVEDP. Compression of the pulmonary venous system may also occur in low-flow states, or may occur secondary to increased intrathoracic pressure.

Clinical conditions that produce large V waves in the PAOP and PAP trace also create problems in data interpretation. The V wave corresponds to the flow of blood into the atrium against a closed mitral valve toward the end of ventricular systole. If tall V waves on the PAOP trace are not recognized the PA catheter may be misinterpreted as being “unwedged,” causing the operator to make further attempts to wedge the catheter, with the attendant risk of pulmonary artery rupture. Overdamping of pressure waveforms by recording systems may occasionally prevent recognition of large V waves on a PAOP trace or cause the mistaking of a damped pulmonary arterial waveform for a PAOP waveform. Recognition of the usual relationships between electrical and mechanical cardiac events is necessary to avoid this pitfall. The normal pulmonary artery waveform consists of a systolic peak and a diastolic trough, with a dicrotic notch associated with closure of the pulmonic valve (Fig 3). The peak of the pulmonary artery systolic wave occurs within the electrocardiographic T wave. The peak of the V wave occurs at the end of ventricular systole at a time when the atrium is maximally filled, and appears in the PAOP tracing after the T wave has been inscribed because of delay in transmitting this left atrial event through the pulmonary vascula-

![Fig 3. Relationship between electrocardiographic and pulmonary artery (occlusion) pressure waveform events. Dashed lines represent LV pressure waveform.](image-url)
Large V waves may be seen with mitral regurgitation, but they are not necessarily diagnostic. They also occur in mitral stenosis when there is poor left atrial compliance, when there is pulmonary venous volume overload as in acute left-to-right shunting, or whenever left atrial/pulmonary vein compliance decreases with increased PAOP. Regurgitant flow across the mitral orifice produces more prominent C and V waves, which usually are fused and defined as a large V wave. When filling volumes are larger, a small stroke volume can result in a large pressure change because the left atrium is no longer on the horizontal portion of its pressure-volume curve. Similarly, if mitral regurgitation is present, the height of the V wave is more dependent on left atrial compliance than on the severity of regurgitation.

The appearance of large V waves in the PAOP tracing has also been associated with the onset of new myocardial ischemia. However, if preload is augmented in a patient with a noncompliant atrium secondary to chronic ischemic heart disease, there will be a large increase in LAP and occurrence of V waves even in the absence of additional ischemia. Reduction of preload with nitroglycerin will diminish LAP and V-wave size and move the patient back to the horizontal portion of the left atrial end-diastolic pressure-volume curve, regardless of whether or not ischemia is present. Because of this, the occurrence of elevations in PAOP with new V waves may be sensitive to the onset of new ischemia, but are not highly specific, even if nitroglycerin reverses these changes. When V waves are present in the PAOP trace, what is the best way to estimate LVEDP? The pressure preceding the large V wave (after the “A” wave) is a close approximation of LVEDP (Fig 3). Digitally, LVEDP is best assessed using the diastolic PAOP when V waves are present because the mean digital electronic PAOP will be greater than the true LVEDP in this circumstance.

Conditions in Which PAOP Underestimates LVEDP

In contrast to conditions that increase PAEDP and PAOP relative to LVEDP, a right bundle branch block causes the PAEDP to be lower than the mean LAP. In the presence of normal pulmonary vascular resistance, the delay in right ventricular systole allows the pulmonary artery pressure to continue to decrease during the X descent of the left atrial pressure contour. The PAOP may underestimate the baseline LAP in patients with a markedly reduced pulmonary vascular bed, such as that occurring after pneumonectomy or large pulmonary thromboembolism. The baseline LAP would be underestimated because inflation of the balloon on the PAC may occlude so much cross-sectional area of the remaining pulmonary vascular bed that the venous return to the left side of the heart is reduced, thus decreasing LAP. The falsely low value for PAOP may mislead the clinician and result in fluid management that causes or worsens pre-existing pulmonary congestion. This phenomenon should be suspected whenever the simultaneously measured systemic arterial pressure and/or arterial oxygen saturation (pulse oximetry) decreases during balloon inflation. If the PAOP is measured without inflating the balloon, ie, by advancing the catheter and wedging it into a smaller peripheral artery, a more accurate estimate of the PAOP may be obtained. However, this method is not recommended in clinical practice because the rigid catheter tip may perforate the pulmonary artery.

Although a noncompliant left atrium will cause overestimation of LVEDP when using mean PAOP, a noncompliant left ventricle may cause the opposite type of discrepancy. The normal left atrial A wave is caused by atrial contraction and generally follows the peak of the electrical P wave by 200 to 250 milliseconds, usually occurring shortly after the QRS complex. When left ventricular compliance decreases, the A wave of the PAOP or PAP trace may increase. These A waves are often difficult to see on the transduced PAOP waveform, even with a high-fidelity recording system. If the preload of the left ventricle is defined as the initial stretch on the myocardial fibers just prior to systole, ie, just after the peak of atrial contraction designated by the A wave, the actual LVEDP may be underestimated, if the pressure is recorded before the peak of the A wave (Fig 3). This can occur if the A wave is not clearly seen. The discrepancy is largest when the left ventricle is noncompliant, the heart is in slow sinus rhythm, and there is no
associated valvular heart disease. Conversely, large pathologic A waves may occur with mitral stenosis (when sinus rhythm is present), complete heart block, atrial myxoma, and early acute heart failure. In these situations, the diastolic PAOP (measured at end-exhalation) will give the most reliable reflection of left ventricular filling pressures. When aortic insufficiency exists, the regurgitant flow back into the left ventricle during diastole causes premature closure of the mitral valve and causes left atrial pressure (hence PAOP) to underestimate the elevated LVEDP.

**Impairment of PAOP for Pulmonary Capillary Hydrostatic Pressure**

Although PAOP may accurately reflect LVEDP, LAP, and pulmonary venous pressure, it may be misinterpreted as the hydrostatic pressure determining the rate of fluid filtration from the pulmonary capillaries into the interstitium and air spaces of the lung. The Starling equation attempts to quantitate the fluid flux across a microvascular membrane and takes into account not only the relative permeability of the membrane, but also the balance between the hydrostatic gradient tending to push fluid out (Pc minus tissue fluid pressure) to the opposing colloid osmotic pressure gradient (plasma minus tissue fluid colloid osmotic pressure). Clinicians often use PAOP to estimate the tendency for hydrostatic pulmonary edema. However, PAOP is an indicator of LAP, not Pc. For blood to flow forward through the lungs, Pc must be higher than PAOP. Under normal conditions, the vascular pressure gradient across the pulmonary circulation is so small that PAOP is very close to Pc. However, when pulmonary vascular resistance increases, Pc exceeds PAOP. Pc can be described in terms of PAP, PAOP, and the part of the pulmonary vascular resistance on the arterial (Ra) and venous (Rv) sides of the capillary:

\[
Pc = \frac{PAP \cdot Rv + PAOP \cdot Ra}{Ra + Rv}
\]

Accordingly, a patient may have a normal or near-normal PAOP and still have hydrostatic pulmonary edema. A method has been described in animals and humans that uses the pulmonary artery pressure profile after balloon occlusion of the large pulmonary vessels (precapillary resistance) changing to a slower decline as blood spreads through the large cross-sectional area of the pulmonary capillaries to the pulmonary veins and the left atrium (postcapillary resistance). The inflection point or transition from fast to slow decline is thought to reflect the Pc, and the Pc-PAOP gradient is thought to reflect postcapillary resistance (Fig 4). Large gradients between estimated Pc and PAOP have been shown in patients with increased pulmonary venous resistance after mitral valve replacement.

In numerous publications, the pressure measured when a balloon-tipped pulmonary artery catheter occludes flow to a lung segment has been called the pulmonary capillary wedge pressure (PCWP). Because it is obviously not a capillary pressure, the term PAOP (pulmonary artery occlusion pressure) is more appropriate. PAOP may underestimate Pc whenever increased pulmonary vascular resistance occurs, and this should be considered whenever levels of exogenous or endogenous pulmonary vasoconstrictors such as catecholamines, histamine, serotonin, or prostaglandins are increased. Simply observing an increased difference between the PAEDP and the PAOP confirms this suspicion, while a difference of less than 2 to 3 mm Hg means the PAOP more closely approximates the Pc. Unfortunately, accumulation of lung water involves transudation not only through pulmonary capillaries but also through small pulmonary arterioles. Thus, effective hydrostatic pressure for accumulation of lung water frequently exceeds not only PAOP but also Pc. Lymphatic function also must be considered when assessing the significance of a particular Pc. In addition to physical disruption of lymphatics by disease processes, increased RAP produces increased lung water by opposing lymph drainage. Finally, many influences, most notably respiratory artifact, make it very difficult to determine Pc with certainty in most patients.

![Fig 4. Estimation of Pc using the pulmonary pressure profile after inflation of PAC balloon (open arrow).](image-url)
Errors in Pressure Measurement Caused by Faulty Transduction

The problems of electronic pressure measurement by a transducer include both overdamping and underdamping of the transduced waveform. The effects of overdamping have been noted above; underdamping similarly produces falsely high systolic and occasionally low diastolic pressures, although the electronically determined mean pressure will be accurate. Most clinicians recognize that large air bubbles along the recording system cause systolic underestimation, but many fail to recognize that very small air bubbles (50 to 100 µL) introduced into the system will cause systolic overshoot. Incorrect transducer placement and inaccurate calibration produce additional reasons for errors in measurement of PAOP. The management of these technical problems has been reviewed elsewhere. Clearly, attention to these technical details is necessary before relying on a pressure measurement to assess left ventricular function.

Validity of PAOP as a Measure of Preload

It is clear, based on the previous discussion, that a variety of pathophysiologic conditions and ventilatory maneuvers can upset the balance of the relationship of PAEDP, PAOP, PVP, LAP, and LVEDP, as well as between intravascular and transmural pressures. If preload is defined as the distending force or stretch of the fibers of the left ventricle at end-diastole, each step away from end-diastolic fiber length further attenuates the validity of that variable as a measure of preload (Fig 1). The net diastolic distending force has been shown to correlate better with the left ventricular end-diastolic volume (LVEDV) than with the left ventricular end-diastolic pressure. Therefore, to reflect left ventricular preload reliably, the PAOP must correlate with the LVEDV reliably. Normally, the relationship between the end-diastolic transmyocardial filling pressure and the end-diastolic volume of the left ventricular filling is curvilinear (Fig 5). Ventricular compliance is defined as the instantaneous relationship between the ventricular end-diastolic volume and pressure (dv/dp). The end-diastolic volume is determined by ventricular distensibility (compliance) and the transmural distending pressure (LVEDP — juxtaocular pressure). Changes in compliance may result from moving along a single diastolic pressure volume curve, ventricular compliance being greater at lower levels of ventricular filling than at higher levels. Thus, changes in LVEDP will result in different changes in LVEDP, depending on the location on the curvilinear diastolic pressure-volume curve (Fig 5). At low preload, a large increase in LVEDP (ΔV) will result in a small change in end-diastolic pressure (ΔP1) or no change. Conversely, regardless of the absolute position of the pressure-volume relationship, at high levels of preload (large EDV), the same change in EDV will be associated with a large variation in EDP (ΔP2).

Changes in left ventricular compliance may also result from changes of the inherent stiffness properties of the myocardium, represented by a shift of the pressure-volume curve to the left (lower compliance) or to the right (higher compliance) (Fig 5). Thus, an elevated LVEDP (or PAOP) may reflect high volume and preload for a ventricle with normal or increased compliance (B, C), or a low end-diastolic volume and preload in a ventricle with decreased compliance (A). In addition, an increase in LVEDP may indicate an increase in preload (B-F) or may reflect a change in the modulus of chamber stiffness and decreased compliance without any change in preload (B-D). These phenomena have been shown in both animals and humans to produce...
significantly discrepancies between PAOP and LVEDV.\textsuperscript{58,59}

Left ventricular compliance is influenced by left ventricular preload, left ventricular wall mass,\textsuperscript{60} myocardial fiber stiffness,\textsuperscript{61} right ventricular end-diastolic volume,\textsuperscript{55} temperature,\textsuperscript{62} heart rate,\textsuperscript{63} and oncotic pressure.\textsuperscript{64} Increased systemic systolic arterial pressure shifts the end-diastolic pressure-volume curve to the left,\textsuperscript{64} as does the administration of exogenous catecholamines.\textsuperscript{65} Individual myocardial fiber stiffness may increase in the presence of myocardial ischemia, myocardial fibrosis, and infiltrative cardiomyopathies such as with amyloidosis. Global changes in the modulus of chamber stiffness may be seen with sepsis,\textsuperscript{66} left ventricular hypertrophy which results in decreased left ventricular compliance, or with chronic left ventricular dilation due to valvular heart disease or dilated cardiomyopathies which result in enhanced left ventricular compliance.\textsuperscript{60} The clinical impact of such alterations in compliance is apparent from studies in patients with acute myocardial infarction where optimal left ventricular filling requires a higher PAOP than would be required in the absence of ischemia.\textsuperscript{67} Vasoadative infusions of drugs such as nitroglycerin,\textsuperscript{68} nitroprusside,\textsuperscript{69} vasopressors,\textsuperscript{69} inotropes,\textsuperscript{65} and \( \beta \)-blockers\textsuperscript{70} all have been shown to shift left ventricular diastolic compliance curves. Drugs such as nitroglycerin may have combined effects on altering LVEDP. If ischemia is present, the majority of the decrease in EDP may be caused by a decrease in the modulus of chamber stiffness with a shift in the pressure-volume curve to the right,\textsuperscript{68} increased ventricular compliance, and little change in preload (EDV), (Fig 5, D-B). If ischemia is not present, nitroglycerin may primarily produce movement along a single pressure-volume curve,\textsuperscript{68} and the decrease in LVEDP will be associated with a large change in preload (Fig 5, E-B). In addition, if the venodilating effect of nitroglycerin reduces right ventricular end-diastolic pressure and right atrial pressure, pericardial pressure will decrease because a linear relationship exists between the right atrial pressure, or the right ventricular end-diastolic pressure, and the pericardial pressure.\textsuperscript{68,71} Reduction in the constraining pericardial pressure will cause the intracavitary EDP-EDV curve to be shifted to the right without any change in the inherent stiffness properties of the left ventricle.\textsuperscript{68} In different patients, equal changes in PAOP after nitroglycerin may be associated with quite different changes in left ventricular end-diastolic filling and preload, and casts doubt on the specificity in changes in PAOP reflecting ischemia in the absence of electrocardiographic changes or other corroborating evidence.\textsuperscript{40}

Because the left and right ventricles are physically joined by the interventricular septum, and are constrained jointly by the pericardium, the end-diastolic pressure-volume curve of the left ventricle is dependent on the diastolic volume of the right.\textsuperscript{55} Because of this ventricular interdependence, any factor that increases right ventricular preload will result in a leftward shift of the left ventricular end-diastolic pressure-volume curve. Additionally, right ventricular preload is dependent on right ventricular afterload, so that disease states or drugs which increase right ventricular afterload, eg, acute pulmonary hypertension,\textsuperscript{59} or drugs which decrease right ventricular afterload, eg, nitroglycerin\textsuperscript{68} and nitroprusside,\textsuperscript{69} may affect the left ventricular pressure-volume relationship because of ventricular interdependence. Therefore, administration of vasoactive drugs that affect right ventricular preload and afterload may alter LVEDP because of shifts in the left ventricular pressure-volume relationship without parallel changes in end-diastolic volume.

Thus, even if the assumption of PAOP \( \approx \) LVEDP is accurate, PAOP may not always provide a reliable index of left ventricular preload.\textsuperscript{56,58,59,69} This was clearly shown in a recent study that compared PAOP and LVEDP with left ventricular cavity size assessed with two-dimensional echocardiography before and after cardiac surgery.\textsuperscript{72} PAOP and LVEDP did not correlate with absolute left ventricular cavity size either before or after bypass. The change in left ventricular cavity size was opposite in direction to the changes in PAOP and LVEDP in 50% and 67% of measurements, respectively. After cardiopulmonary bypass, LVEDP often rose, while echocardiography showed a marked decrease in left ventricular cavity size. Both hypotension and reduced cavity size responded to volume loading, a treatment not suggested by LVEDP or PAOP.
measurements. Currently, there is hope that transesophageal echocardiography will provide continuous measurement of ventricular cavity size in clinical practice.

Changes in left ventricular compliance often result in changes in stroke volume. If PAOP does not change, a decrease in stroke volume is therefore usually interpreted as a decrease in contractility. If a decrease in end-diastolic volume was recognized at the same PAOP, this would be interpreted as decreased ventricular compliance with diminished preload rather than depressed contractility. To determine whether increasing preload or increasing contractility is correct treatment, the clinician must determine where the patient lies on the diastolic pressure-volume curve. This can be done by rapidly administering a volume infusion to determine where a given patient lies on his left ventricular end-diastolic pressure-volume curve. If a rapidly administered volume infusion does not increase PAOP by at least 3 mm Hg, the ventricle is still on the horizontal portion of its diastolic pressure-volume curve; if it rises by more than 7 mm Hg, the ascending limb has been reached. Further volume infusion is not likely to increase preload nor improve stroke volume, and other therapies may be needed. Continued volume infusion may increase PAOP and the risk of hydrostatic pulmonary edema dramatically. Appropriate interventions would be either to increase the contractile state of the myocardium (inotropes) or to reduce the modulus of chamber stiffness (nitroglycerin).

The clinician can construct a family of Starling curves only if he is able to show that end-diastolic volume has changed in response to a rapid volume infusion by noting a substantial increase in PAOP. Slow volume infusions may not allow such a relationship to be tested because intravenously administered fluid (especially crystalloid) may equilibrate rapidly with the extravascular space in critically ill patients, and because the ventricular diastolic pressure-volume relationship may vary abruptly. After showing a change in end-diastolic volume, the corresponding change in stroke volume can be used to determine if the Frank-Starling relationship between stroke volume and PAOP is displaced to the right, as might occur with decreased left ventricular compliance from an ischemic or hypertrophied left ventricle or pericardial disease. Because the usual Frank-Starling relation between the ventricular pressure and volume is not valid when myocardial compliance changes, the PAOP may not be an accurate monitor of left ventricular performance. Clinical assessment of the patient's response to therapy is essential to optimizing treatment. The effects of volume infusion, vasodilating agents, ventilatory changes, and disease states on left ventricular preload are not entirely predictable when PAOP (or even LVEDP) are measured; however, the net effect can usually be surmised from resultant changes in the measured stroke volume.

**Pitfalls in Determination of Stroke Volume**

The ability to rapidly obtain accurate measurements of cardiac output in critically ill patients is one of the principal advantages of the pulmonary artery catheter. Because assessment of optimal left ventricular preload depends in part on measurement of stroke volume, knowledge of the pitfalls in determination of cardiac output using a thermodilution pulmonary artery catheter is essential. Thermodilution method is the most widely used means of measuring cardiac output. Injection of a solution (whose temperature is cooler than body temperature) into the right atrial lumen of the catheter allows cardiac output to be determined by the indicator-dilution technique. As blood flow in the right side of the heart increases, the cooler solution becomes more and more diluted by warmer venous blood, and less temperature drop is detected by the pulmonary artery thermistor. Cardiac output is then calculated by a computer that uses the Stewart-Hamilton equation, incorporating the area under the thermodilution curve obtained by plotting the decline in pulmonary artery temperature v time. A number of important practical considerations must be satisfied for the successful application of the indicator-dilution method. There must be reasonable circulatory stability for serial thermodilution measurements to be reproducible, and the volume and temperature of the injectate must be accurately measured. The duration of injection must be brief (<4 seconds for 10 ml) to enhance the signal-to-noise ratio, particularly if room-temperature injections are used. Recent work
using an in vitro model, however, failed to show any difference in accuracy or reproducibility between iced injectate and room-temperature injectate when injection was performed rapidly. The proximal (RA) port of the pulmonary artery catheter may be within the percutaneous catheter introducer sheath, or very close to it, when the distal tip of the pulmonary artery catheter is correctly positioned in one of the main pulmonary arteries after insertion via the subclavian or innominate route in a patient with a small body habitus and normal-sized heart. This may produce retrograde injection of indicator solution into the introducer catheter sheath and result in a spuriously high cardiac output. Similarly, if cold injectate is used, warming may occur in the operator’s hand or in the catheter, and may produce false overestimations of cardiac output unless injectate temperature is measured in-line at the site of injection into the right atrial port of the PAC. Rapid intravenous administration of cold fluids can also create large errors in thermodilution cardiac output determinations unless the measurement is delayed for at least 30 seconds after stopping the rapid volume infusion. The timing of injection in relation to respiratory events may be important to achieving reproducibility and to differentiate actual physiologic changes in cardiac output from phasic oscillations associated with respiration that are due to fluctuations in venous return, changes in afterload, and alterations in ventricular geometry. When thermodilution cardiac outputs are randomly obtained without reference to the respiratory cycle, they may vary by 1.4 to 1.7 L/min, while injections initiated at end-exhalation will vary only by 0.6 to 0.7 L/min. Nevertheless, determinations made at one point in the respiratory cycle are less representative of true average cardiac output, and undergo phase shifts with PEEP alterations. Thus, accurate cardiac output determinations may require multiple measurements made throughout the respiratory cycle.

Pathophysiologic conditions may also make thermodilution cardiac output measurements inaccurate. Thermodilution cardiac output may be unmeasurable or spuriously underestimated in patients with tricuspid regurgitation or bidirectional intracardiac shunts. Similarly, when atrial fibrillation is present, the beat-to-beat variability in cardiac output may make reproducibility poor, even when determinations are performed sequentially over a short period of time.

Errors in Interpretation of Mixed Venous Oxygen Saturation

Several studies in acute care settings have suggested that \( \text{SvO}_2 \) monitoring is useful as an “early warning indicator” of low cardiac output. Rearranging the Fick equation and ignoring the dissolved portion of blood oxygen content, where \( Hb \) is hemoglobin concentration, shows that:

\[
\text{SvO}_2 = \text{SaO}_2 - (\text{VO}_2/\text{CO} \cdot 1.36 \cdot \text{Hb})
\]

Changes in \( \text{SvO}_2 \), therefore, may not only reflect changes in \( \text{CO} \), but also depend on arterial oxygenation, \( \text{V}_{O_2} \), and \( Hb \). Increases of \( \text{V}_{O_2} \) in shivering or febrile patients produce a decrease in \( \text{SvO}_2 \) for a given \( \text{CO} \). \( Hb \) can change sufficiently during fluid resuscitation (rapid blood transfusion for hemorrhage) to obscure the relationship between \( \text{SvO}_2 \) and \( \text{CO} \). Decreases in \( \text{SaO}_2 \) occur with increased venoarterial admixture during ventilatory manipulations and positioning changes during thoracic surgery. Postoperatively, in the even less-controlled environment of the intensive care unit, it is not surprising that several authors have found poor correlations between changes in \( \text{SvO}_2 \) and changes in \( \text{CO} \). Though a very low \( \text{SvO}_2 \) is always considered ominous, a normal \( \text{SvO}_2 \) does not always imply adequate regional tissue \( P_{O_2} \). Animal and clinical studies have shown that when \( P_{VO_2} \) is less than 28 mm Hg because of decreased \( \text{CO} \), lactic acidosis supervenes. Several other studies, however, have shown lactic acidosis without accompanying mixed venous desaturation in both septic patients and cardiac patients. When \( \text{VO}_2 \) decreases from severe maldistribution of systemic flow in shock or early in the postresuscitation period, ischemic tissues are underrepresented in their contribution to \( \text{SvO}_2 \). In addition, ischemic tissue acidifies its capillary blood, producing a right shift in the \( \text{O}_2 \)-hemoglobin dissociation curve (Bohr effect). This reduces the saturation at a given \( \text{P}_{O_2} \); however, upon mixing with blood from nonischemic regions, the previously acidified hemoglobin is alkalinized, increasing its...
affinity for oxygen and raising the saturation of the mix. Peculiar flow dependence of \( O_2 \) delivery above normal levels also dissociates \( S_iO_2 \) from \( CO \) in patients with adult respiratory distress syndrome. Thus, it is important not to interpret changes in \( S_iO_2 \) (or lack of them) as reflecting changes in \( CO \) (or lack of them), despite stable \( SaO_2 \) and \( Hb \).

On the technical level, fiberoptic \( SvO_2 \) measurement can be affected by the light reflected from arterial walls, despite attempts to minimize the effect by use of a third wavelength for saturation measurements. Fiberoptic \( SvO_2 \) usually increases artifactually when the arterial walls are partially collapsed during PEEP application or when PAOP is measured. Likewise, distal migration of the catheter into a small artery often brings the arterial walls close enough to increase \( SvO_2 \), and simple repositioning of the catheter tip will correct this problem. Introduction of signal processing and an artifact meter into the system allows these problems to be detected. The intensity magnitude alarm is generally out of the acceptable range when these artifactual conditions are present. Occasionally, especially during PEEP manipulations, this can occur with the intensity meter of the oximetry device reading in the working range. Normal or increased \( SvO_2 \) may also be seen in other situations in which \( CO \) is inadequate, but samples are contaminated with more saturated blood from a left-to-right intracardiac shunt. Of course, \( SaO_2 \) and \( Fro_2 \) may not reflect the redox potential of the mitochondria when oxygen utilization is blocked, as in cyanide toxicity. Congenital and acquired hemoglobin abnormalities, such as carboxyhemoglobin and methemoglobin, affect the light reflectance of hemoglobin. Oximetric PACs misread these abnormal hemoglobins as oxyhemoglobin, and may overestimate true \( S_iO_2 \) in their presence. While some of the above problems can be obviated by measuring both \( P_iO_2 \) and \( S_iO_2 \) in mixed venous blood samples drawn from a PAC, switching from a continuous to an intermittent monitor is a disadvantage. Furthermore, it is common that \( S_iO_2 \) and \( P_iO_2 \) be increased factiously by contamination of desaturated mixed venous blood with saturated pulmonary capillary blood, because withdrawal of blood from the distal lumen is too rapid. This situation should be suspected if in vitro (preinsertion) calibration of an oximetric PAC results in a significantly lower \( S_iO_2 \) than that from subsequently coximetered pulmonary arterial blood, or if the hemoglobin saturation of blood drawn from the proximal lumen is commensurately lower.

**Pitfalls in Interpretation of Hemodynamic Indices Derived From Pulmonary Artery Catheter Data**

The physiologic parameter known as systemic vascular resistance (SVR) is commonly calculated from the following relationship:

\[
SVR = (MAP - RAP) \times 79.9/CO
\]

where MAP = mean arterial pressure and RAP = right atrial pressure. The logical basis for this expression arises by analogy from Poiseuille’s law for fluid flow within rigid pipes, which states that flow is proportional to the difference between the upstream and downstream pressures. Although \( CO \), stroke volume, and stroke work are usually indexed to body size, it is not yet common practice to do so for vascular resistance. Because the peripheral arterial tree branches in parallel and is proportional to body size, larger individuals have lower resistance (more vessels in parallel) than smaller individuals. For example, a 110-lb 62-inch female with a MAP of 90 mm Hg, RAP of 5 mm Hg, and a \( CO \) of 4.2 L/min has a calculated SVR of 1617 dynes \( \cdot \sec \cdot \text{cm}^{-5} \), while a 210-lb 72-inch male with the same MAP and RAP and a \( CO \) of 6.16 L/min (same cardiac index) has a calculated SVR of 1,103 dynes \( \cdot \sec \cdot \text{cm}^{-5} \). Calculating SVR index by using cardiac index instead of CO in the denominator normalizes the parameter to body surface area and yields an SVR index (SVRI) of 2,426 dynes \( \cdot \sec \cdot \text{cm}^{-5} / \text{m}^2 \) for both individuals, suggesting that their vascular trees were similarly matched to their \( CO \). It would seem that a method of indexing systemic vascular resistance should be universally adopted into clinical practice.

An additional problem with SVR measurements concerns the choice of the appropriate downstream pressure to be used in the calculation. Although most clinicians use RAP as the downstream pressure, there may be a vascular
waterfall,\textsuperscript{107} or a critical venous closing pressure,\textsuperscript{108} producing a venular pressure 20 to 30 mm Hg above RAP. Thus, increases in SVR may occur without arteriolar vasoconstriction. In shock states (especially with disseminated intravascular coagulation), large beds of capillaries are removed from the circulation so that resistance increases but arteriolar tone may not. When the RAP is much less than the effective downstream pressure, the simplistic assumption of the equation just mentioned not only produces inaccurate estimations of arterial resistance but also obscures the assessment of changes of that resistance at different pressures. For example, if cardiac output decreases from 4.20 L/min to 1.99 L/min, and there is a simultaneous decrease in MAP from 100 mm Hg to 50 mm Hg, without any change in RAP, calculation of SVR using a downstream pressure of 5 mm Hg shows no change in resistance, and suggests that myocardial depression was the sole cause of the change in blood pressure. However, if 20 mm Hg is used for the effective downstream pressure, with the same change in CO and MAP, the calculated resistance decreases 21% (1522 to 1204 dynes \( \cdot \) sec \( \cdot \) cm\(^{-5} \)), and uncovers its contribution to the decrease in blood pressure. Because there is no convenient way for clinicians to assess the effective downstream pressure except under special conditions,\textsuperscript{106} the comparison of SVR at widely different pressures should be made with caution.

**Left Ventricular Stroke Work Index (LVSWI)**

LVSWI may be calculated from measurement of heart rate, CO, LVEDP, and MAP. Because LVEDP must be estimated from PAOP, errors occur whenever PAOP does not equal LVEDP. In general, PAOP artifacts increase PAOP relative to LVEDP (Table 2). Therefore, LVSWI tends to underestimate ventricular performance. In general, artifacts also increase LVEDP relative to LVEDV. Therefore, LVEDP tends to underestimate preload. Both types of artifact produce LVSWI values which underestimate contractility. Therefore, it is recommended that no conclusion be drawn regarding changes in contractility when PAOP and LVSWI are changing in the same direction, especially if SVR is changing in the opposite direction.\textsuperscript{106}

**Right Ventricular Stroke Work Index (RVSWI)**

Analogous to LVSWI, RVSWI may be calculated from measurement of heart rate, CO, RVEDP, and mean pulmonary artery pressure (MPAP). RAP matches RVEDP better than PAOP matches LVEDP, but RAP rarely can overestimate RVEDP. No artifact commonly causes RAP to underestimate RVEDP. Underestimates of contractility are more likely to be produced by reductions in RV compliance. There is another source of artifact that results from the greater difficulty in estimating impedance using MPAP. Multidirectional echo waves of pressure in the pulmonary arteries distort the Poisellian assumption that a given pressure difference corresponds to a definite flow. The ratio of pulmonary oscillatory-to-pulsatile power has been shown to be as high as 40%.\textsuperscript{109} Thus, relatively steady flow during pressure transients in the pulmonary artery may cause gross underestimation of right ventricular performance.

**CONCLUSION**

If careful attention is paid to the mechanics of pressure measurements, the PAC will reliably and reproducibly measure intraluminal pressures. However, considerable clinical judgment is necessary to extrapolate these pressure measurements to estimate preload and the risk of pulmonary edema. The PAC may also help the clinician assess the effects of acute hemodynamic interventions on the balance between oxygen supply and demand. However, he must be cognizant of the many artifacts negating the relationship among PAOP, LVEDP, and LVEDV and wary of the pitfalls in the interpretation of calculated hemodynamic indices. If careful attention is paid to the above details, the PA catheter can provide the clinician with measures of cardiovascular performance not previously available. The data must be interpreted and acted upon using astute clinical judgment to identify an appropriate range for PAOP, optimal preload, and CO in a given patient.

The PAC only provides physiologic data. Systematic clinical investigation of the effect of manipulations of filling pressures, flows, and \( \text{SvO}_2 \) on outcome has not been done and is much needed. If ranges of PAOP, CO, and \( \text{SvO}_2 \) achieved by defined therapeutic regimens can be
shown to improve survival in certain types of patients, indications for the use of the PAC will be confirmed. Currently, in the absence of such studies, arguments over the PAC's usefulness (or lack thereof) are premature. Nevertheless, the PAC will never prove its usefulness unless the clinician gives due consideration to the technical and physiological factors which affect interpretation of the data. It is the goal of this review to help disseminate knowledge of those factors so that the PAC can be applied successfully.

REFERENCES


