CHAPTER 18  ■  HEMODYNAMIC MONITORING: ARTERIAL AND PULMONARY ARTERY CATHETERS

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Since the introduction of the pulmonary artery catheter (PAC) in the 1970s, initial enthusiasm has been tempered by allegation that the use of PAC may cause harm (1). Several academic societies have convened expert panels to review the literature and discuss important issues regarding PAC utilization (2). For most critical care practitioners, the benefits of using PACs has warranted continued use in high-risk patients, but controversy remains.

Inherent in the use of the PAC is the assumption that flow-related variables such as cardiac output/cardiac index (CO/CI) and oxygen delivery (DO2) to the tissues are important for survival. Shoemaker et al. popularized this concept when they observed that the differentiating parameters between survivors and nonsurvivors were flow-related variables such as DO2, oxygen consumption (VO2), and CO instead of the traditional values of blood pressure (BP), heart rate (HR), urine output, and arterial oxygen tension (PaO2) (3,4). In addition, Bihari et al. (5) reported that as DO2 increased, suggesting that oxygenation was dependent. The biggest criticism of the supply dependency concept was the mathematical coupling between DO2 and VO2, since cardiac output is on both sides of the equation. 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. The theory of supply dependency and titrating DO2 until the VO2 slope flattens is an attractive concept but is not practical.

Over the decades, clinicians have progressed from treatment 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. What we currently use is an easy, noninvasive method to measure tissue oxygenation and the energy state of the cells. This would allow “titration” of DO₂ to meet tissue demands rather than aiming for a single global survival value of DO₂ and SvO₂ (3,4). We consider these end points of resuscitation to be NOT mutually exclusive, but to complement each other into what we call “tiers” of resuscitation. Instead of discarding the value of DO₂ manipulation, we should seek better measurements of end points of resuscitation, and search for treatment modalities to improve oxygen transport into the cells.

While literature on use of PAC is vast and confusing, it is important for readers to critically evaluate the studies by asking the following questions:

1. What patient population was chosen for the study and was that an appropriate choice of patients?
2. Were the patients chosen early in the course of illness, or after they developed multisystem organ failure (MSOF)? Was there a time specified to reach the goals? Timing of resuscitation is essential for successful outcome. Studies stressing early resuscitative efforts (7–10) demonstrate better outcomes with DO₂ and SvO₂ goals than studies with no time specification (11,12). Given that early resuscitation 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 (13).
3. What were the deletion criteria? Studies excluding patients with acute cardiac or pulmonary problems would be delimiting patients who would most benefit from PAC use. One study that deleted patients who already had a PAC, chronic obstructive pulmonary disease (COPD), renal failure, acute myocardial infarct, and liver disease reported no advantage of PAC use (13).
4. Did patients with good cardiac function and ability to respond to increased metabolic demands “negate” the average differences in DO₂ between the control and treated groups; that is, did the study enroll patients with good cardiac function who reached their hemodynamic goals with minimum intervention? These patients tend to do well with low mortality. There are two studies that deleted patients with good cardiac function, but these studies had conflicting outcome results, most likely due to differences in treatment and timing of goal achievement (12,14).
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 (DO₂/I) rather than CI since the acceptable CI would vary with hemoglobin levels (15,16).
6. How was the treatment administered? Were fluids given to reach a certain pulmonary arterial occlusion pressure (PAOP)? Is left ventricular stroke work index (LVSWI) an appropriate end point for fluid administration since a high BP would lead to an elevated LVSWI without an adequate preload (12)? How were fluid and blood given? What were the dosages of inotropes and did large amounts of inotropes (i.e., 200 μg/kilogram of dobutamine) possibly contribute to negative outcome (12)?
7. What percent of study patients did not reach the goals? Was failure to reach goals due to inadequate effort by the treating team or due to the inherent inability of the patient’s myocardium to respond to treatment? We demonstrated that in the initial phases of our prospective randomized trials, failure to reach DO₂/I of ≥600 mL/minute/m² occurred 46% of the time but decreased to 19% in the second part of the trial (14). High failure rates (>79%) to reach hemodynamic goals have been reported in some studies with negative outcomes (12,17).

Differences in study design and treatment algorithm may have contributed to the confusion. Nevertheless, our therapeutic regimen is limited, and as clinicians, we continue to optimize DO₂ with the hope of delivering oxygen to the tissues.

### Table 18.1

<table>
<thead>
<tr>
<th>Indications for Pulmonary Artery Catheter Insertion</th>
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<tbody>
<tr>
<td><strong>Precautionary Reasons</strong></td>
</tr>
<tr>
<td>For prevention of multisystem organ failure in high-risk patients (perforated viscus)</td>
</tr>
<tr>
<td>For prophylactic assessment of high-risk patients with cardiac, pulmonary, and renal dysfunction</td>
</tr>
<tr>
<td>For management of high-risk patients postoperatively (major hemorrhage)</td>
</tr>
<tr>
<td>For patients with expected large fluid shifts: sepsis, bleeding, multiple trauma, burns, and circulatory collapse</td>
</tr>
<tr>
<td><strong>Treatment of Shock</strong></td>
</tr>
<tr>
<td>Hypotension not relieved with fluid</td>
</tr>
<tr>
<td>Suspected cardiac event or cardiac compromise contributing to shock</td>
</tr>
<tr>
<td>Oliguria not responding to fluid</td>
</tr>
<tr>
<td>Patients with multiple organ dysfunction</td>
</tr>
<tr>
<td>For continuous SvO₂ monitoring</td>
</tr>
<tr>
<td><strong>To Guide Treatment in Pulmonary Dysfunction</strong></td>
</tr>
<tr>
<td>To differentiate cardiogenic causes of hypoxia from acute respiratory distress syndrome and guide fluid management</td>
</tr>
<tr>
<td>For monitoring cardiac output in patients requiring high positive end-expiratory pressure (≥15 cm H₂O)</td>
</tr>
<tr>
<td><strong>Treatment of Cardiac Dysfunction</strong></td>
</tr>
<tr>
<td>Complicated myocardial infarction</td>
</tr>
<tr>
<td>Congestive heart failure with poor response to afterload reduction and diuretic therapy</td>
</tr>
<tr>
<td>Suspected rapamone or contraindication from blunt chest injury</td>
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<tr>
<td>Pulmonary hypertension with myocardial dysfunction</td>
</tr>
</tbody>
</table>

**Section II: Monitoring**

**PEARLS**

- Identify patients who may benefit from PAC insertion (Table 18.1), and identify what hemodynamic information is needed to guide treatment.
- 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).
INDICATIONS FOR PULMONARY ARTERY CATHETER INSERTION

Indications for PAC insertion (Table 18.1) 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 (2,31). 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 MVO2; (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 and heart rate normal; and (f) prevent myocardial infarct by avoiding wide swings (±15%) in heart rate and blood pressure (21,32,33). 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 exam. Clinical predictors of hemodynamic status in the critically ill patient, such as chest radiograph, jugular venous distention, and urine output, are inaccurate (1,34). Physicians are correctly able to predict PAOP and CI only 30% to 70% of the time. With insertion of the PAC, four types of information may be obtained:

1. Central pressures in relationship to the right ventricle (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 cardiac output and presented as cardiac index
3. SvO2
4. Intrapulmonary shunt (Qp/Qt)

General Considerations

The technical aspects of PAC insertion are presented in “Vascular Catheterization.” It is essential that the catheter be positioned and transduced properly, and a knowledgeable clinician must be able to correctly interpret the data (18,35,36). 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 weren’t 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 or retro calibration is done prior to removing the catheter tip from the casing. The PAC is then flushed to assess the patency of its 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-size heart. For cardiac output monitoring, a thermistor is located proximal to the tip to measure temperature changes (discussed below). The catheter is placed through the protective sheath and the balloon is checked for integrity prior to insertion. The transducer is placed at the level of the patient’s midsagittal line and zeroed to atmospheric pressure (phlebostatic point). If the transducer elevates above the patient level, the readings will be falsely low. Conversely, if the transducer falls below the patient level, the readings will be falsely high. This is typically noted in beds that are designed to rotate patients.

PRESSURE MEASUREMENTS

Normal hemodynamic values are presented in Table 18.2. Pressure changes in the heart or vessels cause movement of the catheter, which is then converted to an electrical signal by a transducer (37). Electrical noise is filtered and the signal is amplified and displayed as a tracing on a 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 1.5 to 20 cm into the introducer in the vein, the balloon is inflated and the catheter is gently advanced. The natural flow of blood from the vena cava...
TABLE 18.2

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 mm Hg</td>
</tr>
<tr>
<td>Diastolic blood pressure</td>
<td>60–90 mm Hg</td>
</tr>
<tr>
<td>Mean arterial pressure (MAP)</td>
<td>70–105 mm Hg</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 mm Hg</td>
</tr>
<tr>
<td>Right ventricle systolic pressure</td>
<td>15–30 mm Hg</td>
</tr>
<tr>
<td>Right ventricular diastolic pressure</td>
<td>0–8 mm Hg</td>
</tr>
<tr>
<td>Pulmonary artery (PA) systolic pressure</td>
<td>15–30 mm Hg</td>
</tr>
<tr>
<td>PA diastolic pressure</td>
<td>4–12 mm Hg</td>
</tr>
<tr>
<td>Mean PA</td>
<td>9–16 mm Hg</td>
</tr>
<tr>
<td>Pulmonary artery occlusion pressure (PAOP, wedge)</td>
<td>6–12 mm Hg</td>
</tr>
<tr>
<td>Left atrial pressure (LAP)</td>
<td>6–12 mm Hg</td>
</tr>
<tr>
<td>Cardiac output (L/min)</td>
<td>Varies with patient size</td>
</tr>
<tr>
<td>Cardiac index (L/min/m²)</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 to body surface area (RVEDVI)</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 mm Hg</td>
</tr>
<tr>
<td>Arterial oxygen saturation</td>
<td>93%–98%</td>
</tr>
<tr>
<td>Mixed venous oxygen tension (PvO₂)</td>
<td>36–42 mm Hg</td>
</tr>
<tr>
<td>Mixed venous oxygen saturation (SvO₂)</td>
<td>70%–75%</td>
</tr>
</tbody>
</table>

through the heart and to the lungs guides the catheter to the pulmonary vasculature (38). While passing from the vena cava to a branch of the pulmonary artery, characteristic waveforms are displayed on the monitor (Fig. 18.1). Once the catheter is advanced to the “wedged” position, 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. The catheter should coil gently in the right ventricle (RV) and sit in a larger branch of the pulmonary artery (Fig. 18.2). A chest radiograph is done to assess for pneumothorax and may help to confirm proper position. If there is not an obvious wedge tracing, blood may be sampled from the distal port with the balloon inflated, after discarding about 15 mL from the distal port. The sample should have a higher PaO₂ and pH with a lower PCO₂ than blood aspirated when the balloon is deflated (39). Proper placement is also indicated by the SvO₂ signal quality (if using a fiberoptic catheter), which is displayed in different ways by different manufacturers. The quality of the signal may be altered by a fibrin clot at the tip of the catheter or by placement of the tip against a vessel wall and may suggest that the catheter is in too far or that the patient is hypovolemic with collapse of the vessel wall around the catheter tip. If the

![FIGURE 18.1. Waveforms seen during pulmonary artery catheter insertion.](image-url)
catheter is in too far, the PAOP tracing will continue to elevate and is called “overwedging” (Fig. 18.3). If this occurs, the balloon should be deflated and the catheter pulled back (≈1 cm) and the balloon inflated again 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 (40). This effect may be reduced by using stiff, noncompliant tubing and the shortest length (<4 feet) 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 (Fig. 18.4). 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 (41). 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. 18.4B). 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. 18.4C). 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. This may be minimized by a high-frequency filter built into the transducer system (40).
FIGURE 18.5. Reading of pulmonary artery occlusion pressure (PAOP) at end expiration. During spontaneous breathing, 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.

Whether the patient is on mechanical ventilation (positive intrathoracic pressure) or spontaneously breathing (negative intrathoracic pressure), all pressure measurements should occur at end expiration when the intrathoracic pressure is closest to atmospheric pressure (Fig. 18.5) (unless the patient is on higher levels of positive end-expiratory pressure [PEEP]). This point can be determined by watching the patient's respiratory movements or displaying the airway pressure tracing on the same monitor where the pulmonary artery pressure 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. 18.6). In this situation, patients may need to be sedated, or if getting a PAOP is crucial, even paralyzed. What is unacceptable is to “guess” what the number may be, leading to wrong conclusions and wrong treatment. Nothing is better than wrong information.

The first characteristic waveform seen when inserting a PAC is the right atrium (RA) tracing (Fig. 18.7). 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 and is due to the pressure increase during atrial systole (Figs. 18.7 and 18.8). 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 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. 18.7). 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 (A-V) 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. 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

FIGURE 18.6. 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.
these patterns may help to suggest a diagnosis before a confirmatory echocardiogram is obtained.

The pressures observed in the right atrium range from 0 to 8 mm Hg. Higher pressures may not necessarily mean fluid overload, but 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 pulmonary artery pressures are elevated (42,43). It is in these situations of right heart failure, severe pulmonary disease, and in most of the critically ill patients when monitoring the CVP only (and not the PAOP) would be misleading.

The next waveform seen is that of the right ventricle (Fig. 18.1). 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 quickly advanced through the ventricle both to avoid dysrhythmias and to keep the catheter from warming and losing its stiffness. The RV systolic pressures generally range from 15 to 30 mm Hg and diastolic pressures from 4 to 12 mm Hg. 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 in patients with pre-existing left bundle branch block (LBBB) (44). However, the incidence of complete heart block appears to be no greater in patients with LBBB than without (45).

Once the catheter enters the pulmonary artery, the waveform shows an increase in diastolic pressure while the systolic pressure remains about the same as in the ventricle, sometimes referred to as the “step up” (Fig. 18.1). 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 30 cm, it may be coilng 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 mm Hg systolic over 8 to 15 mm Hg diastolic. The beginning of diastole is marked by a dicrotic notch on the PA tracing, corresponding to the closure of the pulmonic valve (46). 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 electrocardiogram (ECG). The pulmonary circulation is very dynamic and is affected by acidosis, hypoxia, sepsis, and vasoactive drugs (47). An increase in CO may also seemingly paradoxically lower the PA pressures by a reflexive decrease in pulmonary vascular resistance with fluid resuscitation and decreased sympathetic nervous system discharge (48).

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 mm Hg in normal states. PAOP most closely reflects LVEDP after atrial contraction and before ventricular contraction (Fig. 18.8). 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 (49). 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. 18.9). A prominent v wave may fool the novice into thinking that the catheter is not wedging. It is important to note the change in wave form 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. 18.9). There may be large a waves secondary to decreased left ventricular compliance (50), the point 0.03 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 (51) (Fig. 18.6).

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. Since there is no flow, the pressure between the occluded pulmonary artery segment and the left atrium will equalize (Fig. 18.10), analogous to closing off a pipe with pressures equalizing between the two ends (52). With the closed pipe analogy, there is a list of assumptions: PAOP = PcP = LAP = LVEDP = LVEDV, where PcP is pulmonary capillary pressure, LAP is left atrial pressure, and LVEDP 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 one 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 (53). In 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. 18.11). 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 (54). When pulmonary artery diastolic 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 (PcP) when there is an increase in pulmonary vascular resistance (PVR), the wedge pressure

\[ \text{PAOP} = \text{PcP} = \text{LVEDP} = \text{LVEDV} \]

\[ \text{PAOP} = \text{PcP} = \text{LAP} \]

\[ \text{PAOP} = \text{PcP} = \text{LVEDP} = \text{LVEDV} \]
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FIGURE 18.10. Closed pipe analogy: blocking of flow by the balloon with theoretical equalization of pressure in the conduit. RA, right atrium; RV, right ventricle; PAOP, pulmonary artery occlusion pressure; Pc, pulmonary capillary; LAP, left atrial pressure; LV, left ventricle.

underestimates Pc. A difference of 2 to 3 mm Hg between the PAOP and pulmonary artery diastolic (PAD) pressure is a clue that there may be a discrepancy between PAOP and Pc (35). 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 (35). The point where the rapid decline transitions to a more gradual slope before reaching the PAOP is the Pc (Fig. 18.12).

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 (56). 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 mm Hg, but a greater effect is seen in hypovolemic patients or when the catheter is not in West zone 3 (57). 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 (54). 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

FIGURE 18.11. West’s lung zones. PAP, pulmonary arterial pressure; PalvP, pulmonary alveolar pressure; Ppv, pulmonary venous pressure. Zone 1: PAP < PalvP < Ppv (there is no blood flow across the collapsed pulmonary capillary bed). Zone 2: PAP > PalvP > Ppv (there is some flow when PAP is greater than PalvP); Zone 3: PAP > PalvP = Ppv (pulmonary arteries are patent).
assumptions. In order to more accurately correct for the 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, resulting in a “transmural” pressure. 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 (58). This nadir pressure better reflects LAP than the on-PEEP PAOP. The discontinuation should be brief (<1 second) so that a decrease in PaO\(_2\) from derecruitment of alveoli does not occur (59). 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 FiO\(_2\) may be increased temporarily for patient safety, the balloon reinflated, 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 (58). When done properly, it is extremely rare to cause hypoxia.

2. LAP \(\approx\) 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 above). 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 ventricle compliance such as in ischemic states, left ventricular hypertrophy, and restrictive cardiomyopathies (60). This is especially true when LVEDP is greater than 25 mm Hg.

3. LVEDP \(\approx\) 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. 18.13). 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 (52,61). 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 mm Hg (62,63), some patients require a high pressure to reach the
optimum stroke volume (Fig. 18.14). The Starling curve plotting stroke volume index to PAOP (as an estimate of LVEDP) may help identify the optimum wedge but some patients may have a flat curve. If vasoactive agents are started, the heart may now be on a different curve requiring new assessment of the optimum PAOP. The 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). There are no set numbers to treat to, but each patient must be individually assessed and assessed repeatedly, making the PAC a highly user-dependent tool.

Elevated wedge pressures may help differentiate hydrostatic pulmonary edema from that caused by increased permeability. A PAOP of 24 mm Hg or higher is associated with a tendency for hydrostatic edema (68). Lower pressures may imply increased capillary permeability and traditionally, a PAOP of ≤18 mm Hg 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. 18.12).

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 get 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 as the clinical condition changes (such as vasoactive agents, myocardial compliance, and external forces around the heart). The effects of manipulating preload, afterload, and contractility and shifting to another curve can be seen.

**CARDIAC OUTPUT**

The ability of the heart to meet increasing tissue oxygen demand is perhaps the single most important determinant in oxygen delivery and tissue perfusion. The evolution of cardiac output measurement 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: $\text{CO} = \frac{\text{VO}_2}{\text{CaO}_2 - \text{CvO}_2}$, where $\text{VO}_2$ is oxygen consumption, $\text{CaO}_2$ is arterial content of $\text{O}_2$, and $\text{CvO}_2$ is mixed venous content of $\text{O}_2$. This principle is widely accepted as an accurate though invasive assessment of cardiac output since a pulmonary artery catheter must be placed to obtain accurate mixed venous oxygen content. Its practical use is limited by the cumbersome measurement of oxygen consumption. Stewart (1897) and Hamilton (1932) utilized the concept 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 cardiac output using the following equation: Cardiac output = Amount of dye injected/Integral (dye concentration × function of time). The next revolutionary step in cardiac output measurement was using temperature as the dye. Crystallized solution (usually 10 mL, but 5 mL may be used as 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 (70). The thermistor near the tip of the PAC detects the change in temperature,

![Figure 18.14. Frank-Starling curves (family of curves) showing the relationship between left ventricular end-diastolic pressure (LVEDP) to stroke volume (SV, mL/beat). Augmenting preload increases LVEDP with a concomitant increase in SV (up to a certain point). The effects of manipulating preload, afterload, and contractility and shifting to another curve can be seen.](image-url)
Section II: Monitoring

Modified Stewart-Hamilton equation

\[ Q = \frac{VI (TB - T1) \times SI \times CL \times 60 \times CT \times K}{SB \times CB \times \Delta TB \, dt} \]

- **Q** = Cardiac Output
- **VI** = volume of injectate
- **TI** = injectate temperature
- **TB** = blood temperature
- **CL** = specific heat of the injectate (D5W = 0.965, saline = 0.997)
- **SI** = specific gravity of the injectate (D5W = 1.018, saline 1.005)
- **60** = seconds/minute
- **CT** = correction factor (loss of thermal indicator due to time lost in injecting, catheter length, patient's temperature)
- **TB** = mean temperature of the injectate delivered to the right atrium
- **TB - TI** (pre-injectate temperature)
- **SB** = specific gravity of blood (1.045)
- **CB** = specific heat of the blood (0.87)

\[ \Delta TB \, dt = \text{integral of blood temperature change} \]

Computation constant (K) = \[ \frac{SI \times CL \times 60 \times CT \times VI}{SB \times CB} \]

Changes with VI

*FIGURE 18.15.* The modified Stewart-Hamilton equation for estimating cardiac output.

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 cardiac output (Fig. 18.15).

Although initial studies used iced solutions as injectates, ambient temperature injectate is now the standard solution used with excellent reproducibility and correlation with iced injection (71) and has less likelihood of reflexive bradycardia (72). It is important to note that iced injectates (0°C to 5°C) are associated with higher reproducibility and the highest signal-to-noise ratio (73) 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 (74,75). 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 cardiac output. Although there is no 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 (74). Unlike the manual injection of crystalloid, the measurements are done 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. A word of caution is that the CCO value may not change instantly when the cardiac output changes (e.g., with titration of inotropes), but the effect of treatment can be seen in seconds if using a continuous SvO\textsubscript{2} monitor. Due to the heat-generating wire coil in the distal end, these catheters must be removed before magnetic resonance imaging.

Dr. 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 preload 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, Fig. 18.14), 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 ON VENOUS OXIMETRY)

Specialized PACs with the ability to measure mixed venous oxygen saturation ($SvO_2$) continuously using principles of reflection spectrophotometry are available (Fig. 18.16). Oxygen saturation is the ratio of hemoglobin bound to $O_2$ 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 $SvO_2$ value indicates the balance between oxygen delivery to the tissues and the amount consumed by the tissues before returning to the heart.

Rearranging the Fick Equation

$$\text{SvO}_2 (\%) = \frac{\text{VO}_2}{\text{SaO}_2 - \text{VO}_2 \times \text{CO} \times \text{Hgb}} \times 1.36 \text{ (mL/O}_2/\text{g Hgb)} \times 10$$

Four factors determine the $SvO_2$ value: three parameters contributing to oxygen delivery (CO, hemoglobin, and $\text{SaO}_2$), and one parameter for $O_2$ consumption. Low $SvO_2$ suggests insufficient $O_2$ delivery or increased $O_2$ consumption. $SvO_2$ is also a harbinger of shock and may decrease before overt shock is apparent (14,15,25,76). Mixed venous oxygen saturation has also regained popularity as an end goal of resuscitation with decreased mortality (7,77–79). Inadequate oxygen delivery can be the result of decreased cardiac output, low hemoglobin, or low oxygen saturation. Increased consumption may occur due to activity, fever, hyperthyroid state, or repayment of oxygen debt. High $SvO_2$ 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 $\text{VO}_2$. 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 <1.25 mL of air.

Calibration by lab oximeter on a daily basis is important to check for drifting and whenever the values do not seem to correlate with the patient’s clinical condition. Even if the PAC does not have continuous $SvO_2$ monitoring, $SvO_2$ can be checked by sending a blood sample from the distal PA port (by drawing slowly at the rate of 1 mL over 20 seconds to prevent sampling of pulmonary capillary blood) and sending it to the lab for oximeter analysis (direct saturation measurement and not arterial blood gas [ABG] analysis). If the PAC has continuous $SvO_2$ monitoring, a signal quality indicator is generated. If the signal intensity suggests poor quality, errors include (a) that the PAC is in too far, (b) there are fibrin clots around the tip, (c) the catheter is touching the vessel wall, or (d) the patient may be hypovolemic. Repositioning the PAC or flushing the PA port may resolve this issue.

**INTRAPULMONARY SHUNT (QS/QT)**

Shunt refers to the portion (in %) of blood that flows (CO) from the right side of the heart to the left completely deoxygenated (Fig. 18.17). We will not discuss cardiac shunts in this chapter. Physiologic shunt = Anatomic shunt + Intrapulmonary shunt. Anatomic shunt refers to the direct drainage of the venous system to the left ventricle through the bronchial, thebesian, and pleural veins (∼2%–5% of CO). Intrapulmonary shunt (Qs/Qt) is expressed as the % of cardiac output passing through completely collapsed alveoli with no or little gas exchange so that the ventilation-to-perfusion ratio is zero (i.e., $V/Q = 0$).
Venoarterial admixture or shuntlike states refer to blood flow passing through partially open alveoli (i.e., V/Q <1). Acute shunt will not respond to FiO₂ and the treatment is PEEP to open the alveoli and make them responsive to FiO₂. Venous admixture will demonstrate some response to FiO₂. If the shunt is minimal (normal condition), there is almost a linear relationship between FiO₂ and PaO₂, but as the shunt increases, FiO₂ no longer affects PaO₂ in a linear fashion (Fig. 18.18). This is an important concept as one cannot improve hypoxaemia by increasing FiO₂ alone, but needs to open the alveoli with PEEP. If the shunt equation is calculated on a FiO₂ of <1.0, both the shunt and venous admixture will be captured in the equation. Intrapulmonary shunt (Qs/Qt) is calculated as \( \frac{(CcO_2 - CaO_2)}{(CcO_2 - CvO_2)} \), where \( CcO_2 \) is the pulmonary capillary content of O₂, \( CaO_2 \) is the arterial content of O₂, and \( CvO_2 \) is the mixed venous content of O₂ (Table 18.3). 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 >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/minute, the PaO₂ will decrease from ~80 to 65 mm Hg (Fig. 18.19). In the exact same scenario, if the patient’s hemoglobin is 10 g/dL, the PaO₂ will decrease from 70 to 55 mm Hg. This demonstrates the importance of low SvO₂ contributing to lower PaO₂. If the PEEP had been increased in this patient to treat a low PaO₂, a further decrease in CO would have resulted in worsening SvO₂ and PaO₂. Treatment in this case is to optimize CO first to see if PaO₂ improves. Another example: If a patient is agitated and the arterial saturation decreases, this may be due to increased oxygen consumption 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 cardiac output and PEEP simultaneously in patients...
with life-threatening hypoxia. It is important to understand the interaction of one organ on the other and the relationship between PaO₂, hemoglobin, and cardiac output (Fig. 18.19).

**DERIVED VARIABLES**

See Table 18.3 for the equations and normal values. Once the flow and pressure values have been obtained from the PAC, further hemodynamic calculations may be done to obtain complete information. Most bedside monitors are capable of calculating and displaying the numbers, but clinicians must understand the significance and pitfalls of these values.

**Stroke volume index (SVI)** 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 heart rate’s contribution to CI, and is an important variable because one does not want to augment CI by causing tachycardia (Fig. 18.14). SVI varies with preload, afterload, and contractility. Preload is the theoretical stretch of ventricles at end diastole.

### Table 18.3

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Equation</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stroke volume indexed (SVI)</td>
<td>CIHR</td>
</tr>
<tr>
<td>Left ventricular stroke work index (LVSWI)</td>
<td>EDVI – ESVI × (MAP – PAOP)</td>
</tr>
<tr>
<td>Right ventricular stroke work index (RVSWI)</td>
<td>SVI × (MPAP – CVP)</td>
</tr>
<tr>
<td>Pulmonary vascular resistance indexed (PVRI)</td>
<td>(MAP – CVP) × 80</td>
</tr>
<tr>
<td>Systemic vascular resistance (SVR)</td>
<td>(MAP – CVP) × 80</td>
</tr>
<tr>
<td>Arterial oxygen content of blood (CaO₂)</td>
<td>Hgb × 1.36 mL O₂/gHgb + (0.0031 × PaO₂)</td>
</tr>
<tr>
<td>Mixed venous oxygen content of blood (CvO₂)</td>
<td>(0.0031 × PaO₂)</td>
</tr>
<tr>
<td>Arterial-mixed venous oxygen content difference (AVDO₂)</td>
<td>How much O₂ was consumed by the tissues before returning to the heart</td>
</tr>
<tr>
<td>Delivery of oxygen indexed (DO₂i)</td>
<td>CaO₂ × CI × 10</td>
</tr>
<tr>
<td>Oxygen consumption indexed (VO₂i)</td>
<td>(CaO₂ – CvO₂) × CI × 10</td>
</tr>
<tr>
<td>Intrapulmonary shunt (Qp/Qs)</td>
<td>(CeO₂ – CarO₂)/(CeO₂ – CvO₂)</td>
</tr>
<tr>
<td>Coronary perfusion pressure (CPP)</td>
<td>CPP = DRP – PAOP</td>
</tr>
</tbody>
</table>

CI: cardiac index (mL/min/m²); HR, heart rate (beats/min); EDVI, end-diastolic volume index (mL/min/m²); ESVI, end-systolic volume index (mL/min/m²); MAP, mean arterial pressure; DRP, diastolic blood pressure (mm Hg); PAOP, pulmonary artery occlusion pressure (mm Hg); CPP, coronary perfusion pressure (mm Hg); Hgb, hemoglobin (g/dL); PaO₂, partial pressure of oxygen in the alveolar; RQ, respiratory quotient; VO₂i/VO₂ is 0.8 for a mixed fuel diet.
According to Frank and Starling, the stretch of myocardium augments contractility to a certain point, and then cardiac output is negatively affected by further increases (80). Afterload is 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. 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) 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. Current technology does not allow continuous measurements of both ventricular volumes at the bedside. Stroke work index is low in cardiogenic and hypovolemic shock. In trauma patients, it has been suggested that high LVSWI is associated with decreased mortality (81,82). It should be noted that not all work is alike since “good work” is associated with large volume change with little pressure, and “bad work” is associated with large pressure change with little volume movement.

Right ventricular stroke work index (RVSWI) is the right ventricle’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 (discussed below).

Pulmonary vascular resistance index (PVRI) is the resistance for the right ventricle. Resistance to blood flow is analogous to electrical circuit resistance defined by Ohm’s Law: \( R = \frac{P}{I} \). Physiologically, the pressure change between two vascular beds drives the flow (i.e., cardiac index). PVRI reflects resistance in the pulmonary vasculature. Pulmonary hypertension exists when systolic PAP is >35 mm Hg or mean PAP is >25 mm Hg (83,84). In critically ill patients, the most common causes for elevated PAP are acute respiratory distress syndrome, acute LV dysfunction, and pulmonary embolism (83,85–89). Patients with comorbid conditions such as chronic pulmonary hypertension may suffer from interstitial lung disease, COPD, or liver or cardiac disease. The right ventricle is exquisitely sensitive to increases in afterload and lacks the ability to overcome pulmonary hypertension with PAP >40 mm Hg (84). Subsequent decrease in cardiac output is due to the combination of decreased RVSWI and decreased filling of the left ventricle as a result of interventricular septal deviation (90). Since cardiac output is indexed to BSA, PVR should also be indexed and presented as PVRI.

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

Arterial oxygen content of blood (\( CaO_2 \)) 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 ($CvO_2$) is the amount of oxygen carried in the mixed venous blood. Low mixed venous oxygen content has similar clinical implications as low $SV_O_2$ and suggests decreased oxygen delivery or increased oxygen consumption (see above). Since the blood is sent for oximeter analysis for saturation value and not $PvO_2$, the $PvO_2$ value in the equation (Table 18.3) is usually substituted with the normal $PvO_2$ value of 40 mm Hg since the amount dissolved is so small that a $PvO_2$ substitution of 0 to 70 mm Hg will not make a difference in the calculation of $CvO_2$.

Delivery of oxygen indexed to BSA ($DO_{I}$) is the amount of oxygen delivered to the tissues by hemoglobin, arterial saturation, and flow ($CI$; i.e., the product of cardiac index and arterial oxygen content of blood). The survival benefit of titrating to a specific $DO_{I}$ value has been extensively studied as an end point of resuscitation with conflicting results. The controversy surrounding $DO_{I}$ augmentation is discussed in the beginning of this chapter (10,14,21,22,32,91–93).

Oxygen consumption indexed ($VO_{I}$): There are two methods of assessing oxygen consumption: Fick's principle and indirect calorimetry (94). 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 cardiac output. Consumption can also be assessed by indirect calorimetry and is typically 3.5 mL of oxygen/kg (95). 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 oxygen consumption (96–99). Shoemaker first noted that a higher $VO_{I}$ if desired, but there is individual variation.

**SPECIAL COMMENT ON OBESITY AND DERIVED PARAMETERS**

The validity of derived parameters indexed to body surface area has been questioned in morbidly obese patients. Several studies have demonstrated that derived parameters indexed to body surface area are appropriate and closely approximate indexing to body mass index. The large body surface area in the obese patient does not affect these measurements (103–105).

**COMPLICATIONS OF PULMONARY ARTERY INSERTION**

Pulmonary artery catheter insertion is an invasive procedure and carries inherent risks (106–108). 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, hemoptysis, 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 (109,110). 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%, consequent bacteremia is relatively rare (0.7%–2.2%) (111). Catheters inserted for greater than 3 days may be associated with more infectious and thrombotic complications (112,113). 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, and pulmonary artery rupture (109,110). Pulmonary artery rupture is exceedingly rare with a reported incidence of 0.031% (114) but usually occurs in patients with pulmonary hypertension and can be fatal due to high pulmonary pressures. Slow inflation of the balloon with immediate abortion if there is too much resistance or the waveform shows overwedging are important precautions. As discussed earlier in this chapter, perhaps the most dangerous complication of pulmonary artery catheter insertion is the misinterpretation of information.

**ARTERIAL LINES**

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

Arterial cannulation is relatively safe with nonocclusive thrombosis and hemorhage being the most common complications (115). 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 (118,117). 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 (118,119).

Pressure Measurement

Continuous measures of systolic blood pressure (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 blood pressure monitoring rendering the often large discrepancy between cuff and invasive pressures. The SBP is determined by the ventricular ejection velocity and volume. 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. 18.4). A properly calibrated system produces one overdamped waveform followed by several oscillating overshoot waves (120).

Waveform analysis (Fig. 18.20) 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. 18.20, 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 mean arterial pressure 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 (121). 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 systolic blood pressure and ventricular stroke volume are of greater magnitude in hypovolemic states (122). Theories on the etiology of this phenomenon relate to the characteristics unique to hypovolemia and include (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. 18.21)—delta up (Δup) and delta down (Δdown)—while emphasizing the strong correlation between Δdown and hypovolemic states (123–125). Δup is the difference between maximum SBP and a reference SBP (usually at expiratory pause during mechanical ventilation). Δdown is similarly the difference between minimum SBP and reference SBP and represents a decrease in stroke volume during expiration. SPV > 10 mm Hg indicates hypovolemia and suggests responsiveness to fluid challenge (126). SPV also has significant correlation with the left ventricular end-diastolic area by echocardiogram (127) and PAOP (128). Note that SPV, like stroke volume variation, 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 (122). Pulsus paradoxus describes falls in arterial pressures (≥5 mm Hg) during inspiration and rises in pressures during expiration in spontaneously breathing patients. Reverse pulsus paradoxus occurs in ventilated patients. Proprietary algorithms in new monitor devices analyze the pulse-to-pulse variation in a semi-continuous 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 (128–131). SVV > 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).

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

![Figure 18.20. Arterial waveform analysis (see text for explanation of points 1-6).](image)
Available technology on the market: Several companies market continuous arterial catheter cardiac output 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.

Lidco Plus (Lidco, Cambridge, UK): Lidco Plus combines the previously validated Lidco lithium indicator dilution calibration procedure with continuous pulse contour analysis for real-time cardiac output assessment (132). A small amount of lithium chloride is injected in a vein and the concentration of lithium chloride is measured over time. The algorithm also takes into account the dynamic changes associated with alterations in vascular tone.

FloTrac (Edwards Lifesciences, Irvine, CA, USA) features continuous cardiac output and stroke volume variation 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 patterns to beat-to-beat changes and evaluates cardiac output based on the characteristic changes associated with alterations in vascular tone. Preliminary studies have demonstrated similar cardiac output results compared to intermittent, bolus thermodilution procedures with continuous pulse contour analysis (133). Although the use of SVV has 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.

FloTrac analyzes stroke volume based on pulse pressure variances. Equally important is the concept of early resuscitation before the onset of multisystem organ failure. The next quantum leap in hemodynamic monitoring will come when we are able to treat patients with noninvasive technology with the goal of delivering oxygen to every tissue bed. It is also conceivable that if we can constantly measure with minimally invasive devices cardiac output and cardiac preload, intravascular blood volume status, central SvO₂, and tissue oxygenation (see chapters on noninvasive cardiovascular monitoring, monitoring tissue perfusion, blood volume, and venous saturation monitoring), the PAC could become obsolete in the future. Until that time, it has been difficult to reproduce the continuous reliable information obtained from the PAC.

REFERENCES


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, and that technology is used by trained personnel who understand both the benefits and the limitations of the devices.
Section II: Monitoring


Chapter 18: Hemodynamic Monitoring: Arterial and Pulmonary Artery Catheters

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