The purposes of hemodynamic monitoring are to characterize the cardiovascular state of the individual, identify cardiovascular insufficiency and its most probable causes, and monitor response to targeted therapies aimed at restoring cardiovascular sufficiency.\(^1\)\(^2\) The previous chapters outlined the various forms of circulatory shock. It is within this physiological framework that interpretation of data derived from hemodynamic monitoring arises. Circulatory shock and systemic hypotension are medical emergencies, because if sustained, even for a short time, they will result in end-organ dysfunction and increased morbidity and mortality. The basic tenet of resuscitation is to provide adequate oxygen (O\(_2\)) delivery (DO\(_2\)) to meet metabolic demand and reverse any existing tissue hypoperfusion. Accordingly, the choice of monitoring technique must be individualized for each patient. In general, noninvasive continuous monitoring, if available and accurate, is preferred to invasive intermittent monitoring. In reality, some degree of invasiveness of monitoring is often required to accurately assess the physiological data in a continuous fashion in the monitoring and management of the critically ill patient.

The principal hemodynamic monitoring biomarkers discussed in this chapter are arterial pressure, central venous pressure, pulmonary artery pressure and its occlusion pressure, estimates of cardiac output (CO), and the various ways of assessing oxygenation. Like all vascular monitoring using fluid-filled catheters connected to electronic pressure transducers, hemodynamic monitoring
requires an open tubing system without obstruction at the tip (often due to blood clots), elimination of air bubbles in the tubing that dampen the signal, and hydrostatic zeroing to the isobstetic point (5 cm below the manubrium sterni) in order to measure dynamic and mean pressure and arterial pressure-derived estimates of CO. However, unlike other vascular pressure measures, arterial pressure has large pressure swings associated with rapid acceleration and deceleration, and these demand that stiff and short tubing be used to connect the intra-arterial catheter to the pressure transducer. The technical aspects of measuring pressures and pressure-derived CO are beyond the scope of this chapter. Similarly, measures of CO by indicator dilution require complete indicator mixing, no early recirculation (eg, intracardiac shunts), and an appropriate sensor. All commercially available indicator dilution devices have sufficiently accurate sensors, so most measurement errors come from incomplete indication mixing or early recirculation artifacts.

**ARTERIAL BLOOD PRESSURE**

Arterial blood pressure is the primary force driving blood into the tissues. Thus, hypotension is a medical emergency because it not only causes tissue hypoperfusion but also effectively abolishes normal autoregulation of blood flow distribution. Furthermore, owing to baroreceptor feedback and the increased use of β-adrenergic blocking agents, often neither tachycardia nor hypotension occurs in shock until the final terminal stages.

Blood pressure varies phasically with each heartbeat. Systolic pressure is the maximum pressure during ventricular ejection, and diastolic pressure is the lowest pressure in the blood vessels between heartbeats during ventricular filling as the stored arterial blood runs off into the periphery. The systolic to diastolic pressure difference is called the pulse pressure and is determined by left ventricular (LV) stroke volume, central arterial capacitance, and to a certain extent the rate of LV ejection. Since the arterial circuit is elastic and functions as both a capacitor for the ejected blood and an outflow resistor to prevent rapid decreases in arterial pressure in diastole, both systolic and diastolic pressures vary across the vascular tree. Systolic pressure usually increases from central to peripheral sites whereas diastolic pressure decreases slightly. Mean arterial pressure (MAP), estimated as the sum of diastolic pressure plus one-third of the pulse pressure, is the primary driving pressure for cerebral and peripheral organ perfusion. Coronary perfusion is primarily determined by diastolic arterial pressure because cardiac contraction during systole otherwise stops intramyocardial blood flow. Importantly, MAP throughout the large to medium-sized arteries is constant because these central arteries have almost no measurable resistance.

**Noninvasive Measures of Arterial Pressure**

The most common way of measuring arterial pressure is with a sphygmomanometer. This is often automated using an oscillatory algorithm that senses flows. There is a fundamental distinction between the automatic blood pressure measures (eg, Dynamat) and auscultation-defined measures of blood pressure using Korotkoff sounds. In an unstable patient, one should not rely on noninvasive automatic measures of blood pressure, because the oscillatory sensing algorithm degrades, but rather should measure blood pressure manually using a stethoscope. Sphygmomanometric measurements of blood pressure often give slightly higher systolic pressure and lower diastolic pressure than those reported from simultaneous direct measurement using an intra-arterial catheter. This is because with cuff inflation the reflected pressure waves summate, increasing systolic pressure, whereas the ischemic vasodilation downstream from the occluded cuff decreases cuff-opening diastolic pressure.

**Invasive Measures of Arterial Pressure**

Intra-arterial catheterization is the reference method for blood pressure measurement and should be used in all hemodynamically unstable patients in whom accurate and continuous measures of arterial pressure are required. The reason for this statement, which may be at odds with prior recommendations, is that intra-arterial catheterization provides instantaneous measures of MAP.
arterial pulse pressure, pulse pressure variation, and CO with newer transducer technologies. The arterial catheter allows easily repeated blood sampling for chemistries and blood gas analysis. The most frequently used site for arterial catheterization is the radial artery. Femoral artery catheterization is also used and can be easier to perform in hypotensive patients, although it is associated with more complications when the catheter is left in place. Still, in the profoundly vasoconstricted patient, radial arterial pressure can underestimate central arterial pressure measured more proximally.9

**Arterial Pressure Targets**

Once one measures arterial pressure, the major question is the target range of pressures to be maintained in order to sustain organ perfusion without excessively increasing LV afterload. Hypotension decreases organ perfusion pressure and blood flow, stimulating a sympathetic response to increase vasomotor tone, heart rate, and contractility. The change in local vasomotor tone in response to decreased arterial pressure forms the basis of autoregulation to maintain constant blood flow. Cardiac output is important to sustain an adequate and changing blood flow to match changes in vasomotor tone such that arterial pressure to the organ remains optimal. Since CO is proportional to metabolic demand, which itself can vary 3-fold in a matter of minutes, there is essentially no normal CO in the critically ill patient. However, as MAP decreases below 65 mm Hg in a previously nonhypertensive patient, organ perfusion becomes compromised. Thus, a reasonable arterial pressure target is a MAP between 90 and 60 mm Hg,10 although the optimal MAP will vary depending on the underlying cause of hemodynamic instability.11

To assess the adequacy of MAP to sustain peripheral perfusion, the bedside clinician needs to assess additional measures of organ perfusion, such as mixed venous O₂ saturation (SvO₂), central venous O₂ saturation (ScvO₂), lactic acid levels, urine output, capillary blood flow, or gastric mucosal Pco₂. The first 2 of these parameters are discussed further subsequently. Finally, cerebral perfusion is a function of MAP relative to intracranial pressure. Thus, in the setting of neurotrauma cerebral perfusion pressure must be measured. As a general rule one should target a MAP of 90 mm Hg for traumatic brain injury12 and a MAP greater than 65 mm Hg for other forms of shock.

**CENTRAL VENOUS PRESSURE**

Although central venous access is often used as a secure venous access site for infusion of fluids and vasoactive drugs as well as sampling of blood for various things including Scvo₂, its use in assessing intravascular volume status is poor. Central venous pressure (CVP) is not a measure of central blood volume nor can its values be used to determine whether a patient will be volume responsive.13,14 Still, dynamic decreases in CVP of greater than 2 mm Hg during spontaneous inspiration appear to identify those patients who are volume responsive independent of the absolute CVP value.15 CVP is the back pressure to systemic venous return; thus, high CVP values (>12 mm Hg) indicate a larger than normal mean systemic pressure allowing an adequate perfusion pressure gradient to sustain venous return.16 However, CVP in and of itself does not reflect blood volume status. From the perspective of patient safety, the insertion of a central venous catheter using ultrasound guidance for internal jugular vein insertion has also become part of standard care and has markedly reduced complications.17

**Noninvasive Measures of CVP**

Central venous pressure can be estimated noninvasively by inspection of jugular venous pulsation. With the patient sitting at 45°, the height of the jugular venous distention (JVD) above the sternal angle (itself about 5 cm above the center of the right atrium) can be used to estimate CVP.18 Potentially, this approach is most useful in documenting sustained increases in JVD on phase increases in JVD with spontaneous inspiration, suggesting cor pulmonale or pulmonary hypertension, and periodic cannon waves seen in atrial fibrillation.

**Invasive Measures of CVP**

Although CVP is usually measured from the internal jugular or subclavian vein via an indwelling catheter, it can be estimated from a femoral venous site as long as there
is no intra-abdominal hypertension (ie, intra-abdominal pressure <12 mm Hg). Using the femoral site for a central venous catheter is also associated with a greater incidence of complications. \( ^{30} \) The effects of the respiratory cycle on all intrathoracic vascular pressures must be considered when examining a continuous CVP measurement. To minimize the pressure artifact induced by respiratory pressure changes, all intrathoracic vascular pressures should be measured at end-expiration. In the dyspneic patient or a patient who is fighting the ventilator, determination of end-expiration can be very difficult, if not impossible. Extreme caution needs to be used when estimating CVP under these conditions.

### PULMONARY ARTERY PRESSURE AND PULMONARY ARTERY OCCLUSION PRESSURE

Bedside balloon flotation insertion of a pulmonary artery catheter and subsequent measurement of right atrial, pulmonary arterial, and pulmonary artery occlusion pressure (Ppaoo) plus CO and mixed venous O\(_2\) saturation defined hemodynamic monitoring in critical care for more than 40 years. \( ^{21} \) Pulmonary artery pressure is measured from the tip of a nonoccluded pulmonary artery catheter once this catheter has been floated past the pulmonic valves into the main pulmonary arteries. Mean pulmonary artery pressure measures can be calculated similarly to MAP, as described previously, with the proviso that they are measured at end-expiration. Thus, one can measure pulmonary artery systolic, diastolic, and mean pressures. Mean pulmonary artery pressure is usually used to assess input pulmonary vascular pressure for calculating pulmonary vascular resistance (PVR), whereas systolic pulmonary artery pressure reflects the afterload against which the right ventricle ejects.

By balloon inflation and migration of the catheter tip into a medium-sized pulmonary artery where it is occluded, one can measure Ppaoo. Pulmonary artery occlusion pressure is used most often to assess PVR, pulmonary edema, intravascular volume status and LV preload, and LV performance. \( ^{32,23} \)

### Pulmonary Hypertension and Pulmonary Vascular Resistance

Increased pulmonary arterial pressure impedes right ventricular (RV) ejection, causing RV dilatation and a decreased CO. If pulmonary hypertension occurs rapidly, as with massive pulmonary embolism or marked hyperinflation, acute cor pulmonale and cardiovascular collapse occur. Pulmonary hypertension can be due to an increase in pulmonary vasomotor tone, pulmonary vascular obstruction, or passive increases in Ppaoo due to LV failure. The pulmonary circulation normally has a low resistance, with pulmonary arterial diastolic pressure only slightly higher than Ppaoo. By measuring pulmonary artery pressure, Ppaoo, and CO, one can calculate PVR as the ratio of the pressure gradient divided by flow: (mean pulmonary artery pressure – Ppaoo)/CO. If PVR is increased, then therapies to reduce pulmonary artery pressure are needed (eg, \( O_2 \) inhaled nitric oxide, intravenous pulmonary vasodilator therapy). Patients with pulmonary hypertension who have increased PVR that is not responsive to vasodilator therapy usually have either vascular obstruction (eg, pulmonary embolism), or vascular loss (eg, emphysema). If pulmonary hypertension is associated with a normal PVR, then LV failure is its probable cause.

Unfortunately, PVR is a poor measure of pulmonary vasomotor tone. Pulmonary vascular pressure can vary across lung regions because of lung distention, structural damage, and acute inflammatory processes (eg, hyperinflation, emphysema, pulmonary fibrosis, pulmonary emboli, and acute lung injury). Thus, measuring PVR cannot identify local injury or explain why PVR is elevated. Furthermore, with nonhomogeneous lung disease, pulmonary blood flow will preferentially go to those regions with the lowest resistance, thus masking lung abnormalities.

### Pulmonary Edema

Pulmonary edema can be caused by elevations of pulmonary capillary pressure (hydrostatic or secondary pulmonary edema), increased capillary or alveolar epithelial permeability (primary pulmonary edema),
or a combination of both. If pulmonary capillary pressure increases above 20 mm Hg, hydrostatic vascular forces promote increased fluid flux across the capillary membrane, flooding the alveoli. However, if capillary or alveolar cell injury is present, as in acute lung injury, alveolar flooding can occur at much lower pulmonary capillary pressures. Clinicians usually use Ppao as a surrogate of pulmonary capillary pressure, and if pulmonary venous resistance is not increased, this assumption is valid. Measures of absolute Ppao are used to determine the cause of pulmonary edema. In the setting of pulmonary edema, Ppao values less than about 18 mm Hg suggest capillary leak, whereas values greater than this suggest heart failure. However, these are not hard values. For example, transient severe LV dysfunction can transiently increase Ppao during upper airway obstruction with vigorous inspiratory efforts (inspiratory stridor, obstructive sleep apnea), unstable angina (reversible ischemia), and arrhythmias. Increased pulmonary capillary pressure can occur when massive sympathetic discharge increases pulmonary venous resistance (eg, intracerebral hemorrhage and heroin overdose), which reverses quickly while the pulmonary edema remains. Furthermore, persistently elevated pulmonary capillary pressures can coexist with normal Ppao values if pulmonary venous resistance is increased and CO is also increased, as is often the case in high altitude pulmonary edema and end-stage acute lung injury.

**Left Ventricular Preload and Volume Status**

Pulmonary artery occlusion pressure is often used erroneously to assess intravascular volume status and LV preload. Regrettably, neither absolute Ppao values nor their change in response to fluid infusion trends preload or volume responsiveness. The reasons for this are multiple. First, although increases in LV end-diastolic volume will increase CO in volume-responsive patients, the relation between Ppao and LV end-diastolic volume is curvilinear and may be very different among subjects and within subjects as RV volumes, intrathoracic pressure, and myocardial ischemia vary. Second, Ppao is not the distending pressure for LV filling but is only an estimate of LV intraluminal pressure, whereas pericardial pressure and LV diastolic compliance can vary widely and not be indicated by this or other measures. Hyperinflation, tamponade, and active inspiratory and expiratory muscle activity can rapidly alter pericardial pressure. Myocardial ischemia, arrhythmias, and acute RV dilation can alter LV diastolic compliance and can occur over a few heartbeats. Thus, it is not surprising that Ppao is a very poor predictor of preload responsiveness. The use of Ppao as a measure of LV end-diastolic volume and preload responsiveness is not recommended.

**Assessment of Left Ventricular Performance**

The assessment of LV performance is central to invasive hemodynamic monitoring. The primary determinants of LV performance are preload (LV end-diastolic volume), afterload (LV wall stress, which is itself the product of LV end-diastolic volume and diastolic arterial pressure), heart rate, and contractility. Since LV end-diastolic volume is a fundamental determinant of stroke volume and LV stroke work, Ppao is often used as a surrogate for LV end-diastolic volume and for the calculation of stroke work, which is the product of the difference between MAP to Ppao and stroke volume. Thus, Ppao can be used to construct Starling curves that plot Ppao versus LV stroke work. With this approach, patients with heart failure can be classified by their Ppao and cardiac index values using a Ppao of 18 mm Hg and a cardiac index of 2.2 L/min/m² as cutoff values. Low cardiac indices and high Ppao reflect heart failure, and low Ppao reflects hypovolemia, whereas high cardiac indices and high Ppao reflect volume overloaded and low Ppao reflects increased sympathetic tone.

These constructs are probably too simplistic, as many a clinical study has documented. Still, in the patient without lung or pericardial disease, tamponade, or pulmonary embolism, the relation between Ppao and pulmonary wedge pressure can be used to assess LV performance.
CARDIAC OUTPUT

Shock reflects an inadequate DO₂ to meet the body's metabolic demand, and CO is a primary determinant of DO₂. Indeed, except for extreme hypoxemia and anemia, most of the increase in DO₂ that occurs with resuscitation and normal biological adaptation is attributable to increasing CO. A vast array of devices, both invasive and noninvasive, are available that measure CO accurately enough to drive clinical decision making.

Noninvasive Measures of Cardiac Output

Cardiac output can be reliably measured noninvasively using a variety of techniques including ultrasound, plethysmographic pressure profile analysis, and bioreactance. The most commonly used noninvasive techniques are ultrasound based and include echocardiography, pulsed esophageal Doppler, and continuous-wave suprasternal notch ultrasound.²⁵

Echocardiography

To conduct transthoracic echocardiographic analysis of the aortic root flow, the clinician places the echo probe along the short axis of the aorta using pulse Doppler to measure the aortic valve diameter and then positions the probe from the suprasternal notch looking through the aortic valve to measure aortic velocity using continuous-wave Doppler. Although this procedure requires training and measures only a single point in time, it can reliably estimate CO.²⁶ The accuracy of the measure increases with transesophageal echocardiography, but so does its invasiveness. One can use changes in aortic flow during positive pressure ventilation to predict volume responsiveness, as described subsequently.²⁷,²⁸ Recent consensus conferences of multiple professional societies have stated that basic expertise in ultrasound techniques should be a central part of all critical care medicine training. However, since the transthoracic measures of CO are highly dependent on the expertise of the operator and cannot be measured continuously, this important method will not be discussed further.

Esophageal Doppler Ultrasound

Esophageal Doppler ultrasound uses an esophageal probe similar in size to a nasogastric tube to measure descending aortic flow as it parallels the esophagus. A Doppler transducer probe is inserted orally or nasally to midthoracic level and rotated until the probe senses a characteristic aortic velocity signal profile.²⁹ This technique can be taught to bedside nurses and can be used to drive resuscitation algorithms to improve patient outcomes in patients undergoing high-risk surgery.³⁰ Several studies have documented that esophageal Doppler estimates of CO and its change can be made accurately after minimal training.³¹ Similarly, dynamic increases in CO assessed by esophageal Doppler in response to a passive leg-raising test predict volume responsiveness.³² The only drawback to this technology is that nasogastric insertion is difficult, necessitating oral insertion, so this is not the preferred technique in the awake nonintubated patient. However, when compared with standard therapy in randomized clinical trials, the use of esophageal Doppler ultrasound to achieve targeted DO₂ levels in high-risk perioperative patients resulted in decreased hospital length of stay and decreased postoperative complications.³³ Furthermore, complications with the use of esophageal Doppler measures are very rare.

Transcutaneous Doppler Ultrasound

This modification of the esophageal Doppler technique uses a handheld probe placed at the suprasternal notch with the transducer aimed downward at the aortic valve. This technique is easy to learn and gives accurate measures of LV stroke volume and CO.³⁴,³⁵ However, like echocardiographic techniques, transcutaneous ultrasound cannot be used continuously. This accurate, noninvasive device can be used as a decision support tool to identify patients who are volume responsive and to calibrate minimally invasive devices (discussed later). Potentially, transcutaneous ultrasound will be most readily accepted in the emergency department, during medical transport, and on the general medical floor to rapidly identify cardiovascular instability.³⁶ Still, no clinical trials have been published using this promising technology to guide resuscitation.
**Thoracic Electrical Bioreactance**

Thoracic electrical bioimpedance has been around for decades and is accurate only in highly shielded environments because electrical interference makes the measures of CO unreliable.\(^{39}\) However, using the frequency modulation component of the thoracic impedance signal in a process called bioreactance markedly eliminates this artifact and increases the accuracy of the estimates of CO and its change.\(^{40}\) Both bioimpedance and bioreactance rely on a derivation of Ohm's law, which states that the flow of an electrical current is equal to the voltage drop between 2 ends of a circuit, divided by the resistance or impedance to current flow. Since most of current flow through the thorax occurs in the aorta and vena cava, changes in impedance reflect changes in volume and CO. Validation studies comparing bioreactance and pulmonary artery catheter thermodilution CO conclude that bioreactance has acceptable accuracy.\(^{41}\) Furthermore, the dynamic responsiveness of the bioreactance signal allows it to be used in combination with a passive leg-raising maneuver to assess fluid responsiveness. However, although clinical trials using bioreactance technology to guide resuscitation are ongoing, no clinical studies have yet been published using this promising technology.

**Invasive Measures of Cardiac Output**

**Thermodilution Using the Pulmonary Artery Catheter**

Invasive measurement of CO using the pulmonary artery catheter remains the most common method used clinically, although this trend is rapidly changing.\(^{42}\) The pulmonary artery catheter has a thermistor located 4 cm from the tip and a proximal port located 30 cm from the tip. Cardiac output is measured by injecting cold fluid through the proximal port. Under normal conditions, once the pulmonary artery catheter is placed, the proximal port resides at or above the right atrium such that bolus injections of cold (thermal) fluid are mixed in the contracting right ventricle. The thermistor records the dynamic change in blood temperature. The thermodilution-estimated CO is inversely proportional to the area under the temperature versus time curve. Subsequently, a random thermal (heat) signal using an induction coil in the RV region of the pulmonary artery catheter permits the clinician to estimate CO continuously, albeit less accurately measuring dynamic changes in flow.\(^{43}\) Importantly, measures of stroke volume and CO when coupled with other measured hemodynamic variables allow for the calculation of various important parameters, like LV stroke work and both systemic DO\(_2\) and consumption. Although the clinical utility and outcome benefit of pulmonary artery catheterization have been debated for many years,\(^{44}\) no study has used pulmonary artery catheter–specific measures to drive resuscitation algorithms and compared outcomes with those of other patients not having such information.\(^{45}\) Given the present climate in critical care medicine, it is highly doubtful that such a study will be undertaken.\(^{46}\)

Similar to the pulmonary artery catheter is the transthoracic thermodilution method of estimating CO, the only difference being that the measure of thermal change is made in a central artery instead of the pulmonary artery. This approach does not require pulmonary artery catheter insertion but has a major limitation in that the arterial sample needs to be of a flow-by sensor, since the thermal signal must pass by the sensor to report its change, thus requiring insertion of the thermistor probe into a femoral artery.

**Arterial Pulse Pressure Waveform Analysis**

Pulse pressure waveform analysis is also referred to as minimally invasive monitoring because it requires only the insertion of an arterial catheter. Several commercially available devices use proprietary algorithms that analyze the arterial pressure waveform (or the pulse contour).\(^{47}\) Each estimates central arterial compliance differently, and the techniques that require a standard external measure of CO for their calibration are the most accurate.\(^{48}\) Since arterial compliance varies depending on the patient’s blood pressure, age, sex, and height, these devices usually need to be recalibrated on a regular basis. The 2 common reference standards for calibration are transthoracic lithium dilution\(^{49}\) and thermodilution.\(^{50}\) Recent algorithms have been developed that do not require external
calibration with a CO reference standard. Recent head-to-head comparisons of all the invasive and minimally invasive devices demonstrated significant intra-device variability, suggesting that if one uses these devices, it is best to stick with only 1 or 2 devices and learn to use them well rather than use several over time in the same patient. A list of the available devices commonly used to estimate CO is given in Table 1 on page 128. In general, a minimally invasive device should be externally validated using an independent means if possible, and this should be done often if the cardiovascular tone of the patient varies rapidly.

A central question in the resuscitation of hemodynamically unstable patients is whether they need increased blood flow to the tissues. A patient who is in circulatory shock will need increased DO2. Accordingly, efforts to rapidly increase blood flow are important to minimize tissue ischemia and organ dysfunction. Traditionally, an increase in CO of greater than 15% after a fluid challenge has been considered the gold standard reflecting fluid responsiveness. If CO does not increase in response to fluid challenge, then the presumptive diagnosis of impaired cardiac contractility is made and inotropes are given to increase blood flow. Importantly, there is no absolute CO target that should be taken as optimal. The focus during resuscitation from circulatory shock should be a relative change in CO in response to therapy and the associated change in organ perfusion.

Over the past 15 years, numerous studies have validated that either arterial pulse pressure or stroke volume, referred to as pulse pressure variation (PPV) and stroke volume variation (SVV), respectively, induced by positive pressure ventilation can accurately identify patients who are volume responsive and those who are not. A threshold value of either PPV or SVV greater than 15% defines volume responsiveness when patients are ventilated with a tidal volume of 8 mL/kg or more. These parameters are not accurate during arrhythmias and spontaneous breathing because of varying R-R intervals and ventricular interdependence–induced changes in LV diastolic compliance, respectively. In those cases, one can perform a passive leg-raising maneuver and note the transient increase in CO. Postural changes such as passive leg raising have been used for many years to

### Table 1.

<table>
<thead>
<tr>
<th>Noninvasive</th>
<th>Invasive</th>
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<tr>
<td>Echocardiography</td>
<td>Minimally invasive (external calibration)</td>
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<tr>
<td>Transcutaneous ultrasound</td>
<td>Minimally invasive (self-calibrating)</td>
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<tr>
<td>Esophageal Doppler</td>
<td>Pulmonary artery catheter</td>
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<td>Bioimpedance</td>
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<td>Bioreactance</td>
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<td>Plethysmographic</td>
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<tr>
<td>Various manufacturers</td>
<td>PICCO plus (Pulsion Ltd)</td>
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<tr>
<td>USCOM (USCOM Ltd)</td>
<td>LiDCO plus (LiDCO Ltd)</td>
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<tr>
<td>CardioQ (Deltex Medical)</td>
<td>FloTrac (Edwards Lifesciences)</td>
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<tr>
<td>BioZ (SonoSite)</td>
<td>LiDCO rapid (LiDCO Ltd)</td>
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<td>NICOM (Cheetah Medical)</td>
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<td>Nexfin (BMEYE)</td>
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<td>Bolus thermodilution (Edwards Lifesciences) &amp; Continuous thermodilution</td>
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transiently increase venous return. The legs are raised to 30° above the chest and held for 1 minute, and the maximal increase in CO is recorded. This maneuver approximates a 300-mL blood bolus in a 70-kg patient that persists for approximately 2 to 3 minutes. Changes in heart rate, blood pressure, CVP, or CO are then observed after passive leg raising (PLR). The dynamic increases in CO induced by passive leg raising are as sensitive and specific in predicting volume responsiveness as is PPV during positive pressure mechanical ventilation using any of the commercially available, minimally invasive monitoring devices. Importantly, one cannot use the pulmonary artery catheter because neither bolus nor continuous CO measures by this device are rapid enough in their sensing to detect these small and short-lived changes in CO.

OXYGENATION AND TISSUE PERFUSION

Since there is no specific CO that is considered normal, only one that is adequate or inadequate to meet the metabolic demands of the body, other measures of cardiovascular sufficiency need to be made to assess the adequacy of blood flow. Importantly, maintenance of a normal blood pressure does not equate to adequate tissue blood flow because vasomotor tone varies to keep arterial pressure in an acceptable range. Furthermore, targeting a defined supranormal level of DO₂ does not ensure adequate flow to meet metabolic demand. To assess the adequacy of O₂ delivery, one needs to measure arterial and venous blood O₂ saturations and surrogate measures of tissue hypoxia, such as serum lactate levels and tissue O₂ saturation. Pulmonary artery catheterization identifies true mixed venous O₂ saturation (Svo₂), whereas Scvo₂ taken via a central venous catheter mostly reflects the degree of O₂ extraction from the brain and the upper part of the body.

Pulse Oximetry

Continuous monitoring of arterial blood O₂ saturation (Sao₂) using pulse oximetry is universally used in the ICU although clinical data of its usefulness are lacking despite impressive clinical trials attempting to demonstrate a beneficial effect. The greatest utility of pulse oximetry is in reducing the need for repetitive arterial blood gas analysis. Pulse oximeters estimate Sao₂ saturation by measuring the tissue light absorption at 2 specific wavelengths, 660 nm (red) and 940 nm (infrared). The absorption ratios, based on calibration against known Sao₂ values, allow for the continuous measurement of Sao₂. Importantly, nonphasic O₂ absorption also occurs, so the devices presume that dynamic changes in the density of absorption must reflect the arterial pulse, hence the name pulse oximetry. Accordingly, if no arterial pulsatility is seen from the plethysmographic waveform, Sao₂ cannot be calculated using these devices. Since they actually measure the pulsed O₂ saturation, they are referred to as pulse O₂ saturation (SPO₂), which in practice approximates Sao₂ well.

Detection of Hypoxemia

The most commonly used application of SPO₂ is the detection of hypoxemia. Hypoxemia is usually defined as a SPO₂ less than 90%. Titration of FIO₂ and other ventilatory maneuvers to keep SPO₂ greater than 90% is a common goal in most critically ill patients. However, there is little additional benefit in increasing SPO₂ above 95% because of the shape of the oxyhemoglobin dissociation curve limits O₂-carrying capacity. Taking the usually stated 95% confidence limits of ±4%, an oximeter reading of 95% could represent a PaO₂ between 60 mm Hg (Sao₂ > 91%) and 160 mm Hg (Sao₂ > 99%). Thus, decisions on ventilatory therapy should not be determined solely by SPO₂ values.

Detection of Volume Responsiveness

Another novel use of pulse oximetry is the plethysmographic waveform analysis. Variation in plethysmographic density from beat to beat reflects paired variations in arterial pulse pressure, thus making plethysmographic variability another method of assessing volume responsiveness in the ventilator-dependent patient, as discussed earlier.
Venous Oximetry and the Physiology of S\textsubscript{vo}\textsubscript{2} and Scvo\textsubscript{2}

The DO\textsubscript{2} describes whole-body O\textsubscript{2} supply without reference to blood flow distribution or O\textsubscript{2} uptake. DO\textsubscript{2} is equal to the product of CO and arterial O\textsubscript{2} content (Cao\textsubscript{2}). Arterial O\textsubscript{2} content is the sum of O\textsubscript{2} bound to hemoglobin (Hb) (product of Hb concentration Sao\textsubscript{2}) and dissolved O\textsubscript{2} (Pao\textsubscript{2}). The formula is Cao\textsubscript{2} = Hb × 1.36 × Sao\textsubscript{2} × Pao\textsubscript{2} × 0.0031. Thus, dissolved O\textsubscript{2} in the plasma has minimal effect on overall Cao\textsubscript{2}.

Clinically, DO\textsubscript{2} only has relevance to O\textsubscript{2} demand estimate by whole-body O\textsubscript{2} consumption (VO\textsubscript{2}). Since VO\textsubscript{2} must equal the difference in DO\textsubscript{2} and the amount of O\textsubscript{2} remaining in mixed venous blood, VO\textsubscript{2} can be expressed by the Fick principle as the product of CO and arteriovenous O\textsubscript{2} content difference (Cao\textsubscript{2} – Cvo\textsubscript{2}); VO\textsubscript{2} = CO (Cao\textsubscript{2} – Cvo\textsubscript{2}), where mixed venous O\textsubscript{2} content (Cvo\textsubscript{2}) like Cao\textsubscript{2} is Cvo\textsubscript{2} = Hb × 1.36 × Svo\textsubscript{2} × Pao\textsubscript{2} × 0.0031. By simple algebraic transposition, Cvo\textsubscript{2} = Cao\textsubscript{2} – (VO\textsubscript{2}/CO). Thus, Cvo\textsubscript{2} reflects the relationship between whole-body VO\textsubscript{2} and CO under conditions of a constant Cao\textsubscript{2}. Importantly, Svo\textsubscript{2} is the most important factor to determine Cvo\textsubscript{2} since, like in arterial blood, the dissolved O\textsubscript{2} can be neglected and Hb is usually constant over short time intervals. Thus, Svo\textsubscript{2} correlates well with the O\textsubscript{2} supply-to-demand ratio.

Trending Svo\textsubscript{2} and Scvo\textsubscript{2} to Assess Circulatory Sufficiency

A decrease in Svo\textsubscript{2} and Scvo\textsubscript{2} usually represents an increased metabolic stress. Factors that decrease Svo\textsubscript{2} include low CO or anemia (decreasing DO\textsubscript{2}), exercise (increasing VO\textsubscript{2}), and hypoxemia. In the sedated patient not actively bleeding and in whom Sao\textsubscript{2} is greater than 90%, Svo\textsubscript{2} and CO covary. Importantly, the normal cardiovascular response of increasing VO\textsubscript{2} (exercise) is to increase O\textsubscript{2} extraction and CO. Thus, Svo\textsubscript{2} normally decreases during exercise despite increasing DO\textsubscript{2}. Therefore, a decrease in Svo\textsubscript{2} or Scvo\textsubscript{2} does not necessarily mean that tissue hypoxia occurs. Only if Svo\textsubscript{2} decreases less than 50% can one be sure some degree of tissue hypoxia has occurred. Conversely, a normal Svo\textsubscript{2} (eg, >72% saturation) does not ensure that some vascular beds are not underperfused. In summary, Svo\textsubscript{2} can decrease, suggesting increasing cardiovascular stress due to a decrease in DO\textsubscript{2} (eg, anemia, hypoxia, hypovolemia, or heart failure) or increased VO\textsubscript{2} (eg, fever, pain, stress, shivering).

Since central venous catheterization is commonly performed for a variety of reasons in critically ill patients, direct access to central venous blood is also commonly available in most critically ill patients. Thus, Scvo\textsubscript{2} is often used as a surrogate for Svo\textsubscript{2}. Since most central venous catheters have their distal tip in the superior vena cava, Scvo\textsubscript{2} reflects the venous blood of the upper body but neglects venous drainage from the lower body (ie, intra-abdominal organs). Accordingly, Scvo\textsubscript{2} is usually lower than Svo\textsubscript{2} by 2% to 3% because the lower body extracts less O\textsubscript{2} than the upper body, making inferior vena caval O\textsubscript{2} saturation higher. The primary cause of the lower inferior vena cava O\textsubscript{2} extraction is the oxidative phosphorylation blood flow (eg, renal blood flow, portal flow, hepatic blood flow). Importantly, however, Svo\textsubscript{2} and Scvo\textsubscript{2} change in parallel when the ratio of whole-body O\textsubscript{2} supply and O\textsubscript{2} demand is altered. Still, the difference between the absolute values of Svo\textsubscript{2} and Scvo\textsubscript{2} changes under conditions of shock. In septic shock, for example, Scvo\textsubscript{2} often exceeds Svo\textsubscript{2} by as much as 8%, and the opposite can occur with hypovolemic and cardiogenic shock. During cardiogenic or hypovolemic shock, mesenteric and renal blood flow decreases, which for the same level of metabolic demand increases local O\textsubscript{2} extraction. In septic shock, splanchnic O\textsubscript{2} consumption increases, thus increasing local O\textsubscript{2} extraction despite increased CO. Thus, Scvo\textsubscript{2} cannot be used as surrogate for Svo\textsubscript{2} under conditions of circulatory shock. As a rule, if Scvo\textsubscript{2} is less than 65% then inadequate DO\textsubscript{2} probably exists, but if Scvo\textsubscript{2} is greater than 70% it has no prognostic utility.

Near-Infrared Spectroscopy to Measure Tissue O\textsubscript{2} Saturation

Near-infrared spectroscopy (NIRS) is a noninvasive technique capable of continuous measurement that uses the varying light absorption properties of deoxygenated and oxygenated blood to determine tissue O\textsubscript{2}.
Preload Responsiveness

The use of positive pressure breathing–induced PPV and SVV (discussed earlier) and passive leg-raising–
induced changes in CO accurately predicts volume
responsiveness. Similarly, one can measure inferior vena
caval diameter decreases during inspiration, superior
vena caval decreases during inspiration, and aortic flow
velocity changes and plethysmographic pulse oximetry
change to predict volume responsiveness. Using a simple
SVV minimization can markedly reduce intra-operative
protocol hospital length of stay.

Occult Circulatory Shock

Examining the changes in StO2, deoxygenation and
reoxygenation during a vascular occlusion test reveals
misdistribution of blood flow and occult circulatory shock,
respectively, and the StO2 changes induced by a vascular
occlusion test.

Goal-Directed Therapy

Initial studies suggested that the nonspecific increase
in DO2 to supranormal levels in critically ill patients
improved survival. Subsequent studies using aggressive
resuscitation to increase DO2 to supranormal levels in
patients with existing organ failure to document survival
benefit reported that if anything this procedure increased
mortality. In essence, the benefit of increased flow
cannot be realized by already dead tissues. However,
early aggressive resuscitation driven by hemodynamic
monitoring if done before the onset of organ injury
uniformly improves survival, whether done as early
goal-directed therapy in septic patients presenting to
an emergency department, and as preoptimization
therapy for high-risk surgical patients, but less so in
postoptimization therapy for postoperative patients with
difficult intraoperative courses, suggesting that early
aggressive resuscitation is most useful in the intraoperative
arena but still shows benefit in the ICU, although to a
lesser degree.
SUMMARY

Hemodynamic monitoring is done to determine whether a subject is physiologically stable, to determine whether blood flow to the periphery is adequate to meet metabolic demands, to diagnose specific causes of circulatory shock, to determine specific therapies, and to indicate when cardiorespiratory sufficiency has been reestablished. Using our knowledge of cardiorespiratory physiological changes, disease pathophysiological factors, and the strengths and limitations of device measurement, the bedside caregiver now has an extremely powerful array of monitoring devices to accomplish these goals. However, no hemodynamic monitoring device will improve outcome unless coupled with a treatment that itself improves outcome. What is primarily missing now is a field theory of global cardiorespiratory performance and validated management protocols of sufficient generalizability to be universally applied. Until that time arrives, the need for well-trained bedside applied physiologists in the guise of intensivists will remain a health care priority.

REFERENCES


