CHAPTER
20
Noninvasive Cardiovascular Monitoring
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INTRODUCTION
Circulatory shock in the critically ill commonly occurs from trauma, hemorrhage, high-risk surgery, sepsis, anaphylaxis, burns, tension pneumothorax, and cardiac emergencies such as acute myocardial infarction (AMI) and pulmonary embolism (PE). Up to one-third of patients admitted to the intensive care unit (ICU) are in circulatory shock (1). Early recognition and aggressive therapy of shock by way of hemodynamic support are keys to successful resuscitation and mitigation of worsening organ dysfunction and failure in a broad spectrum of patient subgroups (2–6). Shock results from four potential but not exclusive pathophysioologic mechanisms (7,8): hypovolemia, cardiogenic factors, obstruction, or distributive factors. Identification of the main mechanism responsible for shock by clinical evaluation alone is difficult, especially in complex situations such as cardiac tamponade in a patient with trauma or septic shock in a patient with chronic heart failure (9). Furthermore, clinical assessment of cardiac output (CO) and intravascular volume status are very inaccurate (10). Hemodynamic measurements help with the determination of the type of shock, selecting appropriate therapeutic interventions, and evaluating the patient’s response to therapy.

Hemodynamic monitoring and management has greatly improved during the past decade (11), with technologies evolving from very invasive to noninvasive (12,13), and a philosophical shift from more traditional static approaches to functional and dynamic approaches for monitoring (14). Traditional methods of hemodynamic monitoring—including blood pressure, pulse rate, central venous pressure (CVP), and arterial oxygen saturation—change minimally in early shock and are poor indicators of the adequacy of resuscitation (13,15). The measurements of stroke volume (SV) and CO are the fundamental aspects of current hemodynamic management and will be the focus of this chapter. Noninvasive continuous CO monitoring has potential clinical applications in the ICU, emergency department (ED), and operating room (OR) by way of establishing a diagnosis, risk stratification, and therapy guidance.

There are a variety of modalities that have been developed to provide for hemodynamic monitoring by invasive, minimally invasive, and noninvasive methods. CO measurement with a pulmonary artery catheter (PAC) using the bolus thermodilution method is the gold standard and reference method used to compare newer novel technologies (16,17). The PAC and other invasive methods are presented in detail elsewhere in this textbook. Minimally invasive methods include transesophageal Doppler and pulse contour analysis, with or without a dilution technique; noninvasive methods include CO2 and inert gas rebreathing, transthoracic Doppler, thoracic bioimpedance cardiography, and bioelectance. Each of these methods has its own advantages and limitations; taken together they have the potential to create a robust multidimensional picture of patients’ hemodynamic states and their responses to therapeutic interventions. When only one method is used, its limitations may obscure the real problem and limit clinical usefulness.

CO MONITORING BY ELECTRIC BIOIMPEDANCE
The technique of bioimpedance cardiography is based on the measurements of impedance (or resistance) to transmission of a small electrical current through the chest area (thoracic bioimpedance) or whole body (whole-body bioimpedance). Thoracic bioimpedance systems use pair of injecting and sensing electrodes applied at the base of the neck (thoracic inlet) and the costal margins (thoracic outlet), while whole-body systems use electrodes attached to limb extremities (17,18). Thoracic bioimpedance cardiography relies on the theory that the impedance of the thorax is dependent on the amount of fluid (blood and plasma) in the thoracic compartment; this represents conduits of low impedance, with higher impedance occurring in cardiac muscle, lungs, and fat. The varying amount of blood volume in the aorta during the cardiac cycle is thought related to the observed changes in impedance (19). The outer pairs of electrodes pass a small-amplitude (0.2–5.0 mA) alternating current at 40 to 100 kHz through the patient’s thorax to produce an electrical field. The injected electrical signals travel predominantly down the aorta, which has lower electrical resistance than aerated lung. Each ventricular contraction propels the SV down the aorta, increasing aortic blood volume and aortic flow and lowering impedance. The impedance is sensed by the inner recording electrodes that capture the baseline impedance (Z0) and the first derivative of the impedance waveform (dZ/dt) (20,21). Changes in aortic blood flow throughout the cardiac cycle are quantitatively related to changes in the electrical impedance. CO is computed based on mathematical equations under the assumption that thoracic impedance changes over time are proportional to the SV. Electrical velocimetry—a new bioimpedance method with a different algorithm for processing the impedance signal has demonstrated low accuracy and precision compared with pulmonary artery (22) and transthoracic (23) thermodilution techniques.

Numerous validation studies in diverse patient populations have been carried out to compare the performance of thoracic electrical bioimpedance with well-established reference methods. Data from very early studies showed inconsistent results when compared with the PAC thermodilution CO technique (24–27). Early meta-analyses reported mean percentage error of 37% (28) and poor agreement (29) between CO measurements obtained by thoracic cardiac impedance, a reference method. Improved newer-generation bioimpedance devices with upgraded computer technology and refined mathematical
algorithms for CO calculation have also yielded contradictory results with regard to agreement with reference techniques and the ability to trend CO changes (30–35). A more recent meta-analysis of 13 validation studies of thoracic electrical bioimpedance reported an overall percentage error of 42.9% for thoracic bioimpedance measurements (36).

Thoracic electrical bioimpedance technology has several limitations. The clinical applicability of all thoracic electrical bioimpedance devices is highly dependent on electrode positioning, variations in patient age, gender, body size, and other physical factors like temperature and humidity that impact on electric conductivity between the electrodes and the skin (26,37,38). Its accuracy is further limited by electrical interference (e.g., from electrocautery), fluid in the thoracic compartment—pleural effusions, pericardial tamponade, pulmonary edema—changes in peripheral vascular resistance, cardiac dysrhythmias, and motion artefacts (39–42).

THORACIC BIOREACTANCE

Bioreactance devices were developed to overcome the limitations of bioimpedance devices by processing the impedance signals in a way that improves on the signal-to-noise ratio. In addition to changing resistance to blood flow (Z0), changes in intrathoracic volume also produce changes in electrical capacitive and inductive properties that result in phase shifts of the received signal relative to the applied signal (17,43). Bioreactance represents the phase shift in voltage across the thorax; this almost exclusively depends on pulsatile flow. The bioreactance signal is therefore more closely related to aortic blood flow and less dependent on intra- and extravascular lung water (13,42). The only commercially available monitoring system assessing CO by bioreactance is the NICOM system (Cheetah Medical, Portland, OR).

Several studies have shown high correlation of CO measurements by bioreactance with that measured by thermodilution and pulse contour analysis (43–46). More recent studies have questioned the accuracy of the bioreactance cardiography. For example, a study of surgical patients treated for ovarian cancer showed that thoracic bioreactance did not reliably measure cardiac index (CI) compared with transpulmonary hemodilution (47).

Bioreactance cardiography can be performed in ventilated and nonventilated patients and can compute CO in patients with atrial and ventricular dysrhythmias (13). As with bioimpedance, electrical interference can alter bioreactance measurements.

ARTERIAL PULSE CONTOUR ANALYSIS AND PLETHYSMOGRAPHY

There are several devices being marketed for the noninvasive and continuous assessment of arterial blood pressure (ABP) and CO. The most commonly used are the CNAP, Finapres, and Clear-sight systems. These devices function on two operating principles, the volume clamp method and pulse contour analysis.

In the first portion, the volume clamp method is used and a finger cuff is applied to the middle phalanx; the finger cuff is inflated and deflated so as to maintain a constant level of blood volume (48). The finger’s arterial diameter is measured by sending infrared light through the tissue and measuring the absorption of light by the blood using a light detector integrated in the finger cuff. This photoplethysmographic signal controls the finger cuff pressure and the artery is kept at a constant diameter during the cardiac cycle. When the artery’s diameter is constant, the cuff pressure must be equal to the intra-arterial pressure (42). From these measurements, the arterial waveform and pressures are calculated and used for pulse contour analysis. The brachial artery pressure is mathematically reconstructed from the finger cuff measurements and displayed on the monitor for continual display.

The pulse contour method then analyzes the systolic area under the arterial waveform curve—beginning of waveform to the dicrotic notch—for each beat. The arterial impedance is estimated from a three-element model to compute flow that has nonlinear pressure-dependent properties. The three determining elements are aortic impedance, Windkessel—or buffer—compliance, and peripheral resistance (49). The division of the systolic area by the impedance produces an estimate of the SV (50), and CO is derived.

This method has several drawbacks. Arterial impedance is an estimated parameter based on proprietary algorithms that are based on patient age, gender, height, and weight. The arterial waveform input can be degraded in patients with peripheral arterial disease, severe vasoconstriction, or edema of the extremities thereby yielding CO results that have limited accuracy (51–53). Additionally, studies have mostly been performed in cardiac surgery patients and have not been validated in the setting of valvular heart disease or significant tachydyssrhythmias. Although the technology is promising, due to its noninvasive and continuous nature, the CO measurements do not meet the criteria for clinical interchangeability with the PAC, and further studies are needed to support its use in the critically ill shock patient (9).

RADIAL APPLATION TONOMETRY

Applanation tonometry of the radial artery allows for continuous noninvasive monitoring of ABP without external calibration. Commonly used commercial devices are the T-Line System by Tensys Medical and SphygmoCor by AtCor Medical. The operating principle is the application of external pressure to the radial artery against a noncompressible surface (the radius). Once pressure is applied, the contact stress is transduced by a surface sensor into an electrical signal which provides continuous pulse recording of pressure with direct calibration over time. The degree of pressure that is needed to compress the vessel is in direct correlation to the instantaneous intraluminal pressure (54).

Tonometry studies in the critically ill have produced mixed results, with some studies citing a lack of reliability of systolic blood pressure (SBP) measurement (55), and wide agreement limits (56,57). Better correlation was shown with mean arterial pressure (MAP) and diastolic blood pressure (DBP). Additional draw backs are that systolic and diastolic blood pressures are derived from a scaled BP waveform through proprietary algorithms (T-system); that significant time is required before the initial reading is displayed when the system is initiated; and that readings are not available in
the event of arm motion as the radial artery is being located by the device. Compared to the volume clamp method, this device does not appear to be as affected by arterial vasoconstriction and may yield CO measurements that have reasonable accuracy (58), but further studies are needed to support its use in the critically ill.

PULSE WAVE TRANSIT TIME

CO is estimated using the pulse oximeter waveform, arterial pressure, and electrocardiogram. The current commercial device on the market is eSCCO by Nihon Kohden. The principle is that of an inverse correlation between pulse wave transit time and SV. So far, studies in the critically ill have not shown good correlation with transthoracic echocardiography (TTE) (59).

ECHOCARDIOGRAPHY

Since the early 1950s, when the first ultrasound (US) machine was used to examine the human heart, US has allowed us to “see” inside the human heart and gain a new understanding of its complex workings and physiology. As of today, cardiac US is the most advanced and cost-effective noninvasive hemodynamic monitor that we have at our disposal. There is no other device that can provide the wealth of information the echocardiogram (ECHO) does in a similar cost-effective manner, with real-time image assessment and without any known adverse side effects (60).

Given the appropriate acoustic windows, ECHO provides the intensivist with a complete hemodynamic assessment of the patient. Key hemodynamic and nonhemodynamic parameters such as left ventricular ejection fraction (LVEF), left ventricular end-diastolic volume (LVEDV), left ventricular end-systolic volume (LVESV), right ventricular function, pulmonary artery pressures, CO, and SV variation (SVV) can be obtained. Cardiac US has also been shown to alter the diagnosis and therapeutic management of patients in the ICU by 30% to 60% (61).

Over the last two decades, ECHO has replaced the need for invasive hemodynamic assessment (left and right heart catheterization) in valvular heart disease due to its accuracy, reliability, and noninvasive nature. Only in the setting of conflicting echocardiographic data do patients need to undergo additional hemodynamic testing. It is beyond the scope of this text to give an in-depth description of cardiac ultrasonography and its techniques. Below is a summary of key echocardiographic and hemodynamic parameters that are of importance for the critical care practitioner.

Left Ventricular Volumes and Ejection Fraction (LVEF)

The quantitation of left ventricular systolic function is critical to the proper evaluation of patients with cardiovascular disease (62). LVEF is a predictor of cardiovascular outcomes in the critically ill patient. The recommended method for quantification of left ventricular systolic function is the biplane method of discs (modified Simpson’s rule) (63). The “eyeball” estimation of left ventricular systolic function is not recommended given the lack of reproducibility between readers and its subjective nature. The Simpson’s technique requires the recording of an apical four-chamber view and an apical two-chamber view from which the endocardial borders are outlined in end-diastole and end-systole. Failure to avoid these common mistakes will result in the underestimation of ventricular volumes (Fig. 20.1).

\[
\text{Ejection fraction (LVEF)} = \frac{\text{LVEDV} - \text{LVESV}}{\text{LVEDV}}
\]

Ventricular volumes obtained by 2D-ECHO have usually yielded smaller volumes than those obtained by cardiac MRI or nuclear medicine studies. This is attributed to the difficulty in excluding trabeculae and the improper visualization of the true endocardial border. In the absence of significant mitral regurgitation, SV can be obtained by subtracting the LVESV from the LVEDV.

\[
\text{SV} = \text{LVEDV} - \text{LVESV}
\]

Linear measurements of left ventricular function, such as the Fractional shortening method, can be quite misleading when there are regional wall motion abnormalities present.
and, as such, is not recommended. LVEF is a marker of left ventricular contractility and is affected by conditions that alter the preload, afterload, and contractility of the heart. Severe valvular heart disease can affect these parameters and needs to be taken into consideration when interpreting LVEF.

**Right Ventricular Systolic Function**

ECHO evaluation of the right ventricular function is complex due to its unique crescent shape. Multiple studies have demonstrated the clinical utility of TAPSE, S’ of the tricuspid annulus, and RIMP as surrogate measures of right ventricular ejection fraction (RVEF).

Tricuspid annular plane systolic excursion (TAPSE) is a one-dimensional measurement that represents longitudinal right ventricular function. It is measured by M-mode echocardiography with the cursor optimally aligned along the direction of the tricuspid lateral annulus obtained in the apical four-chamber view. TAPSE values less than 1.7 cm are highly suggestive of right ventricular systolic dysfunction (64). Tissue Doppler S’ of the lateral tricuspid annulus has also been shown to correlate with measures of right ventricular systolic function. An S’ velocity less than 9.5 cm/sec is indicative of RV systolic dysfunction (65).

**CO and SV**

CO is a key measurement in the management of the patient in shock. CO and SV can be obtained by a combination of Doppler and 2D US measurements. Pulse Doppler US allows us to measure intracardiac blood flow velocities; this is termed the velocity time integral or VTI. The VTI is the aggregate sum of blood flow velocities per heartbeat (SV) measured over time. By knowing the cross-sectional area through which blood flows through, we can then estimate blood volume (Figs. 20.2 and 20.3).

\[
\text{Stroke volume} = \text{cross-sectional area (CSA)} \times \text{VTI}
\]

Stroke volume = cross-sectional area (CSA) × VTI

\[
\text{CO} = \text{stroke volume (SV)} \times \text{heart rate (HR)}
\]

The preferred site for CSA measurement is the left ventricular outflow tract (LVOT) due to its minimal size variation during systole (66). CO measurements by the VTI method have been validated against invasive hemodynamic measurements with the PAC (67–70), and are reliable in the absence of valvular pathology.

**Right Atrial Pressure/Central Venous Pressure**

Right atrial pressure (RAP) or mean CVP can be estimated from the IVC diameter and its collapsibility on inspiration. Images are obtained of the intrahepatic portion of the inferior vena cava from the subcostal views. The combination of these two parameters (diameter and collapsibility) results in an accurate estimation of the mean RAP in patients who are not mechanically ventilated or who do not have elevated intra-abdominal pressures. In ventilated patients, an IVC diameter less than 1.2 cm appears to accurately identify patients with a RAP less than 10 mmHg. In this same group, if the IVC is small and collapsed it is suggestive of hypovolemia (64,65).

Another method to estimate RAP is through hepatic vein flow patterns obtained from the same subcostal views. Hepatic vein flow velocities have been validated in mechanically ventilated patients by a small number of patients (71) (Fig. 20.4 and Table 20.1).
Systemic Vascular Resistance

If we know the SBP of a patient (by cuff pressure or arterial line), we can then calculate the MAP and obtain the estimated RAP and CO by cardiac US as already described above. We can then deduce systemic vascular resistance (SVR) by applying Ohm’s law.

\[
SVR = MAP - CVP/CO
\]

Left Atrial Pressure

In the critically ill patient, left atrial pressure (LAP) measurements are not easily derived, nor should they be inherently trusted. Many factors need to be assessed and integrated clinically at the bedside. The parameter that best correlates with elevated LAP is \(E/e’\) ratio greater than 15 (<8 being normal). This ratio is obtained by pulse Doppler interrogation of the early mitral filling velocity \(E\) and then divided into the early diastolic mitral annular velocity \(e’\) as obtained by tissue Doppler velocities. The ratio has been shown to accurately predict elevated filling pressures in the heart failure population. Factors that can cause significant variability are regional wall motion abnormalities affecting the basal myocardium, preload conditions, depressed systolic function, mitral annular calcification, tachyarrhythmia with \(E/A\) fusion, and hypertrophic cardiomyopathy (72).

Pulmonary Artery Systolic Pressure and Mean Pulmonary Artery Pressure

Pulmonary artery systolic pressure (SPAP) can be quantified by measuring the right ventricular pressure through the tricuspid regurgitation peak jet velocity. Because the RV systolic pressure (RVSP) and SPAP are—in the absence of pulmonary stenosis—similar; this approach provides a simple means of quantifying pulmonary artery pressures (71).

\[
RVSP = 4 \times (TR \text{ velocity})^2 + RAP
\]

Mean pulmonary artery pressure (MPAP) can be obtained by pulsed Doppler interrogation of the RV outflow tract and obtaining the acceleration time (AT) of the pulmonic valve VTI. An AT less than 90 ms is suggestive of significant pulmonary hypertension (MPAP >25 mmHg). MPAP can also be deduced from the following formula:

\[
MPAP = 70 - (0.45 \times AT)
\]

Stroke Volume Variation and Fluid Responsiveness

Over the last decade there has been an increase in the number of studies emphasizing the importance of SVV and fluid responsiveness in the critically ill (15,60,73). ECHO can easily measure SV by pulse Doppler interrogation of the LVOT, the preferred site for this. By obtaining the VTI and SV measurements (described in the CO section, above) a fluid challenge is given and SV is remeasured. An increase in SV greater than 10% suggests volume responsiveness in the critically ill.

As with any technology, cardiac US has its own limitations and drawbacks. The first barrier is that of the human body and its acoustic windows. Air, a poor conductor of US waves, can impair and degrade image quality in those patients who have underlying lung disease or are mechanically ventilated. Further, acoustic windows may not be accessible due to postoperative pain, positional changes of the heart, drainage catheters, or skin dressings.

Another important barrier, which must be noted, is the learning curve and skill required in both the acquisition and interpretation of US images. This is a learned skill that requires dedicated training and will also be dependent on the power of the equipment used. Training requirements and expertise needed for analysis and interpretation of ECHO have been well delineated in professional society guidelines (74,75).

Finally, one of the biggest drawbacks of ECHO is its non-continuous nature. Echocardiography provides a “snapshot in time” thereby limiting its ability to serve as a continuous monitoring tool in the critically ill patient.

SUMMARY

The future for hemodynamic monitoring is promising given new technologic advances on the horizon. The ideal hemodynamic monitor will be noninvasive, provide continuous display of key hemodynamic variables, be readily applicable with no significant learning curve, and help guide therapeutic interventions for improved outcomes. At this time the perfect hemodynamic monitor does not exist, either invasive or noninvasive. Our current monitors operate on different physiologic principles and assumptions of which critical care practitioners need to be thoroughly familiarized with. Additionally, most devices do not provide a complete hemodynamic picture but rather a partial one. Failure to understand the physiologic principles on which devices function will lead to misinterpretation and/or misdiagnosis in complex cardiovascular conditions. As of today, echocardiography is the most complete noninvasive hemodynamic monitor at our disposal but is limited by its noncontinuous monitoring capabilities and significant learning curve.

Key Points

- Continuous and noninvasive cardiac monitoring modalities are potentially innovative tools for the bedside assessment of hemodynamic parameters.
- The ideal hemodynamic monitor will be noninvasive, provide continuous display of key hemodynamic variables, be readily applicable with no significant learning curve, and help guide therapeutic interventions for improved outcomes.
- Currently there is no ideal hemodynamic monitor that is able to provide all hemodynamic variables in a reliable and continuous fashion.
• For many modalities, data regarding measurement performance in comparison with reference methods for CO assessment are conflicting.

• Each modality has its specific advantages and limitations regarding usefulness in the clinical setting.

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References


