

# Arterial, Central Venous, and Pulmonary Artery Catheters

Jean-Louis Vincent

## CHAPTER OUTLINE

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### CENTRAL VENOUS CATHETERS

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Pulmonary Artery Occlusion Pressure and Partial Occlusion

Does the Use of a Pulmonary Artery Catheter Improve Outcome?

## ARTERIAL CATHETERS

### WHAT DO THEY OFFER?

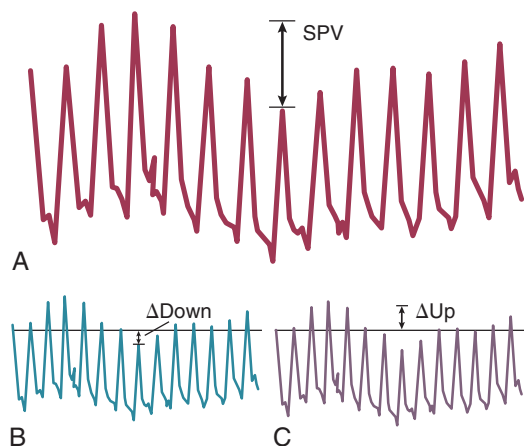
The placement of an arterial catheter permits (1) reliable and continuous monitoring of arterial pressure and (2) repeated blood sampling. Analysis of the arterial pulse pressure curve may also have other applications, including assessment of fluid responsiveness and estimation of cardiac output. The appearance of arterial pressure waves will vary according to the site at which the artery is sampled. As the arterial pressure wave is conducted away from the heart, three effects are observed: The wave appears narrower; the dicrotic notch becomes smaller; and the perceived systolic and pulse pressures rise and the perceived diastolic pressure falls.

### ARTERIAL PRESSURE MEASUREMENT

The optimal range of arterial pressure depends on individual patient characteristics, on underlying diseases, and also on treatment. Hence, it is impossible to give an optimal range of arterial pressure that is applicable in all patients. When arterial pressure needs to be evaluated accurately, oscillometric measurements become unreliable,<sup>1</sup> and insertion of an arterial catheter is indicated.

Four potential indications for insertion of an arterial catheter for measurement of arterial pressure are recognized:

1. *Hypotensive states associated with (a risk of) altered tissue perfusion.* Hypotension that is resistant to fluid administration requires the administration of vasopressor agents, and invasive measurement of arterial pressure is then necessary to titrate this form of therapy. Norepinephrine is the vasopressor agent most commonly used in this setting. A mean arterial pressure (MAP) of 65 to 70 mm Hg is usually targeted, but this level must be adapted to the individual patient and the clinical scenario; in particular, elderly patients with atherosclerotic disease may require higher levels than younger individuals with normal arteries.
2. *Intravenous vasodilator therapy.* Vasodilating therapy (e.g., nitrates and hydralazine) is a mainstay in the management of heart failure, because it can increase cardiac output. Close monitoring of arterial pressure is essential to avoid excessive hypotension.
3. *Severely hypertensive states.* Extreme hypertension may result in organ impairment, especially of the brain and the heart. Sodium nitroprusside or calcium entry blockers usually are used to lower arterial pressure, and careful, accurate monitoring is essential to titrate the antihypertensive therapy.
4. *Induction of hypertension.* Hypertension is sometimes induced in patients with neurologic diseases. Severe cerebral edema with intracranial hypertension, in particular, requires vasopressor support to maintain cerebral perfusion pressure (the gradient between the MAP and the



**Figure 4.1** The systolic pressure variation (SPV) is the difference between the maximum and the minimum systolic blood pressures during one ventilatory cycle (A). The SPV is the sum of the  $\Delta$ Down (B) and the  $\Delta$ Up (C). (From Perel A, Pizov R, Cotov S: Systolic blood pressure variation is a sensitive indicator of hypovolemia in ventilated dogs subjected to graded hemorrhage. *Anesthesiology* 1987;67:498.)

intracranial pressure); likewise, hypertension may be used to treat or prevent the development of vasospasm secondary to subarachnoid hemorrhage, as part of the so-called triple-H therapy (hypertension, hypervolemia, hemodilution). Norepinephrine usually is used for this purpose.

#### FLUID RESPONSIVENESS

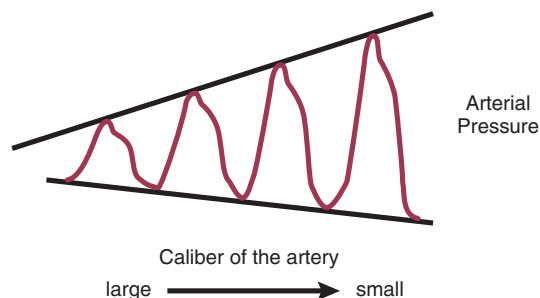
Variations in arterial pressure during positive-pressure ventilation have been used as a measure of fluid responsiveness. The transient increases in intrathoracic pressure influence venous return in patients who are likely to respond to fluid administration. This fluctuation in ventricular filling will translate into fluctuations in arterial pressure a few beats later. Accordingly, the greater the degree of systolic arterial pressure, or pulse pressure, variation during the respiratory cycle, the greater will be the increase in cardiac output in response to fluid administration (Fig. 4.1). However, this observation is valid primarily in patients without spontaneous respiratory movements and without significant arrhythmias, and only when a sufficient tidal volume is applied.<sup>2,3</sup>

#### CARDIAC OUTPUT ASSESSMENT

The pulse contour analysis also can serve to estimate cardiac output less invasively than with the pulmonary artery catheter (PAC). Because the only determinants of arterial pressure are the stroke volume and the resistance and compliance factors of the blood and arteries, analysis of the pulse contour trace can help to monitor cardiac output over time. This can be done with regular calibrations whenever changes in vascular tone or blood volume occur, or even in the absence of calibration. These measurements are still approximations, so further technological developments can be expected to improve accuracy.

#### BLOOD SAMPLING

The presence of an arterial catheter can greatly facilitate blood sampling, especially in terms of enabling easy access



**Figure 4.2** Schematic representation of an arterial pressure tracing showing that the pulse pressure increases as arterial size decreases.

to the circulation for regular monitoring of blood gases, such as may be required in severe respiratory failure or with acute metabolic alterations. Sensors can measure blood gases continuously, but widespread use of such sensors is limited by their cost.

#### ACCESS

For placement of arterial catheters, usually the radial artery is used. The femoral artery can be easily cannulated and gives a better signal, but presence of a femoral catheter interferes more with patient mobility and warrants concern about infection.<sup>4</sup> Use of other sites, such as the brachial or the axillary artery or even the dorsalis pedis artery,<sup>5</sup> can be considered. An important point to keep in mind is that the pulse pressure increases from the core to the periphery. In other words, the systolic pressure is overestimated in smaller arteries (Fig. 4.2). Hence, it may be better to rely more on mean values than on systolic or diastolic pressures.

#### COMPLICATIONS

The most feared complication with use of arterial catheters is ischemia. With any suspicion of ischemia, the catheter must be removed immediately. Allen's test, to determine occlusive arterial lesions distal to the wrist, is unreliable and is no longer widely used. The accidental disconnection of arterial lines can be associated with severe hemorrhage and even exsanguination. Infectious complications are rare.

## CENTRAL VENOUS CATHETERS

### WHAT DO THEY OFFER?

The central venous catheter can facilitate fluid administration. It also allows measurement of the central venous pressure (CVP) and enables access to central venous blood for sampling.

### FLUID ADMINISTRATION

The large-bore central venous catheter allows fluids to be administered fast and reliably in the presence of acute hemorrhage. Placement of a central catheter is therefore essential in patients with hemorrhage due to polytrauma or with other forms of acute bleeding. It also allows irritant or

hypertonic fluids to be administered, such as parenteral solutions, potassium-enriched solutions, and some therapeutic agents. Central venous lines also can be convenient in patients who need prolonged intravenous therapy when peripheral venous access becomes problematic.

#### MEASUREMENT OF CENTRAL VENOUS PRESSURE

CVP is identical to right atrial pressure (RAP) (in the absence of vena cava obstruction) and to right ventricular (RV) end-diastolic pressure (in the absence of tricuspid regurgitation). It is thus equivalent to the right-sided filling pressure. CVP is determined by the interaction of cardiac function and venous return, which is itself determined by the blood volume and the compliance characteristics of the venous system. Hence, an elevated CVP can reflect an increase in blood volume as well as an impairment in cardiac function. Because the CVP evaluates the right-sided filling pressures, CVP can be increased in the presence of pulmonary hypertension, even if left ventricular (LV) function is normal. The normal value in healthy persons is very low, not exceeding 5 mm Hg. Thus, the CVP value may not be much lower than normal in the presence of hypovolemia. In general, a CVP below 10 mm Hg can be considered to indicate that the patient is more likely to respond to fluid resuscitation, but exceptions to this rule exist. A high CVP suggests a certain blood volume but does not guarantee sufficient LV filling.

Clinically, CVP can be assessed by evaluation of the degree of jugular distention<sup>6</sup> or liver enlargement. A single CVP measurement is not very useful and is not a good indicator of a positive response to fluids; an increase in CVP without a concurrent increase in cardiac output is not only useless but also harmful, because it will lead to increased edema formation.

#### ACCESS TO BLOOD IN SUPERIOR VENA CAVA

Measuring the central venous oxygen saturation (ScvO<sub>2</sub>) is a surrogate for measurement of the true mixed venous oxygen saturation (SvO<sub>2</sub>). Although absolute values of ScvO<sub>2</sub> are not identical to single SvO<sub>2</sub> values, trends in ScvO<sub>2</sub> over time follow the same pattern as trends in SvO<sub>2</sub>, making ScvO<sub>2</sub> a useful measure in patients who do not require an arterial catheter.<sup>7</sup> ScvO<sub>2</sub> can be obtained either intermittently (by withdrawal of blood samples) or continuously (with the use of a catheter equipped with fiberoptic fibers).

#### TRACE ANALYSIS

Analysis of the CVP waveform can provide some interesting information. In particular, a large y descent indicates a restrictive cardiac state, but not all restrictive patterns are associated with this finding.

#### ACCESS

The central venous catheter generally is introduced via the internal jugular vein; the subclavian vein also can be used, although the risk of pneumothorax may be somewhat higher with this route. Peripherally inserted central catheters can also be placed, via the cephalic vein, basilic vein, or brachial vein. Introduction of a femoral catheter through the abdominal inferior vena cava to the right atrium can also yield reliable CVP measurements.<sup>8</sup> The use of femoral

catheters, however, is associated with a greater risk of infections and thrombophlebitis.<sup>9</sup>

#### COMPLICATIONS

Complications of central venous catheterization are related primarily to puncture of the central vein: Hemothorax can be life-threatening, especially in the presence of severe respiratory failure. In the presence of unilateral pathology, the catheter must be introduced on the affected side. Arterial puncture resulting in a local hematoma is not uncommon, but hematoma formation usually is without major consequences. Bedside ultrasonography can help guide the introduction of the catheter into the vein. Excessive advancement of a long catheter in a small patient can result in arrhythmias; such arrhythmias have been described with advancement of the catheter tip into the right ventricle, but this problem can be identified by the presence of an RV trace on the monitor display.

Catheter-related infections constitute the major long-term complication. Adherence to basic hygiene guidelines can decrease the incidence of catheter-related sepsis. Triple-lumen catheters may be associated with a higher incidence of catheter-related infection,<sup>10</sup> primarily as a result of increased catheter manipulation. The use of antimicrobial-coated catheters may decrease the risk of infections,<sup>11</sup> but fears remain about the risks of development of resistant organisms.<sup>12</sup> Routine replacement of catheters after 3 to 7 days is not recommended.<sup>13</sup>

### PULMONARY ARTERY CATHETERS

#### WHAT DO THEY OFFER?

PACs allow collection of data on right atrial, pulmonary artery, and pulmonary artery occlusive pressures (Fig. 4.3); flow (cardiac output); and oxygenation (SvO<sub>2</sub>).

#### PRESSURES

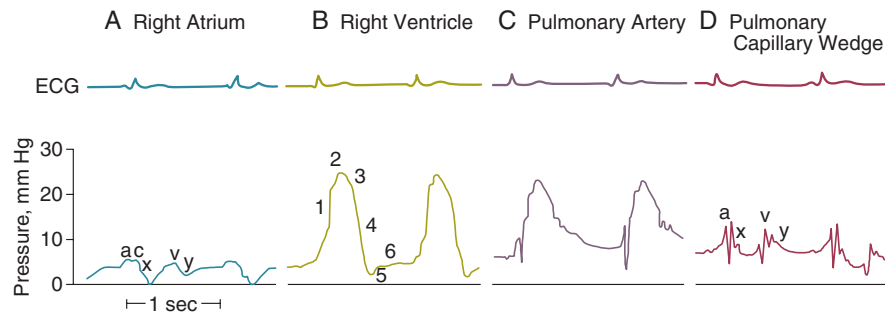
##### Right Atrial Pressure

As indicated earlier, the RAP is identical to the CVP in the vast majority of cases.

##### Pulmonary Artery Occlusion Pressure

When the balloon on the catheter is inflated, it causes an obstruction (becomes wedged) in a small branch of the pulmonary artery, interrupting the flow of blood locally (but blood flow continues normally in the rest of the pulmonary circulation), so that (assuming the absence of an abnormal obstacle) a continuous column of blood is present between the tip of the PAC and the left atrium. This pulmonary artery occlusion pressure (PAOP), or pulmonary artery wedge pressure (PAWP), generally reflects the left atrial pressure well. Nevertheless, a number of steps must be taken to ensure the adequacy of the measurement.

A first question is whether the PAOP reflects the pressure in the pulmonary veins and not the alveolar pressure. The tip of the catheter should be in a West zone III position, where a continuous column of blood exists between the



**Figure 4.3** Pressure waveforms. **A**, The normal right atrial (RA) tracing. The a wave is the RA pressure rise resulting from atrial contraction and follows the P wave of the electrocardiogram (ECG). On simultaneous ECG and RA tracings it usually occurs at the beginning of the QRS complex. The c wave, caused by closure of the tricuspid valve, follows the a wave and is coincident with the beginning of ventricular systole. Atrial relaxation (x descent) is followed by a passive rise in RA pressure resulting from atrial filling during ventricular systole and occurs during the T wave of the simultaneously recorded ECG. The y descent reflects the opening of the tricuspid valve and passive atrial emptying. **B**, The normal right ventricular (RV) tracing. The sharp rise in RV pressure (1) is due to isometric contraction and is followed by a rapid pressure decrease (2) as blood is ejected through the pulmonary valve. This rapid ejection is followed by a phase of more reduced pressure decrease, which is often reflected in a small step in the downslope of the RV pressure waveform (3). The subsequent sharp decline in RV pressure (4) occurs as a result of isometric relaxation and is noted once the RV pressure falls below the pulmonary artery (PA) pressure (with consequent closure of the pulmonary valve). As RV pressure falls below RA pressure, the tricuspid valve opens, and passive refilling (5) of the right ventricle occurs, followed by atrial contraction, causing a biphasic wave of ventricular filling to appear on the RV tracing (6). **C**, The normal pulmonary arterial waveform. A pulmonary artery systolic elevation is caused by ejection of blood from the right ventricle, followed by a decline in pressure as RV pressure falls. As RV pressure falls below pulmonary artery pressure, the pulmonary valve closes, which causes a momentary rise in the declining pulmonary arterial pressure. This is the dicrotic notch characteristic of the pulmonary arterial (and also the systemic arterial) waveform. The pulmonary artery systolic wave usually occurs in synchrony with the T wave of the ECG. Pulmonary artery diastolic pressure (PADP) does not fall below RA pressure and therefore is higher than right ventricular end-diastolic pressure (RVEDP); it is an approximation to left ventricular end-diastolic pressure (LVEDP). **D**, The normal pulmonary artery occlusion pressure (PAOP) waveform. The waveform of the pulmonary capillary wedge pressure (PCWP) is subject to the same mechanical variables as the RA waveform, but because of the damping that occurs through the pulmonary circulation, the waves and descents often are less distinct. Similarly, the mechanical events are recorded later in the cardiac cycle, as seen on the ECG. Thus, the a wave is not seen until after the QRS complex, and the v wave occurs after the T wave of the ECG. The PCWP is a closer approximation to LVEDP than is PADP. (From Grossman W: *Cardiac catheterization*. In Braunwald E (ed): *Heart Disease: A Textbook of Cardiovascular Medicine*, 3rd ed. Philadelphia, WB Saunders, 1992.)

catheter tip and the left atrium (Fig. 4.4). These considerations are less important with fluid optimization and with today's lower positive end-expiratory pressures (PEEPs).

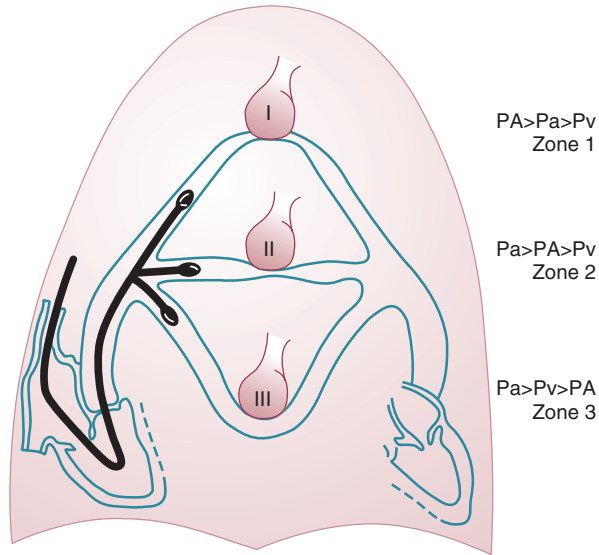
To exclude a possible influence of airway pressure on PAOP readings, the changes in PAOP can simply be compared with the changes in pulmonary artery pressure (PAP) during the respiratory cycle. If PAOP reflects the pressure within the pulmonary veins, these changes should be identical, because the pulmonary artery and vein should be subjected to identical changes in intrathoracic pressures. If, on the other hand, the catheter tip is not in a West zone III, the changes in PAOP will be more significant than the changes in PAP. In these latter conditions, either fluid administration or some reduction in the PEEP level may abolish the differences.

The next question is whether the measured pressure is truly a transmural pressure—that is, the pressure difference between the vascular structures and the environmental structures. In other words, will changes in surrounding intrathoracic pressures influence the pressures measured? In the case of intrathoracic pressures, the changes in pleural pressure are particularly relevant. Hence, all measurements should be performed at end-expiration, when the pleural pressure is closest to zero. A marked fall in intrapleural pressure, as with a Mueller maneuver (forced inspiration against resistance), may dramatically increase the transmural pressures. A more common problem in the intensive care unit (ICU) is the increase in pleural pressure due to

positive-pressure ventilation, sometimes with high PEEP levels.

One method of combating this problem could be to subtract the esophageal pressure from the measured PAOP, but this approach has a number of technological limitations. Simple disconnection of the respirator to measure PAOP after obtaining an equilibration state is not recommended, because the measured PAOP will not represent the real value when PEEP is applied. A better method consists of measuring the lowest (nadir) PAOP, within seconds after a very transient disconnection from the ventilator, to identify the true transmural pressure before a new equilibrium is reached.<sup>14</sup> Such a maneuver suppresses the high intrathoracic pressure and eliminates the influence of the extramural pressure, but the values obtained may not be valid in the presence of intrinsic PEEP. Other methods have been suggested, some relatively sophisticated<sup>15</sup> and others more simple, involving subtraction of approximately one third of the PEEP level from the measured PAOP, for example. The question is whether this is really so important, because absolute PAOP values are not very helpful.

In respiratory failure, PAOP does not represent true capillary pressure, which may be somewhat higher. The true capillary pressure may be estimated from the measurement of the intersection point of the rapid and slow pressure decay curves recorded after a rapid interruption of the blood flow; such measurements of capillary pressure are possible from the pressure trace. This pressure is

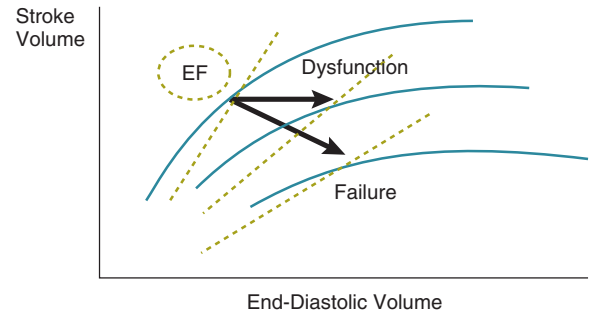


**Figure 4.4** The position of the pulmonary artery catheter tip in relation to the zones of the theoretical lung model described by West. For pulmonary artery occlusion pressure (PAOP) to be a valid estimate of “left heart” filling pressure, a continuous column of blood must exist between the catheter tip and the left atrium. In zones I and II, pulmonary vasculature is either totally or partially compressed by the intra-alveolar pressure, and measurements of PAOP will be misleading. PA, alveolar pressure; Pa, pulmonary artery pressure; Pv, pulmonary venous pressure. (From Marini JJ: Respiratory Medicine and Intensive Care for the House Officer. Baltimore, Williams & Wilkins, 1991.)

well correlated with extravascular lung water in animal experiments. The need to know these values is questionable, however, because the primary goal remains to keep these hydrostatic pressures as low as possible while maintaining adequate cardiac output.

PAOP may not adequately reflect LV end-diastolic pressure. It may be lower in patients with aortic regurgitation or higher in the presence of significant tachycardia or mitral valve disease. LV end-diastolic pressure may not even accurately predict LV end-diastolic volume. This was already demonstrated many years ago with radionuclide techniques.<sup>16</sup> Preload is more directly defined as the end-diastolic volume. A stiff, noncompliant ventricle can result in a relatively high end-diastolic pressure for a given end-diastolic volume. An evaluation of end-diastolic volumes can be obtained less invasively with echocardiographic techniques. Likewise, the use of transthoracic thermodilution techniques allows the estimation of intrathoracic blood volume and global end-diastolic volumes. However, these assessments of end-diastolic volumes do not give additional information about the likelihood of fluid responsiveness.

In sum, then, a given level of cardiac filling pressures does not provide much information about fluid responsiveness, but monitoring can be very helpful to guide a fluid challenge.<sup>17</sup> During fluid administration, the goal is to obtain a significant increase in cardiac output (by the Frank-Starling mechanism), with the least increment in cardiac filling pressures, in order to minimize the risk of edema formation. The goal is not to keep the cardiac filling pressures within predefined arbitrary limits; rather, PAOP is a direct



**Figure 4.5** Ventricular function curve depicting the difference between ventricular dysfunction (decrease in the ventricular ejection fraction [EF] but preservation of stroke volume) and ventricular failure (defined as the inability of the heart to pump enough flow). The EF, the ratio between stroke volume and end-diastolic volume, is represented by the dashed lines; it is decreased in all cases.

determinant of edema formation in the lungs. The key principle is to keep the PAOP as low as possible, provided that all of the other organs are happy.

### Pulmonary Artery Pressures

Normally the pulmonary vasculature is a low-resistance circuit, so that the diastolic PAP should be equal to or only slightly higher than the PAOP. An increased pressure gradient between the diastolic PAP and the PAOP indicates active primary pulmonary hypertension related to pulmonary vascular changes (hypoxia) or diseases (primary pulmonary artery hypertension). Pulmonary hypertension may result in RV dilation with septal shifts that may compromise LV function.

### CARDIAC OUTPUT

The reference method for measurement of cardiac output is use of the Fick equation; however, this equation is difficult to apply in practice. The indicator dilution technique has been used instead. Indocyanine green clearance has been used for many years, but this method is time-consuming and quite difficult to perform. The thermodilution technique developed by Ganz is a convenient technique which today allows the almost continuous measurement of cardiac output. The presence of tricuspid insufficiency is the major limitation to this technique. Other techniques, including transpulmonary and lithium dilution techniques, have been developed but are somewhat less precise.

The thermodilution technique estimates cardiac output over several cardiac cycles, whereas other techniques, including those based on pulse contour analysis, may assess beat-to-beat variations. These techniques may be useful for estimating the influence of changes in intrathoracic pressures on the stroke volume variation, an estimate of fluid responsiveness.

### RIGHT VENTRICULAR VOLUMES

The use of a modified PAC equipped with a fast response thermistor also allows evaluation of the right ventricular ejection fraction (RVEF) (Fig. 4.5). With knowledge of the stroke volume, it becomes easy to calculate the end-systolic and end-diastolic volumes. This measurement can



be particularly useful in the presence of RV failure, but this also is a situation in which the measurement is least reliable: tricuspid regurgitation secondary to pulmonary hypertension.

#### MIXED VENOUS OXYGEN SATURATION

$S\bar{V}O_2$  represents the balance between oxygen consumption and oxygen supply. According to the Fick equation:

$$VO_2 = \text{cardiac output} \times (CaO_2 - CvO_2)$$

where  $VO_2$  is oxygen uptake and  $CaO_2$  and  $CvO_2$  are the arterial and mixed venous oxygen content, respectively. If the dissolved oxygen in the blood is neglected for the purposes of calculation, then

$$VO_2 = \text{cardiac output} \times Hb(SaO_2 - S\bar{V}O_2) \times C$$

and

$$S\bar{V}O_2 = SaO_2 - (VO_2 / \text{cardiac output} \times Hb \times C)$$

where Hb is hemoglobin.

Accordingly, a decrease in  $S\bar{V}O_2$  can reflect either a decrease in  $SaO_2$  (hypoxemia), anemia, or a relative inadequacy of cardiac output in relation to the oxygen demand of the tissues.

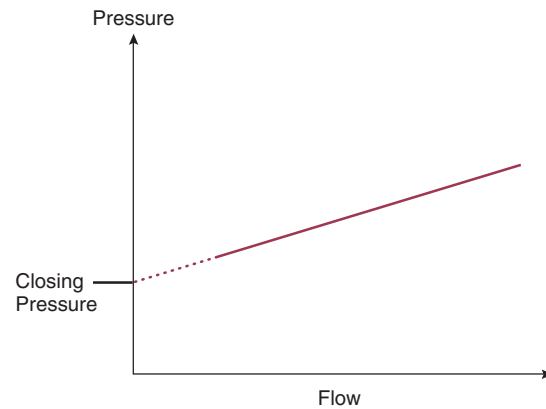
$S\bar{V}O_2$  can be measured continuously using catheters equipped with fiberoptic fibers, and measurements are helpful to guide therapy.  $ScvO_2$  has been proposed as a surrogate for  $S\bar{V}O_2$ , but the relationship between  $ScvO_2$  and  $S\bar{V}O_2$  is rather vague. Indeed, the  $ScvO_2$  is lower than  $S\bar{V}O_2$  in healthy conditions (as a result of the low  $O_2$  extraction by the kidneys) but is higher than  $S\bar{V}O_2$  in critically ill patients (because of relative increase in  $O_2$  extraction in the kidneys and in the gut).<sup>18</sup> Moreover,  $O_2$  extraction is high in the coronary circulation, and this is missed in the measurement of  $ScvO_2$ .

#### DERIVED VARIABLES

Hemodynamic assessment can include a number of derived variables, including resistance, ventricular stroke work, oxygen transport, oxygen consumption, and venous admixture, as described next.

**Resistance.** In steady-flow conditions, Ohm's law indicates that resistance is the ratio between the pressure drop and the flow in the system. In the pulmonary circulation, the inflow pressure would be the mean PAP and the outflow pressure would be PAOP; for the systemic circulation, these would be the MAP and the CVP, respectively. In either case, flow would be cardiac output. This approach is limited, however, by the fact that the extrapolated intercept of the PAP–cardiac output relationship represents the average closing pressure of the small pulmonary arterioles, and the slope represents the upstream arterial resistance. Accordingly, the increased PAP in pulmonary hypertension can be explained by both an increase in pulmonary vascular closing pressure and an increase in vascular tone, and pulmonary vascular resistance (PVR) is not a good reflection of vasomotor tone in the pulmonary vasculature. Pulmonary hemodynamics are therefore best assessed by altering blood flow to better evaluate this relationship (Fig. 4.6).

Calculation of systemic vascular resistance (SVR) is not very helpful either. It is better to base clinical decisions on



**Figure 4.6** Relationship between pressure and blood flow, illustrating the limitations in the calculation of the vascular resistance. Note that the line defining that relationship does not go through the origin, because the pressure is not zero in the absence of blood flow.

primary variables. Simply stated, a relatively high cardiac output in relation to arterial pressure reflects a low SVR state, whereas a relatively low cardiac output reflects a high SVR state.

**Ventricular Stroke Work.** The work developed by the ventricles is determined by the ventricular work as derived from the product of flow and the pressure generated. The relationship between stroke work and the respective filling pressure represents a better assessment of contractility than does the stroke volume. LV stroke work index (LVSWI) can be calculated using the equation

$$LVSWI = SVI(MAP - PAOP) \times 0.0136$$

where SI represents the stroke volume index.

**Oxygen-Derived Variables.** Oxygen transport can be assessed as the product of cardiac output and the arterial oxygen content, according to the equation

$$\begin{aligned} DO_2 &= \text{cardiac output} \times CaO_2 \\ &= \text{cardiac output} \times ([Hb] \times SaO_2 \times 1.39) + 0.0031 \times PaO_2 \end{aligned}$$

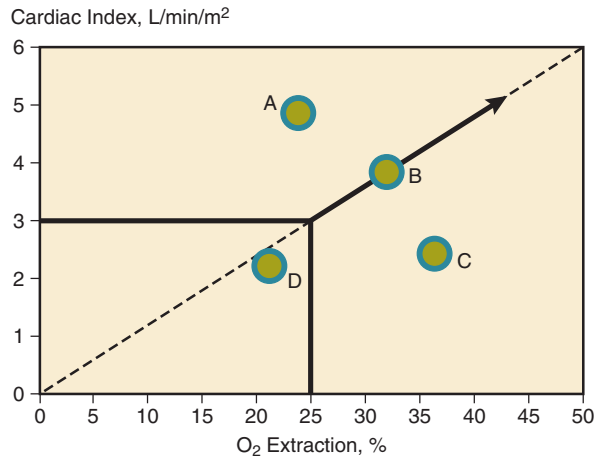
Calculations of this variable benefit from combining measurements of cardiac output, Hb, and  $SaO_2$  but have the limitation that the corresponding oxygen demand is unknown. Some studies suggested that maintaining supra-normal  $DO_2$  in the perioperative period or early after trauma may result in better outcomes.

Oxygen consumption also can be calculated from the product of cardiac output and the arteriovenous oxygen difference, according to the formula

$$VO_2 = \text{cardiac output} \times (CaO_2 - C\bar{v}O_2)$$

where  $C\bar{v}O_2$  is calculated in the same way as for  $CaO_2$ , using the  $SvO_2$  instead of the  $SaO_2$ .

The difficulty is in evaluating the oxygen requirements (or the oxygen demand) of the body.  $VO_2$  assessment may perhaps be useful to evaluate the caloric need of the critically ill patient.



**Figure 4.7** Diagram illustrating the relationship between cardiac index and O<sub>2</sub> extraction. Four typical examples are given: hyperkinetic state (A), anemia (B), low cardiac output state (C), and profound anesthesia (D).

The calculation of oxygen extraction (oxygen extraction ratio, or O<sub>2</sub>ER) is accomplished by determining the ratio of VO<sub>2</sub> to DO<sub>2</sub>, or in a simplified way:

$$\begin{aligned} \text{O}_2\text{ER} &= \text{VO}_2 / \text{DO}_2 = (\text{CaO}_2 - \text{CvO}_2) / \text{CaO}_2 \\ &= (\text{SaO}_2 - \text{SvO}_2) \text{Hb} / \text{SaO}_2 \times \text{Hb} \\ &= (\text{SaO}_2 - \text{SvO}_2) / \text{SaO}_2 \end{aligned}$$

Accordingly, when SaO<sub>2</sub> is close to 100%, in the absence of hypoxemia, O<sub>2</sub>ER mirrors SvO<sub>2</sub>. Its calculation can be useful in the presence of hypoxemia, when SaO<sub>2</sub> is decreased. Also, the relationship between cardiac output and O<sub>2</sub>ER can be helpful to compare the central and the peripheral components of oxygen delivery (Fig. 4.7). This relationship is independent of Hb, so it can be helpful in evaluating the cardiovascular status in the presence of anemia.<sup>19</sup>

**Venous Admixture.** Venous admixture can be calculated by the Berggren equation:

$$\begin{aligned} \text{Qs}/\text{Qt} &= \text{Cc}'\text{O}_2 - \text{CaO}_2 \\ &\quad \text{Cc}'\text{O}_2 - \text{CvO}_2 \end{aligned}$$

in which Qs is shunt flow, Qt is total pulmonary blood flow, and Cc'O<sub>2</sub> represents the capillary oxygen content (assuming an oxygen saturation of 100%). This may help to assess the effects of various interventions (PEEP or other respiratory conditions, administration of vasoactive agents) on pulmonary hemodynamics.

## COMPLICATIONS

Complications of pulmonary artery catheterization can be divided into seven categories as listed in Box 4.1. Many of these can be prevented, and most are relatively uncommon.

Complications of venous access are the same as for the insertion of a central venous catheter. Arrhythmias are

### Box 4.1 Complications of Pulmonary Artery Catheter Insertion

- Complicated vascular access (pneumothorax, hematoma)
- Arrhythmias (e.g., heart block, ventricular tachycardia/fibrillation)
- Catheter knotting
- Pulmonary thrombosis and infarction
- Endothelial or valvular damage
- Colonization and bacteremia
- Pulmonary artery rupture

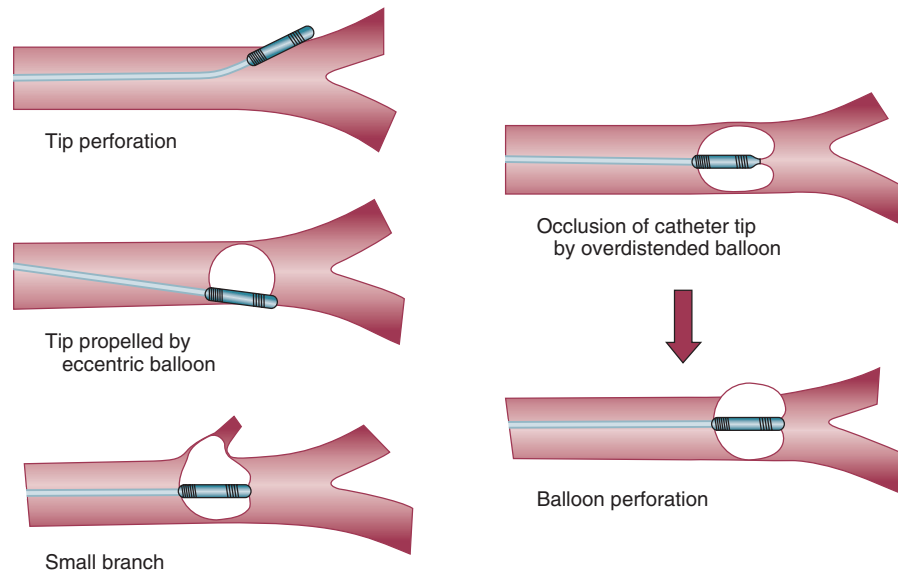
common but usually are without major consequence, except in moribund patients. It has been suggested that lidocaine should be given prophylactically in predisposed patients, but this usually is not necessary. Likewise, complete atrioventricular block may develop in patients with left bundle branch block, but this is exceptional. Knot formation will be rare if the catheter is advanced carefully and if its presence in the pulmonary artery is confirmed before further advancement into the right ventricle. In particular, care should be taken not to advance the catheter by more than 30 to 35 cm into the right ventricle. Thrombotic complications have become rare with the development of heparin-coated catheters. The appearance of an infiltrate beyond the tip of the PAC on the chest film should suggest an evolving thrombotic event, which should lead to consideration of the withdrawal of the catheter. Endothelial lesions have been found at autopsy, but their clinical relevance is doubtful. Endocarditis is very rare. Valvular damage may occur as a result of improper handling of the catheter (in particular, its withdrawal with the balloon still inflated). Catheter-related infections remain a risk, but they do not seem to be any more common than with central venous catheters.

Pulmonary artery rupture is the most feared complication: Although it is exceptionally rare, it is associated with a high mortality rate. The usual cause is overinflating the balloon in the presence of resistance to inflation, particularly in the presence of preexisting PAH; other, less common causes are shown in Figure 4.8. The cardinal sign of rupture is the development of hemoptysis. The reaction to this event should not be to pull out the catheter entirely, but rather to withdraw it slightly and then inflate the balloon. If the hemorrhage does not stop, a thoracotomy may be necessary to repair the pulmonary artery.

## TECHNICAL LIMITATIONS

Invasive measurements of pressures are based on fluid-filled systems with disposable transducers. The transducer includes a deformable membrane with a Wheatstone bridge modifying the electrical resistance and, correspondingly, the intensity of a current. Reliability is ensured by the excellent linearity between the pressure signal and the electrical signal generated by the transducer, in a range of frequency values largely exceeding the range of frequencies found in the human body.

Reliability is less secure in the intermediary system made of tubes and stopcocks along the extent of the pressure



**Figure 4.8** Possible mechanisms of pulmonary artery rupture (other than the most common cause, overinflating the balloon in the presence of resistance to inflation). (From Barash PG, Nardi D, Hammond G, et al: Catheter-induced pulmonary artery perforation. Mechanisms, management, and modifications. *J Thorac Cardiovasc Surg* 1981;82:5.)

system. These can modify the morphology of the trace, resulting in damping. Motion artifacts can further complicate the tracings. The presence of air bubbles also may alter the signal. Use of fluid-filled catheters to measure pressures provides reliable estimates of mean vascular pressures.

Three steps must be followed to guarantee reliable measurements:

1. The first step is appropriate zeroing, which is accomplished by opening the transducer to atmospheric pressure (taken as the zero value). All pressures must be measured with reference to an arbitrary reference point. This zero reference pressure level should ideally be where it is least influenced by location on the body. In humans, it is thought to be at the level of the right atrium, so the reference level usually is placed in the midchest (midaxillary) position at the level of the fourth intercostal space. In healthy persons, the CVP referred to that region does not change with supine versus upright position. An alternative reference is 5 cm vertically below the sternal angle. Obviously, errors in zeroing are relatively more important for measurements of cardiac filling pressures than for arterial pressure measurements, because the errors are quantitatively identical but proportionally greater.
2. The second step is calibration, which is now done automatically by today's electronic systems.
3. The third step is ensuring the good quality of the trace. Shaking the catheter should result in large pressure variations on the screen. A damped system will underestimate systolic pressures. The liquid column should be continuous, without air bubbles in the system. Excessive tubing length, or multiple stopcocks and connectors, may decrease the resonant frequency, resulting in "whipped" traces. Likewise, the presence of bubbles must be carefully avoided. Transient flushing should be

followed by an abrupt return of the pressure trace to its actual value.

## APPLICATIONS: DIAGNOSIS VERSUS MONITORING

Today the PAC is more useful in guiding therapy rather than in identifying abnormalities, this latter role having largely been taken over by noninvasive, mainly echocardiographic techniques (Table 4.1). In the past, analysis of waveforms was used—for example, the abnormal v waves of mitral regurgitation. Likewise, an increase of all pressures to identical levels should suggest pericardial tamponade. These findings should still alert the clinician to possible abnormalities, but echocardiographic techniques have largely replaced the use of the PAC for identifying valvular disease. The use of echocardiographic techniques for monitoring, however, is hampered by the difficulty of keeping the probe in the esophagus for prolonged periods of time, and results are very operator dependent.

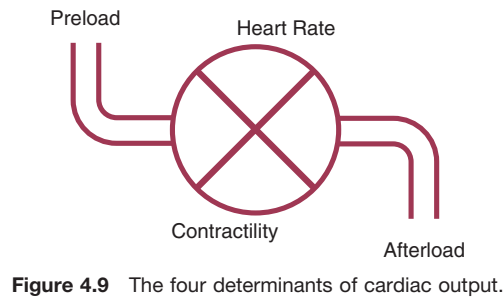
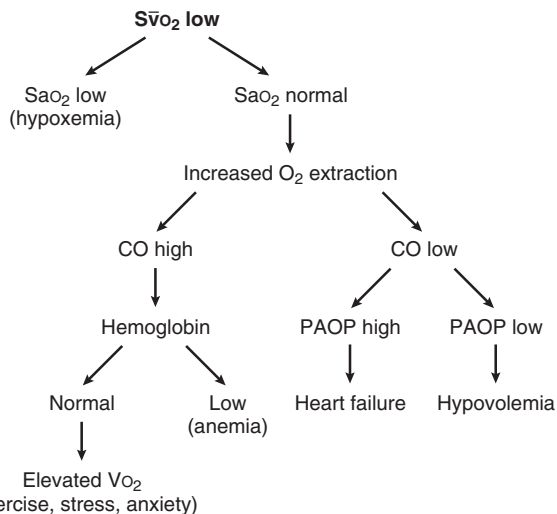
To illustrate the need to integrate different variables, rather than focusing on just one variable, some suggested applications follow:

- *Interpretation of a cardiac output value:* It is important to consider the four determinants of cardiac output (Fig. 4.9).<sup>20</sup> Interpretation should start by considering the stroke volume (cardiac output divided by heart rate) and relating this to an index of ventricular filling (PAOP) and an index of ventricular afterload (arterial and pulmonary artery pressures); an inotropic agent should be added only when preload and afterload have been optimized.
- *Fluid status:* Low cardiac filling pressures may be normal or reflect hypovolemia. The measurement of cardiac output and  $\bar{SvO}_2$  will help determine fluid needs. Indeed, hypovolemia typically is associated with a low



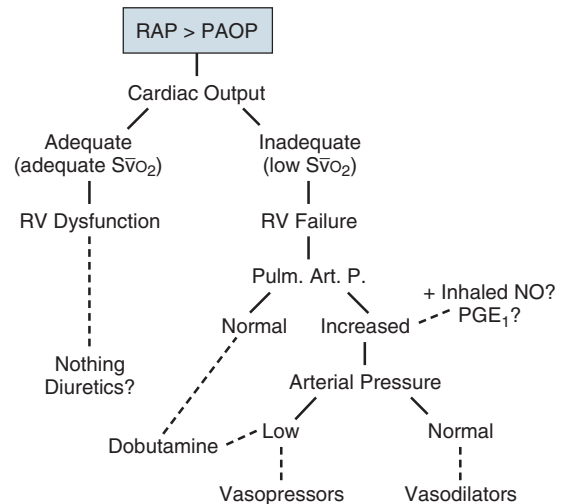
**Table 4.1 Pulmonary Artery (PA) Catheterization Versus Echocardiographic Techniques for Management of Selected Disorders: Relative Value**

Disorder	PA Catheterization		Echocardiography	
	Diagnosis	Monitoring	Diagnosis	Monitoring
Tamponade	+	++	+++	+
Hypovolemia	++	+++	++	+
Valvular disease	+	++	+++	+
Heart failure	++	+++	++	+
Right ventricular failure	++	+++	+++	++
Septic shock	+	+++	+	+

**Figure 4.9** The four determinants of cardiac output.**Figure 4.10** Interpretation of a low  $S\bar{v}O_2$  (mixed venous oxygen saturation). CO, cardiac output; PAOP, pulmonary artery occlusion pressure;  $SaO_2$ , arterial oxygen saturation;  $VO_2$ , oxygen uptake.

cardiac output and a low  $S\bar{v}O_2$  (Fig. 4.10). By contrast, hypovolemia in the presence of normal cardiac function will be manifested by high cardiac filling pressures associated with a relatively high cardiac output and normal or high  $S\bar{v}O_2$ .

- **Hemodynamic versus nonhemodynamic pulmonary edema:** The distinction between hemodynamic and nonhemodynamic types of lung edema no longer requires pulmonary artery catheterization; the clinical history and less invasive (e.g., echocardiographic) measurements usually are sufficient to separate the two. Nevertheless, invasive hemodynamic

**Figure 4.11** Interpretation of a reversed gradient between the right atrial pressure (RAP) and the pulmonary artery occlusion pressure (PAOP). NO, nitric oxide; PGE<sub>1</sub>, prostaglandin E<sub>1</sub>; RV, right ventricular;  $S\bar{v}O_2$ , mixed venous oxygen saturation.

monitoring can reveal that patients thought to meet only the acute lung injury/acute respiratory distress syndrome (ALI/ARDS) criteria sometimes have unexpectedly high PAOP.<sup>21</sup>

- **RV dysfunction versus failure:** A reverse gradient between RAP and PAOP (i.e., a higher RAP than PAOP) indicates RV dysfunction or failure and usually is secondary to pulmonary hypertension, as will be immediately apparent from the measurements of pulmonary artery pressures. RV dysfunction is manifested by RV dilation (and thus a decrease in RVEF) with no limitation on cardiac output. This is the most common situation in patients with ARDS, who usually maintain a hyperkinetic state. Rather, RV failure refers to a state in which cardiac output is no longer sustained at adequate levels.<sup>22</sup> These different entities are illustrated in Figure 4.11.
- **LV dysfunction versus failure:** Here the gradient between PAOP and RAP will be higher than the typical 3 to 5 mm Hg. As with the right ventricle, in LV dysfunction the ventricular volumes may increase (so the ejection fraction will decrease), but the cardiac output may be simultaneously preserved; such a situation may exist in septic shock.

LV failure is more common and is manifested by a decrease in cardiac output and  $\text{S}\bar{\text{v}}\text{O}_2$ .

## ASSESSING EFFECTS OF INTERVENTIONS

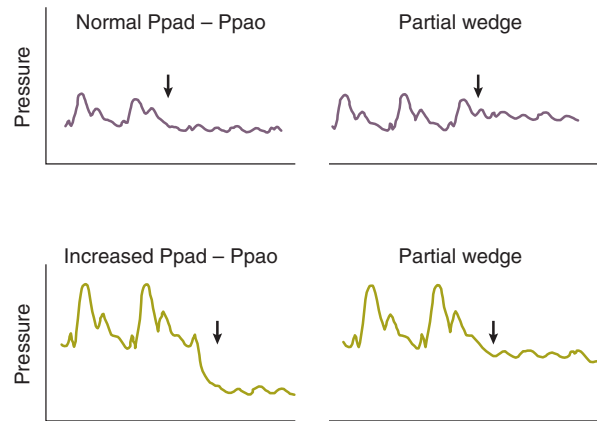
The PAC can be used to monitor the effectiveness of various interventions:

- **Fluid challenge:** A fluid challenge technique is indicated whenever the benefit of fluid administration is in doubt.<sup>17</sup> Ideally, fluid administration will result in increases in cardiac output and tissue perfusion without major increases in cardiac filling. An increase in PAOP in the absence of a significant change in cardiac output and  $\text{S}\bar{\text{v}}\text{O}_2$  indicates that fluid administration will only result in an increased risk of edema and should be discontinued.
- **Inotropic agents:** The use of inotropes aims to increase cardiac output and possibly decrease PAOP. Lack of an increase in cardiac output after administration of a  $\beta_1$ -adrenergic inotropic agent indicates a desensitization of the  $\beta$ -adrenergic receptors and is associated with a worse prognosis.<sup>23</sup>
- **Vasopressors:** The use of pure vasoconstrictors is expected to increase arterial pressure but also cardiac filling pressures. An excessive increase in cardiac filling pressures with use of such an agent suggests the need for addition of an inotropic agent (e.g., dobutamine).
- **Vasodilators:** The administration of vasodilators may rapidly reduce arterial pressure so that continuous arterial pressure monitoring is usually indicated. Moreover, a reduction in vascular tone in the presence of hypovolemia may reduce venous return and thus cardiac output.

## CLINICAL INDICATIONS FOR PULMONARY ARTERY CATHETER INSERTION

The improvement in noninvasive diagnostic techniques means that today the PAC is used primarily for monitoring. Potential indications include the following:

- **Severe circulatory shock:** The PAC can be used to help guide fluid challenges and titrate inotropic agents. Shock due to hypovolemia (as in polytrauma or with other forms of massive bleeding) does not require insertion of a PAC, because management of such patients generally is quite straightforward.
- **RV failure:** The PAC can be used to monitor pulmonary artery pressures, the gradient between RAP and PAOP, cardiac output, and  $\text{S}\bar{\text{v}}\text{O}_2$ .
- **Acute respiratory failure due to pulmonary edema:** Whether lung edema is hemodynamic or nonhemodynamic, the strategy should be to keep the hydrostatic pressures as low as possible, but this requires measurements of cardiac output and  $\text{S}\bar{\text{v}}\text{O}_2$  to make sure the systemic circulation is not compromised.
- **Complex fluid management in the presence of impending renal failure:** Sometimes it is difficult to evaluate the fluid status in oliguric patients, in whom hypovolemia may compromise renal function but hypervolemia obviously must be avoided.
- **Dynamic assessment of cardiac function in specific conditions:** The best example is that of the patient who is difficult to



**Figure 4.12** Schematic depiction of partial occlusion when the gradient between the pulmonary artery diastolic pressure (Ppad) and the pulmonary artery occlusion pressure (Ppao) is normal (top) or is increased (bottom). Arrow denotes balloon inflation. (From Leatherman JW, Shapiro RS: Overestimation of pulmonary artery occlusion pressure in pulmonary hypertension due to partial occlusion. *Crit Care Med* 2003;31:93-97.)

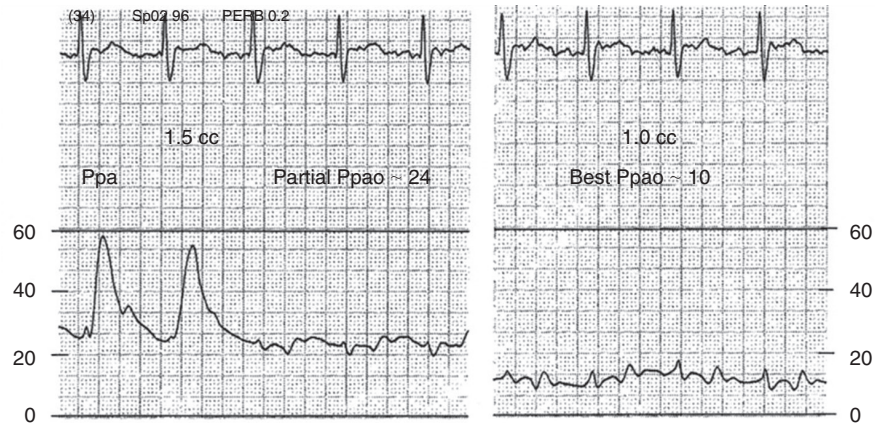
wean from mechanical ventilation, possibly owing in part to cardiac dysfunction.

## PULMONARY ARTERY OCCLUSION PRESSURE AND PARTIAL OCCLUSION

Partial occlusion of the pulmonary artery in the presence of pulmonary hypertension may be difficult to recognize and can lead to significant overestimation of the PAOP, denoted Ppao on [Figure 4.12](#). A useful clue to partial occlusion is occurrence of a substantial increase in the PAOP without a concomitant change in the pulmonary artery diastolic pressure, denoted Ppad on the figure. If the Ppad – PAOP gradient and the underlying disease process would predict increased PVR, partial occlusion should be suspected. Partial occlusion may occur if a catheter is either too proximal or too distal in the pulmonary artery, and appropriate repositioning may be corrective. The best PAOP may be obtained in some circumstances by further advancing the catheter with the balloon fully inflated and at other times by retracting the catheter to the original pulmonary artery position and attempting to occlude with 1.0 to 1.2 mL of air, instead of full inflation. It is imperative never to inflate against resistance. [Figures 4.12](#) and [4.13](#) offer further information on diagnosis and management of partial occlusion.

## DOES THE USE OF A PULMONARY ARTERY CATHETER IMPROVE OUTCOME?

The use of the PAC has been challenged on the basis that it has not been shown to improve outcomes.<sup>21,24-26</sup> An improvement in outcome, however, has not been demonstrated with other monitoring techniques either. Moreover, a number of studies have indicated that the use of the PAC can influence therapy.<sup>27</sup> If use of a PAC does not result in better outcomes, important and challenging questions arise about the beneficial effects of many therapeutic



**Figure 4.13** Partial pulmonary artery occlusion pressure (Ppao) measured when 1.5 cc of air was used to inflate the catheter balloon (*left graph*); a much lower Ppao was obtained with a 1.0-cc inflation (*right graph*). Review of the chest roentgenogram revealed that the catheter tip was too peripheral, so it was withdrawn to a more proximal location. Ppa, pulmonary artery pressure. See text for definition of partial and best Ppao values. Scale in mm Hg. (From Leatherman JW, Shapiro RS: Overestimation of pulmonary artery occlusion pressure in pulmonary hypertension due to partial occlusion. *Crit Care Med* 2003;31:93-97.)

**Table 4.2** Components and Common Errors in Hemodynamic Monitoring

Component	Common Errors
1. Measure	Catheter misplacement Errors in pressure measurements No consideration of $S\bar{v}O_2$
2. Interpret	Interpretation of cardiac output without consideration of $S\bar{v}O_2$ Neglect of PAP/PAOP gradient Neglect of inverted RAP/PAOP gradient
3. Apply	Treating as if the data were not available Giving diuretics for a high PAOP without other consideration

PAP, pulmonary artery pressure; PAOP, pulmonary artery occlusion pressure; RAP, right atrial pressure;  $S\bar{v}O_2$ , mixed venous oxygen saturation.

interventions in the ICU. Some evidence suggests that the use of the PAC may improve outcomes in the most severely ill subsets of critically ill patients.<sup>28,29</sup> Errors in measurements from the PAC were identified many years ago,<sup>30</sup> and another consideration is the considerable interobserver variability in interpretation of PAC tracings.<sup>31</sup> Clearly, not all ICU patients need a PAC and insertion should be reserved for complex cases.<sup>32</sup> If a PAC is considered necessary, it is important to respect the three successive steps (Table 4.2): to take adequate and full measurements; to interpret the results correctly; and to apply the gathered information for the patient's benefit. Unfortunately, potential errors are associated with each step. Some of these issues can be addressed with better teaching and improved basic knowledge of hemodynamics and basic physiology.

#### KEY POINTS

- No simple guidelines for monitoring are available or applicable in all cases; monitoring should be tailored to each patient's needs.
- Each variable, taken individually, has limitations and is subject to error and difficulty in interpretation. Variables should be combined and integrated to provide a global picture of the clinical situation.
- A monitoring technique cannot improve outcome by itself; each of the three components of monitoring is important with any monitoring technique:
  - Accurate collection of data
  - Interpretation of the data
  - Application of the information obtained
- Cardiac output is an adaptive value that must constantly adjust to the oxygen requirements of the organs.
- Separation of the four determinants of cardiac output—heart rate, contractility, preload, and afterload—is useful to consider the various interventions that can be used to increase it.
- Measurements of mixed venous oxygen saturation are essential to interpret cardiac output measurements.
- The relationship between cardiac filling pressures and volumes is relatively weak. Pressure measurements are important, however, because pressures (rather than volumes) are the primary determinant of edema formation.
- The calculation of derived variables, such as vascular resistances, ventricular work, and oxygen-derived variables, is of limited usefulness.

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The complete list of references can be found at [www.expertconsult.com](http://www.expertconsult.com).

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