Noninvasive Hemodynamic Monitoring in the Intensive Care Unit
Paul E. Marik, MD, FCCM, FCCP a,*, Michael Baram, MD a, b

a Division of Pulmonary and Critical Care Medicine, Thomas Jefferson University, 834 Walnut Street, Suite 650, Philadelphia, PA 19107, USA
b Department of Emergency Medicine, Thomas Jefferson University, 834 Walnut Street, Suite 650, Philadelphia, PA 19107, USA

Despite improvements in resuscitation and supportive care, progressive organ dysfunction occurs in a large proportion of patients with acute, life-threatening illnesses. It has been proposed that the multi-organ dysfunction syndrome (MODS) of the critically ill is a consequence of tissue dysoxia attributable to inadequate oxygen delivery, often exacerbated by a microcirculatory injury and increased tissue metabolic demands (distributive hypoxia) [1,2]. This may be further compounded by cytopathic hypoxia attributable to mitochondrial dysfunction [3,4]. Emerging data suggest that early aggressive resuscitation of critically ill patients may limit and/or reverse tissue dysoxia and progression to organ failure and improve outcome [5]. In a landmark study, Rivers and colleagues [6] demonstrated that a protocol of early goal-directed therapy reduces organ failure and improves survival in patients with severe sepsis and septic shock. Traditional goals of resuscitation have included blood pressure, pulse rate, central venous pressure (CVP), and arterial oxygen saturation. These variables change minimally in early shock and are poor indicators of the adequacy of resuscitation [7]. Furthermore, clinical assessment of cardiac output and intravascular volume status are notoriously inaccurate. Consequently, both invasive and noninvasive monitoring tools have been used in critically ill patients in an attempt to optimize resuscitation. Most of these technologies focus on “upstream” markers of resuscitation and provide information on cardiac output and fluid responsiveness. In this respect the pulmonary artery catheter (PAC) is regarded as the gold standard, as it provides an

* Corresponding author.
E-mail address: paul.marik@jefferson.edu (P.E. Marik).

0749-0704/07/$ - see front matter © 2007 Elsevier Inc. All rights reserved.
doi:10.1016/j.ccc.2007.05.002
criticalcare.theclinics.com
accurate estimate of the cardiac output and can be used to determine fluid responsiveness [8]. However, the role of invasive hemodynamic monitoring in critically ill patients is controversial as the PAC has yet to be proven to improve patient outcome [9–14]. Furthermore, the PAC does not provide information as to the adequacy of tissue oxygenation, ie, “downstream” markers. Consequently, the current trend in critical care medicine is to use noninvasive hemodynamic monitoring devices in combination with “downstream” markers of tissue oxygenation. This article provides an overview of those devices currently available for noninvasive hemodynamic monitoring as well as those techniques for indirectly assessing the adequacy of organ perfusion and tissue oxygenation.

**Definitions**

**Shock**

Shock is best defined as end-organ dysfunction as a result of hemodynamic compromise [15]. While hypotension is an important marker of shock, it is clear that blood pressure alone cannot be used as the sole determinant of shock [16]. Shock models have demonstrated that the body can develop an “oxygen debt” in the setting of normal blood pressure [16]. This concept underscores the importance of evaluating organ function and microcirculatory perfusion in patients with hemodynamic compromise [17,18]. Rivers and colleagues [6] has coined the term “cryptic shock” to represent those patients who have normal vital signs despite inadequate organ perfusion.

**Upstream and downstream markers**

The concept of following tissue dysfunction as a guide to resuscitation has led to the concept of “upstream” and “downstream” indicators of organ perfusion (Fig. 1). Upstream markers assess flow and pressure in the heart, vena cava, pulmonary artery, and aorta. Upstream markers include systemic blood pressure, heart rate, central venous pressure (CVP), pulmonary capillary wedge pressure (PCWP), and cardiac output. These are the traditional variables that have been used to assess the hemodynamic status of critically ill patients. However, shock with end–organ dysfunction occurs at the capillary and tissue levels [19]. Tools have therefore been developed that follow alterations in the microvasculature of critically ill patients [20]. These techniques are known as the “downstream” markers of resuscitation. Since patients’ oxygen and metabolic needs vary with different stressors and at different times, monitoring downstream variables can be helpful to determine the adequacy of cardiac output and perfusion pressure at a particular point in time. Currently available downstream markers include urine output, blood lactate, base excess, tissue carbon dioxide levels, and mixed venous oxygen and carbon dioxide levels.
At the current time we have very few therapeutic interventions that can directly improve perfusion and oxygenation at the microcapillary level. The current concept is to use these downstream markers to ensure that interventions that alter upstream variables are improving tissue oxygenation. Downstream markers therefore provide a guide to upstream therapy. Traditionally, shock has been treated with vasopressor and inotropic agents. However, in the setting of normotensive shock, afterload reduction may be necessary. Furthermore, by understanding the interaction and independence of upstream and downstream markers, the use of vasodilator therapy may be appropriate in select circumstances [21].

Fig. 1. The upstream endpoints of resuscitation do not reflect the severity of the microcirculatory injury nor the degree of tissue dysoxia. The downstream variables are markers of tissue perfusion and the adequacy of the resuscitation. The downstream “global” markers are less sensitive markers of tissue dysoxia and less responsive to change.

NONINVASIVE HEMODYNAMIC MONITORING

385
Upstream hemodynamic monitoring: Measurement of cardiac output

The cardiac output is the most important upstream hemodynamic parameter. Adolph Fick [22] described the first method of cardiac output estimation in 1870. This method was the reference standard by which all other methods of determining cardiac output were evaluated until the introduction of the PAC in the 1970s [8]. Despite its limitations, cardiac output measurement with a PAC using the bolus thermodilution method has become the de facto gold standard for measurement of cardiac output and is the reference standard used to compare noninvasive technologies.

Echocardiography

In principle, echocardiography is a simple method of assessing cardiac function since it uses ultrasound waves to generate real-time images of the heart. It can assess chamber size, ventricular contractility, valve function, and with the aid of Doppler can assess flow [23,24]. However a high degree of skill is required in interpreting the images and there can be a large degree of interpreter variation. Additional consideration is the cost of the equipment, which can be considerable.

Newer portable ultrasound devices are making cardiac screening and dynamic evaluation of cardiac function more achievable. Assessment of global ventricular function is helpful in the management of critically ill patients [23,24]. Patients with tissue dysxia and a hyper-contractile left ventricle may benefit from a vasopressor agent and/or fluid administration, whereas those with poor left ventricular function may benefit from an inotropic agent. Similarly, the presence of a dilated right ventricle will alert the intensivist to the presence of right ventricular dysfunction. With basic training this assessment can be learned by noncardiologists, but determining segmental wall motion abnormalities and valvular function remains a highly specialized skill requiring specialized training [25]. Ventricular chamber size can be directly measured allowing calculation of ejection fraction [23]. Flow through the valves (ie, forward flow and regurgitant flow) can be measured using Doppler imaging. This allows calculation of pulmonary artery pressures and cardiac output. However, the difficulty with these techniques is in the acquisition of adequate images in the correct plane, which requires significant experience.

Training tracks for noncardiologists exist; intraoperative transesophageal echocardiography is now commonly performed by anesthesiologists to evaluate cardiac function during open heart surgery and the “FAST” Protocol (Focused Abdominal Sonography in Trauma) is an integral part of the examination of trauma patients in the emergency room [26–29]. Bedside echocardiography as performed by the intensivist holds great promise for the future [23,24,30].
Cardiac output as measured by carbon dioxide rebreathing

Cardiac output can be calculated by the CO₂ partial rebreathing technique using the modified Fick equation [22]. NiCCO, a proprietary device (Respironics, Murraysville, Pennsylvania) measures cardiac output based on this principle. The CO₂ partial rebreathing technique compares end-tidal carbon dioxide partial pressure (etCO₂) obtained during a non-rebreathing period with that obtained during a subsequent rebreathing period. The ratio of the change in etCO₂ and CO₂ elimination after a brief period of partial rebreathing (usually 50 seconds) provides a noninvasive estimate of the cardiac output [31]. A limitation of the rebreathing CO₂ cardiac output method is that it only measures pulmonary capillary blood flow (ie, the nonshunted portion of the cardiac output). To calculate total cardiac output, intrapulmonary shunt and anatomic shunt fractions (Qs/Qt) must be added to the pulmonary capillary blood flow. The NiCCO system estimates Qs/Qt using a shunt correction algorithm, which uses oxygen saturation from pulse oximetry and the fractional concentration of inspired oxygen.

The CO₂ rebreathing technique has a number of significant limitations when used in an ICU setting. Almost all of the validation studies have been performed in patients undergoing anesthesia or in deeply sedated mechanically ventilated ICU patients, where the agreement with thermodilution cardiac output has varied from “poor” to “acceptable” [32–37]. In spontaneously breathing patients, the rebreathing period is associated with an increase in minute ventilation [38]. This reduces the accuracy of the cardiac output determinations [36,39]. Furthermore, a low minute ventilation, a high shunt fraction, and a high cardiac output result in inaccurate measurements [36,37,39]. Considering the limited data in ICU patients and the potential inaccuracies in this patient population, the routine use of the CO₂ rebreathing technique to estimate cardiac output cannot be recommended at this time.

Esophageal Doppler

The esophageal Doppler technique measures blood flow velocity in the descending aorta by means of a Doppler transducer (4 MHz continuous-wave, or 5 MHz pulsed wave, according to manufacturers) placed at the tip of a flexible probe. The probe is introduced into the esophagus of the sedated, mechanically ventilated patients and then rotated so that the transducer faces the descending aorta and a characteristic aortic velocity signal is obtained. The cardiac output is calculated based on the diameter of the aorta (measured or estimated), the distribution of the cardiac output to the descending aorta, and the measured flow velocity of blood in the aorta. As esophageal Doppler probes are inserted blindly, the resulting waveform is highly dependent on correct positioning. The clinician must adjust the depth, rotate the probe, and adjust the gain to obtain an optimal signal
Poor positioning of the esophageal probe tends to underestimate the true cardiac output. There is a significant learning curve in obtaining adequate Doppler signals and the correlations are better in studies where the investigator was not blinded to the results of the cardiac output obtained with a PAC [41].

A recent meta-analysis by Dark and Singer [42] demonstrated an 86% correlation between cardiac output as determined by esophageal Doppler and PAC. Although the correlation between the two methods was only modest, there was an excellent correlation between the change in cardiac output with therapeutic interventions. However, changes in cardiac output in response to a therapeutic intervention are probably more useful than the absolute cardiac output itself (see section on evaluating fluid responsiveness) [43].

While esophageal Doppler has some utility in aiding in the assessment of the hemodynamic status of critically ill patients, this technology has been slow to be adopted. This is likely the consequence of a number of factors including the less than ideal accuracy of the cardiac output measurements, the long learning curve, the inability to obtain continuous reliable measurements, and the practical problems related to presence of the probe in the patients’ esophagus.

Pulse contour analysis

The origin of the pulse contour method of measuring cardiac output is derived from variations in the pulse pressure waveform. In general, the greater the stroke volume, the greater is the amount of blood that must be accommodated in the arterial tree with each heartbeat and, therefore, the greater the pressure rise and fall during systole and diastole, thus causing a greater pulse pressure. The pulse pressure is proportional to stroke volume and inversely related to vascular compliance. The pulse pressure waveform therefore changes predictably with changes in the compliance of arterial wall and stroke volume. As the compliance of the vasculature is difficult to measure directly, this is calculated based on age, sex, ethnicity and body mass index (BMI) [44]. Using complex proprietary formula (PulseCO, LiDCO, London, UK; PiCCO, Pulsion Medical Systems, Munich, Germany) the cardiac output is then calculated from analysis of the pulse contour [45]. Ideally the pulse contour analysis is calibrated to an injection dilution method. Stroke volume is calculated and compared with the stroke volume as determined by the dilution technique, and the cardiac output is then calculated. With beat-to-beat waveform analysis, cardiac output can be determined continuously [46]. External calibration every 6 to 12 hours confirms continued accuracy [45,47]. While the cardiac output as determined by pulse contour analysis shows good agreement with the cardiac output as measured by other techniques, the use of vasoactive agents (pure vasodilators/vasoconstrictors) may result in spurious changes in cardiac output [46,48].
importantly, these devices also calculate the pulse pressure and stroke volume variation with positive pressure ventilation (see section on evaluating fluid responsiveness). A large pulse pressure/stroke volume variation (> 10% to 15%) is indicative of hypovolemia and predictive of volume responsiveness.

**Lithium dilution and pulse contour analysis**

Lithium is the contrast agent most commonly used with the injection dilution method for external calibration of pulse contour analysis devices (PulseCO, LiDCO). The lithium may be injected via a central or peripheral vein [49]. A lithium analyzer is connected to an arterial line, which then measures the wash out curve over time and generates a curve similar to the thermodilution curve of a PAC [45,50]. In an animal model, Kurita and colleagues [51] reported that the cardiac output as measured by the lithium dilution method correlated better with Doppler aortic flow than the cardiac output as determined by thermodilution. Reproducibility was also better with the lithium method as compared with the thermodilution method. In human studies a good correlation between thermodilution and lithium dilution has been reported [46,49,52].

**Transpulmonary thermodilution and pulse contour analysis**

PiCCO (Pulsion Medical Systems) uses the aortic transpulmonary thermodilution curve to calculate cardiac output (TP-TD). For this technique, a thermistor-tipped catheter is typically placed in the descending aorta via a femoral sheath. Iced saline (15 mL) is injected into a central vein and from the temperature change in the aorta, the cardiac output can be calculated and the pulse contour system calibrated [53–55].

**Pulse contour without dilution calibration**

As the concept of pulse contour analysis has become better understood, complex algorithms have been developed that can estimate cardiac output without the need for external calibration [56]. External calibration is replaced by correction factors that depend on the mean arterial pressure and the age, gender, weight, and height of the patient. There are limited published data on the accuracy of this methodology and while promising, additional studies are required before this technology can be used clinically [56–58].

**Plethysmography**

The use of thoracic electrical bioimpedance (TEB) to estimate cardiac output dates back to the early years of manned space exploration. Using low voltage, electrical impedance (or resistance) across the chest is measured. The higher the fluid content, the lower the impedance since fluid conducts electricity. As the heart cycles through systole and diastole the volume of blood in the thorax changes and this can be measured electrically [59].
Early studies demonstrated only a fair correlation between TEB and thermodilution cardiac output [60,61]. In addition, the accuracy of TEB worsened as the degree of volume overload increased. This incongruity occurs since heart failure results in increased pulmonary edema and pleural effusion, which affects conduction. Many of the problems associated with TEB have been overcome with newer generation devices using upgraded computer technology and refined algorithms to calculate cardiac output [62,63]. Van De Water and colleagues [63] demonstrated less variability and more accurate determination of cardiac output using a refined equation as compared with the predecessor equations of Kubicek, Sramek, and Sramek-Bernstein. Recently, a number of investigators have reported a good correlation between TEB and thermodilution in patients following cardiac surgery using these improved devices [63–67]. Similarly, Albert and colleagues [68] demonstrated that cardiac output as measured by TEB and thermodilution were significantly correlated in patients with decompensated chronic heart failure. There are limited data on the use of TEB in critically ill ICU patients; however, the improved TEB technology does hold promise in this group of patients.

**Comparative studies**

A number of studies have been performed comparing the accuracy of the various noninvasive devices reviewed in this paper [46,56,69]. Unfortunately, most of these studies suffer methodological problems in terms of the “gold” standard used and the sample size [70]. In general, no device stands out as being better than another and although not perfectly accurate, all the devices were able to detect changes in cardiac output and reflect appropriate trends.

**Evaluating fluid responsiveness**

Fundamentally the only reason to give a patient a fluid challenge is to increase stroke volume. This assumes that the patient is on the ascending portion of the Frank-Starling curve and has “recruitable” cardiac output. Once the left ventricle is functioning near the “flat” part of the Frank-Starling curve, fluid loading has little effect on cardiac output and only serves to increase tissue edema and to promote tissue dysoxia. In normal physiologic conditions, both ventricles operate on the ascending portion of the Frank-Starling curve [71]. This mechanism provides a functional reserve to the heart in situations of acute stress [71]. In healthy individuals, an increase in preload (with volume challenge) results in a significant increase in stroke volume [72,73]. In contrast, only about 50% of patients with circulatory failure will respond to a fluid challenge [74]. It is therefore crucial during the resuscitation phase of all critically ill patients to determine whether the patient is fluid responsive or not; this determines the optimal strategy of increasing cardiac output and tissue oxygen delivery.
Although cardiac filling pressures (central venous pressure and pulmonary capillary wedge pressure) as measured using a central venous catheter or PAC are widely used to predict fluid responsiveness, this approach is completely devoid of supportive scientific data. Indeed, multiple studies have confirmed that both the CVP and PCWP in healthy controls and in patients with various disease states are unable to predict the hemodynamic response to a fluid challenge \[73–79\]. It is therefore somewhat alarming that the CVP is still widely used as a guide to fluid resuscitation and is incorporated into protocols that are endorsed by professional societies \[6,80,81\]. Traditionally, the PAC has been used to determine fluid responsiveness; a patient is given a fluid challenge and the change in stroke volume and cardiac output is recorded. A number of noninvasive methods to determine fluid responsiveness have been investigated and are further reviewed.

Using heart-lung interactions to assess fluid responsiveness during mechanical ventilation

In mechanically ventilated patients, the magnitude of the respiratory change in left ventricular stroke volume can be used to assess fluid responsiveness. Intermittent positive-pressure ventilation induces changes in the loading conditions of the left and right ventricles. Mechanical insufflation decreases preload and increases afterload of the right ventricle (RV). The decrease in RV preload is attributable to the decrease in the venous return pressure gradient that is related to the increase in pleural pressure. The decrease in RV preload and increase in RV afterload both lead to a decrease in RV stroke volume. The inspiratory reduction in RV ejection leads to a decrease in left ventricular (LV) filling after a phase lag of two or three heartbeats. Consequently, stroke volume, cardiac output, and systemic blood pressure all fall during each mechanical breath. Since venous return depends on the venous pressure gradient, the decrease in venous return with positive pressure ventilation is most marked in hypovolemic patients who have a low mean circulating filling pressure \[82,83\]. Furthermore, patients who are functioning on the ascending portion of the Frank-Starling curve have an exaggerated fall in stroke volume with decreased venous return during each positive pressure breath. Consequently cardiac output and blood pressure falls significantly in these patients with each mechanical insufflation. Multiple experimental and clinical studies have confirmed that large variations (greater than approximately 12\%) in systolic pressure (SPV) and pulse pressure (PPV) as measured using an arterial catheter, predict an increase in cardiac output with fluid loading (ie, volume responsiveness) \[77,78,84–86\]. This dynamic test of “recruitable cardiac output” is highly reproducible and simply performed at the bedside.

Pulse pressure variation and stroke volume variation (SVV) with mechanical ventilation has logically been combined with pulse contour analysis to predict volume responsiveness \[79,87–90\]. In addition, ventilator-induced
changes in aortic blood flow velocity and stroke output as measured with esophageal Doppler [91–93] and changes in stroke volume by echocardiography have been demonstrated to predict fluid responsiveness [94]. Current evidence suggests that the dynamic changes in cardiac performance with positive pressure ventilation are more accurate in predicting volume responsiveness than the static filling pressures that have traditionally been used for this purpose [74,95]. In addition, cyclic changes in inferior vena caval diameter as measured by echocardiography have been used to predict fluid responsiveness [96,97].

**Downstream hemodynamic markers**

*Lactate*

The concept that hypoxic tissues can generate a lactic acidosis has been understood since the 1970s [98]. To generate energy, the body must convert glucose into CO₂ via the Krebs cycle. In anaerobic environments, the Krebs cycle cannot completely metabolize glucose, so instead a partial metabolic pathway is followed, which generates lactate. The greater the oxygen deficit and with increased metabolic demands, the more lactate is produced. Lactic acidosis is, however, not limited to shock. Localized lactic acidosis can occur in muscles from repetitive use such as during exercise, or from a generalized tonic-clonic seizure.

Blood lactate concentration is commonly used as a global “downstream” marker of tissue perfusion and the adequacy of resuscitation [6]. Blood lactate, however, is an insensitive marker of tissue dysoxia [99–102]. If glycolysis occurs at a more rapid rate than is necessary for oxidative metabolism, some pyruvate may not be oxidatively metabolized in the Krebs cycle and will be converted to lactate. The result will be a concomitant increase in both pyruvate and lactate with an unchanged lactate/pyruvate ratio (L/P) [103,104]. James and colleagues [102] provide a compelling argument that a high blood lactate in critically ill patients may be a metabolic manifestation of high blood epinephrine levels (and glycolysis) and a poor indicator of tissue dysoxia. Similarly, Levy and colleagues [105] have elegantly demonstrated that skeletal muscle may be a major source of lactate production during sepsis as a consequence of increased aerobic glycolysis through Na⁺K⁺ ATPase stimulation. Lactate levels may therefore be a marker of illness severity rather than a measure of anaerobic metabolism. In addition, the blood lactate level depends on the rate of production as well as the rate of metabolism by the liver (Cori cycle). Because of decreased splanchnic blood flow and hepatocellular dysfunction, lactate removal may be impaired in critically ill patients.

A lactic acidosis must therefore be assessed in the proper clinical context before it can be used as a tool to assess downstream mitochondrial function. However, a blood lactate concentration in excess of 4 mEq/L is associated with a high risk of death [98,106]. Furthermore the rate of lactate clearance
has been demonstrated to be a good marker of outcome [107]. While an elevated lactate may be a marker of illness severity and an important prognostic marker, this variable has not been studied as an end point of resuscitation. Lactate clearance lags by many hours following therapeutic interventions and is therefore not suited for goal-directed resuscitation. Furthermore, lactate has never been proven to be a surrogate marker for cardiac output [108]. As a marker of index severity it may be appropriate to use an elevated lactate as a trigger to initiate aggressive care; however, that care should not be titrated to the lactate level.

_Gastric tonometry and sublingual capnography_

Because of the flow distribution away from the gastrointestinal tract the development of tissue dysoxia in the gastrointestinal tract appears to be a common and early finding in patients with deranged hemodynamics. Dantzker [109] has suggested that the gastrointestinal tract may be the “canary of the body,” with gastrointestinal dysoxia an “early warning of impending trouble.” It has been demonstrated that changes in gastrointestinal mucosal $p$CO$_2$ mirrors changes in gastrointestinal oxygen uptake during progressive flow stagnation [110,111]. Since the sublingual mucosa is embryologically derived from gut tissue, its perfusion and response to stress is similar to that of the splanchnic bed. The $p$CO$_2$ of the stomach wall (PgCO$_2$) and sublingual tissue (PsiCO$_2$) has been demonstrated to increase predictably during both hemorrhagic and septic shock [112–114]. Gutierrez and colleagues [115] randomized critically ill ICU patients to a standard treatment group or a protocol group in which treatment was titrated to maintain the gastric intramucosal pH (pHi) greater than 7.35. Survival was significantly improved in the protocol subgroup whose initial pHi was greater than 7.35. This study provides further support to the argument that the early detection and treatment of tissue dysoxia may improve the outcome of critically ill patients.

Gastric tonometry is logistically and practically difficult and this may be the main factor that has prevented the widespread use of this technology. The recent introduction of sublingual capnometry has resolved many of the difficulties associated with gastric tonometry. Sublingual capnometry is a technically simple, noninvasive, inexpensive technology that provides near instantaneous information as to the adequacy of tissue perfusion in critically ill and injured patients [112]. Sublingual capnometry may prove to be useful tool for both the risk stratification and as an end point for goal-directed resuscitation. The clinical experience with sublingual capnometry is, however, limited, and additional studies are needed that demonstrate the clinical utility of PsiCO$_2$ monitoring.

_Mixed venous O$_2$, mixed venous PCO$_2$ and base excess_

Mixed venous oxygen saturation (SmvO$_2$) measured in either the pulmonary artery (with a PAC) or the right atrium (with a central venous catheter)
has been used to monitor critically ill patients and their response to therapeutic interventions [6,116–119]. A low SmvO₂ (in the absence of arterial hypoxemia) is usually an indicator of inadequate cardiac output. Mixed venous PCO₂ (PmvCO₂) represents the equilibration of systemic venous CO₂ that has returned to the right side of the heart and as such is a “global” indicator of tissue dysoxia. The major disadvantage of these global measurements is that they lack sensitivity; high tissue PCO₂ and/or low PO₂ draining vital organs will be diluted by blood draining organs with lower metabolic requirements with a lower PCO₂ and higher PO₂. Silva and colleagues [120] measured global and regional indicators of tissue dysoxia in septic patients undergoing fluid challenge. While the gastric intramucosal PCO₂ gradient decreased significantly with volume resuscitation, the SmvO₂ and PmvCO₂ gradient remained unchanged. In addition, in patients with sepsis (and macrocirculatory shunting) the SmvO₂ may be a poor indicator of tissue dysoxia [121].

The base excess (BE) has become the standard end point of resuscitation in trauma patients. Remarkably, while the BE has been demonstrated to be of prognostic value in this patient population, it has never been assessed prospectively in trauma patients [122]. It is likely that tissue hypoperfusion may occur in the absence of a significant change in the BE. Furthermore, as it requires time for the liver and kidney to regenerate bicarbonate, it can be expected that there will be a long lag phase between the correction of intravascular volume deficit and normalization of the BE [123]. While the BE may indicate tissue acidosis, it is a crude indicator of tissue dysoxia that has not been well studied in critically ill patients and should therefore not be used as an end point of goal-directed therapy.

Summary

The quest for the holy grail of noninvasive cardiac output assessment methods continues. Although no tool is perfect, a number of noninvasive methods to determine the cardiac output of critically ill patients are now available. It is, however, important to stress that the cardiac output should be interpreted in conjunction with dynamic indices of volume responsiveness and downstream markers of tissue oxygenation. Furthermore, patients cannot be managed by simplistic algorithms or bundles but rather by thoughtful intensivists, who at the bedside can integrate a body of complex and interrelated information and chart a course based on the best available scientific evidence.

References


