Hemodynamic Monitoring in the Critical Care Environment

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Hemodynamic monitoring is essential to the care of the critically ill patient. In the hemodynamically unstable patient where volume status is not only difficult to determine, but excess fluid administration can lead to adverse consequences, utilizing markers that guide resuscitation can greatly affect outcomes. Several markers and devices have been developed to aid the clinician in assessing volume status with the ultimate goal of optimizing tissue oxygenation and organ perfusion. Early static measures of volume status, including pulmonary artery occlusion pressure and central venous pressure, have largely been replaced by newer dynamic measures that rely on real-time measurements of physiological parameters to calculate volume responsiveness. Technological advances have lead to the creation of invasive and noninvasive devices that guide the physician through the resuscitative process. In this manuscript, we review the physiologic rationale behind hemodynamic monitoring, define the markers of volume status and volume responsiveness, and explore the various devices and technologies available for the bedside clinician.

Key Words: Cardiac output, Hemodynamics, Stroke volume variation, Pulmonary artery catheter

Introduction

The goal of hemodynamic monitoring in the care of critically ill patients is to assess and ensure adequate tissue oxygen delivery and end organ perfusion. This is accomplished by thoughtful management of cardiac output (CO) and systemic vascular resistance (SVR). Often it is difficult to ascertain whether a strategy of volume expansion, vasopressor use, inotropic support, or diuresis is the most appropriate strategy. Moreover, inappropriate volume expansion can lead to volume overload, pulmonary edema, worsening gas exchange, and acidosis. In the setting of chronic kidney disease, volume management is further complicated by impaired kidney autoregulation as well as compromised free water and solute elimination. Several tools have been developed for use in clinical practice that may aid in determining hemodynamic status as well as estimate the effect of volume, diuresis, or manipulation of systemic vascular resistance (from vasopressors). This review article will provide a physiologic basis for hemodynamic monitoring as well as discuss many of the hemodynamic parameters and devices used in the care of the critically ill patient.

Physiology

The relationship among CO, mean arterial pressure (MAP), and SVR (Equation 1) plays an important role in the management of the hemodynamically unstable patient with the goal of optimizing organ perfusion. Clinicians often use systolic blood pressure or MAP as a crude measure of end organ perfusion and central venous pressure (CVP) as a measure of volume status; from a practical perspective, this is an appropriate place to start. However, it is important to note that MAP and CVP are affected by the manipulation of CO and SVR. The manipulation of SVR (via vasopressors or vasodilators) has its limitations because high vasopressor doses ultimately decrease tissue perfusion and increase myocardial oxygen demand. Therefore, it is imperative that a clinician be able to assess CO and its components to optimize perfusion.

\[(\text{MAP} - \text{CVP}) \times 80 = \text{CO} \times \text{SVR} \quad \text{(Equation 1)}\]

\[\text{CO} = \text{HR} \times \text{SV} \quad \text{(Equation 2)}\]

Whereas heart rate is easy to determine, stroke volume is more difficult to measure directly. Stroke volume may be described by its relationship to cardiac filling pressure whereby increases in filling pressures, or preload, potentially correspond to a greater stroke volume. The Frank-Starling curve illustrates this relationship between pressure and volume. Traditionally, cardiac preload has been measured with CVP and pulmonary artery occlusion pressure (PAoP). It is important to recognize that this relationship is not linear because a complex set of factors can alter this relationship (ie, cardiomyopathy). Moreover, preload measurements can actually lead to incorrect assumptions regarding stroke volume, depending...
on where a patient lies along the Frank-Starling curve (Fig 1).

Disease-specific states can confound the relationship between pressure and volume, making volume status difficult to determine accurately. Patients with pulmonary hypertension or right ventricular dysfunction can generate CVPs that are not indicative of left atrial pressures or volume status but rather only reflect the failing right ventricle.³

Septic patients and patients with acute respiratory distress syndrome (ARDS) can also have misleading cardiac filling pressures despite being intravascularly depleted.⁴ The presence of positive end-expiratory pressure may also be implicated in erroneous CVP and PAoP measurements.⁵ All of this may be further complicated by acute or chronic kidney disease, whereby fluid management is an issue of its own.

To optimize end organ perfusion, clinicians have developed a myriad of specialized parameters and devices aimed at monitoring and guiding fluid management. These parameters can be broken down into static measurements that measure CO by exploiting its relationship to pressure and/or volume at one particular point in time. Static measurements include CVP and PAoP as described above briefly. Alternatively, dynamic tools predict a change in CO over time in response to a fluid bolus as a consequence of pressure changes within the respiratory cycle. A discussion of these techniques and their respective measurement devices is provided in the following section.

**Hemodynamic Measurements of Volume Status and Fluid Responsiveness**

**Static Measurements**

Static measurements of volume status include CVP and PAoP as well as left ventricular end diastolic area (LVEDA). CVP and PAoP are typically measured through a central line or a pulmonary artery (PA) catheter, which is inserted via the internal jugular or subclavian vein. The assumption behind CVP as a determinant of volume status is that CVP estimates right atrial pressure and correspondingly right ventricular end diastolic volume. In theory, a higher CVP signifies greater blood volume in the right atrium and thus higher right ventricular preload (a relationship that is questionable in cases of altered ventricular wall compliance).

PAoP utilizes the same set of assumptions for the left side of the heart. PAoP is measured by a PA catheter, which has a balloon tip as well as a distal pressure transducer. When the balloon is inflated and wedged inside of one of the pulmonary arteries, the pressure transducer will measure pressure distal to this balloon (specifically the pressure of the pulmonary capillary bed, which is open to the left atrium). Similar to CVP measurement, left atrial pressure in theory corresponds to left ventricular end diastolic volume, which, according to the Frank-Starling relationship, is related to stroke volume. A schematic of PA catheter tip positions and the corresponding pressure tracings are provided in Figure 2.

Like CVP and PAoP, LVEDA is a static marker used to approximate the volume status of a patient. LVEDA is measured by transthoracic or transesophageal echocardiography. In principle, an increase in LVEDA signifies greater ventricular myocardial stretch and therefore the potential for a larger CO. As explored above, this assumption does not always hold true because myocyte stretch and corresponding myocardial wall tension depend on the shape of the Frank-Starling curve and the position on the curve.

An increasingly large body of evidence suggests that the static markers CVP, PAoP, and LVEDA are poor surrogates for volume status. Despite the classic teaching about the “wedge” pressure (PAoP), this indicator is not an accurate marker of volume status. Moreover, PAOP does not predict whether a fluid challenge will lead to an improvement in cardiac performance (also known as “fluid responsiveness”).³⁻⁵ CVP and LVEDA are similarly poor predictors of fluid responsiveness. A systematic review by Michard concluded that these static measures did not adequately predict or discriminate responders (as defined as an increase in SV or CO to a fluid bolus) from nonresponders.⁶ Even in presumably fluid-responsive patients, evidence suggests poor correlation between cardiac filling pressure and volume status. In a trial of 44 healthy volunteers, initial CVP, PAOP, and LVEDA did not correlate with volume responsiveness. Moreover, changes in CVP and PAOP after a 3-L saline bolus failed to result in changes in cardiac index (CI) or stroke volume index (SVI). However, changes in LVEDA as measured by echocardiography did correlate with changes in SV.³ In comparison to CVP and PAoP, LVEDA is a more accurate measurement of
volume status but is difficult to measure continuously because of its reliance on echocardiogram. Finally, a systematic review of 803 patients concluded that CVP poorly correlated with blood volume, SV, and CO.4 Despite this mounting body of evidence, CVP and PAoP are still widely used to guide fluid management in the critical care setting.

**Dynamic Measurements**

Because static measures do not predict volume responsiveness well, many clinicians have adopted the use of dynamic measures in an effort to predict fluid responsiveness and cardiac performance. These dynamic measures establish a relationship between fluid responsiveness and variations in various cardiac performance measures over time. Dynamic markers are based on the principle of *pulsus paradoxus*, or the variation of stroke volume and blood pressure with respiration. Physiologically, this occurs because blood flow return to the heart varies with the undulation of intrathoracic pressure caused by breathing. In patients who are mechanically ventilated (more specifically, synchronous with the ventilator and/or paralyzed) and who are in normal sinus rhythm, similar respiratory-cycle variations in SV and pulse pressure (SBP-DBP) can be seen. Specifically, positive pressure ventilation causes an increase in intrathoracic pressure, which in turn causes decreased venous return and increased right ventricular afterload. This correlates to a decreased right ventricular and subsequently left ventricular output, which is manifested as a relative decrease in SV or pulse pressure. Because of blood transit time, this decrease is usually seen 2 seconds later, after the cessation of a delivered positive pressure breath (Fig 3).6 The variation in SV or pulse pressure (calculated as the maximum pulse pressure minus the minimum pulse pressure divided by the average of the two values) is exaggerated in periods of relative volume depletion. Specifically, a wide SV or pulse pressure variation indicates that a fluid challenge will result in an increase SV and better cardiac performance.

Many studies have demonstrated that the dynamic parameters of stroke volume variation (SVV) and pulse pressure variation are valid predictors of volume responsiveness. By way of example, a systematic review by Marik highlighted the results of 29 studies in which dynamic changes in arterial waveform outperformed static markers in predicting fluid responsiveness.7

![Figure 1](image1.png)

**Figure 1.** Patient A has a steeper starling curve than patient B. Both patients have identical changes in preload. Administration of a volume challenge will yield different changes in stroke volume. Patient A is more "volume responsive" than patient B.

![Figure 2](image2.png)

**Figure 2.** Schematic of the distal portion of a PA catheter and corresponding pressure tracings as the catheter travels through the heart. The PAoP reflects left atrial pressure. RA, right atrium; RV, right ventricle; PA, pulmonary artery.
Inferior Vena Cava Diameter

Inferior vena cava diameter (IVCd), or more specifically the variation in vena cava diameter during respiration as seen by echocardiography, is another valid dynamic mechanism by which fluid responsiveness can be measured. Much like SVV, IVCd variation during respiration is a function of increasing and decreasing intrathoracic pressures during respiration and has proven to be an accurate metric of volume responsiveness in mechanically ventilated and spontaneously breathing patients.\textsuperscript{8-10} IVCd is measured subcostally, approximately 0.5-4 cm below the junction of the IVC and the right atrium, in the longitudinal direction at a perpendicular angle to the IVC.\textsuperscript{11-15} Variation in IVCd is calculated as “the change” in IVCd during inspiration as compared with baseline (during expiration). Normative values for IVCd have been described in several studies, and, depending on the clinical scenario, range from 8 to 40 mm.\textsuperscript{14,16} Variation of greater than 10-18% in IVCd during a respiratory cycle has been shown to be predictive of volume responsiveness in several studies, (sensitivity ranging from 50% to 100%; specificity ranging from 53% to 100%, predefined variation).\textsuperscript{11,13,15} By way of example, Barbier and colleagues calculated the IVC distensibility index (calculated as the ratio of Dmax-Dmin/Dmin, expressed as a percent) in ventilated septic patients before and after volume challenge.\textsuperscript{11} The authors demonstrated that using an IVC distensibility threshold of 18% differentiated responders (predefined as an increase in CI > 15% after volume expansion) and nonresponders with 90% sensitivity and 90% specificity.\textsuperscript{11} IVCd is measured at the bedside using echocardiography in M-mode during a respiratory cycle.

Determination of vena cava diameter using echocardiography requires operator skill and thus is subject to error. Additionally, interpretation may be difficult in patients with ascites, morbid obesity, and in patients with intra-abdominal hypertension.\textsuperscript{11,13,15}

Passive Leg Raise

In the passive leg raise (PLR) test, the lower extremities are elevated above the heart of a recumbent patient mimicking the effect of a large fluid bolus on the central circulation. The postural maneuver is seen in Figure 4. Static and dynamic measures of CO are evaluated during this maneuver to determine if there is evidence of volume responsiveness. This may include changes in pulse pressure or SV, changes in MAP, or increases in CO or PAoP.

Evidence suggests that the PLR maneuver in critically ill, nonintubated patients not only predicts volume responsiveness but can also serve as a therapeutic intervention. A study by Maizel found that the PLR test induced changes in SV and CO (as measured by echocardiography and Doppler analysis) and was highly predictive of central hypovolemia (sensitivity 63-89% and specificity of 89%). Changes in CO witnessed during the PLR compared with that of a fluid bolus of 500 cc of normal saline correlated well.\textsuperscript{17} A study by Preau concluded that changes in stroke volume, pulse pressure, and femoral artery flow velocity as a result of a PLR were all highly predictive of fluid responsiveness (sensitivity of 79-86% and specificity of 80-90%).\textsuperscript{18} The straight leg raise is limited to those patients who can lay flat and can be put in the appropriate position. It is interesting to note that the PLR position is commonly seen in dialysis units in an effort to alleviate symptoms that arise in patients being dialyzed up to and perhaps beyond their dry weight. However, to date no studies have examined

Figure 3. Illustration of pulse pressure variation. PA, arterial pressure; PAW, airway pressure; PPmax, maximum pulse pressure; PPmin, minimum pulse pressure. Note that tracing occurs during positive = pressure ventilation. Reprinted with permission from Gunn SR, Pinsky MR. Implications of arterial pressure variation in patients in the intensive care unit. Curr Opin Crit Care. 2001;7(3):212-217.\textsuperscript{19}

PLR in the dialysis setting as a tool to optimize volume status.

It should be mentioned that the administration of a fluid bolus for diagnostic purposes (ie, indiscriminately giving a fluid bolus to a patient to determine a patient’s fluid responsiveness) is commonly done in clinical practice. However, this intervention is not always benign. One study by Michard found that 50% of hypotensive, critically ill patients are not fluid responsive.5 Moreover, the consequences of fluid administration to an unresponsive patient can be deleterious, affecting gas exchange and acid base status.19 Dynamic markers provide the clinician with the information to determine if a patient will or will not respond to volume before the fluid challenge, thereby helping to avoid situations of unnecessary and imprudent resuscitation.

Hemodynamic Monitoring Methods and Devices

Thermodilution

Advancements in PA catheter technology have allowed clinicians to calculate CO on the basis of flow of blood through the right ventricle. Thermodilution (TD)-measured CO is based on the Stewart-Hamilton equation (Equation 3).

$$Q = \frac{(V1 \times (Tb-T1) \times K1 \times K2)}{(Tb(t)dt)} \quad (Equation\ 3)$$

where $Q = CO$, $V1 = injectate$ volume, $Tb = blood$ temperature, $T1 = injectate$ temperature, $K1 = density$ factor, $K2 = constant$, and $Tb(t)dt = change$ in blood temperature as a function of time. CO correlates with the temperature gradient between the injectate and the patient’s blood and is inversely related to the change in blood temperature over time. The smaller the temperature change (ie, a higher volume of warm blood mixes with the cold injectate), the higher the CO. In practice, CO is determined after a known volume and temperature of fluid is injected into the proximal end of the PA catheter, which then mixes with the patient’s blood at a known temperature before entry into the right ventricle. Downstream at a given time interval, the blood-injectate temperature is measured again, allowing for an estimation of CO. Newer PA catheters are capable of continuous CO calculation using the same technique by virtue of an embedded proximal heating filament and distal thermistor built into the catheter. Several studies have demonstrated that TD is an accurate and valid way to measure CO.20,21 TD using the PA catheter has emerged as the gold standard in estimating CO and is the method against which all other devices are measured.

A large body of evidence suggests that the use of the PA catheter itself is controversial. A landmark study by Connors showed that for a large population of critically ill patients, PA catheterization resulted in an increased 30-day mortality, increased cost of health care, and a greater length of stay.22 A later trial by Sandham of surgical patients found no difference in mortality or length of stay between patients with and without PA catheters.23 More recently, a Cochrane Database systematic review of PA catheterization found no difference in mortality or length of stay in critically ill or surgical patients, but it did find increased health care costs associated with PA catheters.24 Theories as to why these outcomes exist include direct deleterious effects of the PA catheter itself (ie, arrhythmia, PA rupture, increased incidences of pulmonary embolism), or the harmful effects of the therapies implemented based on inappropriate interpretation of data.22

Transpulmonary Thermodilution

The transpulmonary thermodilution (TPTD) technique utilizes a standard central venous catheter and a thermistor, which is inserted in the femoral artery (Fig 5). A TD analysis, similar to the PA catheter TD methodology, can be generated by using the Stewart-Hamilton
equation (Equation 3). This method is potentially as accurate as the PA catheter TD technique, provided that there is (1) constant blood flow, (2) minimal loss of injectate, (3) complete mixing of the injectate with blood, and (4) only one pass from the proximal thermistor on the central line to the distal thermistor in the aorta. The TPTD is slightly less invasive than the PA catheter TD method and therefore is an attractive alternative.

Several studies have compared TPTD to other methods of hemodynamic monitoring. Sakka compared TPTD and PA catheter TD in 12 critically ill surgical patients and found good agreement between the two methods ($r = 0.98$, $P < 0.001$). Segal likewise found good correlation between TPTD (using an axillary artery as a distal thermistor site) and TD in 22 critically ill patients ($R^2 = 0.82$). A study by Goepfert found that a goal-directed therapy approach using TPTD in 40 cardiac bypass patients led to reduced pressor use, increased colloid administration, fewer days of mechanical ventilation, and a shorter time to achieve the status “fit for ICU discharge.”

Thoracic Electrical Bioimpedance

A known technology for the last 80 years, the thoracic electrical bioimpedance (TEB) has only recently become routinely available in the critical care setting. TEB is founded on the principles of Ohm’s law ($V = IR$), where impedance is based on the electrical resistance ($R$) of a circuit. In the human body, the electrical resistance of an applied current is affected by the relative water content in the descending aorta and will vary with the amount of blood flow through this vessel. Thus, a volume-replete patient will exhibit a low TEB compared with that of a volume-depleted patient. In practice, TEB is calculated using a system of connected electrodes through which a high-frequency, low-amplitude current is passed and is tracked on a recording device (Fig 6). TEB loses accuracy in the setting of increased extravascular lung water.

TEB has been studied in various clinical scenarios and results have been mixed. Resiner compared measuring CO by TEB versus by pulse contour analysis (see below) in healthy patients undergoing lower body negative pressure to simulate central hypovolemia. The two methods correlated well. Gujar studied 35 postoperative cardiac surgical patients and showed that TEB performed similarly to the PA catheter TD ($r = 0.856$, $P < 0.01$). In contrast, Petter found that TEB correlated poorly with TD in 33 heart failure patients. Moreover, a systematic review by Jensen concluded that TEB as a hemodynamic monitoring device is neither accurate nor precise. Further research and advancements in technology are needed before this method becomes widely adopted in the critical care setting.

Esophageal Doppler

Estimation of aortic blood flow through the use of continuous Doppler ultrasound positioned in the esophagus (ED) is a relatively noninvasive method of measuring CO. This technique is based on the principle that the velocity of blood flow travelling through the aorta is inversely related to the aortic diameter and directly related to flow (CO; Equation 4).

$$v = Q/A$$

(Equation 4)

where $v =$ velocity, $Q =$ flow, and $A =$ cross-sectional area. In the setting of reduced CO due to hypovolemia, flow velocity and aortic diameter will fall, as measured by the esophageal probe. As with all other tools used to estimate CO, ED has its limitations. Patients must be intubated to position the esophageal probe. Turbulent flow through the aorta caused by an aneurysm or atherosclerosis may confound calculations. Finally, it should be noted that aortic blood flow is only an estimation of CO because a significant portion of blood (upward of 30%) ejected from the left ventricle never reaches the thoracic aorta but flows to the vessels that stem from the aortic arch.

Despite these limitations, evidence supports the use of ED. A systematic review of 2400 paired measurements from 314 patients calculated a mean bias between TD-calculated and ED-calculated CO of only 0.19 L/min. Moreover, agreement for measuring change in CO during a fluid challenge was 86% ($P < 0.03$). A review of 25 studies by Laupland also showed good correlation between TD and ED ($R = 0.89$). Despite this evidence, this technique has not been widely adopted in critical care settings.
Pulse Contour Analysis

Pulse contour analysis (PCA) has recently emerged as an accurate method for measuring cardiac performance and has gained popularity because of its minimally invasive technique. In addition to measuring cardiac performance (SV, CO, CI), PCA provides dynamic markers (specifically SVV) that assist in determining volume responsiveness. PCA can be determined manually by obtaining routine measurements of an arterial line tracing and performing simple mathematics to determine variability throughout the respiratory cycle. Practically speaking, this can be accomplished by standing at the bedside for 30 seconds and observing the undulation on the arterial line monitor. Associated costs aside, the benefit of using a commercially available proprietary bedside computer to do this allows the clinician to calculate several metrics that would otherwise be difficult to accomplish (such as CO, CI, and SVV). Moreover, manufacturers of this technology argue that their proprietary models integrate several variables aimed at decreasing noise (age, sex, height, weight). However, data on this incremental benefit are lacking. There are several different PCA devices available, including the PICCO (Pulsion Medical Systems, Munich, Germany), the PulsCO (LidCO Limited, Cambridge, United Kingdom), and the FloTrac (Edwards Lifescience LLC, Irvine, CA). Of these devices, only the FloTrac does not require calibration before use. PICCO must be precalibrated with TPTD and thus needs a central venous catheter in addition to an arterial line. The PulsCO uses a lithium indicator and must be calibrated every 8 hours (and is contraindicated in patients on lithium or who are pregnant). All of these systems perform similarly in comparative trials.35

PCA technology has its limitations. The arterial catheter site or the presence of atherosclerosis may adversely affect the accuracy of the technology.6 Additionally, chest wall compliance, tidal volumes, and level of positive end-expiratory pressure reduce the accuracy of PCA.6,7 In patients with open chests, PCA was not found to be helpful in predicting volume status.36 Likewise, Lahner found that SVV determined by the FloTrac system failed to predict volume responsiveness in patients undergoing major abdominal surgery.37 PCA has not been validated in unstable patients, spontaneously breathing patients, or in those with cardiac rhythms other than sinus (although research is ongoing).38-40

There has been some attention paid recently to pulse oximetry waveform variation as a means to calculate volume status. The principle behind this technology is similar to the method using an arterial catheter. The pulse oximetry curve represents the infrared light absorbed by circulating hemoglobin during a cardiac cycle. Variation in the amplitude of this curve can be mathematically related to the amount of blood in the capillary bed, which is in turn related to a patient’s volume status. A systematic review of pulse oximetry waveform variation demonstrated that this method accurately predicted volume responsiveness.41 The less invasive nature of this method makes it an attractive option for future directions of research.

Summary

In summary, there are several hemodynamic monitoring tools available in the critical care setting to assist in determining CO and volume responsiveness. Fluid status and the potential to improve cardiac performance with volume challenge can be measured by static and dynamic measures, respectively. In general, dynamic metrics appear to be more robust in determining volume responsiveness. Monitoring cardiac performance (SV and CO) can also prove invaluable when caring for the hemodynamically unstable patient, and there are various bedside technologies that provide this information. The PA catheter TD technique remains an accurate means of monitoring CO and remains the gold standard. Other less invasive monitoring systems are now available and are in the process of validation. Ultimately, good clinical judgment, appropriate interpretation of values, and judicious use of devices can, in aggregate, improve hemodynamic management and end organ perfusion. Continued research comparing these tools, increased availability in intensive care unit settings, and advancements in technology will further shape the landscape in hemodynamic monitoring.

References


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