A debate continues over the utility of pulmonary artery (PA) catheters in the management of critically ill patients. In 1996, a retrospective study evaluating the use of PA catheters in 5735 critically ill patients suggested that PA catheter use may increase morbidity and mortality. As a result of this research and the lack of studies demonstrating a beneficial effect associated with PA catheter use, several consensus conferences have been held. Based on these consensus conferences, the patient populations for which PA pressure monitoring may be beneficial or additional outcome studies that are needed include: sepsis/septic shock, acute respiratory distress syndrome (ARDS), severe refractory heart failure, high-risk surgical patients, and low/moderate-risk surgical patients undergoing high-risk surgical procedures.

Since the consensus conferences, a meta-analysis and 4 major outcomes trials have been completed to evaluate the effects of the use of PA catheters as a part of care. The meta-analysis of 13 randomized clinical trials found no difference in mortality or length of stay between groups of patients who were randomized to PA catheter versus no PA catheter groups. The Evaluation Study of Congestive Heart Failure and Pulmonary Artery Catheterization Effectiveness (ESCAPE) evaluated the efficacy and safety of PA catheter use in patients with acute heart failure. In this study, endpoints for resuscitation were specified, but there were no standardized recommendations for the use of diuretics or inotropic agents. The study found that the use of a PA catheter did not increase or decrease the mortality or...
length of stay for acute heart failure patients; however, the group that received PA catheter adjusted therapy had a greater improvement in quality of life and a trend toward improved exercise capacity. The Pulmonary Artery Catheter in the Management of ICU Patients (PAC-Man) study evaluated PA catheter versus use of an alternative method for cardiac output monitoring (eg, transesophageal Doppler) in critically ill patients with ARDS, heart failure, and multiorgan dysfunction. No specific treatment guidelines or endpoints were used. There was no significant difference between groups in ICU or 28-day mortality; although, in the PA catheter group, 80% of patients had a change in treatment made within 2 hours of insertion. The Sepsis Occurrence in Acutely Ill Patients (SOAP) study was an observational study that evaluated the possible association between PA catheter use and outcome. Although patients with PA catheters had a higher mortality rate, when confounding factors such as acuity, age, organ dysfunction, and comorbidities were controlled for, the use of a PA catheter was not independently associated with a higher risk of 60-day mortality. Finally, a study of the use of PA catheters in patients with shock, ARDS, or both found no difference in 14 or 28-day mortality in patients treated with routine (nonstandardized) treatment.

A critical point when reviewing these studies is that the insertion of a PA catheter and simply monitoring hemodynamic indices does not improve outcomes. In addition, it may be insufficient to specify endpoints as goals to be met and not specify the most effective treatment regimen to reach these endpoints. Hemodynamic indices (to include perfusion indices) should be used as a part of an evidence-based treatment plan aimed at optimizing tissue perfusion before organ dysfunction occurs, as exemplified by the improved outcomes associated with goal-directed therapy for patients with septic shock, high-risk surgical patients, and postcardiac surgery patients. The challenge remains to identify which hemodynamic indices and types of monitoring devices (eg, PA catheter, noninvasive pressure and cardiac output monitors, perfusion indices) improve outcomes, to identify specifically which patient populations (ie, patient type, etiology of shock, severity of illness, and timing of interventions) will benefit most from the integration of hemodynamic data into their care, and to develop population specific protocols.

Although there are clinical conditions in which the integration of hemodynamic data may improve diagnostic accuracy and integration of hemodynamic data into a plan of care will improve outcomes, there are 2 general areas that limit the utility of PA pressure monitoring. First, critical care clinicians (nurse and physicians) may incorrectly gather and interpret the data. Several excellent resources to improve the knowledge and ability to interpret and use PA pressure data are the Pulmonary Artery Catheter Education Program (PACEP), which is a series of Web-based training modules that cover clinical and technical aspects of care and waveform interpretation and AACN’s Practice Alert: Pulmonary Artery Pressure Monitoring. Second, while the pulmonary artery occlusion pressure (PAOP) provides useful information regarding hydrostatic pressure and the risk for pulmonary edema, static hemodynamic indices (RAP, PAOP) are not sensitive or specific indicators of preload or fluid responsiveness. The remaining sections of this article focus on: (1) a review of factors that affect the reliability and accuracy of hemodynamic indices (ie, zeroing, referencing, dynamic response characteristics, and filter frequency), (2) how to interpret the hemodynamic data within the context of complex care situations (eg, proning, marked respiratory variation, cardiogenic versus noncardiogenic pulmonary edema), and (3) functional hemodynamic measures, which are an alternative to static hemodynamic measurements.

**Factors That Affect the Reliability and Accuracy of Pulmonary Artery/Right Atrial Pressures Zeroing**

Zeroing of the pressure monitoring system is performed by opening the system to air to establish atmosphere as zero. The current transducers have minimal zero drift and do not require routine rezeroing. Of note, rezeroing is not the same as referencing, which is required with any change in the patient’s position relative to the transducer. A recommendation based on older transducer technology was that the pressure system should be rezeroed with changes in barometric pressure, such as those that occur with a change in the weather or ascent to altitude during
aeromedical transport. The current pressure transducers are vented to air and do not require routine rezeroing with a change in barometric pressure.\textsuperscript{30}

**Referencing**

Referencing, which is performed to correct for the changes in hydrostatic pressure above and below the heart, is accomplished by placing the air-fluid interface (stopcock) of the catheter system at the level of the heart to negate the weight effect of the catheter tubing.\textsuperscript{31} Numerous texts identify the midaxillary line as the reference point; however, in the supine position use of the midaxillary line as the reference rather than one-half the anterio/posterior diameter of the chest, may result in a measurement error of 6 mm Hg in patients with varied chest wall configurations.\textsuperscript{32,33} Similarly, the use of angle specific references is also required for the lateral position (see Figure 4).

**Dynamic Response and Filtering**

The dynamic response characteristics of the system affect the ability of the pressure monitoring system to faithfully reproduce a pressure waveform. A method to evaluate and optimize a pressure monitoring system has been previously described.\textsuperscript{34} One key point in optimizing a pressure system is that air bubbles affect the dynamic response characteristics of any pressure monitoring system. Thus, measures must be taken during the set-up and maintenance of the pressure system to remove air bubbles. The “rocket flush,” which is a 10 mL manual rapid flush of the system, starting at the proximal stopcock, is an additional step during line preparation to remove air from the system.\textsuperscript{35} The performance of a rocket flush during line preparation significantly improves the dynamic response characteristics of pressure monitoring systems (Figure 1).\textsuperscript{30} The “rocket flush” should never be performed when the system is attached to a patient because of the risk of retrograde air embolization. A validated algorithm\textsuperscript{36} that decreases air bubble formation and optimizes the dynamic response characteristics of the pressure system is provided (Table 1).

Adjusting the filter frequency limits on the monitor is often incorrectly attempted when the pressure system is underdamped or when there is catheter whip. To understand the function of the filter, it is important to understand how a waveform is created by the pressure monitoring system. The oscillations caused by the pressure wave striking the fluid in the catheter causes distortion of the diaphragm in the transducer and the creation of an electrical signal. The electrical signal from the transducer is sent to the monitor where it is amplified, filtered, and converted to the waveform and digital output observed on the monitor. The waveform that is observed on the monitor is a summation of a series of sine waves or harmonics (Figure 2).\textsuperscript{36} For a patient with a heart rate of 60 beats per minute, the fundamental frequency (first harmonic) is equal to the pulse rate and occurs at one cycle/second (1 Hz). The second harmonic occurs at 2 Hz, the third harmonic at 3 Hz, etc. For a patient with a heart rate of 120 beats per minute, the first harmonic occurs at 2 Hz, the second harmonic at 3 Hz, etc. The important physiological information is contained in the first 6 to 10 harmonics (12 to 20 Hz). Thus, the bandwidth of the filter on the monitor should be set to allow for up to 12 to 20 Hz for a patient with a heart rate of 120 beats per minute.

**Figure 1:** Effect of a “rocket flush” on the dynamic response characteristics of a pressure monitoring system. A, Control: pressure system with a VAMP (PXVMP160; Edwards LifeSciences, Irvine, Calif.) after standard set-up. Fn, 9 Hz; amplitude ratio, 0.3; dynamic response, underdamped. B, Same system after a 10 mL “rocket flush.” Fn, 13 Hz; amplitude ratio, 0.4; dynamic response, adequate.
Decreasing the filter frequency below 12 Hz may cause the waveform to appear cleaner, but important physiological waveform information is being filtered; thus, a true waveform is not being observed. Rather than adjusting the filter to clean up the waveform, attention should be paid to optimizing the dynamic response characteristics of the system.

**Pulmonary Factors**

There are numerous pulmonary factors that add challenges to accurately and reliably obtaining PA pressure measurements: (1) the use of digital versus analog data, (2) how to determine if the PA catheter tip is in the correct lung zone, (3) how to interpret the pressures when there is marked respiratory variation, and (4) interpretation of invasive pressures when there is increased pleural pressure.

**Table 1: Invasive Pressure Monitoring Line Preparation**

1. Wash hands.
2. Gather supplies (isotonic sodium chloride solution IV bag, pressure monitoring kit, 10 mL syringe, pressure bag).
3. Prime pressure monitoring system to remove all air.
   a. Remove pressure monitoring kit from package, open blood salvage reservoir (if present), tighten connections, close roller clamp, turn stopcock OFF to patient (off toward distal end of the catheter), and remove vented stopcock caps.
   b. Vent all air from the IV bag.
   c. Invert bag (orient upside down) and using sterile technique insert spike into IV bag.
   d. Leave the spiked bag upside down, open roller clamp, and simultaneously activate the fast-flush device continuously while gently applying pressure to IV bag to slowly clear air from the IV bag and drip chamber. Completely fill the drip chamber with IV fluid.
   e. Turn the IV bag upright once the fluid is sufficiently past the drip chamber.
   f. Apply gentle pressure (50 mm Hg or hang the IV bag approximately 30 inches above distal end of tubing) and activate fast-flush device, advance fluid, priming the tubing.
   g. Orient the blood collection device (if using a closed-system line) so that all air is removed by the advancing fluid (tilt distal end up 45°).
   h. Complete flushing the line and all stopcocks.
   i. Inspect line for any air bubbles.
   j. Rocket Flush (never perform when the system is attached to a patient)
   1. Turn the stopcock off to the distal end of the catheter
   2. Attach 10 mL syringe to the stopcock near the transducer using sterile technique and slowly withdraw 10 mL of IV fluid into the syringe.
   3. Turn the stopcock off to the transducer.
   4. Flush the pressure line quickly with 10 mL isotonic sodium chloride solution from the syringe to remove any remaining air bubbles; avoid instilling any air into the line.
   5. Turn the stopcock off and remove the syringe.
   6. Cap the stopcock with a solid cap using sterile technique.
4. Inspect the line, remove any remaining air by flushing the line with the fast-flush. and repeat the Rocket Flush.
5. Place IV bag into pressure bag and inflate to 250 to 300 mm Hg and recheck for air in the line.

**Figure 2:** A pressure waveform reflects the summation of a series of sine waves or harmonics.

**Analog Versus Digital Data**

An assumption of invasive pressure monitoring is that the measured pressure (eg, pulmonary artery end diastolic pressure [PAEDP]/
PAOP) is an indicator of atrial and ventricular distending pressure (transmural pressure). To meet this assumption, cardiac pressures are measured at end-expiration when juxtacardiac pressure is close to 0 mm Hg. The most accurate method for correctly identifying end-expiratory pressure is data interpretation from the analog (hard copy) strip with a corresponding electrocardiogram (ECG), followed by the stop-cursor method, with digital data from the monitor being the least reliable and accurate method. For example, with digital data, the PAEDP may be incorrectly identified as the lowest pressure on the PA waveform rather than the pressure immediately before the systolic upstroke and without controlling for ventilatory-induced changes in pressure. (Note: To accurately identify the PAEDP, the pressure should be measured 0.08 seconds after the onset of the QRS complex from a simultaneously recorded ECG strip.) The decrease in accuracy for digital values compared to analog data is important when deciding whether to automate the downloading of hemodynamic data into a clinical information system.

**Lung Zones**

One of the first questions to ask when determining if the PAOP accurately reflects left atrial/ventricular pressures is if the PA catheter tip is in a Zone III vascular field (pulmonary arterial pressure [Pa] > pulmonary venous pressure [Pv] > pulmonary alveolar pressure [Pa]). In a Zone III vascular bed during diastole, there is a continuous column of blood between the PA catheter and the left heart; thus end-diastolic pressures measured by the PA catheter reflect end-diastolic left atrial and ventricular pressures (ie, preload). Clinical clues to detect if the catheter is not in a Zone III vascular bed are summarized in Table 2. Monitoring for non-Zone III catheter tip placement should be undertaken whenever there is an increase in alveolar pressure (PEEP) and/or a decrease in intravascular volume. One technique to increase the likelihood of a Zone III vascular measurement is to laterally rotate the patient such that the catheter tip is below the left atrium. For example, in a patient with the PA catheter in the right pulmonary artery, positioning the patient in the right lateral position will place the catheter tip below the left atrium. PA pressure measurements obtained in the 30° and 90° lateral positions are comparable to supine values as long as an angle-specific reference is used.

**Respiratory Variation**

Active exhalation may increase end-expiratory pressure. An increase in end-expiratory pressure and the overestimation of the PAOP by as much as 10 mm Hg should be suspected if there is a greater than 10 to 15 mm Hg respiratory-induced fluctuation in the PAOP. With marked respiratory variation, the PAOP measured at the mid-point between end-expiration and the end-inspiratory (nadir) pressure is independent of the degree of respiratory variation and is a better estimate of left ventricular pressure than the end-expiratory value (Figure 3).

Different mechanical ventilator strategies may also affect PA pressure measurements. For example, inverse ratio ventilation, which decreases end-expiratory time and increases end-expiratory lung volumes, may cause an overestimation of the PAOP. In this case, use of the airway pressure waveform may help to identify the end-expiratory phase and consideration should be given to the need to correct for PEEP and auto-PEEP. With airway pressure release ventilation (APRV), the PAOP should be measured at the end of the positive pressure plateau, which can be observed on the ventilator and is the point immediately before release of airway pressure and the initiation of inspiration. The addition of an airway pressure signal to the analog tracing may also improve the accuracy of pressure interpretation.
Intraabdominal hypertension (IAH) (intraabdominal pressure [IAP] >15 mm Hg), which increases pleural pressure, is present in approximately 20% of critically ill patients on admission.\textsuperscript{15-17} Intraabdominal hypertension decreases venous return, cardiac output, and ventricular compliance and increases intrathoracic and pleural pressures, causing an artifactual increase in RAP and PAOP, despite a decrease in transmural filling pressures and preload.\textsuperscript{16,17} Possible solutions for interpreting hemodynamic pressure data in the presence of intraabdominal hypertension (if resolution of IAH is not possible) include correction for the effect of the IAP on pleural pressure (approximately 60% to 70% of the IAP is transmitted to the pleural space),\textsuperscript{48} the use of volumetric measurements or the use of functional hemodynamic indices.\textsuperscript{49} The correction for increased IAP may not be necessary if the IAP is <15 mm Hg; although, further studies are needed in this area.

Finally, position induced changes in intrathoracic pressure (eg, Trendelenburg) have lead to the misinterpretation that head down position is beneficial for patients with decreased blood pressure.\textsuperscript{50} In a study\textsuperscript{51} of patients placed in a 30° Trendelenburg position, the RAP increased from 9 to 12 mm Hg and the PAOP from 8 to 11 mm Hg, despite a relatively small increase (20 to 40 mL/m\textsuperscript{2}) in intrathoracic blood volume. The increase in the RAP and PAOP was due primarily to a position-induced increase in intrathoracic pressure and not an increase in cardiac volume. Failure to recognize this artifactual increase in RAP or PAOP may lead to inadequate resuscitation.

**Position**

The practice of positioning the patient flat/supine for PA pressure measurements continues despite numerous well-designed studies demonstrating clinically insignificant changes in PA pressures in a variety of patient populations in the supine and backrest elevated position between 30° to 60°.\textsuperscript{52} One argument frequently cited as a rationale for using the supine position is that for cardiac patients, the initial pressure measurements were obtained in the cardiac catheterization lab where the patient was supine; and for comparison, all pressures should be standardized to this position. The following questions should be addressed in informing clinical practice related to patient positioning for hemodynamic measures: (1)
Are there studies in a given patient population (heart failure, ARDS, sepsis, cardiac surgery) that describe the differences in hemodynamic indices (eg, PA pressures, cardiac output) in the supine versus backrest elevated position or supine versus lateral position? (2) Are there physiologically important changes that occur with repositioning from head of bed elevated to the supine position? For example, what are the clinical consequences of the increased orthopnea observed in patients with heart failure in the supine position? (3) Are the observed pressure differences in the supine compared to head of bed elevated or lateral position greater than the normal variability of the pressures given the patient’s underlying ventricular function? (Normal ventricular function: PAS/PAEDP ± 5 mm Hg; PAOP ± 4 mm Hg; left ventricular dysfunction: PAS ± 7 mm Hg or < 8%, PAEDP ± 6 mm Hg or < 11%; PAOP ± 5 mm Hg or <12%.) A decision-making algorithm outlines a process to systematically evaluate a patient’s hemodynamic response to various positions (Figure 4.)

Proper positioning is used to treat patients with acute respiratory distress syndrome (ARDS). Hemodynamic measurements are most often obtained with the patient in the supine position, which may be the most physiologically unstable position for these patients. The patients are subsequently rotated to the prone position for prolonged periods of time, and therapeutic decisions may be made based on the supine measurements. In several studies, there were no significant differences in PAOP, RAP, or CO measured 30 to 60 minutes after rotation from supine to prone on standard hospital or air-suspension beds. A concern with proning is the potential negative effect of abdominal compression and increased intraabdominal pressure. In non-ARDS medical and surgical patients, proning caused a decrease in venous return and thus cardiac output, despite an artifactual increase in measured PA and RAP pressures. However, these findings have not been found in ARDS patients, as demonstrated in a study of the hemodynamic response to manual proning on an air flotation mattress without relief of abdominal pressure. In this study, pressure measurements were obtained 60 minutes after the position change (supine [S]/prone [P]). The intrathoracic pressure increased from S: 10 ± 3 mm Hg to P: 13 ± 4 mm Hg (NS), the MAP increased from S: 75 ± 10 mm Hg to P: 81 ± 11 mm Hg (P = .05) and CI increased from S: 3.8 ± 0.9 L/min/m² to P: 4.2 ± 0.6 L/min/m² (P < .05). There was no significant change in heart rate (S: 78 ± 16 bpm; P: 82 ± 16 bpm), RAP (S: 16 ± 5 mm Hg; P: 15 ± 5 mm Hg), or intrathoracic blood volume (S: 1008 ± 187 mL/m²; P: 1036 ± 180 mL/m²).

This study, and a second study with similar results, are important as they demonstrate a minimal increase in intraabdominal pressure and no significant change in intrathoracic volume in the prone position; both of which are factors that affect PA pressures. Areas that require further exploration are the time for stabilization of hemodynamic pressures after proning, the effects of proning patients with preexisting intraabdominal hypertension on PA pressures, the effect on PA pressures of proning beds that encase the patient with padding and may increase thoracic/abdominal pressure (eg, RotoProne Bed, KCI, San Antonio, TX), and methods to obtain hemodynamic measurements in patients undergoing combined proning and kinetic therapy.

**Clinical Presentation and Cardiac Index/PAOP**

Many critical care nurses are taught a general assessment of the patient’s perfusion status (cold/warm) and pulmonary congestion (wet/dry), but may not be aware of the exact relationship between this clinical characterization and the patient’s cardiac index and PAOP. In 1977, the clinical subsets (ie, Forrester subsets) for patients with acute myocardial ischemia/infarction were described. According to the subsets (Figure 5), a cardiac index (CI) ≤2.2 L/min/m² is consistent with clinical hypoperfusion (hypotension, tachycardia, confusion, oliguria, and cyanosis) and a PAOP >18 mm Hg is consistent with pulmonary congestion (crackles, abnormal chest radiograph). A key point regarding the CI and PAOP cutoff points is that they were derived in patients with an acute myocardial infarction, who most likely had an intact alveolar capillary membrane and normal colloid oncotic pressure. The importance of these factors with regard to hypoperfusion and pulmonary congestion is explained by the Starling equation for fluid flux:

\[
Q = k [P_{\text{cap}} - P_{\text{int}}] - \sigma [\pi_{\text{cap}} - \pi_{\text{int}}]
\]

where:

- **Q**: Blod flow
- **P_{\text{cap}}**: Capillary hydrostatic pressure
- **P_{\text{int}}**: Interstitial hydrostatic pressure
- **\pi_{\text{cap}}**: Capillary oncotic pressure
- **\pi_{\text{int}}**: Interstitial oncotic pressure

The Starling equation describes how blood flow is determined by the balance between the hydrostatic and oncotic pressures within the capillary and interstitial spaces. In chronic heart failure or sepsis patients, the Starling forces favor fluid retention, which explains the observed changes in blood flow and hemodynamics.
where $Q$ is the outward flow of fluid across the capillary membrane, $k$ is the filtration coefficient (reflecting membrane permeability), $P_{\text{cap}}$ and $P_{\text{int}}$ are the intravascular and interstitial hydrostatic pressure, $\sigma$ is the reflection coefficient for proteins and $\pi_{\text{cap}}$ and $\pi_{\text{int}}$ are the intravascular and interstitial colloid osmotic pressures. In critically ill patients, such as those with acute respiratory distress syndrome or septic shock, the alveolar capillary membrane may be damaged, which affects the filtration coefficient, and the colloid osmotic pressure may be decreased. In addition, in ARDS or any condition that increases pulmonary venous resistance, the PAOP may underestimate the pulmonary capillary pressure ($P_{\text{cap}}$) by 6 to 8 mm Hg. A rough approximation is that the $P_{\text{cap}}$ is greater than the PAOP by approximately 40% of the difference between the mean PA pressure and the PAOP ($P_{\text{cap}} = PAOP + 0.4[PAEDP – PAOP]$) or 2/3 of the difference between the PAEDP and PAOP ($P_{\text{cap}} = PAOP + 0.66[PAEDP-PAOP]$). Thus, under conditions, such as ARDS or septic shock, hydrostatic pulmonary edema may occur at a lower intravascular pressure (ie, PAOP < 18 mm Hg). In addition, in septic shock, myocardial contractility may be also decreased despite a normal or increased CI. Thus, in ARDS and septic shock, the PAOP and CI values must be interpreted with an understanding of the contrast in pathophysiology compared to an acute MI.

### Functional Hemodynamic Monitoring

The RAP and PAOP are the traditional preload indices used to guide decisions regarding fluid volume therapy. Various recommendations have been set regarding optimal filling pressures. For example, in the initial volume resuscitation phase of septic shock, a PAOP between 12 to 15 mm Hg is recommended. However, several assumptions must be met if the RAP or PAOP are to be used as indicators of end-diastolic volume (Table 3). The primary assumption (pressure = volume) is problematic because the relationship between PAOP and left ventricular end-diastolic volume is curvilinear and different for each individual; thus, neither an absolute PAOP nor a change in PAOP is reflective of an absolute end-diastolic volume. Given the required assumptions to establish a relationship between pressure and volume, it is not surprising that there is a poor relationship between RAP/PAOP and CI/stroke volume (SV). Although measurement of the PAOP remains an indicator of the patient’s risk for the development of hydrostatic pulmonary edema, changes in more direct volumetric indices (eg, stroke volume variation, right and left ventricular end-diastolic volume, and intrathoracic blood volume) have a better relationship to changes in CI or SV, and may be better measures of preload.

Functional hemodynamic indices have been suggested to better predict which patients will respond to fluid challenges. These dynamic measurements provide insight into the effect of changes in intrathoracic pressure on cardiac function. To understand why dynamic measurements may be more accurate indicators of preload dependence than static indices (RAP/PAOP), a review of the relationship between ventilatory-induced changes in intrathoracic pressure and right- and left-heart SV is provided.

During spontaneous inspiration, pleural and intrathoracic pressures decrease, with a resultant decrease in RAP. With a decrease in RAP, which is the back pressure to venous filling, venous return increases transiently. This increase in venous return results in an inspiratory increase in right ventricular (RV) preload and output (assuming the right ventricle is on
the steep portion of the ventricular function curve). However, if the right ventricle cannot further dilate (eg, RV failure), the RAP will not decrease during inspiration, which indicates that the right atrium/ventricle are on the flat portion of the cardiac function curve, and the administration of additional volume will not increase RV output (nonresponder).\textsuperscript{81,82}

### Table 3: Assumptions Underlying Use of Pulmonary Artery Occlusion Pressure as Indicator of End-Diastolic Volume

<table>
<thead>
<tr>
<th>Assumption</th>
<th>Examples of Factors That Negate Assumption</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. ΔPressure = ΔVolume</td>
<td>Pressure-volume relationship is curvilinear Alteration in ventricular compliance • Myocardial ischemia/infarction • Position on the ventricular function curve (steep vs flat portion) • Intropic drugs • Cardiac tamponade/effusion</td>
</tr>
<tr>
<td>2. PAOP = LAP</td>
<td>Pulmonary venous obstruction • Atrial myxoma • Pulmonary venous thromboembolism</td>
</tr>
<tr>
<td>3. LAP = LVEDP</td>
<td>• Mitral stenosis • Decreased LV compliance</td>
</tr>
<tr>
<td>4. Measured pressure = transmural pressure (intrathoracic pressure = 0 mm Hg)</td>
<td>• Increased intrathoracic pressure (PEEP or auto-PEEP) • Increased intraabdominal pressure causes increase in intrathoracic pressure</td>
</tr>
</tbody>
</table>

PAOP, pulmonary artery occlusion pressure; LAP, left atrial pressure; LVEDP, left ventricular end diastolic pressure; PEEP, positive end expiratory pressure.

### Table 4: PAOP and RAP Values in Responders and Nonresponders

<table>
<thead>
<tr>
<th>Indicator</th>
<th>Responder</th>
<th>Nonresponder</th>
<th>P</th>
</tr>
</thead>
<tbody>
<tr>
<td>RAP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Critically ill sepsis/cardiac\textsuperscript{90}</td>
<td>5 ± 1</td>
<td>5 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>Critically ill patients\textsuperscript{109}</td>
<td>9 ± 4</td>
<td>8 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>Sepsis/septic shock\textsuperscript{77}</td>
<td>9 ± 3</td>
<td>9 ± 4</td>
<td>NS</td>
</tr>
<tr>
<td>PAOP</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Critically ill sepsis/cardiac\textsuperscript{90}</td>
<td>8 ± 1</td>
<td>7 ± 2</td>
<td>NS</td>
</tr>
<tr>
<td>Critically ill patients\textsuperscript{109}</td>
<td>10 ± 4</td>
<td>10 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>Trauma patients\textsuperscript{110}</td>
<td>16 ± 6</td>
<td>15 ± 5</td>
<td>NS</td>
</tr>
<tr>
<td>Septic shock\textsuperscript{90}</td>
<td>10 ± 4</td>
<td>12 ± 3</td>
<td>NS</td>
</tr>
<tr>
<td>Postcardiac surgery\textsuperscript{111}</td>
<td>12 ± 2</td>
<td>16 ± 3</td>
<td>&lt;.01</td>
</tr>
<tr>
<td>Sepsis/septic shock\textsuperscript{77}</td>
<td>10 ± 3</td>
<td>11 ± 2</td>
<td>NS</td>
</tr>
</tbody>
</table>

PAOP, pulmonary artery occlusion pressure; RAP, right atrial pressure; NS, not significant.
During positive pressure mechanical ventilation, the inspiratory increase in intrathoracic pressure decreases venous return to the heart and increases RV afterload (Figure 6). These changes lead to a decrease in RV stroke volume during inspiration. The decreased RV output causes a decrease in left ventricular (LV) preload, which after a few heartbeats, decreases LV stroke volume, usually during expiration. Thus, the LV stroke volume increases during inspiration due to compression of the pulmonary bed and decreases during expiration, primarily due to decreased RV output.83–85 Observation of the ventilatory-induced changes in SV can be exploited, based on the finding that RV preload and SV changes are greater when the ventricle is on the steep versus the flat portion of ventricular function curve.86–88

The increased RV output is transmitted to the left heart, and if both ventricles are preload dependent, the increased LV preload will be observed as a cyclic change in LV stroke volume. The assumption underlying the interpretation of the cyclic SV changes is that a greater cyclic change is indicative of preload dependence (ie, a patient that will respond to volume loading with an increase in SV), whereas a smaller SV change indicates preload independence. Patients who are preload independent will not increase their SV in response to volume loading and may in fact be compromised by the excess fluid. The cyclic changes in LV stroke volume are particularly important, as the SV is a primary contributor to the systolic blood pressure (SBP) and pulse pressure (PP); thus, variations in SBP, PP, or SV may indicate preload dependence or independence.

**Respiratory Variation in Right Atrial Pressure**

Although the absolute RAP has not been found to be predictive of which patients will respond to a volume challenge,27,81 the inspiratory change in RAP (ΔRAP) may be a useful predictor. In medical and cardiac surgery patients who demonstrated an adequate spontaneous inspiratory effort (defined as an inspiratory decrease in PAOP >2 mm Hg), a

![Figure 6: Hemodynamic effects of mechanical insufflation. The left ventricle (LV) stroke volume is maximal at the end of inspiratory period and minimum 2 to 3 heartbeats later during the expiratory period. The cyclic changes in LV stroke volume are mainly related to the expiratory decrease in LV preload due to the inspiratory decrease in RV filling and output. Reprinted with permission from Michard F, Teboul JL. Using heart-lung interactions to assess fluid responsiveness during mechanical ventilation. *Critical Care*. 2000;4(5):282.](image-url)
spontaneous inspiratory decrease in RAP >1 mm Hg is a positive response (responder), whereas a decrease <1 mm Hg is a negative response (nonresponder) (Figure 7). For example, in response to a 250 to 500 mL saline fluid bolus, 16 of 19 patients in the responder group demonstrated an increase in cardiac output of greater than 250 mL. Conversely, only 1 of 14 patients in the nonresponder group demonstrated an increase in cardiac output. Of note, there were no differences in the baseline RAP, PAOP, or cardiac output between the 2 groups (ie, these values did not aid in determining which patients would or would not respond to volume loading). Similar results were observed in a second study involving postcardiac surgery patients. The authors of these studies suggest that the particular value of the ΔRAP is in identifying patients with low cardiac output who will not respond to fluid volume expansion, thus avoiding potentially deleterious volume overload. An advantage of the ΔRAP, unlike the other functional indices discussed in the following sections, is that it can be measured in a spontaneously breathing patients, rather than requiring that the patient be mechanically ventilated and heavily sedated and/or paralyzed.

**Respiratory Variation in Arterial Systolic Pressure**

With positive pressure inspiration, the RV stroke volume decreases during inspiration. After several beats, the decrease in RV stroke volume is transmitted to the left heart, with a subsequent decrease in LV stroke volume. Stroke volume affects SBP; thus, the ventilatory-induced change in SV may be observed as a change in SBP. In mechanically ventilated patients, the systolic pressure variation (ΔPs) is normally 8 to 10 mm Hg (Figure 8). The ΔPs is described as an absolute value (mm Hg) or a percentage (ΔPs%), which is described by the following equation:

\[
\Delta P_s\% = 100 \times (P_{max} - P_{min})/(P_{max} + P_{min})/2\]

The ΔPs is equivalent or more sensitive to volume-induced changes in CI than the PAOP. For example, in a group of a ventilated patients (VT 6 to 12 mL/kg) with acute circulatory failure related to sepsis, the ΔPs% was significantly higher in responders (15 ± 5%) than nonresponders (6 ± 3%). In another group of patients undergoing abdominal aortic surgery, a ΔPs >12 mm Hg was only observed in patients with overt hypovolemia. In one patient in this study, during the postoperative period, the ΔPs increased to >10 mm Hg, which led to the suspicion of intraabdominal hemorrhage and subsequent return to the operating room. In contrast, in a study of cardiac surgery patients, although the ΔPs was greater in responders (8.2 ± 3.9 mm Hg) than nonresponders (5.3 ± 2.6 mm Hg), only a pulmonary artery occlusion pressure (PAOP) <10 mm Hg was predictive of volume response. Note in this latter study that although the responders had a higher ΔPs, the ΔPs did not exceed 10 mm Hg; thus, the lack of predictive ability would be expected.

The ΔPs may also be a useful indicator of blood loss, particularly occult blood loss that occurs before changes occur in the heart rate or blood pressure. In a study of mechanically ventilated patients (VT = 10 mL/kg) who had 500 and 1000 mL of blood phlebotomized, the ΔPs increased from 9.5 ± 4.6 mm Hg (ΔPs% = 9.1 ± 5.3%) at baseline to 14.3 ± 6.5 mm Hg (ΔPs% = 15.2 ± 7.5%) at 500 mL blood loss, and 19.6 ± 7.5 mm Hg (ΔPs% = 21.2 ± 13.1%) at 1000 mL blood loss. In this study, a ΔPs of 5 mm Hg or less was considered indicative of an absence of hypovolemia. Similar results were observed in cardiac surgery patients on mechanical ventilation (VT = 8 mL/kg) who were phlebotomized 500 mL over 10 minutes. In this study, the ΔPs increased from 14 ± 6 mm Hg at baseline to 18 ± mm Hg postbleed. That is, an increase in the ΔPs of approximately 4 mm Hg was indicative of a significant blood loss.
and in all patients whose blood loss exceeded 20% of their circulating volume the ΔPs exceeded 15 mm Hg.82

Questions remain if the ΔPs solely reflects a change in SV or whether other factors (eg, lung and chest wall compliance, transmural pressure, and tidal volume) contribute to the observed change.91,95,96 However, if these factors are kept constant, the ΔPs may be a useful indicator of preload dependence and occult hemorrhage.

**Respiratory Variation in Arterial Pulse Pressure**

Arterial pulse pressure is the difference between the arterial systolic and diastolic pressure. Three factors affect the pulse pressure: LV stroke volume, arterial resistance, and arterial compliance. Of note, the latter 2 factors do not change enough during a single breath to change the beat-to-beat pulse pressure82,87; therefore, the beat-to-beat changes in pulse pressure reflect changes in LV stroke volume. Additionally, unlike the SBP, which is affected by pleural pressure changes, the pulse pressure is affected only by the SV, as the pleural pressure affects the systolic and diastolic pressure equally.

Pulse pressure variation (ΔPp) is the variability in the difference in the pulse pressure during mechanical ventilation as defined by the following equation:98

\[
\Delta Pp\% = \frac{[(P_{max} - P_{min})/(P_{max} + P_{min})/2]}{\times 100}
\]

In a study77 of patients with septic shock on mechanical ventilation (Vt = 8 to 12 mL/kg), a ΔPp of 13% of the baseline pulse pressure (eg, if the baseline pulse pressure = 40 mm Hg, a 13% change is equal to approximately 5 mm Hg) discriminated between responders and nonresponders (CI increased >15%) to a 500 mL colloid bolus with 94% sensitivity and 96% specificity, and was a more sensitive indicator than a change in ΔPs, PAOP, or RAP. An important finding in this study was that the greater the ΔPp before volume expansion, the greater the CI response to the fluid bolus. After volume expansion, the ΔPp decreased, indicating less preload dependence. This latter finding indicates that the change in the ΔPp from before to after a fluid bolus may be useful to determine if the patients requires additional volume expansion.

There are limitations to the use of functional measurements. The ΔPs and ΔPp can be determined only in patients who are on controlled ventilation and deeply sedated and/or paralyzed. Changes in tidal volume and pulmonary compliance will alter the magnitude of the response. Most of the studies have been conducted with tidal volumes (10 mL/kg). Research is ongoing to evaluate the sensitivity and specificity of these indices under conditions of lower tidal volumes (6 mL/kg) as demonstrated in Figure 8. Significant cardiac dysrhythmias may negate the utility of these indices and a majority of the studies were conducted in patients with relatively normal ventricular function; thus, recommendations for patients with decreased right or left ventricular function are limited.85

**Stroke Volume Variation**

A change in LV stroke volume is the primary factor that affects beat-to-beat changes in pulse pressure. The LV stroke volume also affects the aortic flow, which can be observed as beat-to-beat changes in SV. Stroke volume variation (SVV), which can be continuously measured using pulse contour analysis (a new technique using a specialized transducer placed in the femoral, brachial, or axillary artery), is defined as the change in SV over a 30-second period:

\[
SVV = \frac{SVV_{max} - SVV_{min}}{SVV_{mean}}
\]

The assumption underlying SVV is that the observed SV changes are respiratory-induced variations. As with other volumetric measurements, the SVV is more closely associated with changes in SV than are changes in the PAOP and RAP.99,100

The SVV is predictive of fluid response in a various patient populations. In patients undergoing brain surgery (Vt = 10 mL/kg), a SVV of 9.5% discriminated between responders and nonresponders (defined as an increase in SV ≥5% in response to a 100 mL colloid bolus), with a sensitivity of 79% and a specificity of 93%.101 In cardiac surgery patients (Vt = 13 to 15 mL/kg), SVV decreased from 11.8 ± 7.5% to 5.4 ± 4.2% after a bolus of 500 mL colloid, and was correlated with the change in CI (ΔSVV r = −0.64, P <.005).102 In another group of off-pump bypass surgery patients with preserved left ventricular function, the SVV and ΔPp were the most sensitive and
Figure 8: (Continues).
specific indices of fluid volume responsiveness. Additionally, unlike other functional (ΔPs) or volumetric indices (intrathoracic blood volume), in patients with decreased left ventricular function (EF < 35%), SVV was related to changes in SVI, although no predictive cut-off value has been identified.

Concerns regarding the measurement of SVV include the method used (direct measurement versus pulse contour analysis). Because the technology to perform SVV analysis is proprietary and it has changed over the past few years, comparison of results from the various methods is difficult. Additionally, caution must be taken when interpreting absolute predictive values, as the SVV% varies depending on the tidal volume. For example, the SVV before volume loading for 3 different tidal volumes were significantly different (SVV 5 mL/kg = 7 ± 0.7%; SVV 10 mL/kg = 15 ± 2%, SVV 15 mL/kg = 21 ± 2.5%); thus, interpretation of the sensitivity and specificity of exact cutoff point can only be performed in the context of a standardized tidal volume.

To achieve a stable tidal volume, SVV analysis can be performed only in patients who are on controlled mechanical ventilation and are heavily sedated/paralyzed.

Clinical Example
You are caring for a patient with fibrotic lung damage due to chemotherapy and radiation and pulmonary tumor metastasis complicating ARDS and septic shock. The patient is mechanically ventilated (VT = 7 mL/kg, PEEP = 12 cm H2O) and sedated. SBP: approximately 80 mm Hg; PAOP: 16 mm Hg; CI: 2.4 L/min/m2, and SaO2 89%. Overnight, the interpretation of the patient’s hemodynamic status was challenging, given the potential artifactual increase in PAOP and optimizing the patient’s preload without causing hydrostatic pulmonary edema. The challenging clinical interpretation resulted in the patient being treated first with fluids, followed by diuretics in attempt to increase his blood pressure and cardiac output without compromising his tenuous pulmonary status. Currently, his ΔPs is 4 mm Hg. Should this patient be given a fluid bolus to improve his SBP and CI? Answer: No. The ΔPs indicates that the patient will not respond to fluids. In this case, 2 decisions were made. First, the altered pulmonary status was primarily related to the underlying pulmonary disease, which could not be resolved. Adjustments to the ventilatory parameters would be used to optimize the patient’s oxygenation. Second, if the patient
demonstrated signs of hypoperfusion, vasoactive/inotropic agents would be used to treat the low blood pressure/CI. Diuresis would not be appropriate, as the corrected PAOP (approximately 14 mm Hg) was not excessively high and a diuresis-induced decrease in intravascular volume in conjunction with the high levels of PEEP would potentially decrease cardiac output.

References


