CHEST Postgraduate Education Corner
CONTEMPORARY REVIEWS IN CRITICAL CARE MEDICINE

Hemodynamic Monitoring

Steven M. Hollenberg, MD, FCCP

Hemodynamic assessment is a key component of the evaluation of the critically ill patients and has both diagnostic and prognostic utility. This review outlines a general approach to assessment of hemodynamics and perfusion, and then discusses various hemodynamic parameters: heart rate, BP, intravascular (central venous and pulmonary artery) pressures, cardiac output, and myocardial performance, within the context not only of how they are best measured but also how they should be used in a clinical context. Hemodynamics are best assessed using a combination of not only different hemodynamic parameters but also those with the inclusion of clinical indices of perfusion. The benefits of these techniques, as with all medical testing and interventions, must be weighed against any potential risks. Although what to measure and how to measure it is important, what is most important is how to use the information. Evaluating the response to therapeutic interventions is frequently the most useful way to employ hemodynamic monitoring techniques. For the practitioner, learning how to select from a robust set of hemodynamic tools and how to tailor their use to individual clinical settings will allow for optimal patient care.

CHEST 2013; 143(5):1480–1488

Abbreviations: CO = cardiac output; CVP = central venous pressure; HR = heart rate; PA = pulmonary artery; PAC = pulmonary artery catheter; PAOP = pulmonary artery occlusion pressure; SV = stroke volume; Svo_2 = mixed venous oxygen saturation

Hemodynamics (from the words "blood" and "power" in Greek) refers to the forces that determine blood flow in the circulation. The goal of circulation is to delivery oxygen and other nutrients to the tissues, and, thus, the ultimate concern is tissue and cellular perfusion. Clinicians caring for patients therefore need to focus not only on hemodynamic forces, but also on hemodynamic parameters that address the amount and adequacy of blood flow within the circulatory system.

Although entitled "Hemodynamic Monitoring," this review will consider hemodynamic assessment as well. The distinction is that hemodynamic assessment evaluates the current state of blood flow and perfusion with consideration of potential causes for abnormalities, whereas hemodynamic monitoring is a serial assessment that can establish clinical trajectories and assess the response to therapeutic interventions. Although the terms are often used interchangeably, and monitoring is clearly a form of assessment, consideration of their differences can help clarify some of the confusion about how to assess their effects. An assessment is diagnostic and would not by itself be expected to alter outcome. Monitoring is intended to detect physiologic abnormalities at their earliest stages, when interventions would be expected to be most effective, and to evaluate the effects of therapeutic maneuvers.

The effects of hemodynamic measurements on outcomes depend more on the efficacy of the therapies than on the techniques chosen to make those measurements. Nonetheless, the point of either assessment or serial monitoring is to aid treatment. The risks of any hemodynamic monitoring technique must be weighed against these potential benefits. This brief review will take a physiologic and clinical approach to hemodynamic monitoring, considering what parameters to monitor, when to measure them, and how to use the information. Techniques tend to evolve rapidly,

Manuscript received July 29, 2012; revision accepted October 30, 2012.
Affiliations: From the Cooper Medical School of Rowan University; and Coronary Care Unit, Cooper University Hospital, Camden, NJ.
Correspondence to: Steven M. Hollenberg, MD, FCCP, Divisions of Cardiovascular Disease and Critical Care Medicine, Cooper University Hospital, One Cooper Plaza, 366 Dorrance, Camden, NJ 08103; e-mail: Hollenberg-Steven@cooperhealth.edu.

DOI: 10.1378/chest.12-1901

another factor that suggests the merits of a conceptual approach to hemodynamic monitoring.

Technical Considerations and Monitoring Systems

Detailed consideration of technical aspects of monitoring devices is beyond the scope of this article; the reader is referred to a number of excellent reviews that address these in detail.[1-4] This article is also not specifically intended to guide the choice among various devices that measure the same or similar parameters. Nonetheless, a thorough understanding of the technology is crucial for assessment of the data obtained.

The first requirement of a monitoring system is accuracy. Clinicians must be able to assess the reliability of a given measurement, and in particular, to discern when a device may not be providing accurate data.

Specific techniques must be chosen to fit the patient and the clinical setting. The overall milieu may be the most important factor of all; hemodynamic monitoring entails serial measurements by a team of multidisciplinary practitioners, all of whom need to have a certain level of comfort with the data being obtained. There is a learning curve with all devices, and thus a requisite investment of time and effort if a device is to be used. Local expertise and comfort level with a particular technique may be as important as any other factor in the choice of a hemodynamic monitoring modality.

General Approach

The key issue in hemodynamic assessment is perfusion. Clinical evaluation (history, physical examination, imaging, laboratory data) is crucial and may be sufficient in some cases. Clinical indices of insufficient perfusion include oliguria, clouded sensorium, delayed capillary refill, and cool skin.[5-7] Other measures, such as serum lactate levels and mixed venous oxygen saturation (Svo_2), provide additional information about perfusion. Nonetheless, hemodynamic therapies for shock are usually (and appropriately) targeted at the determinants of perfusion, namely pressure and cardiac output (CO), and clinical assessment may not always be optimally sensitive and specific. This review will focus on monitoring of pressure and flow, with subsequent consideration of how to assess perfusion. When perfusion is compromised, an assessment of its components may be crucial to formulate treatment strategies. There is no intent to minimize the value of clinical judgment in planning and implementing those strategies.

Monitors can be thought of as measuring either hemodynamic parameters themselves, or the effects of those parameters, with obvious overlap. Hemodynamic parameters that determine perfusion include heart rate (HR), BP, intravascular pressures, CO/stroke volume (SV), and ventricular performance (Table 1). The adequacy of perfusion is difficult to measure directly, but it may be possible to get a sense of the adequacy of global perfusion by measuring mixed or central venous oxygen saturation. This review is organized by parameter, with consideration of various techniques to assess that parameter, and the strengths and weaknesses of each technique.

Monitoring techniques should be used in a clinical context—with a clear sense of what the clinician is going to do with the information. An initial hemodynamic assessment is most often useful for diagnostic purposes, usually in the setting of perfusion failure, which may have been occult. That assessment can measure the extent of the hemodynamic abnormalities and may help determine their cause. Subsequent hemodynamic monitoring is most often used to assess the effects of interventions, something best accomplished by using techniques in a dynamic, rather than a static, fashion. This last point is crucial and sometimes underappreciated. It should come as no surprise that a single measurement is less useful than serial assessments of the trend over time or of the response to interventions. When assessments are inadequate, what may be needed is not a better method but a better understanding of how to use an available technique.

Hemodynamic Parameters

Heart Rate

Although HR is relatively simple to assess, its importance should not be underestimated. HRs that are too rapid or too slow can compromise CO. Whether the HR is appropriate to the physiology and degree of illness is also worth consideration. Finally, whether a vasoactive agent is increasing CO via a chronotropic mechanism (by increasing HR) or inotropic mechanism (by increasing SV) can be a crucial distinction.

HR is usually assessed using a bedside electrocardiographic monitor, which gives an instantaneous reading of beats per minute over the prior several seconds. Many bedside monitors are not designed to

Table 1—*Hemodynamic Parameters*

Hemodynamic Parameters
Heart rate
BP
Vascular pressures
Central venous pressure
Pulmonary artery pressure
Cardiac output
Ventricular performance

allow display of the HR trend over the past hours or even minutes and, in this sense, monitoring technology limits the availability of potentially useful data to clinicians, a state of affairs that might merit some rethinking. Bedside flow sheets can trend HR and other parameters, but their use is waning with the move to fully electronic medical records.

Recent data suggest the potential utility of beat-to-beat HR variability as a nonlinear index of degree of illness in shock.[8,9] This remains investigational, but there is no technical reason why trends or nonlinear parameters could not be displayed on a monitor if clinicians declared them useful in patient care.

BP

Noninvasive arterial BP monitoring is widely employed, but estimation of BP using a cuff in the setting of shock, especially an automated measurement system, may be inaccurate. Use of an arterial cannula provides a more appropriate and reproducible measurement of arterial pressure.[6,10] An important benefit of invasive arterial BP monitoring is that it allows beat-to-beat analysis, so that decisions regarding therapy can be based on immediate BP information.[11]

BP by itself is not a particularly good index of perfusion. A number of randomized studies have shown that raising mean arterial pressure from 65 to 85 mm Hg has no demonstrable effect on renal function or metabolic measures of perfusion.[12,13] Below a certain BP, however, autoregulation in vascular beds is compromised, and flow is dependent on pressure. Although the precise BP goal to target in shock remains uncertain, targeting a mean arterial pressure of 60[6] or 65[14] mm Hg is recommended.

Variations in arterial pressure with respiration can be used as an index of fluid responsiveness (also termed preload responsiveness, meaning that SV increases with fluid administration). If the changes in venous return consequent to changes in intrathoracic pressure with respiration are sufficient to influence SV, this suggests relative hypovolemia. In intubated and sedated patients, variability of systolic pressure, pulse pressure, or SV $>$ 10% to 12% is correlated with an increase in CO of at least 15% in response to fluid administration.[15,16] In spontaneously breathing patients, this measure is much less reliable,[16] although some studies suggest that the response to straight leg raising can be used in a similar fashion, albeit with lower accuracy.[17] There are a number of potential challenges in assessment of arterial pressure variation. The tidal volume must be adequate and constant, so sedation must be sufficient to suppress spontaneous ventilation. Irregular rhythms such as atrial fibrillation limit the use of pulse pressure variation as well. These measures may not perform well in patients with ARDS.[18] What is clear, however, is that such dynamic monitoring is superior to static measurements of intravascular pressures in predicting preload responsiveness,[19] and arterial cannulation can enable assessment of these dynamic parameters. Assessment of beat-to-beat variability of pressure obviously requires technology that measures BP on every heart beat.

Vascular Pressures

Central Venous Pressure: Central venous pressure (CVP) can be measured readily by placing a transducer in series with any catheter in central venous circulation. CVP is equivalent to right atrial pressure (except in the uncommon setting of vena caval obstruction), and provides an estimate of right ventricular preload. Use of CVP to measure fluid responsiveness, however, is complicated by a number of factors. First, the waveform is complex, with an *a* wave resulting from atrial contraction, a *c* wave coincident with the closing of the tricuspid valve in end-diastole, and a *v* wave caused by atrial filling, with an *x* descent after the *c* wave and a *y* descent after the *v* wave (Fig 1). Strictly speaking, CVP should be measured in end-expiration at the base of the *c* wave, but if the *c* wave is not visible then the average of the *a* wave is used.[20] The second, and even more daunting challenge, is that the physiologic determinants of CVP are multiple and do not remain constant. CVP is determined by interactions among venous return, which is a function of blood volume and compliance of the venous

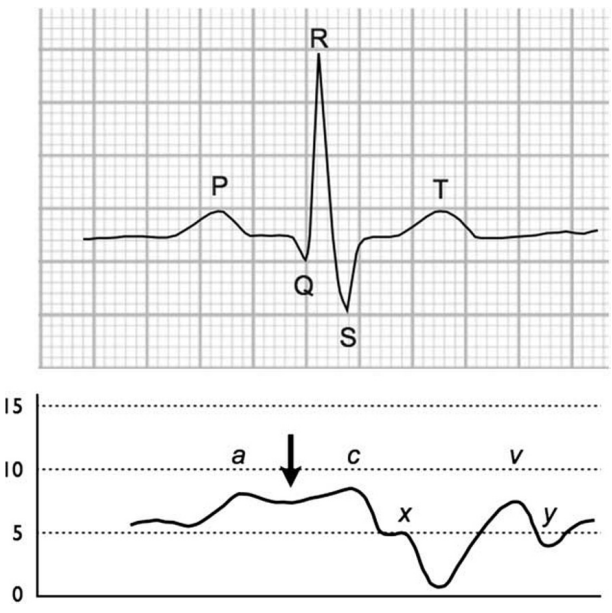

FIGURE 1. A typical central venous pressure (CVP) waveform (bottom tracing) and accompanying ECG (top). The a, c, and v waves and the x and y descents are shown. CVP should be measured at end expiration at the base of the C wave (see arrow).

system, right ventricular function, and pulmonary arterial pressure. In critically ill patients, these are rarely static, and so it should come as little surprise that a single measurement of CVP is a poor predictor of fluid responsiveness.[21] Tricuspid regurgitation, which produces large v waves, can also complicate interpretation of the CVP. Finally, use of CVP as an index of left ventricular preload implies that right-sided pressures are normal; in an ICU population with a high incidence of both acute and chronic pulmonary abnormalities, this is often not the case.

Pulmonary Artery Pressure: Pulmonary artery (PA) pressure can be measured at the bedside using a balloon-tipped flow-directed (Swan-Ganz) catheter.[22] On insertion, pressures are measured in the right atrium, right ventricle, and PA. A PA occlusion ("wedge") pressure (PAOP) is typically obtained by inflating the balloon at the end of the catheter (Fig 2). When there is no pulmonary venous obstruction (something that is rare), a continuous column of blood is present between the left atrium and the tip of the catheter, and thus measured pressure reflects left atrium pressure. On catheter insertion, blood can be withdrawn for determination of oxygen saturation in the right atrium, right ventricle, and PA, which can be useful for the evaluation of intracardiac shunts, and SvO_2 can be measured, either continuously or in blood samples drawn from the PA. The PA catheter can also be used to measure CO by thermodilution; cold saline is injected in the proximal port, and the temperature change is measured at the tip of the catheter. This can be done by injection at the bedside, and some catheters enable continuous measurement of CO.

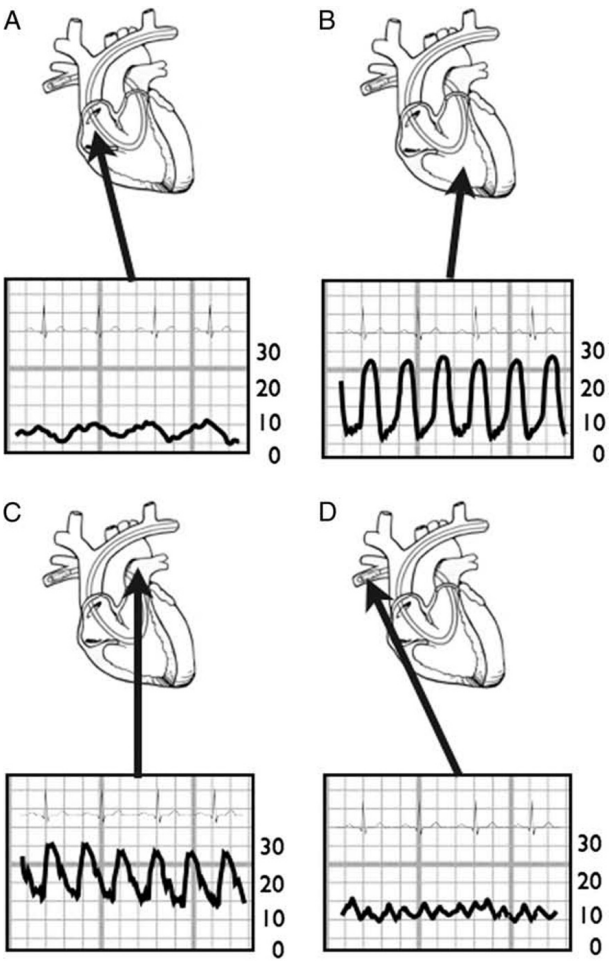

FIGURE 2. Pulmonary artery catheterization waveforms. A, Right atrium. B, Right ventricle. C, Pulmonary artery. D, Pulmonary artery occlusion.

There are a number of pitfalls to be considered in using PAOP as a surrogate for left ventricle preload. Use of the PAOP for this purpose assumes that there is no significant mitral valve disease (or pulmonary venous obstruction, something that is quite uncommon), and a constant relationship between pressure and volume in the left ventricle. These assumptions may not be satisfied in many critically ill patients. In addition, there are a number of technical pitfalls in measurement, including not ensuring that the transducers are correctly positioned and calibrated, not considering the effects of positive end-expiratory pressure on transmural pressures, positioning the PA catheter in a zone of the lung in which alveolar pressures exceed vascular pressures (in which case catheter pressure may not reflect vascular pressure throughout the respiratory cycle), and incorrectly interpreting the waveforms.[23,24]

Evaluating the utility of the PA catheter solely on the ability of a single determination of PAOP to measure left ventricle preload seems short-sighted. As noted previously, other measurements, including right-sided pressures, CO, and SvO_2 can be obtained from the pulmonary artery catheter (PAC) and used to generate a more complete hemodynamic profile. But more to the point, while a single assessment can be used for diagnostic purposes, the PAC can and should be used in a serial fashion, to assess changes in multiple parameters, including filling pressures, SV, and SvO_2, either in response to physiologic stress or during the course of therapeutic interventions. For example, while a given level of PAOP does not provide much information about fluid responsiveness, the PAC, by measuring changes in pressures, SV, and SvO_2 after fluid challenge, can help guide clinicians about how to optimize oxygen delivery while minimizing the risk of pulmonary edema. In this context, it is perhaps as important to know when not to give additional fluids as when to give them; when fluid challenges raise filling pressures but do not result in improvements in SV or clinical status, the clinician has received a clear indication to stop.

There has been considerable controversy concerning the use of PACs since the publication of a retrospective report suggesting their use may be associated with patient harm.[25] This finding has not been confirmed in subsequent randomized trials, and is likely to have been influenced by several factors, including inaccurate acquisition and interpretation of the data. More importantly, the propensity matching used in that study did not account for the selection bias caused by the fact that PACs are more likely to be used in patients with a downward clinical trajectory. Nonetheless, there are important risks, including infection, perforation, occlusion, thrombosis and embolism with indwelling catheters, arrhythmia when catheters are placed in or transit the right ventricle, and a risk of rupture of the PA with balloon occlusion to obtain a PAOP, particularly in patients with pulmonary hypertension. Overall, it is clear that routine use of PACs in a number of settings is not associated with improved outcomes,[26-32] something that perhaps should not come as a surprise. And it should be clear from this review that PAC is not the only available tool for hemodynamic assessment. However, regardless of the opinion of an individual clinician on the balance between the risks and benefits of the PAC, it is important not to take away from the controversy the lesson that hemodynamic assessment has no value. An appropriately focused hemodynamic assessment, regardless of the method by which the information is obtained, can assist in patient management in ways that may not be entirely measurable in clinical trial outcomes.

CO/Stroke Volume

CO is reported more commonly than SV, but one might make a persuasive case that SV is the more relevant hemodynamic parameter. CO is calculated as the product of HR and SV, but changes in HR do not necessarily lead to linear changes in CO since cardiac filling is influenced by filling time. Evaluation of the response of SV to hemodynamic optimization is desirable since the goal is usually to increase CO by increasing SV, not by increasing HR.

Pulmonary Artery Catheters: The reference method for measurement of CO involves use of the Fick principle,[33] where uptake equals flow times the difference between inflow and outflow concentrations; this assumes a steady state. Indicator dilution with indocyanine green was the original technique, but this method is difficult and cumbersome, so thermodilution is most commonly performed.[34] Thermodilution measures CO over several cycles, and has a number of technical limitations that can limit its accuracy, most prominently tricuspid regurgitation, which is not uncommon in critically ill patients with pulmonary disease (Fig 3). Thermodilution CO is usually measured using a PAC; with repeated bolus injections it is considered accurate to ± 5% to 10%.[35] Continuous CO can be measured using a modified PAC with an embedded heating filament; this displays continuous trended CO, but it is important to realize that the displayed measurement represents an average flow over the prior 3 to 6 min.[36] With either method, effective net forward SV can be calculated by dividing CO by HR. In patients with significant valvular regurgitation or intracardiac shunting, the actual SV may be significantly underestimated.

Pulse Contour Analysis: CO can also be estimated by analysis of the pulse contour from an arterial waveform, since the systolic portion of the waveform reflects SV. This also provides information about changes in SV on a beat-to-beat basis. Several different systems are available. The LIDCO device uses a radial arterial waveform and was designed to be calibrated using lithium dilution (although a recent model uses a calibration equation).[37] The PICCO device uses a femoral artery catheter calibrated using transpulmonary thermodilution, in which a bolus is injected into the central veins while temperature is measured from the femoral catheter; this technique also allows for estimation of intrathoracic blood volume.[38] The PICCO device can be used in axillary arteries as well. Both of these devices may need to be recalibrated over time and with changes in clinical status. A third system (Vigileo) uses a blood-flow sensor connected to an arterial line, and is not calibrated externally but using

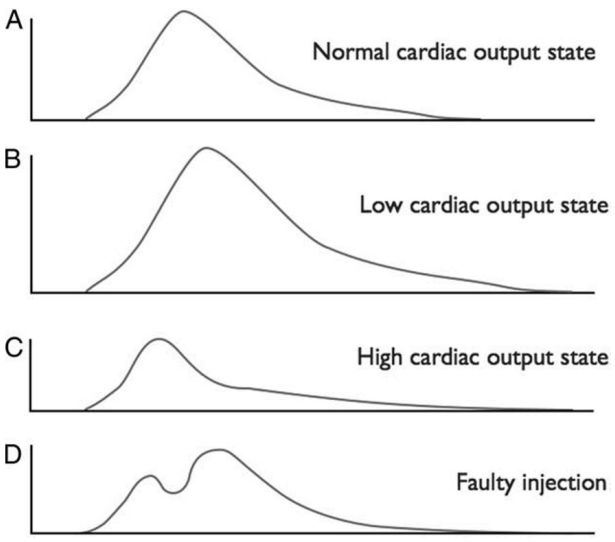

FIGURE 3. Thermodilution cardiac output curves. A, Normal cardiac output. B, High cardiac output. Rapid dilution of cold injectate with warm blood leads to a smaller area under the temperature-time curve. C, Low cardiac output. Slower dilution of cold injectate with warm blood. D, Faulty injection with double dip. The measurement should be disregarded.

an equation that incorporates arterial pulsatility (the SD of the pressure wave over a 20-s interval) and a constant derived from the patient's biometric values (sex, age, height, and weight) and the skewness and kurtosis of the arterial waveform.[39] This calibration constant is updated every minute. The software algorithms to perform this calibration have been updated over time.

The need for recalibration of the LIDCO and PICCO devices can pose interpretive challenges for the clinician when recalibration yields CO values that differ from previous determinations; the question then becomes whether the new values represent changes in clinical status or in calibration. The Vigileo system recalibrates itself, but the clinician has no way to assess the influence of calibration changes on the measured CO. Another important assumption when using pulse waveform analysis is that arterial compliance has remained constant. It is clear that this assumption may be correct over short time periods but is unlikely to remain valid with clinical instability.[36] Recalibration after events that cause profound changes in vasomotor tone, such as sepsis or cardiopulmonary bypass, is recommended. CO measured using pulse contour analysis has been found to be reasonably comparable to values obtained with thermodilution.[40,41]

Ultrasound: SV can also be assessed by echocardiography and then multiplied by HR to yield CO. SV can be measured by multiplying the velocity-time integral of a Doppler signal across the left ventricular outflow tract by the outflow tract area. Whereas transthoracic or transesophageal echocardiography usually gives a SV at only one time point, esophageal Doppler probes can be used for continuous monitoring.[42] Using this technique, the diameter of the descending aorta is either estimated from biometric parameters or measured using M-mode echocardiography built into the device. The Doppler signal needs to be measured at the same angle, so the signal must be audited carefully, something that requires experience and judgment. In addition, this measurement is performed in the descending aorta, excluding blood flow to the upper part of the body. The limits of agreement with this technique are broad when compared with other techniques, but esophageal Doppler may be easier to implement, and its use has in fact been shown to decrease perioperative morbidity and shorten ICU length of stay.[43,44]

Other Techniques: Bioimpedance cardiography can be used to estimate CO. Blood is conductive of electricity, and thus cardiac contraction produces a cyclic change in transthoracic impedance of about 0.5%. Conversion from those changes to SV is done using mathematical algorithms that have evolved since the method was first developed. Bioreactance has been more thoroughly tested and is more accurate in relatively stable patients, often outpatients with heart failure, than in critically ill patients, in whom numerous factors may be contributing to changes in thoracic impedance.[45] Newer techniques under development may show some promise.

Changes in CO_2 in expired air can be used to calculate CO by the Fick principle, thus providing a noninvasive measure of CO. The technique actually measures effective lung perfusion, the capillary blood flow through ventilated parts of the lung, and thus its accuracy is reduced in patients with ventilation-perfusion abnormalities, the latter being common in critically ill patients.[46]

Use of Stroke Volume/CO: CO monitoring is used to assess hemodynamic responses to therapeutic interventions such as fluid resuscitation and vasoactive therapies, but the key clinical question is often not what the CO is but rather whether that output is sufficient to meet a patient's metabolic needs. Mixed venous oxyhemoglobin saturation reflects the balance between oxygen delivery and consumption and can provide an indication of the adequacy of global oxygen delivery; low values indicate increased oxygen extraction and therefore potentially inadequate resuscitation (Fig 4). Mixed venous oxyhemoglobin saturation should be measured in fully mixed blood, as saturation in the superior vena cava can differ from that in the inferior vena cava, and so is measured in

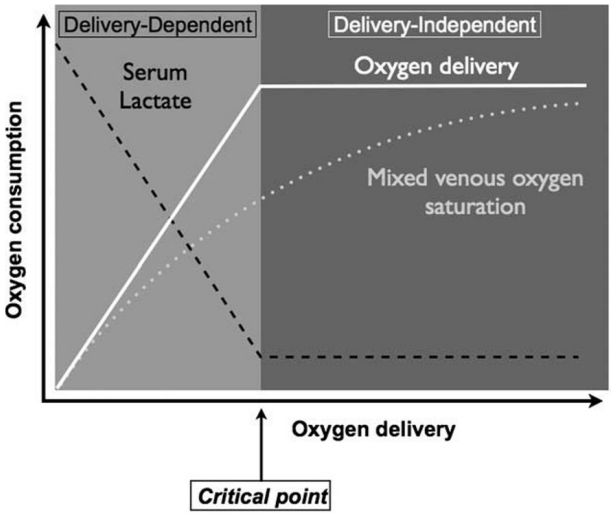

FIGURE 4. Relationship between oxygen delivery and oxygen consumption. When oxygen delivery is adequate, oxygen consumption is autoregulated and does not vary with delivery. Tissues extract more or less oxygen to meet their needs, and this may be reflected in mixed venous oxygen saturation. When delivery drops below a certain critical point, consumption begins to drop. Mixed venous oxygen saturation falls more rapidly, and serum lactate levels increase.

blood drawn from the distal port of a PAC, or using a PAC with a continuous saturation monitor at the distal tip. A clinical study showed that monitoring of central venous oxygen saturation can be a valuable guide to early resuscitation.[47] This measure, while not precisely equivalent to Svo_2,[48] does trend in the same direction and correlates reasonably well.[49] Oxygen saturation in the femoral vein, however, is not a good measure of global tissue perfusion.[50] The ability to get a sense of the adequacy of a given CO in addition to measuring that output in a serial fashion is an important feature of use of a PAC.

Global oxygen delivery may or may not accurately reflect delivery to tissue at the cellular level, which is the most important determinant of organ function. Part of the issue is distribution within the microcirculation. Direct visualization of the sublingual circulation has shown microcirculatory perturbation in patients with cardiogenic[51] and septic shock.[52] Sublingual capnometry correlates with microcirculatory findings,[53] and other techniques under investigation, such as near infrared spectroscopy, may potentially address the adequacy of microcirculatory flow. These techniques are better measures of the degree of microcirculatory perfusion than its adequacy, but changes appear to track the clinical course.[54,55] All of these techniques, however, pose technical challenges, and their utility in the ICU is under investigation.

Ventricular Size and Function: Evaluation of cardiac performance is a key component of a hemodynamic assessment. Echocardiography with color-flow Doppler is noninvasive, low risk, and can help to provide an expeditious assessment of cardiac chamber size, both left and right ventricular function, valvular structure and motion, atrial size, and the anatomy of the pericardial space. Echocardiography can be used to ascertain overall and regional systolic function as well as diastolic function.[56] If transthoracic echocardiographic images are suboptimal, contrast may be used to improve image quality, or transesophageal echocardiography may be performed.

Echocardiography can also be used to provide a hemodynamic assessment. As previously noted, Doppler across the aortic outflow tract can be used to measure SV. Diastolic ventricular dimensions give a sense of chamber size and allow inferences into the adequacy of ventricular filling.

Imaging can also be useful to predict the response to a fluid challenge. The dimension of the inferior vena cava and its response to respiration on transthoracic echo,[57] or a similar assessment of the superior vena cava on transesophageal echo,[58] have been shown to predict increased SV after a fluid bolus.[59]

Although the hemodynamic assessment possible with echocardiography is probably comparable to that using more invasive techniques, its use for monitoring is more problematic. The first obstacle is the technical challenge of image acquisition, a skill that is operator dependent and may not be evenly distributed among the staff at different times. Serial imaging is also time-consuming. A focused cardiac ultrasound examination can provide some of the previously mentioned parameters,[60] and this may suffice in some clinical settings, but serial imaging, even focused serial imaging, takes time and effort. Comparisons between echocardiography and other techniques need to take into consideration these logistic factors and the goals of the hemodynamic assessment.

Conclusion

Hemodynamic assessment is a key component in the evaluation of critically ill patients. It has potential diagnostic and prognostic utility, and, in the opinion of this author, need not necessarily be proven to improve hard outcomes to be considered beneficial. Nonetheless, all diagnostic techniques should be used in a clinical context, with a clear sense that their results have the potential to alter management, and their benefits must be weighed against their potential risks.

Technical aspects of hemodynamic monitoring techniques are vital to their proper understanding. Hemodynamics are best assessed using a combination not only of different hemodynamic parameters but with the inclusion of clinical indices of perfusion.

Consideration of different determinants of flow should guide which parameters to assess and which to monitor. In some contexts, intravascular volumes are important, in others SV and CO are important, and a sense of overall cardiac performance may be desirable. Evaluating the response to therapeutic interventions is frequently the most useful way to employ hemodynamic monitoring techniques.

The key point is that what to measure is important, and how to measure is also important, but what is most important is how to use the information. Seen in this way, debates about the relative utility of some techniques over others, and the failure of some techniques to alter outcomes in selected population become less important. For the practitioner, learning how to select from a robust set of hemodynamic tools and how to tailor their use to individual clinical settings will allow for optimal patient care.

Acknowledgments

Financial/nonfinancial disclosures: The author has reported to *CHEST* that no potential conflicts of interest exist with any companies/organizations whose products or services may be discussed in this article.

References

1. Polanco PM, Pinsky MR. Practical issues of hemodynamic monitoring at the bedside. *Surg Clin North Am*. 2006;86(6):1431-1456.
2. Montenij LJ, de Waal EE, Buhre WF. Arterial waveform analysis in anesthesia and critical care. *Curr Opin Anaesthesiol*. 2011;24(6):651-656.
3. Vincent JL, Rhodes A, Perel A, et al. Clinical review: update on hemodynamic monitoring—a consensus of 16. *Crit Care*. 2011;15(4):229.
4. Alhashemi JA, Cecconi M, Hofer CK. Cardiac output monitoring: an integrative perspective. *Crit Care*. 2011;15(2):214.
5. Practice parameters for hemodynamic support of sepsis in adult patients in sepsis. Task Force of the American College of Critical Care Medicine, Society of Critical Care Medicine. *Crit Care Med*. 1999;27(3):639-660.
6. Hollenberg SM, Ahrens TS, Annane D, et al. Practice parameters for hemodynamic support of sepsis in adult patients: 2004 update. *Crit Care Med*. 2004;32(9):1928-1948.
7. Hollenberg SM. Vasopressor support in septic shock. *Chest*. 2007;132(5):1678-1687.
8. Seely AJ, Macklem PT. Complex systems and the technology of variability analysis. *Crit Care*. 2004;8(6):R367-R384.
9. Arnold RC, Greene G, Glaspey L, Hollenberg SM, Seely AJ. Continuous heart rate predicts worsening organ failure and mortality in sepsis [abstract]. *Shock*. 2010;33:81.
10. Cohn JN. Blood pressure measurement in shock. Mechanism of inaccuracy in ausculatory and palpatory methods. *JAMA*. 1967;199(13):118-122.
11. Hollenberg SM, Parrillo JE. Shock. In: Fauci AS, Braunwald E, Isselbacher KJ, et al, eds. *Harrison's Principles of Internal Medicine*. 14 ed. New York, NY: McGraw-Hill; 1997:214-222.
12. LeDoux D, Astiz ME, Carpati CM, Rackow EC. Effects of perfusion pressure on tissue perfusion in septic shock. *Crit Care Med*. 2000;28(8):2729-2732.
13. Bourgoin A, Leone M, Delmas A, Garnier F, Albanèse J, Martin C. Increasing mean arterial pressure in patients with septic shock: effects on oxygen variables and renal function. *Crit Care Med*. 2005;33(4):780-786.
14. Dellinger RP, Carlet JM, Masur H, et al; Surviving Sepsis Campaign Management Guidelines Committee. Surviving Sepsis Campaign guidelines for management of severe sepsis and septic shock [published correction appears in *Crit Care Med*. 2004;32(6):1448. Dosage error in article text. *Crit Care Med*. 2004;32(10):2169-2170]. *Crit Care Med*. 2004;32(3):858-873.
15. Bendjelid K, Romand JA. Fluid responsiveness in mechanically ventilated patients: a review of indices used in intensive care. *Intensive Care Med*. 2003;29(3):352-360.
16. Marik PE, Cavallazzi R, Vasu T, Hirani A. Dynamic changes in arterial waveform derived variables and fluid responsiveness in mechanically ventilated patients: a systematic review of the literature. *Crit Care Med*. 2009;37(9):2642-2647.
17. Monnet X, Rienzo M, Osman D, et al. Passive leg raising predicts fluid responsiveness in the critically ill. *Crit Care Med*. 2006;34(5):1402-1407.
18. Lakhal K, Ehrmann S, Benzekri-Lefèvre D, et al. Respiratory pulse pressure variation fails to predict fluid responsiveness in acute respiratory distress syndrome. *Crit Care*. 2011;15(2):R85.
19. Michard F, Teboul JL. Predicting fluid responsiveness in ICU patients: a critical analysis of the evidence. *Chest*. 2002;121(6):2000-2008.
20. Magder S. Central venous pressure: a useful but not so simple measurement. *Crit Care Med*. 2006;34(8):2224-2227.
21. Antonelli M, Levy M, Andrews PJ, et al. Hemodynamic monitoring in shock and implications for management. International Consensus Conference, Paris, France, 27-28 April 2006. *Intensive Care Med*. 2007;33(4):575-590.
22. Swan HJC, Ganz W, Forrester J, Marcus H, Diamond G, Chonette D. Catheterization of the heart in man with use of a flow-directed balloon-tipped catheter. *N Engl J Med*. 1970;283(9):447-451.
23. Pulmonary Artery Catheter Consensus Conference Participants. Pulmonary Artery Catheter Consensus conference: consensus statement. *Crit Care Med*. 1997;25(6):910-925.
24. Iberti TJ, Daily EK, Leibowitz AB, Schecter CB, Fischer EP, Silverstein JH. Assessment of critical care nurses' knowledge of the pulmonary artery catheter: The Pulmonary Artery Catheter Study Group. *Crit Care Med*. 1994;22(10):1674-1678.
25. Connors AF Jr, Speroff T, Dawson NV, et al; SUPPORT Investigators. The effectiveness of right heart catheterization in the initial care of critically ill patients. *JAMA*. 1996;276(11):889-897.
26. Sandham JD, Hull RD, Brant RF, et al; Canadian Critical Care Clinical Trials Group. A randomized, controlled trial of the use of pulmonary-artery catheters in high-risk surgical patients. *N Engl J Med*. 2003;348(1):5-14.
27. Harvey S, Harrison DA, Singer M, et al; PAC-Man study collaboration. Assessment of the clinical effectiveness of pulmonary artery catheters in management of patients in intensive care (PAC-Man): a randomised controlled trial. *Lancet*. 2005;366(9484):472-477.
28. Rhodes A, Cusack RJ, Newman PJ, Grounds RM, Bennett ED. A randomised, controlled trial of the pulmonary artery catheter in critically ill patients. *Intensive Care Med*. 2002;28(3):256-264.
29. Richard C, Warszawski J, Anguel N, et al; French Pulmonary Artery Catheter Study Group. Early use of the pulmonary artery catheter and outcomes in patients with shock and acute respiratory distress syndrome: a randomized controlled trial. *JAMA*. 2003;290(20):2713-2720.
30. National Heart, Lung, and Blood Institute Acute Respiratory Distress Syndrome (ARDS) Clinical Trials Network; Wheeler AP, Bernard GR, Thompson BT, et al. Pulmonary-artery versus central venous catheter to guide treatment of acute lung injury. *N Engl J Med*. 2006;354(21):2213-2224.
31. Binanay C, Califf RM, Hasselblad V, et al; ESCAPE Investigators and ESCAPE Study Coordinators. Evaluation study of congestive heart failure and pulmonary artery catheterization effectiveness: the ESCAPE trial. *JAMA*. 2005;294(13):1625-1633.
32. Shah MR, Hasselblad V, Stevenson LW, et al. Impact of the pulmonary artery catheter in critically ill patients: meta-analysis of randomized clinical trials. *JAMA*. 2005;294(13):1664-1670.
33. Cournand A, Riley RL, Breed ES, et al. Measurement of cardiac output in man using the technique of catheterization of the right auricle or ventricle. *J Clin Invest*. 1945;24(1):106-116.
34. Ganz W, Donoso R, Marcus HS, Forrester JS, Swan HJ. A new technique for measurement of cardiac output by thermodilution in man. *Am J Cardiol*. 1971;27(4):392-396.
35. Nishikawa T, Dohi S. Errors in the measurement of cardiac output by thermodilution. *Can J Anaesth*. 1993;40(2):142-153
36. Critchley LA, Lee A, Ho AM. A critical review of the ability of continuous cardiac output monitors to measure trends in cardiac output. *Anesth Analg*. 2010;111(5):1180-1192.
37. Pearse RM, Ikram K, Barry J. Equipment review: an appraisal of the LiDCO plus method of measuring cardiac output. *Crit Care*. 2004;8(3):190-195.
38. Oren-Grinberg A. The PiCCO Monitor. *Int Anesthesiol Clin*. 2010;48(1):57-85.
39. Manecke GR Jr, Auger WR. Cardiac output determination from the arterial pressure wave: clinical testing of a novel

algorithm that does not require calibration. *J Cardiothorac Vasc Anesth*. 2007;21(1):3-7.
40. Costa MG, Della Rocca G, Chiarandini P, et al. Continuous and intermittent cardiac output measurement in hyperdynamic conditions: pulmonary artery catheter vs. lithium dilution technique. *Intensive Care Med*. 2008;34(2):257-263.
41. Goedje O, Hoeke K, Lichtwarck-Aschoff M, Faltchauser A, Lamm P, Reichart B. Continuous cardiac output by femoral arterial thermodilution calibrated pulse contour analysis: comparison with pulmonary arterial thermodilution. *Crit Care Med*. 1999;27(11):2407-2412.
42. Roche AM, Miller TE, Gan TJ. Goal-directed fluid management with trans-oesophageal Doppler. *Best Pract Res Clin Anaesthesiol*. 2009;23(3):327-334.
43. Wakeling HG, McFall MR, Jenkins CS, et al. Intraoperative oesophageal Doppler guided fluid management shortens postoperative hospital stay after major bowel surgery. *Br J Anaesth*. 2005;95(5):634-642.
44. McFall MR, Woods WG, Wakeling HG. The use of oesophageal Doppler cardiac output measurement to optimize fluid management during colorectal surgery. *Eur J Anaesthesiol*. 2004;21(7):581-583.
45. de Waal EE, Konings MK, Kalkman CJ, Buhre WF. Assessment of stroke volume index with three different bioimpedance algorithms: lack of agreement compared to thermodilution. *Intensive Care Med*. 2008;34(4):735-739.
46. Tachibana K, Imanaka H, Takeuchi M, Takauchi Y, Miyano H, Nishimura M. Noninvasive cardiac output measurement using partial carbon dioxide rebreathing is less accurate at settings of reduced minute ventilation and when spontaneous breathing is present. *Anesthesiology*. 2003;98(4):830-837.
47. Rivers E, Nguyen B, Havstad S, et al; Early Goal-Directed Therapy Collaborative Group. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med*. 2001;345(19):1368-1377.
48. Varpula M, Karlsson S, Ruokonen E, Pettilä V. Mixed venous oxygen saturation cannot be estimated by central venous oxygen saturation in septic shock. *Intensive Care Med*. 2006; 32(9):1336-1343.
49. Reinhart K, Kuhn HJ, Hartog C, Bredle DL. Continuous central venous and pulmonary artery oxygen saturation monitoring in the critically ill. *Intensive Care Med*. 2004;30(8): 1572-1578.
50. van Beest PA, van der Schors A, Liefers H, et al. Femoral venous oxygen saturation is no surrogate for central venous oxygen saturation. *Crit Care Med*. 2012;40(12):3196-3201.
51. De Backer D, Creteur J, Dubois MJ, Sakr Y, Vincent JL. Microvascular alterations in patients with acute severe heart failure and cardiogenic shock. *Am Heart J*. 2004;147(1):91-99.
52. Trzeciak S, Dellinger RP, Parrillo JE, et al. Early microcirculatory perfusion derangements in patients with severe sepsis and septic shock: relationship to hemodynamics, oxygen transport, and survival. *Ann Emerg Med*. 2007;49:88-98.
53. Creteur J, De Backer D, Sakr Y, Koch M, Vincent JL. Sublingual capnometry tracks microcirculatory changes in septic patients. *Intensive Care Med*. 2006;32(4):516-523.
54. Sakr Y, Dubois MJ, De Backer D, Creteur J, Vincent JL. Persistent microcirculatory alterations are associated with organ failure and death in patients with septic shock. *Crit Care Med*. 2004;32(9):1825-1831.
55. Trzeciak S, McCoy JV, Phillip Dellinger R, et al; Microcirculatory Alterations in Resuscitation and Shock (MARS) investigators. Early increases in microcirculatory perfusion during protocol-directed resuscitation are associated with reduced multi-organ failure at 24 h in patients with sepsis. *Intensive Care Med*. 2008;34(12):2210-2217.
56. Beaulieu Y. Bedside echocardiography in the assessment of the critically ill. *Crit Care Med*. 2007;35(5 suppl):S235-S249.
57. Feissel M, Michard F, Faller JP, Teboul JL. The respiratory variation in inferior vena cava diameter as a guide to fluid therapy. *Intensive Care Med*. 2004;30(9):1834-1837.
58. Vieillard-Baron A, Chergui K, Rabiller A, et al. Superior vena caval collapsibility as a gauge of volume status in ventilated septic patients. *Intensive Care Med*. 2004;30(9): 1734-1739.
59. Mandeville JC, Colebourn CL. Can transthoracic echocardiography be used to predict fluid responsiveness in the critically ill patient? A systematic review. *Crit Care Res Pract*. 2012;2012:513480.
60. Beaulieu Y. Specific skill set and goals of focused echocardiography for critical care clinicians. *Crit Care Med*. 2007; 35(suppl 5):S144-S149.