
HEMODYNAMIC MONITORING

Not everything that counts can be counted. And not everything that can be counted counts.

Albert Einstein
The arterial blood pressure is one of the most popular measurements in modern medicine. However, as emphasized in the introductory quote made over a half century ago, the standard method of recording arterial pressure with an inflatable cuff (sphygmomanometer) is not expected to produce accurate results. The imprecision of the indirect blood pressure measurement (which makes one wonder about the diagnosis of hypertension) can be corrected by cannulating a peripheral artery and recording direct intra-arterial pressures. This is a common method of recording arterial blood pressure in the ICU, but direct arterial blood pressure measurements can (like their indirect counterparts) be misleading.

This chapter provides a brief description of both indirect and direct methods of arterial blood pressure recording and highlights the important shortcomings of each method.

INDIRECT MEASUREMENTS

The indirect method of measuring blood pressure that is used today was first introduced in Italy in 1896 (by an Italian physician named Riva-Rocci) and was brought to this country at the turn of the century by the famed neurosurgeon, Dr. Harvey Cushing. This method employs a device called a sphygmomanometer (sphygmos is a Greek term for pulse, and a manometer measures pressure), which consists of an inflatable bladder covered by a cloth sleeve and a gauge or column to measure pressure. The cloth sleeve is wrapped around the arm or leg in an area that overlies a major artery, and the bladder in the sleeve is inflated until it reaches a pressure that should compress the underlying artery. The bladder is then slowly deflated, allowing the compressed artery to open,
and the arterial pressure is determined by recording either the sounds (auscultation method) or the vascular pulsations (oscillometric method) that are generated as the artery opens.

**Influence of Bladder Size**

The sounds or vibrations created by the opening of the artery are more reproducible when the artery is compressed uniformly for a short distance. Therefore to ensure a reliable blood pressure recording, the inflatable bladder in the blood pressure cuff should produce a uniform compression of the underlying artery. This is determined by the size of the inflatable bladder relative to the size of the limb being compressed. Figure 8.1 shows the optimal dimensions of the cuff bladder for indirect measurements of brachial artery pressure. The length of the bladder should be at least 80% of the circumference of the upper arm (measured midway between the shoulder and elbow), and the width of the bladder should be at least 40% of the upper arm circumference (1). If the bladder is too small for the size of the arm, the pressure measurements will be falsely elevated (1–5).

**Misreading**

Misreading is the term used to describe the use of inappropriately sized cuffs for the blood pressure measurement (1). This is considered the most common source of errors in the blood pressure measurement, so it deserves some attention. Table 8.1 shows the appropriate cuff sizes for upper arm circumferences ranging from 22 cm (about 9 inches) to 52 cm (about 21 inches). Since this information is not always available when measuring blood pressures, a simple bedside method of determining appropriate cuff size is described next.

**Bedside Assessment of Cuff Size**

Before wrapping the cuff around the arm, align the cuff so that the long axis is parallel to the long axis of the arm. Then turn the cuff over so the bladder on the underside is facing you, and wrap the cuff lengthwise around the upper arm. The bladder (width) should encircle half of the upper arm circumference. If the bladder encircles less than half of the upper arm, the cuff is too small, and the blood pressure measurement may be spuriously high. If the cuff encircles most of the upper arm and seems too big for the arm, no change in cuff size is necessary (i.e., a cuff that is larger than needed will not produce spurious pressure recordings) (1).

**Auscultatory Method**

The standard method of measuring blood pressure involves manual inflation of an arm cuff placed over the brachial artery. The cuff is then gradually deflated, and the pressure is determined by sounds (called Korotkoff sounds) that are generated when the artery begins to open.

**The Korotkoff Sounds**

The Korotkoff sounds are very low frequency sounds (25 to 50 Hz) and are just above the normal threshold for human hearing, which is 16 Hz (6). Human speech is generally in the frequency range of 120 to 250 Hz, and the human ear detects sounds optimally when they have frequencies of 2,000 to 3,000 Hz. (6). What this means is the room should be quiet when listening for Korotkoff sounds (because you will hear people talking more easily than you hear these sounds), and even then, the sounds will be faint to the human ear.

**Stethoscope Head**

The bell-shaped head of a stethoscope is a low frequency transducer, while the flat, diaphragm-shaped head is designed to detect high frequency sounds. Therefore to optimize detection of the low-frequency Korotkoff sounds, the bell-shaped head of the stethoscope should be used to measure blood pressure (1). This is often neglected, and some stethoscopes are manufactured without a bell-shaped head!

**Low Flow States**

Because Korotkoff sounds are generated by blood flow, low flow states can diminish the intensity of these sounds. When this occurs, the sounds may not be heard at first (i.e., at the systolic pressure), and this will result in falsely low recordings for the systolic blood pressure. The tendency
TABLE 8.2 Discrepancy Between Direct and Indirect Blood Pressure Measurements in Shock

<table>
<thead>
<tr>
<th>Systolic BP Difference (Direct BP—Cuff BP)</th>
<th>% Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–10 mm Hg</td>
<td>0</td>
</tr>
<tr>
<td>11–20 mm Hg</td>
<td>28</td>
</tr>
<tr>
<td>21–30 mm Hg</td>
<td>22</td>
</tr>
<tr>
<td>&gt;30 mm Hg</td>
<td>50</td>
</tr>
</tbody>
</table>

From Cohn JN. Blood pressure measurement in shock. JAMA 1987; 119:118.

to underestimate the systolic blood pressure in low flow states is shown in Table 8.2. This is from a study comparing direct and indirect measurements of systolic blood pressure in patients with a low flow state and hypotension (3). In half of the patients, the indirect auscultatory method underestimated the actual systolic blood pressure by more than 30 mm Hg. According to the American Association for Medical Instrumentation, indirect pressure measurements should be within 5 mm Hg of directly recorded pressures to be considered accurate (4). Using this criterion, there was not a single pressure recording with the auscultatory method that could be considered accurate. Observations like these are the reason that direct blood pressure measurements are preferred in hemodynamically compromised patients.

Oscillometric Method

The oscillometric method uses the principle of plethysmography to detect pulsatile pressure changes (oscillations) in an underlying artery. When an inflated cuff is placed over an artery, the pulsatile pressure changes in the artery will be transmitted to the inflated cuff, producing similar changes in cuff pressure. The periodic changes in cuff pressure (i.e., oscillations) are then processed electronically to derive a value for the mean, systolic, and diastolic blood pressures (5).

Performance

Oscillometric devices first appeared in the mid 1970s and since then have gained widespread acceptance for monitoring blood pressure in operating rooms, ICUs, and emergency rooms. However, the accuracy of oscillometric blood pressure measurements is disturbingly low. This is demonstrated in Figure 8.2, which shows a comparison of directly measured systolic pressures with oscillometric measurements in patients undergoing major surgery. The dark line is the line of unity (where the measurements using both techniques would be identical) and the area bounded by the lighter lines (which are 5 mm Hg on either side of unity) is the zone of acceptable accuracy for oscillometric pressure measurements. Note that most of the oscillometric measurements (closed squares) fall outside the zone of acceptable accuracy, indicating that a majority of the oscillometric measurements in this study were inaccurate.

FIGURE 8.2 Comparison of direct (open squares) and oscillometric (closed squares) measurements of systolic pressure in the brachial artery. (From Gravlee GP, Brockeheim JK. Accuracy of four indirect methods of blood pressure measurement, with hemodynamic correlations. J Clin Monit 1990;6:284–298.)

Other studies in ICU patients have shown that oscillometric measurements are consistently lower than direct blood pressure measurements (6,7). Some of this discrepancy is due to "miscalculation" (6,7), so attention to proper cuff sizes is important for oscillometric measurements, as it is for auscultatory measurements. However, until accuracy and reliability improve, oscillometric blood pressure measurements should not be regarded as an adequate substitute for direct blood pressure measurements in the ICU.

DIRECT MEASUREMENTS

Direct recording of intravascular pressures is recommended for all patients in the ICU who are hemodynamically unstable or are at risk for hemodynamic instability. Unfortunately, direct arterial pressure recordings have their own shortcomings, and some of these will be described in the remainder of the chapter.

Pressure Versus Flow

The distinction between pressure and flow is important to recognize because there is a tendency to equate pressure and flow in certain situations. This is
most evident in the popularity of pressor or vasoconstrictor agents in the management of clinical shock. In this setting, an increase in blood pressure is often assumed to indicate an increase in systemic blood flow, but the opposite effect (a decrease in flow) is also possible.

One of the important distinctions between pressure and flow is the transmission of pressure and flow waves through the circulatory system. Ejection of the stroke volume from the heart is accompanied by a pressure wave and a flow wave. Under normal conditions, the pressure wave travels 20 times faster than the flow wave (10 m/second versus 0.5 m/second), and thus the pulse pressure recorded in a peripheral artery precedes the corresponding stroke volume by a matter of seconds (8). When vascular impedance (i.e., compliance and resistance) is increased, the velocity of the pressure wave is increased, while the velocity of the flow wave is decreased. (When vascular impedance is reduced, pressure can be diminished while flow is enhanced.) Thus when vascular impedance is abnormal, the arterial pressure is not a reliable index of blood flow.

The Arterial Pressure Waveform

The contour of the arterial pressure waveform changes as the pressure wave moves away from the proximal aorta. This is shown in Figure 8.3.

Note that as the pressure wave moves toward the periphery, the systolic pressure gradually increases, and the systolic portion of the waveform narrows. The systolic pressure can increase as much as 20 mm Hg from the proximal aorta to the radial or femoral arteries. This increase in peak systolic pressure is offset by the narrowing of the systolic pressure wave, so that the mean arterial pressure remains unchanged. Therefore, the mean arterial pressure is a more accurate measure of central aortic pressure.

Systolic Amplification

The increase in systolic pressure in peripheral arteries is the result of pressure waves that are reflected back from the periphery (9). These reflected waves originate from vascular bifurcations and from narrowed blood vessels. As the pressure wave moves peripherally, wave reflections become more prominent, and the reflected waves add to the systolic pressure wave and amplify the systolic pressure. Amplification of the systolic pressure is particularly prominent when the arteries are noncompliant, causing reflected waves to bounce back faster. This is the mechanism for systolic hypertension in the elderly (9). Because a large proportion of patients in the ICU are elderly, systolic pressure amplification is probably commonplace in the ICU.

RECORDING ARTIFACTS

Fluid-filled recording systems can produce artifacts that further distort the arterial pressure waveform. Failure to recognize recording system artifacts can lead to errors in interpretation.

Resonant Systems

Vascular pressures are recorded by fluid-filled plastic tubes that connect the arterial catheters to the pressure transducers. This fluid-filled system can oscillate spontaneously, and the oscillations can distort the arterial pressure waveform (10,11).

The performance of a resonant system is defined by the resonant frequency and the damping factor of the system. The resonant frequency is the inherent frequency of oscillations produced in the system when it is disturbed. When the frequency of an incoming signal approaches the resonant frequency of the system, the resident oscillations add to the incoming signal and amplify it. This type of system is called an underdamped system. The damping factor is a measure of the tendency for the system to attenuate the incoming signal. A resonant system with a high damping factor is called an overdamped system.

Waveform Distortion

Three waveforms obtained from different recording systems are shown in Figure 8.4. The waveform in panel A, with the rounded peak and
The Flush Test

A brief flush to the catheter-tubing system can be applied to determine whether the recording system is distorting the pressure waveform (11,13). Most commercially available transducer systems are equipped with a one-way valve that can be used to deliver a flush from a pressurized source. Figure 8.4 shows the results of a flush test in three different situations. In each case, the pressure increases abruptly when the flush is applied. However, the response at the end of the flush differs in each panel. In panel A, the flush is followed by a few oscillating waveforms. The frequency of these oscillations is the resonant frequency (f) of the recording system, which is calculated as the reciprocal of the time period between the oscillations. When using standard strip-chart recording paper divided into 1 mm segments, f can be determined by measuring the distance between oscillations and dividing this into the paper speed (11); that is, \( f = \frac{\text{distance between oscillations}}{\text{paper speed (in mm/second)}} \). In the example shown in panel A, the distance (d) between oscillations is 1.0 mm, and the paper speed is 25 mm/second, so \( f = \frac{25}{25} \text{ Hz} = 1 \text{ Hz} \).

Signal distortion is minimal when the resonant frequency of the recording system is five times greater than the major frequency in the arterial pressure waveform. Because the major frequency in the arterial pulse is approximately 5 Hz (14), the resonant frequency of the recording system in panel A (25 Hz) is five times greater than the frequency in the incoming waveform, and the system will not distort the incoming waveform.

The flush test in panel B of Figure 8.4 reveals a resonant frequency of 12.5 Hz (f = 25/2). This is too close to the frequency of arterial pressure waveforms, so this system will distort the incoming signal and produce systolic amplification.

The flush test shown in panel C of Figure 8.4 does not produce any oscillations. This indicates that the system is overdamped, and this system will produce a spuriously low pressure recording. When an overdamped system is discovered, the system should be flushed thoroughly (including all stopcocks in the system) to release any trapped air bubbles. If this does not correct the problem, the arterial catheter should be repositioned or changed.

Mean Arterial Pressure

The mean arterial pressure has two features that make it superior to the systolic pressure for arterial pressure monitoring. First, the mean pressure is the true driving pressure for peripheral blood flow. Second, the mean pressure does not change as the pressure waveform moves distally, nor is it altered by distortions generated by recording systems (10).

The mean arterial pressure can be measured or estimated. Most electronic pressure monitoring devices can measure mean arterial pressure by integrating the area under the pressure waveform and dividing this by the duration of the cardiac cycle. The electronic measurement is preferred to the estimated mean pressure, which is derived as the diastolic
pressure plus one-third of the pulse pressure. This formula is based on the assumption that diastole represents two-thirds of the cardiac cycle, which corresponds to a heart rate of 60 beats/minute. Therefore heart rates faster than 60 beats/minute, which are common in critically ill patients, lead to errors in the estimated mean arterial pressure.

**Cardiopulmonary Bypass**

In most circumstances, the mean pressures in the aorta, radial artery, and femoral artery are within 3 mm Hg of each other; however, in patients undergoing cardiopulmonary bypass surgery, the mean arterial pressure can be significantly (more than 5 mm Hg) lower than the mean pressures in the aorta and femoral artery. This condition may be caused by a selective decrease in vascular resistance in the hand, because compression of the wrist often abolishes the pressure difference. An increase in radial artery pressure of at least 5 mm Hg when the wrist is compressed (distal to the radial artery catheter) suggests a discrepancy between radial artery pressure and pressures in other regions of the circulation.

**A FINAL WORD**

> I would venture to guess that, of all the procedures done in clinical medicine that have important consequences, measurement of blood pressure is likely one that is done most haphazardly.

Norman Kaplan, M.D.

There are an estimated 50 million people in the United States with the diagnosis of hypertension (17). This represents about 25% of the adult population (210 million) and indicates that hypertension is the number one health problem in this country. Hypertension is clearly an enormous health burden, and the source of this burden is a single diagnostic test: the (indirect) blood pressure measurement. Yet, as indicated by the hypertension expert Dr. Norman Kaplan, this measurement receives little attention and is usually performed haphazardly. This means that a diagnostic test that is performed poorly (i.e., the blood pressure measurement) is responsible for the number one health problem in this country. The implications are obvious.

The consequences of an improperly performed blood pressure measurement are illustrated in the following scenario. About 180 million adults (85% of the adult population) have their blood pressure measured each year. If an improperly performed measurement results in a falsely elevated blood pressure reading in just 1% of these subjects, 1.8 million new cases of (erroneously diagnosed) hypertension would be created each year. This might explain why there are so many people with hypertension in this country.

Regardless of whether you are working in the ICU or elsewhere, it is imperative that you learn all you can about the indirect blood pressure measurement to obtain the most accurate readings possible. You owe this to your patients and to our overburdened healthcare system.

**REFERENCES**

**Indirect Measurements**


**Direct Measurements**

THE PULMONARY ARTERY CATHETER

A searchlight cannot be used effectively without a fair knowledge of the territory to be searched.

Fergus Macartney, FRCP

The birth of critical care as a specialty is largely the result of two innovations: positive-pressure mechanical ventilation and the pulmonary artery catheter. The latter device is notable for the multitude of physiologic parameters that can be measured at the bedside. Prior to the introduction of the pulmonary artery catheter, the bedside evaluation of cardiovascular function was essentially a "black box" approach that relied on indirect, qualitative markers provided by sounds (e.g., pulmonary rales, cardiac gallops, cuff-based blood pressures), visual cues (e.g., edema, skin color), and tactile cues (e.g., pulse, skin temperature). The pulmonary artery catheter improved dramatically on this approach, allowing physicians to measure quantitative physiologic parameters at the bedside and to apply the basic principles of cardiovascular physiology to the bedside management of patients with cardiovascular disorders.

This chapter describes the multitude of parameters that can be measured with pulmonary artery catheters (1-5). Most of these parameters are described in detail in Chapters 1 and 2, so it may help to review these chapters before proceeding further.

CAVEAT. The value of the pulmonary artery catheter is not determined solely by the measurements it allows but is also dependent on the clinician’s ability to understand the measurements and how they are obtained. This deserves mention because surveys indicate that physicians have an inadequate understanding of the measurements provided by pulmonary artery catheters (6,7).
CATHETER DESIGN

The balloon-flotation pulmonary artery (PA) catheter was conceived by Dr. Jeremy Swan, who was Chief of Cardiology at Cedars-Sinai Hospital when the following experience occurred.

In the fall of 1967, I had occasion to take my (then young) children to the beach in Santa Monica. It was a hot Saturday, and the sailboats on the water were becalmed. However, about half-a-mile offshore, I noted a boat with a large spinnaker well set and moving through the water at a reasonable velocity. The idea then came to put a sail or parachute on the end of a highly flexible catheter and thereby increase the frequency of passage of the device into the pulmonary artery (1).

Three years later (in 1970), Dr. Swan introduced a PA catheter that was equipped with a small inflatable balloon at its tip. When inflated, the balloon acted like a sail to allow the flow of venous blood to carry the catheter through the right side of the heart and out into one of the pulmonary arteries. This “balloon flotation” principle allows a right-heart catheterization to be performed at the bedside, without fluoroscopic guidance.

Basic Features

The basic features of a PA catheter are shown in Figure 9.1. The catheter is 110 cm long and has an outside diameter of 2.3 mm (7 French). There are two internal channels: One runs the entire length of the catheter and opens at the tip of the catheter (the PA lumen), and the other ends 5 cm from the catheter tip, which should place it in the right atrium (the RA lumen). The tip of the catheter is equipped with a small (1.5 mL capacity) inflatable balloon. When fully inflated, the balloon creates a recess for the tip of the catheter that prevents the tip from coming into contact with (and damaging) vessel walls as the catheter is advanced. The catheter also has a small thermistor (i.e., a transducer device that senses changes in temperature) that is located 4 cm from the catheter tip. The thermistor can measure the flow of a cold fluid that is injected through the proximal port of the catheter, and this flow rate is equivalent to the cardiac output. This is the thermodilution method of measuring cardiac output and will be described in more detail later in the chapter.

Additional Accessories

Other accessories that are available on specially-designed PA catheters include:

- An extra channel that opens 14 cm from the catheter tip that can be used to thread temporary pacemaker leads into the right ventricle (8)
- A fiberoptic system that allows continuous monitoring of mixed venous oxygen saturation (9)
- A rapid-response thermistor that can measure the ejection fraction of the right ventricle (10)

A thermal filament that generates low-energy heat pulses and allows continuous thermodilution measurement of the cardiac output (11)

With such a large variety of accessories, the PA catheter is the Swiss Army knife of the critical care specialist.

CATHETER INSERTION

The PA catheter is inserted into the subclavian or internal jugular veins. A large-bore introducer catheter is inserted first (see Figure 6.4), and the PA catheter is then passed through the introducer catheter. Just before the PA catheter is inserted, the distal (PA) lumen is attached to a pressure transducer, and the pressure is monitored continuously during insertion. When the PA catheter is passed through the introducer catheter and enters the superior vena cava, a venous pressure waveform appears. When this occurs, the balloon is inflated with 1.5 mL of air, and the
catheter is advanced with the balloon inflated. The location of the catheter tip is determined by the pressure tracings recorded from the distal (PA) lumen, as shown in Figure 9.2.

1. The superior vena cava pressure is identified by a venous pressure waveform, which appears as small amplitude oscillations. This pressure remains unchanged after the catheter tip is advanced into the right atrium. The normal pressure in the superior vena cava and right atrium is 1 to 6 mm Hg.

2. When the catheter tip is advanced across the tricuspid valve and into the right ventricle, a pulsatile waveform appears. The peak (systolic) pressure is a function of the strength of right ventricular contraction, and the lowest (diastolic) pressure is equivalent to the right-atrial pressure. The systolic pressure in the right ventricle is normally 15 to 30 mm Hg.

3. When the catheter moves across the pulmonic valve and into a main pulmonary artery, the pressure waveform shows a sudden rise in diastolic pressure with no change in the systolic pressure. The rise in diastolic pressure is caused by resistance to flow in the pulmonary circulation. The pulmonary artery diastolic pressure is normally 6 to 12 mm Hg.

4. As the catheter is advanced along the pulmonary artery, the pulsatile waveform disappears, leaving a venous-type pressure waveform at the same level as the pulmonary artery diastolic pressure. This is the pulmonary artery occlusion pressure, also called the pulmonary capillary wedge pressure (PCWP), or simply the wedge pressure. This pressure is obtained in the absence of flow between the catheter tip and the left atrium and is a reflection of the venous pressure in the left side of the heart (i.e., left atrial pressure and left-ventricular diastolic pressure). The wedge pressure is equivalent to the pulmonary artery diastolic pressure.

5. When the wedge pressure tracing appears, the catheter is left in place (not advanced further), and the balloon is deflated. The pulsatile pulmonary artery pressure should reappear when the balloon is deflated. If this occurs, the PA catheter should be secured in place (usually with a single suture that anchors the catheter to the skin), and the balloon should be left deflated.

The Balloon

Sustained periods of balloon inflation creates a risk of pulmonary artery rupture or pulmonary infarction, so the balloon should be deflated at all times while the catheter is in place. Balloon inflation is reserved only for measurements of the wedge pressure. When obtaining a wedge pressure, do not fully inflate the balloon with 1.5 mL air at all once (catheters often migrate into smaller pulmonary arteries, and a fully inflated balloon could result in vessel rupture). The balloon should be slowly inflated until a wedge pressure tracing is obtained. Once a satisfactory wedge pressure is recorded, the balloon should be fully deflated. (Detaching the syringe from the balloon injection port will help prevent undetected balloon inflation while the catheter is in place.)

Troubleshooting

The following are some common problems encountered during advancement of a PA catheter.

Catheter Will Not Advance into the Right Ventricle

Most catheters should enter the right ventricle after they are advanced a distance of 20 to 25 cm (see Figure 6.5). Difficulty advancing a catheter into the right ventricle (which can occur with tricuspid regurgitation or right heart failure) can sometimes be corrected by filling the balloon with sterile saline instead of air (12) and positioning the patient with the left side down. The fluid adds weight to the balloon, and with the left side down, the balloon can fall into the right ventricle. When the right ventricle is entered, the saline should be removed and replaced with air.

Catheter Will Not Advance into the Pulmonary Artery

Catheters can become coiled in the right ventricle and fail to enter the pulmonary circulation. This problem is sometimes corrected by withdrawing the catheter into the superior vena cava and re-advancing the catheter using a slow, continuous motion (allowing the venous flow to carry the
catheter into the pulmonary circulation) and avoiding rapid thrusts. This problem can persist in patients with pulmonary hypertension.

Atrial and ventricular arrhythmias can appear in over half of PA catheter placements (13), but they are almost always benign and self-limited and require no treatment. Complete heart block that appears during catheter placement should prompt immediate withdrawal of the catheter and, if necessary, a brief period of transthoracic pacing. Prolonged heart block could indicate injury to the AV node, and might require transvenous pacing.

Unable to Obtain a Wedge Pressure
In about 25% of PA catheter placements, the pulsatile PA pressure never disappears despite maximum advancement of the catheter in the pulmonary circulation. This can be the result of nonuniform balloon inflation, but in most cases, the phenomenon is unexplained. If this occurs, the pulmonary artery diastolic pressure can be used as a substitute for the pulmonary capillary wedge pressure (in the absence of pulmonary hypertension, the two pressures should be equivalent).

THERMODILUTION CARDIAC OUTPUT
The addition of a thermodilution catheter to the PA catheter increased the monitoring capacity of the catheter from 2 parameters (i.e., central venous pressure and wedge pressure) to over 10 parameters (see Table 9.1 and Table 9.2).

### TABLE 9.1 Cardiovascular Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Abbreviation</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Central venous pressure</td>
<td>CVP</td>
<td>1–6 mm Hg</td>
</tr>
<tr>
<td>Pulmonary capillary wedge pressure</td>
<td>PCWP</td>
<td>6–12 mm Hg</td>
</tr>
<tr>
<td>Cardiac index</td>
<td>CI</td>
<td>2.4–4 L/min/m²</td>
</tr>
<tr>
<td>Stroke volume index</td>
<td>SVI</td>
<td>40–70 mL/beat/m²</td>
</tr>
<tr>
<td>Left-ventricular stroke work index</td>
<td>LVSWI</td>
<td>40–60 g · m/m²</td>
</tr>
<tr>
<td>Right-ventricular stroke work index</td>
<td>RVSWI</td>
<td>4–8 g · m/m²</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>RVEF</td>
<td>46–50%</td>
</tr>
<tr>
<td>End-diastolic volume</td>
<td>RVEDV</td>
<td>80–150 mL/m²</td>
</tr>
<tr>
<td>Systemic vascular resistance index</td>
<td>SVRI</td>
<td>1,600–2,400 dynes · sec¹ · cm²/m²</td>
</tr>
<tr>
<td>Pulmonary vascular resistance index</td>
<td>PVRI</td>
<td>200–400 dynes · sec¹ · cm²/m²</td>
</tr>
</tbody>
</table>

### TABLE 9.2 Oxygen-Transport Parameters

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Symbol</th>
<th>Normal Range</th>
</tr>
</thead>
<tbody>
<tr>
<td>Mixed venous oxygen saturation</td>
<td>SvO₂</td>
<td>70–75%</td>
</tr>
<tr>
<td>Oxygen delivery</td>
<td>DO₂</td>
<td>520–570 mL/min/m²</td>
</tr>
<tr>
<td>Oxygen uptake</td>
<td>VO₂</td>
<td>110–160 mL/min/m²</td>
</tr>
<tr>
<td>Oxygen-extraction ratio</td>
<td>O₂ER</td>
<td>20%–30%</td>
</tr>
</tbody>
</table>

The Method
Thermomediation is an indicator-dilution method of measuring blood flow, and is based on the premise that when an indicator substance is added to circulating blood, the rate of blood flow is inversely proportional to the change in concentration of the indicator over time (14,15). The indicator substance in this case is not a dye but a fluid with a different temperature than blood.

The thermomediation method is illustrated in Figure 9.3. A dextrose or saline solution that is colder than blood is injected through the proximal port of the catheter in the right atrium. The cold fluid mixes with blood in the right heart chambers, and the cooled blood is ejected into the pulmonary artery and flows past the thermistor on the distal end of the catheter. The thermistor records the change in blood temperature with time and sends this information to an electronic instrument that records and displays a temperature-time curve. The area under this curve is inversely proportional to the rate of blood flow in the pulmonary artery. In the absence of intracardiac shunts, this flow rate is equivalent to the (average) cardiac output.

Thermomediation Curves
Examples of thermomediation curves are shown in Figure 9.4. The low cardiac output curve (upper panel) has a gradual rise and fall, whereas the high output curve (middle panel) has a rapid rise, an abbreviated peak, and a steep downslope. Note that the area under the low cardiac output curve is greater than the area under the high output curve; that is, the area under the curves is inversely related to the flow rate. Electronic cardiac monitors integrate the area under the temperature-time curves and provide a digital display of the calculated cardiac output.

Technical Considerations
The indicator solution can be cooled in ice or injected at room temperature, and the volume of injectate is either 5 mL or 10 mL. In general, higher-volume, lower-temperature injectates produce the highest signal-to-noise ratios and thus the most accurate measurements (16). However, room temperature injectates (which require less preparation than iced injectates) produce reliable measurements in most critically ill patients (17,18). When the indicator fluid is injected at room temperature, the larger (10 mL) injection volume produces the most reliable results.
Serial measurements are recommended for each cardiac output determination. Three measurements are sufficient if they differ by 10% or less, and the cardiac output is taken as the average of all measurements. Serial measurements that differ by more than 10% are considered unreliable (19).

Variability

Thermodilution cardiac output can vary by as much as 10% without any apparent change in the clinical condition of the patient (20). This means that a baseline cardiac output of 5 L/min can vary from 4.5 to 5.5 L/min without the change being clinically significant. A change in thermodilution cardiac output must exceed 10% to be considered clinically significant.

Other Considerations

The following clinical conditions can affect the accuracy of thermodilution cardiac output measurements.

TRICUSPID REGURGITATION. This condition may be common during positive-pressure mechanical ventilation. The regurgitant flow causes the indicator fluid to be recycled, producing a prolonged, low-amplitude thermodilution curve similar to the low-output curve in the bottom frame of Figure 9.4. This results in a falsely low thermodilution cardiac output (21).

INTRACARDIAC SHUNTS. Intracardiac shunts produce falsely high thermodilution cardiac output measurements. In right-to-left shunts, a portion of the cold indicator fluid passes through the shunt, thereby creating an abbreviated thermodilution curve (similar to the abbreviated high-output curve). In left-to-right shunts, the thermodilution curve is abbreviated because the shunted blood increases the blood volume in the right heart chambers, and this dilutes the indicator solution that is injected.

Continuous Cardiac Output

The thermodilution method has been adapted to allow automatic, minute-by-minute measurements of cardiac output without the tedious
of intermittent bolus injections of indicator fluid (22). This method uses
a specialized PA catheter (Baxter Edwards Critical Care, Irvine, CA)
equipped with a 10-cm thermal filament located 15 to 25 cm from the cath-
eter tip. The filament generates low-energy heat pulses that are transmit-
ted to the surrounding blood. The resulting change in blood temperature
is then used to generate a thermodilution curve. This method records an
average cardiac output over successive 3-minute time intervals.

The continuous thermodilution method provides reliable measure-
ments of cardiac output in critically ill patients (23), and it is more accu-
rate than the intermittent bolus-injection thermodilution method (24).
Because the continuous method of monitoring cardiac output is less
time consuming and more accurate than the intermittent bolus-injection
method, this method should be preferred for cardiac output determina-
tions in the ICU.

HEMODYNAMIC PARAMETERS

The value of the PA catheter is the multitude of hemodynamic parameters
that can be generated: there are 10 parameters used to describe different
aspects of cardiovascular function (see Table 9.1), and 4 parameters that
describe systemic oxygen transport (see Table 9.2). A detailed description
of these parameters can be found in the first two chapters of this book.

Body Size

Bsa meters of mass weighted the body index of body size for hemodynamic mea-
surements is the body surface area (BSA), which can be determined with
the simple equation shown below (25).

\[
BSA (m^2) = \frac{Ht (cm) + Wt (kg)}{100} - 60
\]  

The average-sized adult has a body surface area of 1.6 to 1.9 m².

Cardiovascular Parameters

Table 9.1. Size-adjusted parameters (expressed in relation to body surface
area) are identified by the term index.

Central Venous Pressure

When the PA catheter is properly placed, the proximal port of the cath-
eter should be situated in the right atrium, and the pressure recorded
from this port should be the right atrial pressure. As mentioned previ-
ously, the pressure in the right atrium is the same as the pressure in the
superior vena cava, and these pressures are collectively called the central
venous pressure (CVP). In the absence of tricuspid valve dysfunction, the
CVP should be equivalent to the right atrial pressure (RAP) and the right-
ventricular end-diastolic pressure (RVEDP).

\[
CVP = RAP = RVEDP
\]  
Pulmonary Capillary Wedge Pressure

The measurement of the pulmonary capillary wedge pressure (PCWP) is
described earlier in the chapter (and the next chapter is devoted almost
exclusively to this measurement). The PCWP is measured when there is
no flow between the catheter tip and the left atrium (because the balloon
on the PA catheter tip is inflated), so the PCWP will be the same as the left-
atrial pressure (LAP). When the mitral valve is normal, the LAP should
be equivalent to the left-ventricular end-diastolic pressure (LVEDP).

\[
PCWP = LAP = LVEDP
\]  

Cardiac Index

The thermodilution cardiac output is usually corrected for body size as
shown below. The size-corrected cardiac output is called the cardiac index
(CI).

\[
CI = \frac{CO}{BSA}
\]  

Stroke Volume

The stroke volume is the volume of blood ejected by the ventricles dur-
ing systole. It is derived as the cardiac output divided by the heart rate
(HR). When cardiac index (CI) is used, the parameter is called the stroke
volume index (SVI).

\[
SVI = \frac{CI}{HR}
\]  

Right-Ventricular Ejection Fraction

The ejection fraction is the fraction of the ventricular volume that is ejected
during systole and is equivalent to the ratio of the stroke volume and the
ventricular end-diastolic volume. This parameter provides an indication
of the strength of ventricular contraction during systole. The ejection
fraction of the right ventricle (RVEF) is the ratio of the stroke volume (SV)
to the right-ventricular end-diastolic volume (RVEDV).

\[
RVEF = \frac{SV}{RVEDV}
\]  

As mentioned earlier, the right ventricular ejection fraction can be
measured with a specialized PA catheter that is equipped with a rapid-
response thermistor (see Reference 10 for a description of this tech-
nique).

Right-Ventricular End-Diastolic Volume

Ventricular end-diastolic volume is the true measure of ventricular pre-
load (see Chapter 1). The end-diastolic volume of the right ventricle can
be determined when the RVEF is measured using the specialized PA catheter mentioned above. The equation below is derived by rearranging the terms in Equation 9.6.

\[
RVEDV = \frac{SV}{RVEF}
\]  
(9.7)

**Left-Ventricular Stroke Work Index**

Left-ventricular stroke work (LVSW) is the work performed by the ventricle to eject the stroke volume. Stroke work is a function of the systolic pressure load (afterload minus preload), which is equivalent to the mean arterial pressure minus the wedge pressure (MAP - PCWP) and the stroke volume (SV). The equation below is corrected for body size (so LVSW becomes LVSWI), and the factor 0.0136 converts pressure and volume to units of work.

\[
LVSWI = \frac{(MAP - PCWP) \times SVI \times 0.0136}{(9.8)}
\]

**Right-Ventricular Stroke Work Index**

The right-ventricular stroke work (RVSW) is the work needed to move the stroke volume across the pulmonary circulation. It is derived as the systolic pressure load of the right ventricle, which is equivalent to the mean pulmonary artery pressure minus the CVP (PAP - CVP), and the stroke volume (SV). The equation below is corrected for body size and includes the same unit correction factor as in Equation 9.8.

\[
RVSWI = \frac{(PAP - CVP) \times SVI \times 0.0136}{(9.9)}
\]

**Systemic Vascular Resistance Index**

The systemic vascular resistance (SVR) is the vascular resistance across the systemic circulation. It is directly proportional to the pressure gradient from the aorta to the right atrium (MAP - CVP) and is inversely related to blood flow (CI). The equation below is corrected for body size, and the factor of 80 is necessary to convert units.

\[
SVRI = \frac{(MAP - RAP) \times 80}{CI}
\]  
(9.10)

**Pulmonary Vascular Resistance Index**

The pulmonary vascular resistance index (PVRI) is directly proportional to the pressure gradient across the entire lungs, from the pulmonary artery (PAP) to the left atrium (LAP). Because the wedge pressure (PCWP) is equivalent to the LAP, the pressure gradient across the lungs can be expressed as (PAP - PCWP). The PVRI can then be derived using Equation 9.11, which is corrected for body size. As in Equation 9.10, the factor of 80 is used to convert units.

\[
PVRI = \frac{(PAP - PCWP) \times 80}{CI}
\]  
(9.11)

**Oxygen-Transport Parameters**

The transport of oxygen from the lungs to the systemic organs is described using the parameters in Table 9.2. These parameters are described in detail in Chapter 2 and are presented only briefly here.

**Oxygen Delivery**

The rate of oxygen transport in arterial blood is called the oxygen delivery (DO₂) and is a function of the cardiac output and the oxygen concentration in arterial blood. The determinants of DO₂ are shown in Equation 9.12. This equation is explained in Chapter 2.

\[
DO₂ = CI \times 13.4 \times Hb \times \text{SaO}_2
\]  
(9.12)

**Mixed Venous Oxygen Saturation**

The oxygen saturation of hemoglobin in pulmonary artery (mixed venous) blood (SvO₂) can be monitored continuously with a specialized PA catheter, or it can be measured in vitro with a blood sample obtained from the distal port of the PA catheter. (See Chapter 20 for a description of how O₂ saturation of hemoglobin is measured.) The SvO₂ is used as an indirect marker of systemic blood flow. A decrease in cardiac output is accompanied by an increase in O₂ extraction from the capillaries, and this will decrease the SvO₂. Therefore a decrease in SvO₂ can signal a decrease in cardiac output. If O₂ extraction is fixed and does not vary with changes in blood flow (which can happen in sepsis), the SvO₂ is unreliable as an index of blood flow.

**Oxygen Uptake**

Oxygen uptake (VO₂), also called oxygen consumption, is the rate at which oxygen is taken up from the systemic capillaries into the tissues. The determinants of VO₂ are shown in Equation 9.13. This equation is explained in detail in Chapter 2.

\[
VO₂ = CI \times 13.4 \times Hb \times (\text{SaO}_2 - \text{SvO}_2)
\]  
(9.13)

**Oxygen-Extraction Ratio**

The oxygen extraction ratio (O₂ ER) is the fractional uptake of oxygen from the systemic microcirculation and is equivalent to the ratio of O₂ uptake to O₂ delivery. Multiplying the ratio by 100 expresses it as a percent.

\[
O₂ \text{ ER} = \frac{VO₂}{DO₂} \times 100
\]  
(9.14)

**HEMODYNAMIC SUBSETS**

The parameters just described can be organized into groups or subsets that are tailored to specific problems. Some examples of hemodynamic subsets are presented below.
Hypotension

The mean arterial pressure is a function of the cardiac output and the systemic vascular resistance: MAP = CI \times SVRI. The cardiac output, in turn, depends on the venous return. If the CVP is used as an index of venous return, there are three variables that can be used to describe any patient with hypotension: CVP, CI, and SVRI. This 3-variable subset is used below to describe the three classic forms of hypotension.

<table>
<thead>
<tr>
<th>Hypovolemic</th>
<th>Cardiogenic</th>
<th>Vasogenic</th>
</tr>
</thead>
<tbody>
<tr>
<td>Low CVP</td>
<td>High CVP</td>
<td>Low CVP</td>
</tr>
<tr>
<td>Low CI</td>
<td>Low CI</td>
<td>High CI</td>
</tr>
<tr>
<td>High SVRI</td>
<td>High SVRI</td>
<td>Low SVRI</td>
</tr>
</tbody>
</table>

These three hemodynamic parameters can be used to identify the hemodynamic problem in any patient with hypotension. For example, suppose a patient with hypotension has a low CVP, a normal CI, and a low SVRI. This pattern is closest to the vascular dysfunction (vasogenic) category shown above, except that the CI is normal instead of high. Therefore the hemodynamic problem in this patient is a combination of vascular dysfunction and cardiac dysfunction. There are 3^3 or 27 possible combinations of these 3 variables (CVP, CI, SVRI), and each of these combinations identifies a distinct hemodynamic problem. Therefore this hemodynamic subset of 3 variables will identify the hemodynamic problem in any patient with hypotension.

Clinical Shock

The three-variable hemodynamic subset just presented will identify a hemodynamic problem but not the consequences of the problem on tissue oxygenation. The addition of the oxygen uptake (VO₂) will correct this shortcoming and can help identify a state of clinical shock. Clinical shock can be defined as a condition where tissue oxygenation is inadequate for the needs of aerobic metabolism. Since a VO₂ that is below normal can be used as indirect evidence of oxygen-limited aerobic metabolism, a subnormal VO₂ can be used as indirect evidence of clinical shock. The following example shows how the VO₂ can add to the evaluation of a patient with a low output state.

<table>
<thead>
<tr>
<th>Heart Failure</th>
<th>Cardiogenic Shock</th>
</tr>
</thead>
<tbody>
<tr>
<td>High CVP</td>
<td>High CVP</td>
</tr>
<tr>
<td>Low CI</td>
<td>Low CI</td>
</tr>
<tr>
<td>High SVRI</td>
<td>High SVRI</td>
</tr>
<tr>
<td>Normal VO₂</td>
<td>Low VO₂</td>
</tr>
</tbody>
</table>

Without the VO₂ measurement in the above profiles, it is impossible to differentiate a low-output state from cardiogenic shock. This illustrates how oxygen transport monitoring can be used to determine the consequences of hemodynamic abnormalities on peripheral oxygenation. The uses and limitations of oxygen transport monitoring are described in more detail in Chapter 11.

A FINAL WORD

The PA catheter has been maligned in recent years because of clinical studies showing that mortality is not reduced (26) and can be higher (27) in patients who have PA catheters. As a result of these studies, use of the PA catheter in the western world has dropped about 10% in the past few years (28), and the most zealous critics of the catheter have called for a moratorium on its use.

There are two fundamental problems in the criticism of the PA catheter based on mortality data. The first is the simple fact that the "PA catheter is a monitoring device, not a therapy." If a PA catheter is placed to evaluate a problem, and it uncovers a disorder that is untreatable and fatal (e.g., cardiogenic shock), the problem is not the catheter, it is the lack of effective therapy. Mortality rates should be used to evaluate therapies, not measurements.

The second problem is the seemingly prevalent notion that everything we do in the ICU must save lives to be of value. Mortality should not be the dominant outcome measure in ICUs because there are too many variables that can influence mortality in critically ill patients, and also because mortality is an eventual outcome in all patients admitted to the ICU. Management decisions should be based on the scientific rationale for an intervention—those who expect their management decisions to consistently save lives are doomed to failure.

REFERENCES

Reviews


Selected References

CENTRAL VENOUS PRESSURE AND WEDGE PRESSURE

It is what we think we know already that often prevents us from learning.

Claude Bernard

The central venous pressure (CVP) and pulmonary artery occlusion (wedge) pressure, which are clinical measures of right and left ventricular filling pressures, respectively (1-3), have been popularized as hemodynamic measures because of the Frank-Starling relationship of the heart, which identifies ventricular filling volume (preload) as the major determinant of cardiac stroke output (see Figure 1.1). Unfortunately, the CVP and pulmonary artery wedge pressure share two major shortcomings: they are often misleading as measures of ventricular preload (4), and the pressure waveforms are often misinterpreted (5-7). Attention to the information in this chapter will help reduce errors in the interpretation of these measurements.

SOURCES OF VARIABILITY

Body Position

The zero reference point for venous pressures in the thorax is a point on the external thorax where the fourth intercostal space intersects the mid-axillary line (i.e., the line midway between the anterior and posterior axillary folds). This point (called the phlebostatic axis) corresponds to the position of the right and left atrium when the patient is in the supine position. It is not a valid reference point in the lateral position, which means that central venous and pulmonary artery wedge pressures should not be recorded when patients are placed in lateral positions (8).
Changes in Thoracic Pressure

The pressure recorded with a vascular cannula is the intravascular pressure [i.e., the pressure in the vessel lumen relative to atmospheric (zero) pressure]. However, the physiologically important vascular pressure (i.e., the one that determines distention of the vessels and the rate of edema formation) is the transmural pressure [i.e., the difference between the intravascular and extravascular pressures]. The intravascular pressure is an accurate reflection of the transmural pressure only when the extravascular pressure is zero (atmospheric pressure).

When vascular pressures are recorded in the thorax, changes in thoracic pressure can be transmitted across the wall of blood vessels, resulting in a discrepancy between intravascular and transmural pressures. This is illustrated by the respiratory variations in the CVP tracing shown in Figure 10.1. The transmural pressure changes in this tracing are caused by respiratory variations in intrathoracic pressure that are transmitted into the lumen of the superior vena cava. In this situation, the transmural pressure (i.e., the cardiac filling pressure) may be constant despite the phasic changes in intravascular pressure. (It is not possible to determine how much of the change in thoracic pressure is transmitted into the blood vessel in an individual patient, and thus it is not possible to determine whether transmural pressure is absolutely constant.) Thus respiratory variation in intravascular pressures in the thorax is not an indication that the transmural pressure (the cardiac filling pressure) is also changing (9).

End-Expiration

Intravascular pressures will be equivalent to transmural pressures when the extravascular pressure is zero. In healthy subjects breathing at normal rates, this occurs at the end of expiration, when intrathoracic (extravascular) pressure returns to atmospheric or zero pressure. Therefore intravascular pressures should be measured at the end of expiration, when they are equivalent to the transmural pressure (1,9). Intravascular and transmural pressures will differ at end-expiration only if there is positive intrathoracic pressure at the end of expiration, as explained next.

Positive End-Expiratory Pressure (PEEP). There are two situations, where the intrathoracic pressure is above atmospheric pressure at the end of expiration. In one situation, positive end-expiratory pressure (PEEP) is applied during mechanical ventilation to prevent alveolar collapse. In the other situation, incomplete alveolar emptying (e.g., due to airflow obstruction) does not allow alveolar pressure to return to atmospheric pressure at the end of expiration. These two conditions are referred to as extrinsic PEEP (see Chapter 25) and intrinsic PEEP (see Chapter 26), respectively. In both conditions of PEEP, intravascular pressures measured at the end of expiration will exceed the transmural pressure.

When external PEEP is applied, intravascular pressures should be measured at end-expiration when the patient is briefly disconnected from the ventilator (10). In the presence of intrinsic PEEP, accurate recording of intravascular pressures can be difficult (11). See Chapter 26 for a description of how the CVP and wedge pressure can be corrected in the presence of intrinsic PEEP.

Pressure Monitors

If the bedside monitors in the ICU have oscilloscope display screens with horizontal grids, the CVP and wedge pressures should be measured directly from the pressure tracings on the screen. This provides more accurate measurements than pressures that are digitally displayed (12). Most ICU monitors have a digital display that includes systolic, diastolic, and mean pressures; each measured over successive 4-second time intervals (the time for one sweep across the oscilloscope screen). The systolic pressure is the highest pressure, the diastolic pressure is the lowest pressure, and the mean pressure is the integrated area under the pressure wave in each time period. During spontaneous breathing, the pressure at the end of expiration is the highest pressure (i.e., systolic pressure), and during positive-pressure mechanical ventilation, the end-expiratory pressure is the lowest pressure (i.e., diastolic pressure). Therefore systolic pressure should be used as the end-expiratory vascular pressure in patients who are breathing spontaneously, whereas diastolic pressure should be used in patients receiving positive-pressure mechanical ventilation. The mean pressure should never be used as a reflection of transmural pressure when there are respiratory variations in intravascular pressure (9).

Units of Measurement

Most intravascular pressures are measured with electronic transducers that record the pressure in millimeters of mercury (mm Hg). Water-filled manometers that record pressure in cm H₂O are occasionally used to measure CVP (13). Because mercury is 13.6 times more dense than water, pressures measured in cm H₂O can be divided by 13.6 × 1/10 = 1.36 to be expressed in mm Hg (the factor 1/10 converts cm to mm).
pressure in cm H₂O ÷ 1.36 = pressure in mm Hg. A table of conversions for these units is included in Appendix 1.

Spontaneous Variations
Like any physiologic variable, vascular pressures in the thorax can vary spontaneously, without a change in the clinical condition of the patient. The spontaneous variation in wedge pressure is 4 mm Hg or less in 60% of patients, but it can be as high as 7 mm Hg in any individual patient (14). In general, a change in CVP or wedge pressure of less than 4 mm Hg should not be considered a clinically significant change.

PULMONARY ARTERY WEDGE PRESSURE

Few pressures in the ICU are misinterpreted as frequently, and as consistently, as pulmonary capillary wedge pressure (5-7,15). Probably the most important feature of the wedge pressure is what it is not:

Wedge pressure is not left-ventricular preload.
Wedge pressure is not the pulmonary capillary hydrostatic pressure.
Wedge pressure is not a reliable measure for differentiating cardiogenic from noncardiogenic pulmonary edema.

These limitations are explained in the description of the wedge pressure that follows.

Wedge Pressure Tracing
When the pulmonary artery catheter is properly positioned, inflation of the balloon at the tip of the catheter causes the pulsatile pressure to disappear. This is demonstrated in Figure 10.2. The nonpulsatile or “wedged” pressure is equivalent to the pulmonary artery diastolic pressure, and represents the pressure in the venous side of the pulmonary circulation. The magnified section of the wedge pressure in Figure 10.2 shows the individual components of the pressure: the a wave is produced by left atrial contraction, the c wave is produced by closure of the mitral valve during isometric contraction of the left ventricle, and the v wave is produced by systolic contraction of the left ventricle against a closed mitral valve. These components (which are also present in the central venous pressure tracing) are often not distinguishable in a normal wedge pressure tracing, but they can become evident in conditions where one component is magnified (e.g., mitral regurgitation produces large v waves, which can be identified in a wedge pressure tracing).

Principle of the Wedge Pressure
The wedge pressure is a measure of the filling pressure in the left side of the heart, and the basis for this is shown in Figure 10.3 (13). Inflation of the balloon at the tip of pulmonary artery catheter creates a static column of blood between the catheter tip and the left atrium. In this

FIGURE 10.2 Pressure tracing showing the transition from a pulsatile pulmonary artery pressure to a balloon occlusion (wedge) pressure. The magnified area shows the components of the wedge pressure: a wave (atrial contraction), c wave (mitral valve closure), and v wave (ventricular contraction).

FIGURE 10.3 The principle of the wedge pressure measurement. When flow ceases because of balloon inflation (Q = 0), the pressure at the catheter tip (Pc) is the same as the pressure in the left atrium (PLa). This occurs only in the most dependent lung zone. The lung is divided into three zones based on the relationship between alveolar pressure (Ppa), mean pulmonary artery pressure (Pc), and pulmonary capillary pressure (Pc). Wedge pressure is an accurate reflection of left-atrial pressure only in zone 3, where Ppa is greater than Psa.
situation, the pressure at the tip of the pulmonary artery catheter is the same as the pressure in the left atrium. This can be demonstrated using the simple hydraulic relationship \( Q = \Delta P/R \), which indicates that steady flow in a tube \((Q)\) is directly proportional to the pressure drop along the tube \((\Delta P)\) and is inversely proportional to the resistance to flow in the tube \((R)\). Rearranging terms yields the following relationship: \( \Delta P = Q \times R \). This relation is expressed below for the venous side of the pulmonary circulation, where \( P_c \) is capillary pressure, \( P_{LA} \) is left-atrial pressure, \( Q \) is pulmonary blood flow, and \( R_v \) is pulmonary venous resistance.

\[
P_c - P_{LA} = Q \times R_v \tag{10.1}
\]

if \( Q = 0 \), \( P_c - P_{LA} = 0 \), and \( P_c = P_{LA} \)

Thus when the balloon is inflated, the pressure at the tip of the pulmonary artery catheter \((P_c)\) is equal to the pressure in the left atrium \((P_{LA})\). Because left-atrial pressure is normally the same as the left-ventricular end-diastolic pressure (LVEDP), the pulmonary capillary wedge pressure can be used as a measure of left-ventricular filling pressure. What the wedge pressure actually measures is the focus of the remainder of this chapter.

**Wedge Pressure as Preload**

The wedge pressure is often used as a reflection of left-ventricular filling during diastole (i.e., ventricular preload). In Chapter 1, preload was defined as the force that stretches a muscle at rest, and the preload for the intact ventricle was identified as end-diastolic volume (EDV). However, the pulmonary capillary wedge pressure (like the CVP) is a measure of end-diastolic pressure, and end-diastolic pressure may not be an accurate reflection of preload (EDV). The graph in Figure 10.4 shows the relationship between pulmonary capillary wedge pressure and left-ventricular end-diastolic volume in a group of normal subjects (4). Note the poor correlation between the two measurements \((r = 0.04)\). In fact, only 7 of the 12 wedge pressure measurements (58%) are within the normal range (shaded area). This shows that the pulmonary artery wedge pressure is not an accurate reflection of left-ventricular preload (4,16). Similar results have also been reported with the central venous pressure (4).

**Wedge Pressure as Left-Atrial Pressure**

The following conditions can influence the accuracy of the wedge pressure as a measure of left-atrial pressure.

**Lung Zones**

If the pressure in the surrounding alveoli exceeds capillary (venous) pressure, the pressure at the tip of the pulmonary artery catheter may reflect the alveolar pressure rather than the left-atrial pressure. This is illustrated in Figure 10.3. The lung in this figure is divided into three zones based on the relationship between alveolar pressure and the pressures in the pulmonary circulation (1,3). The most dependent lung zone (zone 3) is the only region where capillary (venous) pressure exceeds alveolar pressure. Therefore, wedge pressure is a reflection of left-atrial pressure only when the tip of the pulmonary artery catheter is located in zone 3 of the lung.

**Catheter-tip Position**

Although the lung zones shown in Figure 10.3 are based on physiologic rather than anatomic criteria, the lung regions below the left atrium are considered to be in lung zone 3 (1,3). Therefore the tip of the pulmonary artery catheter should be positioned below the level of the left atrium to ensure that the wedge pressure is measuring left-atrial pressure. Because of the higher blood flow in dependent lung regions, most pulmonary artery catheters are advanced into lung regions below the level of the left atrium. However, as many as 30% of PA catheters are positioned with
TABLE 10.1 Criteria for Wedge Pressure Validation

<table>
<thead>
<tr>
<th>Condition</th>
<th>Criteria</th>
</tr>
</thead>
<tbody>
<tr>
<td>Wedge PO₂</td>
<td>Arterial PO₂ ≥ 19 mm Hg</td>
</tr>
<tr>
<td>Arterial PCO₂</td>
<td>Wedge PCO₂ ≥ 11 mm Hg</td>
</tr>
<tr>
<td>Wedge pH</td>
<td>Arterial pH ≥ 0.008</td>
</tr>
</tbody>
</table>


their tips above the level of the left atrium (3). When patients are supine, routine portable (anteroposterior) chest x-rays cannot be used to identify the catheter-tip position relative to the left atrium. Rather, a lateral view of the chest is needed. An alternative approach is to assume that catheter tips are in zone 3 of the lung in all but the following conditions: when there are marked respiratory variations in the wedge pressure and when PEEP is applied and wedge pressure increases by 50% or more of the applied PEEP (3).

Wedge Blood Gases

As many as 50% of the nonpulsatile pressures produced by balloon inflation represent damped pulmonary artery pressures rather than pulmonary capillary wedge pressures (17). Aspiration of blood from the catheter tip during balloon inflation can be used to identify a true wedge (capillary) pressure using the three criteria shown in Table 10.1. Although this is a cumbersome practice that is not used routinely, it seems justified when making important diagnostic and therapeutic decisions based on the wedge pressure measurement.

Wedge Pressure as Left-Ventricular End-Diastolic Pressure

Even when wedge pressure is an accurate reflection of left-atrial pressure, there may be a discrepancy between left-atrial pressure and left-ventricular end-diastolic pressure (LVEDP). This can occur under the following conditions (3).

- **Aortic insufficiency**: LVEDP can be higher than PCWP because the mitral valve closes prematurely while retrograde flow continues to fill the ventricle.

- **Noncompliant ventricle**: Atrial contraction against a stiff ventricle produces a rapid rise in end-diastolic pressure that closes the mitral valve prematurely. The result is a PCWP that is lower than the LVEDP.

- **Respiratory failure**: PCWP can exceed LVEDP in patients with pulmonary disease. The presumed mechanism is constriction of small veins in lung regions that are hypoxic (18).

Wedge Pressure as Capillary Hydrostatic Pressure

The wedge pressure is often assumed to be a measure of hydrostatic pressure in the pulmonary capillaries. The problem with this assumption is the fact that the wedge pressure is measured in the absence of blood flow. When the balloon is deflated and flow (Q) resumes, the pressure in the pulmonary capillaries will remain the same as the left-atrial (wedge) pressure only if the resistance to flow in the pulmonary veins is negligible. This is expressed below, where \( P_c \) is capillary hydrostatic pressure, \( R_v \) is the hydraulic resistance in the pulmonary veins, \( Q \) is blood flow, and wedge pressure (PCWP) is substituted for left-atrial pressure.

\[
P_c - PCWP = Q \times R_v
\]

If \( R_v = 0 \), \( P_c - PCWP = 0 \), and \( P_c = PCWP \).

**Pulmonary Venous Resistance**

Unlike the systemic veins, the pulmonary veins contribute a significant fraction to the total vascular resistance across the lungs. (This is a reflection more of a low resistance in the pulmonary arteries than of a high resistance in the pulmonary veins.) As shown in Figure 10.5, 40% of the pressure drop across the pulmonary circulation occurs on the venous side of the circulation, which means that the pulmonary veins contribute 40% of the total resistance in the pulmonary circulation (19). Although this is derived from animal studies, the contribution in humans is probably similar in magnitude.

The contribution of the hydraulic resistance in the pulmonary veins may be even greater in critically ill patients because several conditions that are common in ICU patients can promote pulmonary venuconstriction. These conditions include hypoxemia, endotoxemia, and the acute respiratory distress syndrome (18,20). These conditions further magnify differences between wedge pressure and capillary hydrostatic pressure, as demonstrated below:

**FIGURE 10.5.** The distinction between capillary hydrostatic pressure (\( P_c \)) and wedge pressure (PCWP). When the balloon is deflated and flow (Q) resumes, \( P_c \) and PCWP are equivalent only when the hydraulic resistance in the pulmonary veins (\( R_v \)) is negligible. \( P_c \) = pulmonary artery pressure. If the pulmonary venous resistance (\( R_v \)) is greater than zero, the capillary hydrostatic pressure (\( P_c \)) will be higher than the wedge pressure.
Wedge–Hydrostatic Pressure Conversion

Equation 10.3 can be used to convert wedge pressure (PCWP) to pulmonary capillary hydrostatic pressure (Pc). This conversion is based on the assumption that the pressure drop from the pulmonary capillaries to the left atrium (Pc - PLa) represents 40% of the pressure drop from the pulmonary arteries to the left atrium (Pa - PLa). Substituting wedge pressure for left-atrial pressure (i.e., PLa = PCWP) yields the following relationship:

\[
P_c - PCWP = 0.4 \times (P_a - PCWP)
\]

\[
P_c = PCWP + 0.4 \times (P_a - PCWP)
\]  \hspace{1cm} (10.3)

For a normal (mean) pulmonary artery pressure of 15 mm Hg and a wedge pressure of 10 mm Hg, this relationship predicts the following:

Normal lung: \( P_c = 10 + 0.4 \times (15 - 10) \) \hspace{1cm} (10.4)

\( P_c = 12 \text{ mm Hg}, P_c - PCWP = 2 \text{ mm Hg}. \)

Thus in the normal lung, wedge pressure is equivalent to capillary hydrostatic pressure. However, in the presence of pulmonary venoconstriction and pulmonary hypertension (e.g., in acute respiratory distress syndrome), there can be a considerable difference between wedge pressure and capillary hydrostatic pressure. The example below is based on a mean PA pressure of 30 mm Hg and a venous resistance that is 60% of the total pulmonary vascular resistance.

ARDS: \( P_c = 10 + 0.6 \times (30 - 10) \) \hspace{1cm} (10.5)

\( P_c = 22 \text{ mm Hg}, P_c - PCWP = 12 \text{ mm Hg}. \)

Unfortunately, pulmonary venous resistance cannot be measured in critically ill patients, and this limits the accuracy of the wedge pressure as a measure of capillary hydrostatic pressure.

Occlusion Pressure Profile

The transition from pulsatile pulmonary artery pressure to nonpulsatile wedge pressure in Figure 10.6 shows an initial rapid phase followed by a slower, more gradual pressure change. The initial rapid phase may represent the pressure drop across the pulmonary arteries, while the slower phase may represent the pressure drop across the pulmonary veins. If this is the case, the inflection point marking the transition from the rapid to the slow phase represents the capillary hydrostatic pressure. Unfortunately, inflection points are often not recognizable following balloon occlusion (21,22).

A FINAL WORD

Despite their popularity, the central venous pressure and pulmonary artery wedge pressure provide limited and often misleading information about intravascular volume, cardiac filling volumes, and capillary hydrostatic pressure. What this means is that these pressures should **not** be used (at least in isolation) to determine if a patient is dehydrated or fluid overloaded (23), and the wedge pressure should not be used to diagnose hydrostatic pulmonary edema. The pulmonary artery catheter provides much more important measurements, particularly cardiac output and systemic oxygen transport variables, and these, together with other methods of assessing tissue oxygenation (see next chapter) make the CVP and wedge pressures outdated measures that are not necessary in the hemodynamic assessment of critically ill patients.

REFERENCES

Reviews

Selected References