Since the introduction of the pulmonary artery catheter (PAC) in the 1970s, initial enthusiasm has been tempered by studies suggesting equivocal or potentially increased mortality when employed in the critically ill (1). Academic societies have convened expert panels to review the literature, poll society membership, and discuss important issues regarding PAC utilization; the usual conclusion calls for more rigorous, adequately powered, randomized, controlled trials (2). For many critical care practitioners, the benefits of PAC warrant its continued use in high-risk patients; interestingly, the PAC continues to be the gold standard by which noninvasive cardiac output (CO) monitoring is judged.

Inherent in the use of the PAC to improve outcomes is the assumption that flow-related variables such as cardiac output/cardiac index (CO/CI) and oxygen delivery (DO2) are important for survival. Shoemaker et al. observed that survivors and nonsurvivors did not differ in traditional values of blood pressure (BP), heart rate (HR), urine output, and arterial oxygen tension (PaO2). Contrary to conventional wisdom of the time, flow-related variables such as DO2, oxygen consumption (VO2), and CO were superior determinants of mortality (3,4). In addition, Bihari et al. (5) reported that as DO2 was augmented in critically ill patients, VO2 of the tissues increased suggesting, at least in part, that tissue consumption can become supply dependent. Mathematically, DO2 and VO2 are coupled because CO is on both sides of the equation; is this a true physiologic dependency? In a study using independent measurement of VO2, Yu et al. (6) demonstrated that some but not all critically ill patients showed an increase in VO2 as DO2 improved. Titration of DO2 until the VO2 slope plateaus is an attractive but impractical theory as measurement of VO2 is cumbersome. Perhaps an easier, and more clinically useful, end point of resuscitation is the mixed venous oxygen saturation (SvO2)—a reflection of the balance between supply (DO2) and demand (VO2) (7).

The current body of literature regarding resuscitation to CI, DO2, and SvO2 goals (both for and against) is vast and beyond the scope of this chapter; nevertheless, these are important concepts and are summarized in the practice guidelines for PAC (2). Although global values of DO2 are important, the exact threshold is unknown because they may not reflect oxygen transport at the cellular level. Until we have a tool to measure tissue-level oxygenation states (or one tissue bed that is a surrogate marker for the rest of the body), the amount of DO2 necessary for tissue perfusion will remain controversial.

Over the decades, clinicians have progressed from treatment goals of BP, HR, and urine output to markers of anaerobic metabolism such as lactic acid and base deficit, and then to flow-related variables such as DO2, VO2, and SvO2. While we have progressed in our goal-directed therapy, the whole concept has been called into question recently (8). Ideally, a user-friendly, noninvasive device could measure tissue oxygenation and the energy state of cells, thereby allowing clinicians to “titrate” DO2 to meet tissue demands rather than aiming for a single global survival values of DO2 and SvO2 (3,4). Hemodynamic monitoring affords us the ability to treat the critically ill in a goal-oriented manner; we see these goals (BP, HR, urine output, DO2, VO2, and SvO2) as complementary. While no existing technology is perfect in our pursuit to titrate resuscitation to cellular demands, we continue our quest to define superior end points of resuscitation and the modalities with which to improve cellular oxygen transport.

Once a standard of intensive care medicine, much controversy has befallen the PAC and its use in modern critical care. While literature on use of PAC is extensive and confusing, it is important for readers to critically evaluate the studies by asking the following questions.

1. Was the patient population appropriate to study design?
2. When was the PAC utilized during the course of illness and was there specific time-dependent goals? Timing of resuscitation is essential for successful outcome and studies stressing early resuscitative efforts (7,9–11) demonstrate better outcomes with DO2 and SvO2 goals than studies with no time specification (12,13). Given that early resuscitation potentially improves outcome, studies that demonstrate lack of benefit from PAC may be flawed if enrollment occurs within 48 hours of respiratory failure since resuscitation should be completed by 24 hours (14). Even among studies that call into question the utility of goal-directed therapy, early intervention itself seems to improve outcome (8).
3. What were the exclusion criteria? Studies excluding patients with acute cardiac or pulmonary problems would be deleting patients who would likely most benefit from PAC use. As an example, one study reporting no PAC advantage excluded patients who already had a PAC, chronic obstructive pulmonary disease (COPD), renal failure, acute myocardial infarct, and liver disease reported (14).
4. Patients with good cardiac function and reserve likely did not benefit from PAC use compared to high-risk patients. Their expected mortality would be lower and potentially “negate” benefits realized for the high-risk patients. Two studies that excluded patients with good cardiac function resulted in conflicting outcomes, most likely due to differences in treatment and timing of goal achievement (13,15).
5. What were the hemodynamic goals of the study: CI, DO2, or SvO2, or any one or combination of these? Our preference has been to use oxygen delivery indexed (DO2I) rather than CI since the acceptable CI would vary with hemoglobin levels (16,17).
imbalance of supply and demand at the tissue level.

•

•

•

•

•

•

•

DO2 with the aid of PAC—the ultimate goal is to satisfy the clinicians, there may still be benefit in continuing to optimize nevertheless, our therapeutic armamentarium is limited, and as clinicians, there may still be benefit in continuing to optimize DO2 with the aid of PAC—the ultimate goal is to satisfy the imbalance of supply and demand at the tissue level.

Differences in study design and treatment algorithm may contribute to different outcomes reported with PAC use. Nevertheless, our therapeutic armamentarium is limited, and as clinicians, there may still be benefit in continuing to optimize DO2 with the aid of PAC—the ultimate goal is to satisfy the imbalance of supply and demand at the tissue level.

**PEARLS**

- Identify patients who may benefit from PAC insertion (Table 21.1).
- Insert the PAC early. The best treatment for MSOF is prevention. The majority of successful outcome studies suggest that timing is of the essence.
- Ensure proper readings. No information is better than wrong information leading to erroneous treatment. Studies have demonstrated an alarming degree of user error (18–20). A corollary is that infrequent use of PAC may lead to more error (both nursing and physician related).
- Have specific goals for the patient (i.e., DO2, SvO2). The goals may vary with disease process and clinical changes (17). For example, the preoperative patients may have a modest DO2 goal (19) compared to shock patients (11,20). Older patients (>75 years of age) may have lower metabolic rates and need less DO2 to meet tissue demands (6,15).
- Use of the PAC requires judgment. The “optimum” PAOP may vary compliance of the heart and degree of ventilator support. Similarly, a DO2 goal is not fixed either.
- Individualize DO2 to achieve normal values of traditional parameters (BP, HR, urine output), lactate levels, and SvO2 levels. Additional monitoring of peripheral tissues is currently available such as gastrointestinal tonometry (21), transcutaneous pressure of O2 (PtcO2) and CO2 (PtcCO2) (22–24), and near-infrared spectroscopy (25). While orthogonal polarization spectral imaging of oral capillaries holds promise (26,27), further validation studies are needed.
- The PAC is a diagnostic tool that provides many parameters, but interpretation of the data is important. For example, central pressure may not give accurate information about the intravascular volume (28). Central and global values such as DO2 and SvO2 may not reflect adequate oxygenation state or cellular viability and function in all the tissue beds.

**INDICATIONS FOR PULMONARY ARTERY CATHETER INSERTION**

Indications for PAC insertion (Table 21.1) have been broadly categorized to (a) precautionary measures in high-risk patients, (b) shock states, (c) pulmonary problems, and (d) cardiac dysfunction.

Preoperative intervention of high-risk surgical patients using PACs remains a controversial area and recommendations are vague in the American College of Cardiology/American Heart Association (ACC/AHA) guidelines (2,29). The key points are as follows: insert the PAC with enough time to achieve the hemodynamic goals (usually the day before), communicate with the anesthesiologist regarding the information obtained while in the intensive care unit (ICU), and monitor the patient in the ICU beyond 24 hours to allow for fluid shifts to occur.

The goals of preoperative invasive monitoring are to (a) optimize preload (plot the Starling curve, see below); (b) optimize CI and stroke volume index (SVI) by adjusting preload, afterload, and contractility (possibly by using inotropes); (c) maintain DO2 to perfuse the rest of the body and prevent MSOF; (d) perfuse the coronaries by maintaining coronary perfusion pressure (CPP) and DO2; (e) decrease myocardial work and myocardial oxygen consumption (MVO2) by keeping systolic blood pressure (SBP) and HR normal; and (f)
prevent myocardial infarct by avoiding significant changes (>15%) in HR and BP (19,30,31). Further details in utilization of PACs for treatment of high-risk patients, shock, and cardiopulmonary failure are covered in other chapters.

The PAC is inserted to obtain information beyond the physical examination. Clinical predictors of hemodynamic status in the critically ill patient, such as chest radiograph, jugular venous distention, and urine output, are inaccurate (1,32). Physicians are correctly able to predict PAOP and CI only 30% to 70% of the time. The PAC provides the following information to guide therapy.

1. Central pressures in relationship to the right ventricle (RV) (i.e., central venous pressure [CVP] or right atrial [RA] pressure); central pressures in relationship to the left ventricle (i.e., PAOP to estimate left ventricular end-diastolic pressure [LVEDP])
2. Cardiac function measured as CO and presented as CI
3. SvO2
4. Intrapulmonary shunt (Qs/Qt)

**General Considerations**

The technical aspects of PAC insertion are presented in “Vascular Cannulation.” It is essential that the catheter be positioned and transduced properly, and a knowledgeable clinician must be able to reliably interpret the data (33–35). Physicians should understand the basic physical principles involved in catheterization, know the design of the catheter, and be able to recognize and remedy technical errors.

Although the modern PAC has features that were not available when it was introduced, the general principles of placement have not changed. If the PAC has fiberoptic bundles at the tip for continuous SvO2 monitoring, external in vitro calibration is done prior to removing the catheter tip from the casing. The PAC is then flushed to remove any lumens and to fill the catheter with a noncompressible column of fluid capable of transmitting pressures. There is a distal port for monitoring the pulmonary artery pressures (PAPs) and a central port approximately 30 cm from the tip that will lie in the right atrium in the average, adult heart. For CO monitoring, a thermistor is located proximal to the tip to measure temperature changes (see Cardiac Output section). The catheter is then advanced. The natural flow of blood from the vena cava to a branch of the pulmonary artery, characteristic waveforms are displayed on the monitor (Fig. 21.1). Once the catheter is advanced to the “wedged” position (PAOP), the balloon is inflated and the catheter adjusted until 1.25 to 1.5 mL of inflation is needed to produce the PAOP tracing. The balloon should only be inflated long enough to record a measurement in order to avoid rupture of the artery or infarction of the downstream segment of lung. The balloon should always be deflated when withdrawing the catheter to avoid vascular and valvular injury. A chest radiograph is performed to assess for pneumothorax and may help to confirm proper position, the catheter should curve gently in the RV and sit in a larger branch of the pulmonary artery (Fig. 21.2). If there is not an obvious PAOP tracing, blood may be sampled from the distal port with the balloon inflated. The sample should have a higher PaO2 and pH with a lower PaCO2 than blood aspirated without occlusion (37). Proper placement is also indicated by the SvO2 signal quality if using a fiberoptic catheter with continuous measurement. The quality of the signal may be altered by (1) fibrin clot at the tip of the catheter, (2) the tip situated against a vessel wall, (3) inserting the catheter too far, or (4) a hypovolemic with collapse of the vessel wall around the catheter tip. If the catheter is in too far, the PAOP tracing will continue to elevate and is called “overwedging” (Fig. 21.3). If this occurs, the balloon should be deflated and the catheter pulled back (≥1 cm) and the balloon inflated again.

**Presssure Measurements**

Normal hemodynamic values are presented in Table 21.2.

Pressure changes in the heart or vessels cause movement of the catheter, which is then converted to an electrical signal by a transducer. Electrical noise is filtered and the signal is amplified and displayed as a tracing on a bedside monitor. Before insertion, the function of the system is checked by shaking the catheter and seeing good waveforms on the monitor. If the waveform is dampened, the system should be flushed to rid the catheter and tubing of all air bubbles, and all connections should be tightened. After inserting the PAC 15 to 20 cm into the introducer sheath, the balloon is inflated and the catheter is gently advanced. The natural flow of blood from the vena cava through the heart and to the lungs guides the catheter to the pulmonary vasculature (36). While passing from the vena cava to a branch of the pulmonary artery, characteristic waveforms are displayed on the monitor (Fig. 21.1). Once the catheter is advanced to the “wedged” position (PAOP), the balloon is deflated and the catheter adjusted until 1.25 to 1.5 mL of inflation is needed to produce the PAOP tracing. The balloon should only be inflated long enough to record a measurement in order to avoid rupture of the artery or infarction of the downstream segment of lung. The balloon should always be deflated when withdrawing the catheter to avoid vascular and valvular injury. A chest radiograph is performed to assess for pneumothorax and may help to confirm proper position, the catheter should curve gently in the RV and sit in a larger branch of the pulmonary artery (Fig. 21.2). If there is not an obvious PAOP tracing, blood may be sampled from the distal port with the balloon inflated. The sample should have a higher PaO2 and pH with a lower PaCO2 than blood aspirated without occlusion (37). Proper placement is also indicated by the SvO2 signal quality if using a fiberoptic catheter with continuous measurement. The quality of the signal may be altered by (1) fibrin clot at the tip of the catheter, (2) the tip situated against a vessel wall, (3) inserting the catheter too far, or (4) a hypovolemic with collapse of the vessel wall around the catheter tip. If the catheter is in too far, the PAOP tracing will continue to elevate and is called “overwedging” (Fig. 21.3). If this occurs, the balloon should be deflated and the catheter pulled back (≥1 cm) and the balloon inflated again.

### Table 21.2 Normal Hemodynamic Values

<table>
<thead>
<tr>
<th>Hemodynamic Parameter</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Systolic blood pressure</td>
<td>100–140 mmHg</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>60–90 mmHg</td>
</tr>
<tr>
<td>Mean arterial pressure (MAP)</td>
<td>70–105 mmHg</td>
</tr>
<tr>
<td>Heart rate</td>
<td>60–100 beats/min</td>
</tr>
<tr>
<td>Right atrial (RA) or central venous pressure (CVP)</td>
<td>0–8 mmHg</td>
</tr>
<tr>
<td>Right ventricle systolic pressure</td>
<td>15–30 mmHg</td>
</tr>
<tr>
<td>Right ventricular diastolic pressure</td>
<td>0–8 mmHg</td>
</tr>
<tr>
<td>Pulmonary artery (PA) systolic pressure</td>
<td>15–30 mmHg</td>
</tr>
<tr>
<td>PA diastolic pressure</td>
<td>4–12 mmHg</td>
</tr>
<tr>
<td>Mean PA</td>
<td>9–16 mmHg</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure (PAOP, wedge)</td>
<td>6–12 mmHg</td>
</tr>
<tr>
<td>Left atrial pressure (LAP)</td>
<td>Varies with patient size</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>2.8–4.2 L/min/m²</td>
</tr>
<tr>
<td>Right ventricular ejection fraction (RVEF)</td>
<td>40–60%</td>
</tr>
<tr>
<td>Right ventricular end-diastolic volume indexed (RVEDVI) to body surface area</td>
<td>60–100 mL/m²</td>
</tr>
<tr>
<td>Hemoglobin</td>
<td>12–16 g/dL</td>
</tr>
<tr>
<td>Arterial oxygen tension (PaO2)</td>
<td>70–100 mmHg</td>
</tr>
<tr>
<td>Arterial oxygen saturation</td>
<td>93–98%</td>
</tr>
<tr>
<td>Mixed venous oxygen tension (PvO2)</td>
<td>36–42 mmHg</td>
</tr>
<tr>
<td>Mixed venous oxygen saturation (SvO2)</td>
<td>70–75%</td>
</tr>
</tbody>
</table>
until a good tracing is seen with 1.25 to 1.5 mL of balloon inflation.

Because there is a tendency for materials to oscillate at their natural frequencies, the pressure signal may be distorted (38). This effect may be reduced by using noncompliant, short (<4 ft) tubing in the setup of the catheter and monitoring system. The loss of transmitted signal is referred to as damping, and catheters may be over- or underdamped. The degree of damping can be determined by a “fast-flush” device while demonstrating an optimal waveform (Fig. 21.4A). When the catheter is rapidly flushed, a square wave is produced, followed by a series of oscillations before the tracing returns to the baseline pressure reading (39). The appearance of the oscillations demonstrates the degree of damping. Underdamping, which occurs more frequently, is identified by several sharp oscillations and produces higher systolic pressure readings (Fig. 21.4B). Overdamping results in a rounded oscillation and results in lower readings, and may be due to clots, air bubbles, or kinking in the catheter (Fig. 21.4C).

Another factor that may interfere with the signal is catheter whip, which results from contraction of the heart. The tracing will show high-frequency distortion and may be minimized by a high-frequency filter built into the transducer system (38).

Whether the patient is on mechanical ventilation (positive intrathoracic pressure at end inspiration) or spontaneously breathing (negative intrathoracic pressure at end inspiration),
all pressure measurements should occur at end expiration when the intrathoracic pressure is closest to atmospheric pressure (Fig. 21.5). This point can be determined by watching the patient’s respiratory movements or displaying the airway pressure tracing on the same monitor where the PAP is displayed. If respiratory variation is so pronounced that there is no flat end expiration, then it is best NOT to record a number (Fig. 21.6). In this situation, patients may need to be sedated, or if getting a PAOP is crucial, even paralyzed. Note that the approximation to atmospheric pressure is clearly affected by patients on extreme PEEP. It is ill advised to guess the PAOP; no information is better than wrong information.

The first characteristic waveform seen when inserting a PAC is the right atrium (RA) tracing (Fig. 21.7). The tracing can be seen while inserting the catheter or by transducing the right atrial pressure once the PAC is in position. There are two main positive pressure deflections, called the “a” and “v” waves. The “a” wave follows the P wave of the electrocardiogram (ECG) and is due to the pressure increase during atrial systole (Figs. 21.7 and 21.8). The “v” wave results from atrial filling against a closed tricuspid valve during ventricular systole. Between these two positive deflections is a small “c” wave due to tricuspid closure. Two negative deflections called the x and y descents occur when pressure in the atrium decreases. The x descent occurs during atrial relaxation. The y descent is

**FIGURE 21.4** Checking transducer reliability by the “fast-flush square wave testing.”

**FIGURE 21.5** Reading of pulmonary artery occlusion pressure (PAOP) at end expiration. During spontaneous breaths, PAOP dips down during peak inspiration due to negative intrathoracic pressure. During mechanical ventilation, PAOP goes up during peak inspiration due to positive pressure ventilation and intrathoracic pressure. In both situations, PAOP should be read at end expiration.

**FIGURE 21.6** Excessive variation in pulmonary artery occlusion pressure with forced inspiratory and expiratory efforts precluding accurate measurement due to absence of a stable end-expiratory point.
seen when the tricuspid valve opens and blood flows from the atrium to the ventricle.

The best estimate of CVP and PAOP is at end diastole when the atrium contracts. For CVP, where the “c” wave emerges from the “a” wave (also called the z-point) is the optimum reading point. If this point is not clear, read the pressure at the middle of the x descent. Certain patterns in the RA tracing may be seen in disease states (Fig. 21.7). For example,

- “a” waves may not be seen in patients with atrial fibrillation.
- Sawtoothed “a” waves will be present during atrial flutter.
- Large “a,” or “cannon” waves occur during atrioventricular (AV) dissociation when the atrium contracts against a closed valve, or during complete heart block.
- A steep y descent is seen in tricuspid regurgitation and the x descent is not apparent.
- Both descents are prominent in RV infarction.
- Cardiac tamponade tends to cause loss of the y descent due to impairment of ventricular filling.
- In pericarditis, sharp a and v waves are followed by steep x and y descents.

- Large v waves are seen in mitral regurgitation, congestive heart failure, and ventricular septal defect due to the increase in atrial pressure.

Recognizing these patterns may suggest a diagnosis before a confirmatory echocardiogram is obtained.

The pressures observed in the right atrium range from 0 to 8 mmHg. Higher pressures may not necessarily mean fluid overload, but may reflect the volume of the right heart and the ability of the ventricle to eject that volume. There is little relationship between CVP and PAOP or left heart pressures in patients with valvular or coronary artery disease or when PAPs are elevated (40,41). Monitoring only the CVP can be misleading in patients with right heart failure, severe pulmonary disease, and most critically ill patients.

The next waveform seen is that of the RV (Fig. 21.1). The pressures here are higher with a wider difference between systolic and diastolic. If no RV waveform is seen after inserting the catheter 30 cm from the internal jugular or subclavian vein entry site, the catheter may be curling in the atrium or passing into the inferior vena cava. The catheter should be
expeditiously advanced through the ventricle both to avoid dysrythmias and to keep the catheter from warming and softening. The RV systolic pressures generally range from 15 to 30 mmHg and diastolic pressures from 4 to 12 mmHg. In right heart failure, the RV diastolic pressures may be high enough that the waveform mimics the PA. Low RV pressures will be seen in hypovolemic shock and they will also be close to PA pressures. One concern at this point of insertion is causing a right bundle branch block (RBBB), or even complete heart block (LBBB) (42). However, the incidence of complete heart block appears to be no greater in patients with LBBB than without (43).

Once the catheter enters the pulmonary artery, the waveform shows an increase in diastolic pressure while the systolic pressure remains similar to the ventricle, sometimes referred to as the “step up” (Fig. 21.1). This transition may be difficult to discern when there is hypovolemia, tamponade, RV failure, or catheter whip. If there is no change in waveform after inserting the catheter 50 cm, it may be coiling in the ventricle and is at risk of knotting. A chest radiograph will discern the problem and fluoroscopy may be used to guide placement.

Normal PA pressures range from 15 to 25 mmHg systolic over 8 to 15 mmHg diastolic. The beginning of diastole is marked by a dicrotic notch on the PA tracing, corresponding to the closure of the pulmonic valve (44). This incisura distinguishes the PA from the RV when RV diastolic pressures are elevated. As blood flows through the lungs to the left atrium, the PA pressure drops until it reaches a nadir at the end of diastole. Since the pulmonary circulation has low resistance, the diastolic pressure is able to decrease until it is just higher than PAOP. The highest PA systolic pressure occurs during the T wave of the corresponding ECG. The pulmonary circulation is very dynamic and is affected by acidosis, hypoxia, sepsis, and vasoactive drugs (38). An increase in CO may also paradoxically lower the PA pressures by a reflexive decrease in pulmonary vascular resistance (PVR) with fluid resuscitation and decreased sympathetic nervous system discharge (45).

The transition to the wedge position is noted by a drop in mean pressure from the PA. The PAOP usually ranges from 6 to 12 mmHg in normal states. PAOP most closely reflects LVEDP after atrial contraction and before ventricular contraction (Fig. 21.8). There are often no clear “a,” “c,” or “v” waves. The point 0.05 seconds after onset of QRS of the ECG is where the pressure best estimates LVEDP (46). When “v” waves are prominent such as in mitral insufficiency, the bottom of the “v” wave or the “a” wave may be used to measure the PAOP (Fig. 21.9). A prominent “v” wave may fool the novice into thinking that the catheter is not wedging. It is important to note the change in waveform from PA to v-wave tracing (although the two waves may look remarkably similar). One way of differentiation is that the “v” wave occurs later in the ECG cycle after the T wave while the PA wave occurs right after QRS (Fig. 21.9). There may be large “a” waves secondary to a decrease in left ventricle compliance (47); the point 0.05 seconds after initiation of QRS again best reflects LVEDP. Even though the measurements are correlated with the ECG and are done during end expiration, the PAOP may be exaggerated by respiratory muscle activity, especially during active or labored exhalation. Once the patient is adequately sedated, a short-acting paralyzing agent may be necessary to eliminate this effect (48) (Fig. 21.6).

**Principles of Measuring Pulmonary Artery Occlusion Pressure**

When the balloon is inflated, the blood flow in that segment of the pulmonary artery is occluded and the PAOP is measured. The pressure between the occluded pulmonary artery segment and the left atrium will equalize upon flow cessation (Fig. 21.10) which is analogous to closing off a pipe with pressures equalizing between the two ends (49). With the closed pipe analogy, there is a host of assumptions: PAOP ≅ PcP ≅ LAP ≅ LVEDP ≅ LVEDV, where PcP is pulmonary capillary pressure, LAP is left atrial pressure, and LVEDV is left ventricular end-diastolic volume. As long as there is no obstruction in this conduit, the relationship between PAOP and LVEDP may hold. The final assumption is equating pressure to volume by estimating LVEDV or “preload” with LVEDP. We will now assess the pitfalls with each of these assumptions.

1. **PAOP ≅ PcP ≅ LAP.** In the “closed pipe” analogy, the column of blood between the catheter tip and the left atrium should be patent and not narrowed by alveolar pressures. This occurs in the dependent areas of the lung, where the pressures from blood flow in the right atrium and pulmonary artery are greater than the alveolar pressure, or zone 3 in the West classification (50). In

![FIGURE 21.9 Regurgitant mitral valve generating a v wave seen during wedging. PA, pulmonary artery.]
other areas of the lung (zone 1 or 2), the pulmonary arteries may collapse from higher alveolar pressures and the wedge pressure may partially reflect alveolar pressure (Fig. 21.11). Because the PAC is directed by blood flow, it is more likely to pass into zone 3, where pulmonary arterial and venous pressures exceed alveolar pressures. This is especially true when the patient is supine, since there is greater volume of lung located in a dependent position (51). When pulmonary artery diastolic (PAD) pressure is lower than the PAOP, this implies incorrect positioning of the PAC (i.e., blood cannot flow in reverse direction), and may be due to transmission of alveolar pressures on the PAOP in non-zone 3 catheter position. Other factors that may cause errors in estimation of PAOP to LAP are pulmonary venous obstruction and respiratory variation as well as high ventilator support (PAOP reads higher than LAP).

The PAOP usually closely approximates the pulmonary capillary hydrostatic pressure (Pc). When there is an increase in PVR, the wedge pressure underestimates Pc. A difference of 2 to 3 mmHg between the PAOP and PAD pressure is a clue that there may be a discrepancy between PAOP and Pc (34). Hydrostatic pulmonary edema may therefore occur at lower wedge pressures. A method of calculating the Pc has been described by recording the rapid drop in pressure decline when the catheter balloon is inflated in the wedge position (52). The point where the rapid decline transitions to a more gradual slope before reaching the PAOP is the Pc (Fig. 21.12).

Increased intrathoracic pressure secondary to respiratory failure and the addition of PEEP in ventilated patients affects pulmonary vascular pressures. Up to about 15 cm H₂O, PAOP closely correlates with LAP (53). During higher PEEP states, the PAOP may not
reflect the true filling pressure of the heart (i.e., pressure outside minus pressure inside the heart). Although the heart is seeing the high PEEP support at all times, on-PEEP PAOP is not giving the information that we need from the PAOP, which is the cardiac filling pressure. In general, 5 cm H₂O of PEEP is said to raise the measured PAOP by 1 mmHg, but a greater effect is seen in hypovolemic patients or when the catheter is not in West zone 3 (54). High PEEP may also turn zone 3 status to zone 2 or 1 by compressing the pulmonary artery and/or pulmonary vein. Another formula predicts that 50% of PEEP is transmitted to the pleural space (51). However, in noncompliant lungs, such as in the acute respiratory distress syndrome, the alveolar pressure is not effectively transmitted to the vasculature. Also, pulmonary disease is not homogeneous. In complicated cases, it is best to avoid formulas or assumptions. In order to more accurately correct for the effect of pressure transmitted during high PEEP ventilation, the intrapleural pressure may be measured directly with a catheter in the pleural space or distal esophagus and then subtracted from the PAOP. However, this is cumbersome and not often done.

Another method is to measure an “off-PEEP” wedge pressure by temporarily disconnecting the patient from the ventilator circuit and recording the nadir of the tracing (55). This nadir pressure better reflects LAP than the on-PEEP PAOP. The discontinuation should be brief (<1 second) so that a decrease in PaO₂ from derecruitment of alveoli does not occur (56). The brief off-PEEP state will not change physiologic conditions such as venous return and cardiac function. The procedure should be coordinated and done by trained personnel only when the PAOP is needed to make a clinical decision. The balloon is inflated first to ensure good position, then deflated. The FiO₂ may be increased temporarily for patient safety, the balloon reinfated, and at end expiration, the patient is disconnected from the ventilator for 1 second and then reconnected while the PAOP tracing is being recorded. The drop in PAOP upon ventilator disconnection is the off-PEEP PAOP (55). When done properly, it is extremely rare to cause hypoxia.

2. LAP ≅ LVEDP. LAP (and thus PAOP) will overestimate LVEDP if there is an obstruction between the left atrium and the left ventricle such as a myxoma or mitral stenosis. Mitral valve regurgitation also causes the PAOP to read higher than the true LVEDP because of the additional pressure of the retrograde flow of blood across the valve resulting in a large v wave (see Pressure Measurements Section). LAP (and thus PAOP) will underestimate LVEDP when severe aortic regurgitation causes premature closure of the mitral valve when the left ventricle is still filling. LAP (and PAOP) is higher when there is a left atrial kick in a failing heart and decreased ventricular compliance such as in ischemic states, left ventricular hypertrophy, and restrictive cardiomyopathies (57). This is especially true when LVEDP is greater than 25 mmHg.

3. LVEDP = LVEDV. The pressure-volume relationship depends on the compliance of the ventricle and the transmural ventricular distending force. The compliance of the ventricles will change with ischemia, infarct, and hypertrophy. A stiff heart (myocardial hypertrophy) will need higher pressures to obtain the same amount of volume as a normal heart (Fig. 21.13). The transmural ventricular distending force (intracavitary pressure minus juxtacardiac pressure) will depend on the pressure inside and outside the heart. External forces elevating juxtacardiac pressures may be high ventilator support or pericardial tamponade, which may cause elevation of PAOP but may not reflect ventricular filling pressure.

Clinical Use of the Pulmonary Artery Occlusion Pressure

As long as the previously mentioned assumptions regarding the relationship between PAOP, LAP, and LVEDP have been evaluated, the PAOP may be used as an estimate of LAP with reasonable correlation (49,53). The optimum wedge pressure depends on the patient, but has been defined as the pressure where there is minimal increase in stroke volume or left ventricular stroke work. Although the normal PAOP values may be 10 to 14 mmHg (58,59), some patients require a high pressure to reach the optimum stroke volume (Fig. 21.14). Such optimization can be graphically illustrated using a Starling curve plotting SVI to PAOP (as an estimate of LVEDP). If vasoactive agents are started, the heart may now be on a different curve requiring new assessment of the optimum PAOP. The

![FIGURE 21.12 Estimation of pulmonary capillary pressure. PA, pulmonary artery pressure; Pc, pulmonary capillary pressure; PAOP, pulmonary artery occlusion pressure.](image)

![FIGURE 21.13 The same pulmonary artery occlusion pressure of 20 mmHg reflecting three different clinical conditions. A: Distended hypervolemic ventricle in a normal heart. B: Normal volume in a noncompliant heart (ventricular hypertrophy). C: Low volume in a normal ventricle with high juxtacardiac pressures such as high positive end-expiratory pressure.](image)
optimum PAOP varies not only from patient to patient, but also temporally within the same patient as the clinical condition changes (such as vasoactive agents, myocardial compliance, and external forces around the heart). No set of values is broadly appropriate for all patients; in practice, each patient must be assessed repeatedly, further stressing the importance of judgment and expertise of the end user.

Elevated wedge pressures may help differentiate hydrostatic pulmonary edema from that caused by increased permeability. A PAOP of 24 mmHg or higher is associated with a tendency for hydrostatic edema (60). Lower wedge pressures may imply increase capillary permeability and traditionally, a PAOP of <18 mmHg has implied a pulmonary (or noncardiogenic) cause of lung edema. When there is an increase in PVR, the wedge pressure underestimates Pc and hydrostatic pulmonary edema may therefore occur at lower wedge pressures (see Fig. 21.12).

Volumetric PACs are designed with the ability to measure right ventricular ejection fraction (RVEF), from which the right ventricular end-diastolic volume indexed (RVEDVI) to body surface area (BSA) is calculated. Traditionally, the right heart function was deemed unimportant and thought to merely act as a conduit to funnel blood to the left ventricle. However, right heart dysfunction with septal deviation may impact LV compliance and contractility, and the function of RV is important when PAPs are elevated. The volumetric PACs have two additional electrodes that provide continuous measurement of the ECG and a thermistor with a rapid response. From beat-to-beat measure, the ejection fraction (EF) is calculated. EF (%) = SVI/EDI × 100, where SVI is stroke volume indexed and EDVI is end-diastolic volume indexed to BSA. CI/HR = EDVI – ESVI, where ESVI is end-systolic volume indexed to BSA (61). RVEDVI has been shown to be a more accurate measure of cardiac preload than pressure measurements in certain patient populations (62,63). The measurement of RVEF has been validated by comparisons with transesophageal echocardiography (64). The RVEDVI in healthy individuals falls between 60 and 100 mL/m². The information obtained from the volumetric catheter has been used to predict response to fluid challenge when the values are relatively low (<90 mL/m²) (62). Much like other measurements of fluid responsiveness, the validity of the RVEF measurement is compromised by tachycardia (pulse >120 beats/min) and atrial fibrillation (irregular HR) (65).

# Cardiac Output

One’s ability to meet increasing tissue oxygen demand by improving cardiac function is perhaps the single most important determinant in DO₂ and tissue perfusion. The evolution of CO measurement is fascinating in its simplicity and started with Adolf Fick, who in the 1870s proposed that uptake or release of a substance by an organ is the product of blood flow through that organ and the difference between arterial and venous values of that substance. The original “dye” was oxygen and the organ studied was the lung. Fick’s equation stated: CO = VO₂/(CaO₂ – CvO₂), where VO₂ is oxygen consumption, CaO₂ is arterial content of O₂, and CvO₂ is mixed venous content of O₂. This principle is widely accepted as an accurate though invasive assessment of CO since a PAC must be placed to obtain accurate mixed venous oxygen content. Additionally, its practical use is limited by the cumbersome measurement of VO₂. Stewart (1897) and Hamilton (1932) utilized Fick’s principles but used a known amount of dye injected into central circulation followed by serial peripheral arterial measurements of dye concentration (i.e., change in dye concentration over time), and calculated the flow. The area under the curve after plotting time (x axis) versus dye concentration (y axis) reflected the CO using the following equation: Cardiac output = Amount of dye injected/integral (dye concentration × function of time). The next revolutionary step in CO measurement was using temperature as the dye. Crystalloid solution (usually 10 mL, but 5 mL may be used in volume-restricted patients) is injected into the RA port at similar parts of the respiratory cycle (end expiration), within 4 seconds in a smooth manner (66). The thermistor near the tip of the PAC detects the change in temperature, and the change in blood temperature over time is proportional to the blood flow from the ventricle. Several measurements (three to five) should be taken and the average of the values (within 10% of each other) used. Principles of the modified Stewart–Hamilton equation calculate the CO (Fig. 21.15).

![Frank-Starling curves](image-url)
Although initial studies used iced solution as injectate, ambient temperature injectate is now the standard counterpart (67) and has less likelihood of reflexive bradycardia (68). It is important to note that iced injectate (0° to 5°C) is associated with higher reproducibility and the highest signal-to-noise ratio (69) and may be necessary in hypothermic patients. Falsely low CO will occur if an error in the system increases the change in temperature (which is in the denominator of the Stewart–Hamilton equation): the temperature probe reading the injectate is cooler than the actual injectate (or the solution is warmer than the temperature reading of the injectate), more than allotted “dye” amount is injected (>10 mL fluid), there is too rapid an injection, or the injection occurs during positive pressure ventilation. Falsely high CO may occur if the temperature probe measuring injectate reads warmer than the actual injectate (if the solution is cooler than the temperature reading of the injectate), less than the allotted amount of “dye” (<10 mL) is used, or the catheter has migrated distally with less change in temperature difference. Most institutions use temperature probes at the site of injection (RA port) so that variations in injectate temperature should not contribute to errors in CO measurements.

Another development in the evolution of measuring CO is the PAC with continuous cardiac output (CCO) monitoring (70,71). A heat element is embedded in the PAC to deliver small pulsations of heat, which is detected by a rapid-response thermistor placed distally to the heat source. The change in temperature detected is then used to calculate CO. Although intermittent bolus technology is still gold standard for measuring CO at the bedside, CCO values are reproducible and close to manual CO measurements, although discrepancies are observed at extremely low-flow states (70). Unlike the manual injection of crystalloid, the measurements are obtained at random parts of the respiratory cycle and are less subject to human error, which may account for some of the differences in the two techniques. It is important to note that the CCO value may not instantaneously reflect CO changes (e.g., while titrating inotropes), but the effect of treatment can be seen in seconds if using a continuous SvO2 monitor. Due to the heat-generating wire coil in the distal end, these catheters must be removed before magnetic resonance imaging.

### Starling Curves

Drs. Frank and Starling described the relationship between myocardial stretch and contractility. Myocardial stretch is an independent determinant of stroke work and the actin–myosin interaction has a linear correlation with the strength of systolic contraction up to a certain point. Given the heart’s dynamic environment, a family of curves is more representative of the true preload-to-stroke volume relationship. Increasing afterload or decreasing contractility shifts the curve down and to the right (i.e., more stretch is necessary to produce a similar difference in stroke volume). One cannot stress enough the importance of reassessment after each therapy. For example, initiating afterload reduction may put the heart on a different Starling curve (to the left and up; see Fig. 21.14), but may decrease the preload. Unless more fluid is given to optimize the LVEDP (i.e., PAOP), the best stroke volume may not be achieved.

## Mixed Venous Oxygen Saturation (See Chapter 24)

Specialized PACs with the ability to measure SvO2 continuously using principles of reflection spectrophotometry are available (Fig. 21.16). Oxygen saturation is the ratio of hemoglobin bound to O2 divided by total hemoglobin, and when measured at the tip of the PAC, reflects mixing of deoxygenated blood from superior and inferior vena cavae and coronary vessels. The SvO2 value indicates the balance between DO2 to the tissues and the amount consumed by the tissues before returning to the heart.

### Rearranging the Fick’s Equation

\[
\text{SvO}_2(\%) = \frac{\text{SaO}_2 - \text{VO}_2}{\text{CO} \times \text{Hgb}} \times 1.36 \times 10
\]

Four factors determine the SvO2 value: three parameters contributing to DO2 (CO, hemoglobin, and SaO2), and one parameter for O2 consumption. Low SvO2 suggests insufficient O2 delivery or increased O2 consumption. SvO2 is also a harbinger of shock and may decrease before overt shock is apparent (15,16,23,72). SvO2 has also regained popularity as an end goal of resuscitation with decreased mortality (7,73–75). Inadequate DO2 can be the result of decreased CO, low hemoglobin, or low oxygen saturation. Increased consumption may occur due to activity, fever, hyperthyroid state, or repayment of oxygen debt. High SvO2 suggests low cellular consumption such as in late sepsis, arteriovenous shunts (cirrhosis), or excessive inotrope use. Hypothermia, sedation, paralysis, anesthesia, hypothyroidism, and cyanide poisoning can also reduce VO2. The catheter should also be checked to ensure that distal migration has not occurred leading to sampling of pulmonary capillary blood that is normally highly saturated (~100%). Inflating the balloon (wedging) should determine that the catheter is in too far if the PAOP tracing is seen with less than 1.25 mL of air.
Intrapulmonary shunt ($Q_s/Q_t$) is the percentage of cardiac output (CO) not involved with gas exchange (goes through collapsed alveoli), with ventilation/perfusion $V/Q = 0$. LA, left atrium; RV, right ventricle.

**FIGURE 21.17**

Intrapulmonary shunt ($Q_s/Q_t$) is calculated as $(CcO_2 - CaO_2)/(CcO_2 - CvO_2)$, where $CcO_2$ is the pulmonary capillary content of $O_2$, $CaO_2$ is the arterial content of $O_2$, and $CvO_2$ is the mixed venous content of $O_2$ (Table 21.3). Since pulmonary capillary blood cannot be sampled, the saturation is assumed to be 100%, which usually holds true when the FiO2 is 1.0. It is important to understand the contribution of a low SvO2 to PaO2; if there is a moderate shunt (>20%). Any decrease in SvO2 in a patient with more than 20% shunt will allow more deoxygenated blood to go into the arterial circulation, resulting in a lower PaO2. This is called nonpulmonary cause of hypoxia. For example, if a patient with a 20% intrapulmonary shunt and a hemoglobin of 15 g/dL has an acute cardiac event, and the CO decreases from 5 to 3 L/min, the PaO2 will decrease from approximately 80 to 65 mmHg (Fig. 21.19). If the same patient's hemoglobin is 10 g/dL, the PaO2 will decrease from 70 to 55 mmHg. This demonstrates the importance of low SvO2 contributing to lower PaO2 (recall that hemoglobin affects the delivery portion of the SvO2 equation). If the CO was not optimized prior to increasing PEEP, a potential further decrease in CO, SvO2, and PaO2 may have ensued. Another example: If a patient is agitated and the arterial saturation decreases, this may be due to increased VO2 and low SvO2 in a patient with a moderate intrapulmonary
shunt, and not from an acute pulmonary event. Treatment is to decrease agitation and VO₂. There are times when severe cardiorespiratory compromise warrants titration of both CO and PEEP simultaneously in patients with life-threatening cardiorespiratory compromise. Preload, afterload, and contractility. Preload is the theoretical stretch of ventricles at end diastole. According to Frank and Starling, the stretch of myocardium augments contractility to a certain point, and then CO is negatively affected by further increases (76). Afterload reflects the interplay between aortic compliance, peripheral vascular resistance, viscosity of blood, aortic impedance, and aortic wall resistance. Afterload is therefore the force that myocytes must overcome during each contraction and clearly more complex than an arbitrary division of the heart into right and left components. Contractility is the maximum velocity of myocardial fiber contraction; it is the myocytes’ inherent ability, independent of preload. All these parameters are extremely dynamic and require frequent reassessment.

**Derived Variables**

See Table 21.3 for the equations and normal values. Once flow and pressure variables have been obtained from the PAC, further hemodynamic calculations may be performed to ascertain a more complete clinical picture. Clinicians must understand the significance and pitfalls of these calculated values (most of which are automatically calculated by monitoring devices).

**Stroke volume index (SVI):** It is the quantity of blood ejected from the ventricle with each contraction (i.e., the difference between end-diastolic and end-systolic volumes). SVI accounts for the effect of the HR’s contribution to CI, and is an important variable because one does not want to augment CI by causing tachycardia (Fig. 21.14). SVI varies with preload, afterload, and contractility. Preload is the theoretical stretch of ventricles at end diastole. According to Frank and Starling, the stretch of myocardium augments contractility to a certain point, and then CO is negatively affected by further increases (76). Afterload reflects the interplay between aortic compliance, peripheral vascular resistance, viscosity of blood, aortic impedance, and aortic wall resistance. Afterload is therefore the force that myocytes must overcome during each contraction and clearly more complex than an arbitrary division of the heart into right and left components. Contractility is the maximum velocity of myocardial fiber contraction; it is the myocytes’ inherent ability, independent of preload. All these parameters are extremely dynamic and require frequent reassessment.

**Left ventricular stroke work index (LVSWI):** This estimates the work of the left ventricle in one beat. Work is the product of force and distance. Physiologically, this translates to the product of change in pressure and change in volume. Unfortunately,
It the RVSWI must compensate accordingly. Pulmonary hypertension and consequent right heart failure, a relatively low pulmonary pressure system. In patients with the work generated by RV is markedly less than LV due to circulation and estimates external work for the RV in one beat. Right ventricular stroke work index (RVSWI): It is the right heart’s ability to produce forward flow against the pulmonary circulation and estimates external work for the RV in one beat. The work generated by RV is markedly less than LV due to a relatively low pulmonary pressure system. In patients with pulmonary hypertension and consequent right heart failure, the RVSWI must compensate accordingly.

Pulmonary vascular resistance index (PVRI): It is the resistance for the RV. Resistance to blood flow is analogous to electrical circuit resistance defined by Ohm’s Law. Resistance = Pressure/Flow. Physiologically, the pressure change between two vascular beds drives the flow (i.e., CI). PVRI reflects resistance in the pulmonary vasculature. Pulmonary hypertension exists when systolic PAP is greater than 35 mmHg or mean PAP is greater than 25 mmHg (79,80). In critically ill patients, the most common causes for elevated PAP are acute respiratory distress syndrome, acute LV dysfunction, and pulmonary embolism (79,81–85). Patients with comorbid conditions such as chronic pulmonary hypertension may suffer from interstitial lung disease, COPD, or liver or cardiac disease. The RV is exquisitely sensitive to increases in afterload and lacks the ability to overcome pulmonary hypertension with PAP greater than 40 mmHg (84). Subsequent decrease in CO is due to the combination of decreased RVSWI and decreased filling of the left ventricle as a result of interventricular septal deviation (86). Since CO is indexed to BSA, PVR should also be indexed and presented as PVRI.

Systemic vascular resistance index (SVRI): It is the resistance for the left ventricle. In the context of hyperdynamic states with high CI and decreased SVRI, the patient may be in distributive shock. Patients with low CI and high SVRI are in hypovolemic or cardiogenic shock. It is important to recognize that SVRI represents the interaction of vascular diameter and viscosity, neither variable is easily and reliably measured. SVRI is calculated; therefore, error is introduced if any of its subcomponents carries inaccuracy. Since CO is indexed to BSA, SVRI should also be indexed and presented as SVRI.

Arterial oxygen content of blood (CaO\(_2\)): It is the amount of oxygen carried in arterial blood. When evaluating delivery of oxygen, the CaO\(_2\) is of critical importance. Each gram of hemoglobin carries 1.36 to 1.39 mL of oxygen if it is 100% saturated. Oxygen is poorly soluble in plasma and the dissolved oxygen contribution to arterial oxygen content is negligible. Therefore, saturation (SaO\(_2\)) plays a more important role than pressure of oxygen (PaO\(_2\)).

Mixed venous oxygen content of blood (CvO\(_2\)): It is the amount of oxygen carried in the mixed venous blood. Low mixed venous oxygen content has similar clinical implications as low SvO\(_2\) and suggests decreased DO\(_2\) or increased VO\(_2\). Since the blood is sent for oximeter analysis for saturation value and not PvO\(_2\), the PvO\(_2\) value in the equation (Table 21.3) is usually substituted with the normal PvO\(_2\) value of 40 mmHg since the amount dissolved is so small that a PvO\(_2\) substitution of 0 to 70 mmHg will not make a difference in the calculation of CvO\(_2\).

Delivery of oxygen indexed to BSA (DO\(_2I\)): It is the amount of oxygen delivered to the tissues by hemoglobin, arterial saturation, and flow (CI) (i.e., the product of CI and arterial oxygen content of blood). The survival benefit of titrating to a specific DO\(_2I\) value has been extensively studied as an end point of resuscitation with conflicting results. The controversy surrounding DO\(_2\) augmentation is discussed in the beginning of this chapter (11,13,19,20,23,30,87–89).

Oxygen consumption indexed (VO\(_2I\)): There are two methods of assessing VO\(_2\): Fick’s principle and indirect calorimetry (90). Fick’s principle states that the rate of diffusion of a known indicator (oxygen) is proportional to the product of concentration gradient and flow. Physiologically, this translates to the difference between arterial and mixed venous oxygen content multiplied by the CO. Consumption can also be assessed by indirect calorimetry and is typically 3.5 mL of oxygen/kg (91). Indirect calorimetry compares the difference between inspired and expired oxygen to carbon dioxide ratios. There is usually a discrepancy (either way) of up to 11% between Fick’s principle and indirect calorimetry, partially explained by Fick’s method not accounting for pulmonary VO\(_2\) (92–95). Shoemaker first noted that a higher VO\(_2I\) of greater than 160 mL/min/m\(^2\) was associated with survival (3,4). It may reflect cells’ ability to increase metabolic rate and utilize oxygen during stressed states. The concept of “critical DO\(_2I\)” where VO\(_2\) becomes delivery dependent has been described in certain disease states with values occurring at DO\(_2\) of less than 450 mL oxygen/min/m\(^2\) (96–98). Although the concept is attractive, it is difficult to
Coronary perfusion pressure (CPP): In compliant vessels, flow to the coronary arteries can be augmented via coronary artery dilation. However, in patients with coronary artery disease with fixed vessels, flow depends on the pressure gradient between the two ends. Due to high LV pressures, coronaries feeding the LV fill during diastole, and maintenance of diastolic pressure is important. In patients undergoing preoperative optimization, nitroglycerin can be used to preferentially dilate healthy coronary vessels, thereby augmenting DO₂ to the myocytes. Generally CPP greater than 50 mmHg is desired, but there is individual variation.

SPECIAL COMMENT ON OBESITY AND DERIVED PARAMETERS

The validity of derived parameters indexed to BSA has been questioned in morbidly obese patients. Several studies have demonstrated that derived parameters indexed to BSA are appropriate and closely approximate indexing to body mass index. The large BSA in the obese patient may not affect these measurements (99–101).

COMPLICATIONS OF PULMONARY ARTERY INSERTION

PAC insertion is an invasive procedure and carries inherent risks (102–104). Complications related to central venous access are discussed in other chapters. The overall complication rate associated with PACs can be as high as 25%. The procedural risks are pneumothorax, hemothorax, and knotting of catheters. Multiple prospective and retrospective studies have reported the most common complications including infection, thrombosis, arrhythmias, new bundle branch blocks, and pulmonary artery rupture (105,106). Serious complications (PA rupture and cardiac perforation with tamponade) are infrequent, but they can be fatal if unrecognized. Although reports of PAC-related infection are up to 22%, consequential bacteremia is relatively rare (0.7%–2.2%) (107). Catheters inserted for greater than 3 days may be associated with more infectious and thrombotic complications (108,109). Arrhythmias were relatively common, occurring in up to 75% of insertions. However, clinically significant arrhythmias requiring treatment were rare; 3% developed new bundle branch blocks, but this complication was not associated with increased mortality (105,106). Pulmonary artery rupture is exceedingly rare with a reported incidence of 0.031% (110) but usually occurs in patients with pulmonary hypertension and can be fatal due to high pulmonary pressures. To prevent rupture, it is important to: 1) slowly inflate the balloon, 2) stop inflating when resistance is encountered, and 3) deflate when the waveform tracing demonstrates overdwelling. Placement of PACs requires skilled operators who are trained to troubleshoot and recognize complications when they occur. As discussed earlier in this chapter, perhaps the most dangerous complication of PACs is the misinterpretation of information.

ARTERIAL LINES

Indications for invasive pressure monitoring are (a) hyperdynamic states including all forms of shock and (b) frequent blood sampling for blood gas analysis and laboratories. Other indications include monitoring of response to vasoactive agents and severe peripheral vascular disease precluding noninvasive BP monitoring. There are no true absolute contraindications.

Arterial cannulation is relatively safe with nonocclusive thrombosis and hematoma being the most common complications (111). Selection of anatomic site is an important consideration; percutaneous arterial catheters can be introduced in the radial, brachial, axillary, femoral, and dorsalis pedis arteries. Placement in brachial arteries is ill advised; it is an end artery and patients may develop ipsilateral hand ischemia in up to 40% of insertions (112,113). The radial artery remains the most popular placement site due to its ease of access and relatively low complication rates. A preprocedure Allen test assesses the patency of collateral arteries, but this test has poor correlation with distal flow and likelihood of hand ischemia (114,115).

Pressure Measurement

Continuous measures of SBP, diastolic blood pressure (DBP), and mean arterial pressure (MAP) are displayed with invasive arterial catheters. Four elements must be considered in direct pressure measurement: (a) energy content, (b) transformation of pressure pulse, (c) reflection of pressure wave, and (d) recording system. Each element can introduce error in invasive BP monitoring rendering the often large discrepancy between cuff and invasive pressures. The SBP is determined by the ventricular ejection velocity and volume and this pulse wave meets increasing impedance as the caliber of vessels decrease. Additionally, pulse amplification is proportional to distance from aorta; consequently, the radial artery pressure tends to be higher than aortic pressure.

Volume and velocity of left ventricular ejection, peripheral resistance, distensibility of arterial wall, and viscosity of blood determine the peak SBP. Usage of long tubing (≥4 feet) or microbubbles in the closed system can result in inaccurate measurements, specifically underdamping and falsely high SBP. Underdamping produces characteristic waveforms with sharp and overshooting upstroke and small, artifact pressure waves along the waveform. Overdamped tracings are caused by kinking, macrobubbles, or mechanical obstruction of tubing. Overdamped waveforms are characteristically diminished in their upstroke and exhibit loss of the dicrotic notch. The dynamic response of arterial monitoring circuits is assessed by a fast-flush square wave test (Fig. 21.4); a properly calibrated system produces one overdamped waveform followed by several oscillating overshoot waves (116).

Waveform analysis (Fig. 21.20) demonstrates the typical points associated with (a) systolic upstroke, (b) systolic peak, (c) systolic decline, (d) dicrotic notch, (e) diastolic runoff, and (f) end diastole. Examination of the arterial waveform provides useful information regarding a patient’s clinical status. Left ventricular ejection produces the first, sharp upstroke at the beginning of aortic valve opening (Fig. 21.20, points 1 and 2). As the ventricular flow is dispersed peripherally, the waveform declines (point 3); this is also when the heart is in
isovolumetric relaxation and diastolic filling. Just prior to closure of the aortic valve and as a result of isovolumetric relaxation, there is a slight drop in pressure known as the incisura (at the aorta) or dicrotic notch (at the periphery) (point 4). Further decrease in the pressure waveform reflects the runoff to distal arterioles (points 5 and 6). More peripheral arteries exhibit narrower waveforms and higher systolic pressures and wider pulse pressures, although the MAP remains similar to central vessels. The etiology of varying pulse contours in the periphery is related to the elasticity, amplification, and distortion of smaller arteries (117). Various cardiac conditions produce characteristic arterial waveforms. In aortic stenosis, narrow waveform and loss of the dicrotic notch secondary to diseased valve are seen. Aortic regurgitation may exhibit widened pulse pressures and a sharp upstroke, sometimes accompanied by two peaks.

**Systolic pressure variation (SPV):** Variations in SBP and ventricular stroke volume are of greater magnitude in hypovolemic states (118). Theories on the etiology of this phenomenon relate to the characteristics unique to hypovolemia and include the following: (a) the superior vena cava is more easily collapsible, (b) there is an increased effect of transmural pressures in the right atrium, and (c) the preload and stroke volume relationship is on the steep portion of the Frank–Starling curve. Usually, a decrease in left ventricular stroke volume occurs with inspiration due to the positive pressure ventilation. Originally, Perel described SPV as two components (Fig. 21.21)—delta up ($\Delta_{\text{up}}$) and delta down ($\Delta_{\text{down}}$)—while emphasizing the strong correlation between $\Delta_{\text{down}}$ and hypovolemic states (119–121). $\Delta_{\text{up}}$ is the difference between maximum SBP and a reference SBP (usually at expiratory pause during mechanical ventilation). $\Delta_{\text{down}}$ is similarly the difference between minimum SBP and reference SBP and represents a decrease in stroke volume during expiration. SPV greater than 10 mmHg indicates hypovolemia and suggests responsiveness to fluid challenge (122). SPV also has significant correlation with the left ventricular end-diastolic area by echocardiogram (123) and PAOP (124). Note that SPV, like stroke volume variation (SVV), may be sensitive to changes in volume status, but may not necessarily equate to actual intravascular blood volume.

**Stroke volume variation (SVV):** Arterial pressure variation during the respiratory cycle is a well-documented phenomenon (118). There exists normal fluctuations of arterial pressure during spontaneous respirations; however, pulsus paradoxus is observed when the ebb and flow of arterial pressures exceeds 10 mmHg. Reverse pulsus paradoxicus occurs in ventilated patients. Proprietary algorithms in new monitor devices analyze the pulse-to-pulse variation in a semicontinuous fashion with updates at 20-second intervals. SVV is not a measurement of absolute preload; rather, it is an assessment of response to fluid resuscitation (124–127). SVV greater than 9.5% to 15% is associated with fluid responsiveness. SVV is only approved for use in sedated, mechanically ventilated patients who are in sinus rhythm (rhythm must be regular or the variation may be due to irregular rate rather than volume status).

$$\text{SVV} = \frac{(\text{SV maximum} - \text{SV minimum})}{(\text{SV maximum} + \text{SV minimum})/2} \times 100$$

**Available technology on the market:** Several companies market continuous arterial catheter CO monitoring with several important distinctions. The main difference between the aforementioned technologies is that Lidco analyzes areas under a concentration curve, whereas FloTrac analyzes stroke volume based on pulse pressure variances. Each manufacturer has its own, largely proprietary algorithms. Over the years, manipulation of the algorithms attempted to mitigate errors introduced by various hyperdynamic states such as sepsis, no definitive outcome trial is available to guide the clinician in its use (128–130).

**LidcoPlus** (Lidco, Cambridge, UK): LidcoPlus combines the previously validated Lidco lithium indicator dilution calibration procedure with continuous pulse contour analysis for real-time CO assessment (131). A small amount of lithium chloride is injected in a vein and the concentration of arterial sampling over time produces a concentration-time curve. The area under this curve provides the CO by integral mathematics. The manufacturer recommends calibration every 8 hours. Studies have
shown that LidcoPlus provides similar CO results as traditional PACs over a wide range of hemodynamic states (132,133).

**FloTrac** (Edwards Lifesciences, Irvine, CA, USA): This features CCO and SVV without need for calibration utilizing an existing arterial catheter. Assumptions are made regarding the patient’s vascular compliance given his or her age, weight, and height. Proprietary software analyzes waveform contours beat to beat and evaluates CO based on the concept that stroke volume is related to beat-to-beat pressure changes. The algorithm also takes into account the dynamic changes in vascular compliance by assessing the characteristic changes associated with alterations in vascular tone. Preliminary studies have demonstrated similar CO results compared to intermittent, bolus titrations (134). Although the use of SVV has only been validated in heavily sedated patients without spontaneous respirations and in sinus rhythm, preliminary data confirm that SVV may be utilized in spontaneously breathing patients with quiet respirations.

The search for new, noninvasive, emerging technologies continues. The literature on noninvasive hemodynamic monitoring devices is extensive; as of 2015, more than 1,500 articles attempt to compare various novel devices to address the complexities of hemodynamic monitoring. Devices encompassing bioimpedance, spectroscopy, pulse contour and wave analysis, and indwelling transesophageal echo all fail to address the clinical demand in a reliable manner and none has withstood the rigorous, randomized controlled clinical testing that demonstrates improved outcome (135). For now, the thermodilution technique is still gold standard.

**SUMMARY**

The value of any monitoring system is to impact outcomes. Although surrounded by controversy, there remains a group of patients who may benefit from invasive monitoring. It is imperative that technology is used by trained personnel who understand both the benefits and the limitations of the devices. Equally important is the concept of early resuscitation before the onset of multisystem organ failure. The next quantum leap in hemodynamic monitoring will allow us to optimize DO2 to the tissue bed in a noninvasive platform. But until we can noninvasively and continuously monitor CO, cardiac preload, intravascular blood volume status, central SvO2, and tissue oxygenation (see chapters 20, 22, 24, and 27), the PAC will likely continue to have a place in modern ICUs (136–138). To minimize the heterogeneity and often difficult to interpret data, future research should be focused on specific patient populations, particularly in high-risk surgical patients (139,140).

**Key Points**

- Misinterpretation and inexperience in PAC likely contribute to lack of outcome benefit from its use.
- Clinicians should titrate flow variables during resuscitation to meet cellular level oxygen demand.
- The preload to stroke volume relationship governed by Frank–Starling’s Law is a dynamic response to patient’s clinical status.
- SVV has limited utility in patients who have spontaneous respiration and/or tachycardia.
- Until we can noninvasively and continuously monitor CO, cardiac preload, intravascular blood volume status, central SvO2, and tissue oxygenation (see chapters 20, 22, 24, and 27), the PAC will likely continue to have a place in modern ICUs.

**References**