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Methods of Monitoring Shock

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Abstract

Intensive monitoring is a crucial component of the management of shock. However, there is little consensus about optimal strategies for monitoring. Although the pulmonary artery catheter has been widely used, conflicting data exist about the utility of this device. A variety of other techniques have been developed in hopes of providing clinically useful information about myocardial function, intravascular volume, and indices of organ function. In addition, there is evolving evidence that targeting and monitoring certain physiological goals may be most important early in the course of shock. In this chapter, we examine many of the available monitoring techniques and the evidence supporting their use.

Keywords

Shock; monitoring; critical illness; intensive care unit; pulmonary artery catheter

The syndrome of circulatory shock involves an inciting event that results in a curtailment of effective blood flow to vital organs. If untreated, tissue hypoperfusion leads to organ dysfunction and a cycle of inflammation and worsening organ failure. The treatment of shock has centered on reversing the initial insult (if possible) and providing supportive care. Attempts at altering the pathophysiology of shock, mainly by immune modulation in septic shock, have proven unsuccessful,[1] with the notable exception of drotrecogin alfa (activated).[2] Thus the focus has been on ensuring adequate blood pressure for organ perfusion and adequate oxygen delivery (DO_2) to tissues.[3]

Beyond a set of standard or "basic" monitoring techniques (Table 1), there is little consensus on what types of monitoring are appropriate or what the goals of monitoring should be. Even among the standard techniques, there remains a great deal of uncertainty in application. For example, it is widely accepted that blood pressure should be supported above a mean arterial pressure of ~60 mm Hg or a systolic blood pressure of 90 mm Hg, but there are few data suggesting what the optimal level should be.[4]

A variety of other "advanced" monitoring techniques have been employed, generally attempting to track cardiac function or adequacy of tissue perfusion. Until recently, there has been little or no evidence that following these variables and using them to guide therapy produces any benefit in outcomes. This has led to a reappraisal of the utility of monitoring hemodynamic variables in shock, and the emergence of several new trends in the field. In light of suggestions that guiding management according to pulmonary artery catheters (PACs) was not helpful and potentially harmful,[5] one trend has been toward seeking

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noninvasive methods that can provide equivalent information while reducing the opportunity for iatrogenic harm. Another trend has been inspired by the observation that much of the benefit in optimizing hemodynamics appears to come within hours after the onset of sepsis.[6] Later in the course, mounting evidence suggests that tissue injury may persist despite normal or supranormal DO_2.[7] This has led to the recognition of the "cytopathic hypoxia" syndrome, a situation in which damaged tissues are unable to utilize oxygen adequately despite restoration of DO_2.[8,9] This condition is surmised to be a result of the breakdown of intracellular metabolism and suggests a dual-phase model of the pathophysiology of shock. The early reversible phase is characterized by insufficient DO_2 and if not corrected in a timely fashion leads to a second phase characterized by intracellular damage and dysfunctional oxygen utilization. This model is supported by clinical evidence, including observations that goal-directed therapy may be beneficial early in the course of sepsis[6] but does not appear to be of benefit after the early phase,[10] and by observations that patients with evidence of anaerobic metabolism early in presentation appear to have a worse prognosis.[11]

This article provides an overview of the major methods currently available for monitoring physiological variables of interest in both early and established shock. We have divided these into categories according to the physiological information they provide; that is, the measurement of cardiac output and function, measurement of cardiac preload (intravascular volume), and measurement of global and organ-specific tissue perfusion. In light of evidence that the information provided by these techniques may not be of use late in the course of shock, we will also review evolving technologies that may in the future provide insight into the molecular mechanisms of shock and suggest directions for therapy beyond reestablishment of oxygen delivery.

VARIABLES OF INTEREST

Cardiac Output and Myocardial Function

CLINICAL ASSESSMENT—Physical examination and overall clinical assessment present a difficult problem in critically ill patients. Existing data have mainly focused on physical examination in heart failure. Analysis of one cohort of patients with chronic heart failure showed that the presence of elevated jugular venous pulsations and an S3 gallop were associated with worsened outcomes.[12] However, other attempts to clinically assess systolic function in heart failure have been disappointing.[13] One study evaluated 264 critically ill patients and compared subjective assessments of skin temperature with a variety of collected physiological data, including data from pulmonary artery catheters. Cool skin temperature was found to be significantly associated with other markers of decreased cardiac function and end-organ perfusion. In particular, cool skin was an indicator of significantly lower cardiac output (CO).[14] This appears to support the classic findings of Joly and Weil, who found that temperature of the great toe was significantly correlated with cardiac output and appeared to correctly predict patient outcome 67% of the time.[15] In contrast, other evidence has suggested that clinical assessment is generally a poor predictor of actual hemodynamics.[16]

ECHOCARDIOGRAPHY—Echocardiography has evolved as a highly useful tool for evaluating cardiac structure and function. As techniques have been refined over the past 15 years, a great deal of interest has focused on the use of echocardiography in the intensive care unit (ICU). Based on two-dimensional transthoracic echocardiography alone, substantial information about ventricular wall motion, chamber size, valvular function, and other cardiac parameters can be obtained (Table 2). The addition of Doppler techniques also allows further definition of hemodynamics by measuring blood flow. Because obtaining adequate images can be technically difficult in ICU patients, much attention has focused on

the use of transesophageal echocardiography (TEE), which can also provide measurement of aortic blood flow and thus cardiac output. However, adapting echocardiography for ideal use in ICUs has been challenging. In part, this is due to the cost of advanced ultrasound equipment as well as the expense involved in having highly trained personnel readily available to perform and interpret the studies. Another challenge has been defining what data are useful for routine ICU monitoring.

Several studies have attempted to show the utility of performing echocardiography on a routine basis or in response to clinical scenarios such as unexplained hypotension. For example, Poelaert and colleagues retrospectively analyzed a series of 108 ICU patients who had undergone TEE. They found that TEE provided information that led to changes in therapy in a substantial proportion of cases. This was true even in many patients with pulmonary arterial catheters in place.[17] Overall, the data do suggest that employing TEE diagnostically can be helpful in identifying a cardiac abnormality, as in cases of unexplained hypoxemia or shock, in detection of occult mitral regurgitation, and in a general population of hemodynamically unstable patients.[18–20]

Ideally, echocardiography could be used to follow cardiac performance in shock and identify targets for therapies aimed at improving cardiac output, particularly during resuscitation of early shock. Part of the difficulty in achieving this purpose lies in the inability thus far to select which markers of function are most important to monitor as surrogates for cardiac output or adequacy of volume loading.[21] Cardiac output can be determined from color Doppler TEE,[22] but these measurements have not been shown to correlate consistently with thermodilution measurements.[23] Another group has published multiple observations made from a series of 183 patients with septic shock and established parameters for monitoring ventricular systolic function and intravascular volume; however, this approach has not been tested in clinical trials.[24]

Esophageal Doppler probes have been developed that are dedicated to determining cardiac output by utilizing the same principles.[25] These devices measure blood flow and diameter of the descending aorta, thereby providing a measure of descending aortic blood flow. Multiplying this by a correction factor allows estimation of total cardiac output. This method has the advantage of being simpler and cheaper than performing a full TEE study. The probe can also be left in place to provide continuous monitoring of cardiac output. In addition, this technique of esophageal Doppler monitoring (EDM) allows measurement of an index of left ventricular ejection time (corrected flow time), which provides a surrogate for preload.[26] A study of one such device showed good correlation (r=0.80) with thermodilution measurements in 20 ICU patients.[27] A limited amount of clinical data has also indicated that EDM may be useful as a guide to therapy. For example, in a randomized controlled trial, a group of 40 patients undergoing high-risk femur fracture repair were assigned to either optimization of cardiac output via EDM or conventional therapy. EDM was associated with significantly shorter recovery and hospital stay.[28]

VENOUS OXYGEN SATURATION—Mixed venous oxygen saturation ($S_{MV}O_2$) is typically measured by sampling blood from the pulmonary artery. As such, it represents a flow-weighted average of the venous return from the entire body after sufficient mixing of blood from the inferior and superior vena cava has occurred. Decreases in $S_{MV}O_2$ can be caused by a decrease in DO_2 or an increase in oxygen consumption (VO_2) or both. In the absence of major derangements of tissue metabolism, decreases in $S_{MV}O_2$ will mainly be due to decreased cardiac output. $S_{MV}O_2$ can either be intermittently measured by sampling blood from a PAC or continuously measured by a fiber-optic sensor in a PAC.

A large amount of data has been compiled on the physiological significance of $S_{MV}O_2$, including data suggesting that changes in the variable reflect changes in cardiac output[29] and global oxygen transport.[30] In clinical use, several small studies have met with mixed results regarding the prognostic value and clinical utility of $S_{MV}O_2$ monitoring.[31,32] Pearson and colleagues compared $S_{MV}O_2$, conventional pulmonary arterial catheterization and central venous pressure (CVP) monitoring in a trial of invasive ICU monitoring. They found that monitoring $S_{MV}O_2$ did not improve length of ICU stay or length of vasopressor treatment and was associated with significantly higher cost.[33]

$S_{MV}O_2$ was also subjected to scrutiny in a large, multicenter, prospective, randomized trial done by Gattinoni and colleagues. In this study, 762 critically ill patients were randomized to goal-directed therapy aimed at achieving either a normal cardiac index (CI) (the control group), a supranormal CI, or a normal $S_{MV}O_2$, which was defined as either $S_{MV}O_2 > 70\%$ or < 20% lower than arterial oxygen saturation. There were no significant differences in mortality, organ dysfunction, or ICU length of stay between the three groups. Although significantly fewer patients met the hemodynamic targets in the two experimental groups, outcomes did not appear to be different in the patients who did achieve the desired targets. The results of this study did not support the use of $S_{MV}O_2$ as a guide to therapy.[10]

Because of risks and costs associated with monitoring of mixed venous oxygenation, others have evaluated the feasibility of using central venous oxygen saturation ($S_{CV}O_2$) to obtain similar information. Central venous catheterization is commonly performed in patients with shock to deliver vasopressors and other medications. If the central venous catheter could provide useful information about cardiac function (via the $S_{CV}O_2$) and volume status (via the CVP) without increasing morbidity, it would represent a valuable approach to monitoring of shock. Early data suggested that $S_{CV}O_2$ was lower than $S_{MV}O_2$ by ~5 to 10%.[34] A correlation study in dogs showed a very high correlation coefficient (r=0.96) between the two variables.[35]

A recent study attempted to exploit the correlation between $S_{MV}O_2$ and $S_{CV}O_2$ to reevaluate the concept of using venous oxygenation to guide treatment. This study added the twist of attempting to intervene early in the course of shock, presumably before injury had occurred that could not be reversed by resuscitation[6] (reviewed in Chapter 6). Multiple interventions were employed to ensure tissue oxygenation by providing volume resuscitation, dobutamine, packed red blood cells, along with vasopressors to achieve an $S_{CV}O_2$ of > 70%. A 16% absolute reduction of in-hospital mortality was seen in the treatment group. Differences in $S_{CV}O_2$ may have played a significant part because there was no significant difference in $S_{CV}O_2$ between the groups at enrollment, but the treatment group had a significantly higher $S_{CV}O_2$ at each subsequent time point. Average $S_{CV}O_2$ in the control group was below 70% through the course of the protocol. However, the multifaceted nature of the study makes it difficult to assess which interventions had the most relative impact on outcome. For example, patients in the treatment group had a significantly higher mean arterial pressure than patients in the control group, opening the possibility that the same effect could have been achieved by resuscitating according to this (much simpler) parameter.

PULSE CONTOUR ANALYSIS—Another method of monitoring cardiac output continuously involves mathematical analysis of the pressure waveform of the arterial pulse. Data for pulse contour analysis can be obtained from invasive or noninvasive monitoring of arterial blood flow. The technique is based on the assumption that the stroke volume is proportional to the area under the curve of the pressure tracing during systole. Therefore, a single independent measurement of cardiac output, taken via another modality, can be used to quantify the relationship between cardiac output and the arterial pulse waveform. Subsequently, this calibrating measurement can be used to follow changes in the waveforms

and calculate the resulting changes in cardiac output. Commercially available systems use computer algorithms to make the necessary calculations. In practice, the technique has been shown to correlate well with measurements obtained by thermodilution,[36,37] but it has been suggested that the relationship may break down if vasoconstrictors are administered.[38] Minimal data exist to evaluate clinical outcomes when pulse contour analysis is used as a guide to treatment. Although pulse contour analysis systems are available, they have not entered wide clinical usage. A major drawback to acceptance is the necessity of having another means of measuring cardiac output available for calibration purposes and it is not clear if continuous measurement of cardiac output offers a significant advantage over the intermittent measurements required for calibration. An alternative approach would be to forgo calibration measurements and follow trends in analysis.

THORACIC BIOIMPEDANCE—The thoracic bioimpedance technique involves the application of a low-magnitude electrical current across the thorax. Sensing electrodes placed in the path of the current measure the electrical resistance of the thorax. Bioimpedance decreases with increased fluid in the chest, providing the basis for measuring cardiac output using this technique. To make this technology useful, it must be able to distinguish aortic blood flow from other sources of thoracic fluid movement. This is accomplished by simultaneously monitoring the electrocardiogram and using computer algorithms to filter out interfering signals. This also results in some limitations to the device, as it cannot be used effectively in situations where the ventricular rhythm is irregular. Also, conditions in which there are acute changes in thoracic fluid content, as with pulmonary edema or pleural effusions, can theoretically introduce important error into measurements.

There have been a large number of studies attempting to validate thoracic bioimpedance by comparison with “gold standard” methods, mainly thermodilution. A meta-analysis looked at over 100 of these trials and came to the conclusion that correlation was highly variable, ranging from a correlation coefficient (r) of 0.74 to 0.44, with the least reliable results coming in patients with cardiac disease in whom a single measurement was obtained. In noncardiac patients in whom serial measurements were done, the correlation appeared to be reasonably good.[39] Better results were obtained from a recent multicenter trial in which a large, heterogeneous group of patients was studied by measuring thoracic bioimpedance and comparing with thermodilution.[40] Measurements were found to be very well correlated (r =0.85). When patients with high thoracic fluid volume (such as with pulmonary edema) were excluded, strength of the correlation improved (r=0.93). However, the value of targeting or monitoring thoracic bioimpedance during resuscitation of shock for improving clinical outcomes has not been well studied. Several systems are commercially available, and given the noninvasiveness and ease of use of the technique, it may be useful in monitoring trends in cardiac output, with limitations as described.

TRANSPULMONARY DILUTION—Measurement of cardiac output can also be accomplished by injecting a substance into the central venous circulation and measuring the resulting concentration that appears in the arterial circulation. Cardiac output can then be calculated by generating a dilution curve, using the same physiological principles as pulmonary artery thermodilution. Most commonly, the substance used has either been indocyanine-green dye (dye-dilution) or cold saline (thermodilution). The dye-dilution technique has been in use for ~40 years.[41] With the development of the PAC, the technique lost prominence, but it has been shown to be accurate compared with electromagnetic flowmetry[42] and thermodilution[43] measurements. The transpulmonary thermodilution technique is less well studied but does appear to correlate with measurements taken by traditional thermodilution.[44]

The major limitations to these techniques are the lack of widespread availability (although commercial systems are available for performing the necessary measurements) and the need for invasive arterial monitoring. Currently, clinical evidence supporting their use to guide interventions is limited, although one study attempted to improve splanchnic perfusion in eight patients by increasing cardiac output, as determined by the dye-dilution technique. Although the investigators were unable to improve splanchnic perfusion, they did successfully increase cardiac output.[45]

PULMONARY ARTERY CATHETERIZATION—There are few issues in clinical medicine that have engendered as much controversy as the debate about pulmonary artery catheterization. The flow-directed, balloon-tipped catheter described by Swan et al in 1970[46] revolutionized critical care medicine by making considerable hemodynamic information available at the bedside. Since that time, the technique has become a mainstay of critical care practice worldwide. One estimate of PAC use placed yearly sales at 1.2 million units, with related costs exceeding $2 billion.[47]

Early evidence suggested the utility of catheterization for detecting complications of acute myocardial infarction.[48] It was also suggested that the technique allows for more accurate assessment of hemodynamic status than clinical assessment.[16] However, despite widespread use, there has never been convincing proof that PAC use, either as a diagnostic tool or as a method for monitoring therapy, improves clinical outcomes.

Gore and colleagues studied a group of 3263 patients hospitalized with acute myocardial infarction (MI). Of these patients, 454 received a PAC, in most cases for management of hemodynamically significant complications. The groups were followed for 5 years, at which time period survival was not significantly different between them. In addition, hospital stay was longer for patients who were catheterized, and unadjusted case-fatality rates were higher in patients with heart failure or hypotension who were catheterized.[49] A similar observational study done by Zion and colleagues in 1990 of 5841 patients with acute MI showed that overall mortality in patients who received a PAC was higher. However, as with the first trial, analysis was not adjusted for level of acuity, which seemed to be higher in the patients receiving PAC.[50] In 1996, Connors and colleagues studied 5735 patients in a retrospective cohort study. They attempted to match ICU patients by diagnosis and severity of illness. Thirty-eight percent of their patients received a PAC. The authors found that patients who had received a PAC had significantly higher 30-day and 180-day mortality.[51] The results of this study led to a widespread reevaluation of the use of PA catheterization, including calls for a moratorium on PAC use.[5] Despite state-of-the art propensity scoring in an attempt to control for indication bias, there may have been important differences between the groups. Indications for use have also been poorly defined, and in some cases use of a PAC may have reflected a more aggressive or invasive management style of clinicians, for better or worse. Fears of increased mortality caused by use of the PAC have been quelled somewhat by a prospective randomized controlled trial that demonstrated no difference in morbidity or mortality in patients with either or both shock and ARDS who were randomized either to receive or not to receive a PAC.[52]

Subsequent efforts have focused on conducting randomized controlled clinical trials to evaluate the effects of PA catheterization in specific populations. The largest of these that has been completed evaluated the benefit of perioperative catheterization in high-risk surgical patients to guide attempts at achieving hemodynamic treatment goals. This study showed no benefit in terms of mortality but did show a significant increase in the incidence of pulmonary embolism over a standard-care group.[53]

Measurement of cardiac output via PA catheterization generally involves use of the thermodilution technique, which was described soon after the development of the Swan-Ganz catheter.[54,55] The gold standard for measuring cardiac output is direct electromagnetometry of aortic blood flow, which is not practical for general use in humans. Thermodilution has been shown to be well correlated to electromagnetometry[56] and is generally considered to be the gold standard for practical purposes. In practice, however, variations in technique throw this distinction into question.

Despite the large amounts of data that have been generated describing the clinical use of PACs, it is unclear whether monitoring cardiac output by this method is beneficial. Several randomized trials have attempted to use PAC data to guide therapy aimed at reaching physiological or supraphysiological goals in critically ill patients. In 1996, Heyland and colleagues performed a meta-analysis of these trials and found that, overall, mortality was not reduced by using PAC data to guide therapy.[57] However, the methodology employed in some trials has been problematic. In one of the studies included, Tuchschmidt and colleagues randomized 70 patients with suspected sepsis to "treatment" or "control" groups, with the only difference being the goal CI. The treatment group had a goal CI of >6.0 L/min/m^2, whereas in the control group, the goal was a CI of >3.0 L/min/m^2. After resuscitation, the protocol group had a significantly higher CI compared with the control group. The mortality rate was 72% in the control group and 50% in the experimental group, which did not achieve statistical significance (p =0.14). However, the study suffered from a high degree of crossover, with several patients in the control group achieving supranormal values of CI, and several treatment group patients who did not achieve treatment goals. Furthermore, there were a large number of patients excluded after randomization.[58] In the study by Gattinoni and colleagues,[10] a total of 762 patients were randomly allocated to one of three groups designed to achieve different hemodynamic goals. As previously mentioned, one of the groups was targeted to a normal $S_{MV}O_2$. In the other two groups, patients were either targeted to a normal CI (2.5–3.5 L/min/m^2, the control group) or a supranormal CI (>4.5 L/min/m^2, the CI group). In addition, mean arterial pressure (MAP), pulmonary artery occlusion pressure (PAOP), urine output, CVP, and arterial pH were all maintained at levels considered standard of care in all three groups. Overall, the hemodynamic targets were reached by 94.3% of the control CI group, but only 44.9% of the supranormal CI group. In the final analysis, there were no significant differences between the two groups in terms of length of ICU stay, organ dysfunction, and mortality rates at the time of ICU discharge or at 6 months follow-up. However, over half of the patients in the treatment group failed to achieve the goal CI. Overall, data regarding use of the PAC have been negative or inconclusive, but there are several reasons why existing trials may have failed to show a benefit.

OTHER METHODS—The Fick method is based on physiological principles described by Adolph Fick over 100 years ago. As he put it, "the total uptake or release of a substance by an organ is the product of the blood flow to the organ and the arteriovenous concentration of the substance." As it relates to the determination of cardiac output, the principle states that cardiac output is equal to VO_2 divided by the difference between arterial and mixed venous oxygen concentration. VO_2 can be determined from the difference between inspired and expired oxygen. Although this method does provide accurate measurements of cardiac output,[59] the technique is somewhat cumbersome and the multiple calculations involved introduce potential for error. Because pulmonary artery catheterization is required for data collection, the thermodilution method is generally preferred unless thermodilution is considered unreliable, as with tricuspid insufficiency.

The "indirect Fick" method utilizes CO_2 instead of oxygen measurements and can be performed without the need for invasive monitoring. A device utilizing this principle is

commercially available, but the technique does not appear to correlate well with measurements taken by thermodilution[60] and may be more suitable for monitoring trends than for measuring absolute values.

Intravascular Volume

The Frank-Starling principle states that a major determinant of contractility is the length of a muscle fiber prior to contraction, also known as preload. Thus, in determining cardiac contractility, left ventricular end-diastolic volume (LVEDV) will have a major impact on the strength of the ensuing systolic contraction. Hence, preload is a major determinant of cardiac output. Because of this relationship, optimizing preload is an important part of improving cardiac output, and hence, maximizing tissue perfusion. Measuring preload is complicated by the fact that even knowing the exact value of LVEDV does not necessarily provide clinically useful information. It is more important to know where current preload lies in relation to optimum preload, and thus whether adding or removing volume will produce changes in cardiac output. Because of the difficulties involved in achieving this goal attempts have been made to find useful surrogates for LVEDV, such as estimation of intracavitary pressure in other parts of the hemodynamic system, or estimation of global "volume status."

CLINICAL ASSESSMENT—Classically, a variety of clinical findings have been utilized in the determination of volume status. However, for the most part, the value of individual findings has not been well validated. Physical examination findings used to assess systemic volume status include height of jugular venous pulsations (JVPs) and for assessing intrathoracic blood volume, auscultation of pulmonary crackles or wheezes. However, pulmonary findings may be due to multiple causes unrelated to volume status, such as pneumonia or noncardiogenic pulmonary edema. This lack of specificity was illustrated in one study of patients with pulmonary edema, in which clinical assessment was found to be a poor predictor of volume status determined by measurement of intracavitary pressures.[61]

Capillary refill has been shown in one study to be an insensitive predictor of hypovolemia in patients presenting to an emergency department.[62] The study also suggested that orthostatic vital signs were significantly more sensitive and specific. An extensive review of the literature regarding the use of the physical exam in hypovolemia found that there was no evidence supporting the utility of capillary refill and that orthostatic vital signs were more useful but still insensitive.[63]

Clinical measurement of the height of JVPs above the right atrium has historically been used for estimation of CVP. A systemic review of the literature[64] revealed that among three major studies done on this topic, results varied widely, and in one study, physicians correctly predicted the CVP only ~55% of the time.[65]

CENTRAL VENOUS PRESSURE—Measurement of CVP provides and is a surrogate for the loading conditions of the right ventricle (RV), and by extension a surrogate for the loading conditions of the left ventricle as well. Although the superior vena cava (SVC) is usually used for this measurement, it appears that cannulation of the abdominal vena cava or common iliac vein correlates with SVC pressures, in both spontaneously breathing[66] and mechanically ventilated[67] patients, although caution must be used in extreme cases of high intraabdominal pressure, as with abdominal compartment syndrome. Because CVP can be transduced through any standard port of a central venous catheter, it is usually readily available and involves no additional risk to the patient beyond that involved in placing the catheter.

Although this measurement can be used as an assessment of volume status, historically the PAOP has been preferred. The main rationale for this argument has been the nature of the Frank-Starling relationship, which suggests that stroke volume is dependent on left ventricular preload. Hence, the loading conditions of the left ventricle may be the most pertinent factor in determining cardiac output. Additional arguments favoring the use of PAOP over CVP are unreliability of CVP to estimate left ventricular preload in patients with pulmonary vascular disease (e.g., parenchymal lung disease including late ARDS, or pulmonary embolism) or right ventricular infarction.

The counterpoint to this argument is the (Guytonian) theory that the gradient between mean circulatory pressure and right atrial pressure determines venous return and cardiac output.[68] Therefore, right atrial pressure (and by extension, CVP) acts as the downstream pressure for venous return and thus would be a major determinant of cardiac output and a suitable target for resuscitation.[69,70]

This view has been supported by physiological observations made by Magder and colleagues in small groups of patients, suggesting that guiding interventions by the CVP can result in meaningful changes in cardiac output. In patients whose CVP recordings showed an inspiratory decline in pressure (indicating that the right atria was relatively underfilled), they were able to decrease cardiac output by applying positive end-expiratory pressure (PEEP),[71] and increase cardiac output with fluid loading,[72] indicating that the use of the effect of respiration on the CVP to detect hypovolemia may be justified.

Clinical data validating use of the CVP as a guideline to resuscitation are lacking. As already discussed, Rivers and colleagues utilized a resuscitation protocol designed to maximize hemodynamic support early in the course. CVP was included in the combined hemodynamic endpoint, with a desired goal of 8 to 12 mm Hg. Although the treatment group in this study had a significantly lower mortality, it is not clear that differences in CVP were responsible because CVP was only significantly higher in the treatment group at one time point (6 hours after enrollment), and CVP goals were on average met in both groups.

PULMONARY ARTERY OCCLUSION PRESSURE—The PAOP is an estimate of left atrial and left ventricular end-diastolic pressure (LVEDP) assuming no pressure gradient exists across the mitral valve in diastole (e.g., no mitral stenosis). Assuming that the left ventricle has a direct pressure–volume relationship, LVEDP should represent LVEDV. The clinician can then attempt fluid challenges to increase the PAOP and observe resulting changes in cardiac output and stroke volume. It has long been known that many of the physiological assumptions underlying these concepts are problematic. In order for the PAOP to accurately reflect preload, it must accurately reflect the LVEDP and there must be a direct and predictable relationship between LVEDP and LVEDV. However, in practice, this relationship is not straightforward and may be affected by several factors that occur commonly in patients. For example, myocardial ischemia, sepsis, hypertension, and diabetes may all alter the compliance of the ventricle. In addition, intrathoracic pressure changes due to PEEP, increased abdominal pressure, or changes in right ventricular volume may all alter the LVEDP/LVEDV relationship. Furthermore, accurate measurement of PAOP is often unreliable due to technical reasons. In one oft-quoted study, evaluation of 2711 examples of PAOP recording revealed that 30% were not adequate for evaluation. Among the problems cited were poor dynamic response or damped pressure tracings and balloon overinflation.[73] Even when readings are correctly obtained, there is a great deal of variability in expertise among physicians, and readings may often be misinterpreted.[74]

Multiple studies involving patients in a variety of clinical settings have shown a rather poor ability of the PAOP to predict changes in stroke volume or left ventricular volume in

response to volume loading.[75–77] Based on available data, it appears that using the PAOP as a measure of preload must be done with caution.

ECHOCARDIOGRAPHY—In addition to measuring global cardiac function and cardiac output as already discussed, echocardiography has been used to estimate volume status, with little success. In part, this results from the lack of a true echocardiographic standard for determining hypovolemia, although certain findings have been correlated with hemodynamic measurements.[78] Two studies comparing echocardiographic evaluation of hypovolemia with invasive hemodynamic monitoring were also not encouraging. In one group of 60 postoperative cardiac patients with unexplained hypotension, echocardiographic findings were poorly associated with hypovolemia.[79] In the other, mitral blood flow and annular displacement were measured echocardiographically and used as a surrogate for left ventricular loading conditions. When compared with pulmonary artery catheterization, they found that PAOP could only be roughly estimated as either < 8 mm Hg or > 13 mm Hg.[80] Another group measured changes in chamber sizes during challenge with military antishock trousers, and suggested that changes in systolic function as a result of altering preload and afterload might be useful in guiding therapy.[81]

OTHER METHODS—Measurements of right ventricular compliance are currently receiving attention as markers of preload. Using modified thermodilution techniques with a rapid-response pulmonary artery thermistor, the relative temperature drop between systolic contractions of the RV can be plotted. The RV ejection fraction can then be computed from the results. This also allows computation of RV end-systolic and end-diastolic volumes.[82] The resulting right-ventricular end-diastolic volume index (RVEDVI) may be superior to the PAOP in determining preload. The rationale behind this argument is the fact that RVEDVI appears to predict the ability of CO to increase in response to a fluid challenge. This was shown by Diebel and colleagues, who evaluated PAOP, RVEDVI, and CI in 32 trauma patients. They found that RVEDVI and PAOP provided different assessments of preload in 35% of patients, and RVEDVI appeared to better predict whether CI would increase following fluid challenge.[83] Other studies have shown benefit to monitoring RVEDVI in patients who are being mechanically ventilated with PEEP, a situation in which PAOP is difficult to interpret.[84] Although measurement of RVEDVI requires special equipment and expertise that are not yet widely available, it may receive more attention based on the promising early results.

Extravascular lung water (EVLW) and intrathoracic blood volume (ITBV) are variables that can be calculated using dye-dilution techniques, or derived from pulse contour analysis. These techniques have recently piqued interest for their applicability in quantifying pulmonary edema, particularly in the management of ARDS. One study of treatment guided by EVLW suggested a decreased length of mechanical ventilation and ICU stay in comparison to treatment guided by PAOP.[85] Sakka and colleagues evaluated a dye-dilution technique for determining EVLW and suggested the measurement might provide useful prognostic information.[86] Other evidence has emerged that the ITBV may be a better predictor of preload and response to fluid challenge than CVP and PAOP.[87] A device is currently available that can measure cardiac output via pulse contour analysis and simultaneously estimate ITBV. The ability to simultaneously follow these variables noninvasively would be a potent addition to the ICU monitoring arsenal. Finally, Nuckton and colleagues have suggested that increased pulmonary dead space (measuring through inspired and expired gases) connotes worse mortality in ARDS.[88] Because one of the causes of increased dead space is decreased cardiac output, this technique might be useful for detecting occult hemodynamic compromise.

Indices of Organ Perfusion

PHYSICAL EXAMINATION AND CLINICAL ASSESSMENT—Among the classic findings attributed to hypoperfusion or an ineffective arterial circulation are skin changes (pallor, cyanosis, or mottling), decreased urine output, and altered mental status.

As mentioned previously, one study evaluated subjective skin temperature (defined as warm or cool) in conjunction with more objective findings, and found that cool skin temperature was associated with significantly lower cardiac output, CI, $S_{MV}O_2$, and pH, and was also associated with significantly higher lactate levels. This suggests that the subjective finding of cool skin temperature has value as a screen for occult hypoperfusion.[14]

Decreased urine output is widely viewed as a harbinger of decreased organ perfusion, based on years of clinical observations. Restoration of urine output is also commonly used as a goal for resuscitation. Decreased urine output is often the first sign of impending renal failure, which connotes a worsened prognosis in shock.[89] However, caution must be used when interpreting the cause of oliguria because factors other than decreased splanchnic perfusion may be at work, such as drug toxicity, ureteral obstruction or interruption following trauma, or immune-mediated renal failure. Similarly, restoration of urine output does not necessarily indicate adequate renal perfusion. An osmotic diuresis following intravenous contrast administration or during hyperglycemia may occur in the presence of early renal injury. In addition, cerebral salt wasting or the diuretic phase of acute tubular necrosis may “uncouple” urine flow from parenchymal renal function or renal blood flow. For these and other reasons, diminished urine output may present as a late indicator of hypoperfusion, by which time irreversible injury has already occurred.[90] In their study of early goal-directed therapy, Rivers and associates targeted urine output as a measure of adequate tissue perfusion in their combined hemodynamic endpoint. Although the treatment group had less mortality, the authors did not report whether urine output was significantly different between the groups.[6]

Altered mental status (sensorium and/or cognition) has long been associated with shock, and may be due in part to poor tissue perfusion. However, in septic shock the pathogenesis of mental status changes appears to be complex, including alterations in neurotransmitter function and cytokine-mediated changes in brain function (so-called septic encephalopathy) that cannot be attributed to hypoperfusion alone.[91] Other complicating factors may include the presence of primary neurological injury or altered mental status due to the administration of sedatives or analgesics.

LACTATE—Lactate is a product of pyruvate metabolism and is primarily produced by tissues that undergo a high rate of glycolysis. Because lactate is a product of anaerobic metabolism, lactate is produced by tissues that are in oxygen debt. In the clinical setting, lactate has proven value in predicting outcomes. For example, it has been shown in one study that survivors of shock had a 10% or greater decrease in lactate levels after 1 hour of therapy, whereas nonsurvivors did not.[11] In another study of 48 patients with septic shock, increased initial and late lactate levels proved to be a better predictor of adverse outcome than oxygen-derived variables, and survivors were noted to have a decrease in lactate levels whereas nonsurvivors did not.[92] The same investigators more recently showed that the duration of hyperlactatemia appeared to best differentiate survivors from nonsurvivors, and increased serial lactate levels predicted the onset of multi–organ system failure.[93]

Lactate is useful as a global marker of tissue hypoperfusion but cannot be used to measure perfusion of specific tissues. It may be useful in the initial assessment of shock, and throughout the course for prognostic value and following response to therapy. However, caution must be exercised in interpreting lactate levels. Elevated lactate represents a

relatively delayed response to hypoperfusion and may result in making treatment decision in response to conditions that may not reflect the patient's current clinical status. Also, lactate levels may remain elevated for a period of time after the insult that caused their elevation, particularly in liver failure, where lactate metabolism is impaired. Furthermore, current understanding of lactate metabolism suggests that lactate formation in shock occurs via a complex process that is not explained by tissue hypoperfusion alone. Failure to clear lactate within 24 hours appears to connote a poor prognosis, and persistent elevation of lactate levels appears to be associated with almost total mortality.[95] However, it is not clear that altering therapy in response to these elevations will affect outcome, particularly in established septic shock. Rather, the persistent elevation of lactate may reflect the presence of tissue injury that cannot be reversed by conventional means of hemodynamic resuscitation. However, because of its prognostic value, low cost, and ease of monitoring, serum lactate remains a useful part of overall assessment and monitoring and may identify a subset of patients who benefit from early, goal-directed resuscitation.[6]

ARTERIAL BLOOD GASES AND BASE DEFICIT—Arterial blood gases (ABGs) are commonly used for monitoring of respiratory and acid–base status in the critically ill. To some extent, the association of shock with metabolic acidosis makes ABG monitoring useful in shock. However, overall acid–base status involves a complex interplay between various metabolic and respiratory influences and may be deceiving if used as a surrogate for tissue perfusion.

The blood base excess concentration refers to the amount of acid (or base) that must be added to a liter of human blood under standardized conditions of temperature, O_2 concentration, and CO_2 concentration to correct the pH to 7.40. In acidemia, this number is referred to as the base deficit. Actual titration is not required because the number can be calculated from any blood gas sample using the Astrid and Siggard-Anderson nomogram. The potential advantage of using this method is that it allows "isolation" of the metabolic component of the acid–base status from the respiratory component.

When used clinically, base deficit correlates with lactate levels and appears to provide similar information.[96] In particular, increased initial base deficit and failure to improve on serial measurements appear to be associated with a worse prognosis.[97] Because both lactate and base deficit measurements are readily available, some attention has focused on comparing them. Results in this regard have been conflicting, with some suggesting that persistently increased base deficit despite normalized lactate indicates incomplete resuscitation,[98] whereas others favor lactate as a better predictor.[99] Overall, the data appear to favor the use of lactate. Given the low cost and ease of obtaining measurements, it may be reasonable to utilize base deficit, but available data do not support using values to guide therapy, especially values taken in isolation.

OXYGEN DELIVERY AND CONSUMPTION—Experimental data in animals show that as tissue DO_2 falls, oxygen extraction increases until a critical value of O_2 delivery is reached, below which tissues cannot extract any more oxygen, and tissue hypoxia ensues.[100] This phenomenon is known as supply-dependent O_2 consumption and occurs when DO_2 is very low. In comparison, clinical studies in critically ill humans have suggested that the critical value (sometimes known as the dysoxic point) is increased, and supply-dependent O_2 consumption can occur even when DO_2 is normal or high.[101]

With this in mind, techniques have been developed for making global measurements of changes in DO_2 and consumption, with the goal of detecting occult tissue hypoxia. The optimal methods for accomplishing this have been hotly debated and there is no single method of making these determinations that has been universally accepted. Furthermore,

because the same variables appear in the calculation of both DO_2 and oxygen uptake in some research methods, the mathematical phenomenon known as coupling of shared measurement error raises serious questions about the very presence of supply-dependent VO_2 at normal or supra-normal levels of DO_2.[102]

Monitoring DO_2 and VO_2 for use in guiding therapy has not proven clinically useful, mainly because of the uncertain nature of the DO_2/VO_2 relationship. A promising early study measured DO_2 and VO_2 in critically ill patients and showed that increasing DO_2 by prostacyclin infusion increased VO_2 in nonsurvivors, but not survivors, suggesting that occult tissue hypoxia was present in the nonsurvivors.[103] This suggested a potential strategy of evaluating the DO_2/VO_2 relationship and attempting to therapeutically increase DO_2 in hopes of passing the dysoxic point. However, because of the unclear relationship and the difficulty in precisely describing the dysoxic point, the alternative strategy of increasing cardiac output to a specified supranormal levels has been employed. This has not proven to be a useful approach, as already discussed.[10] At present there does not appear to be a role for routine measurement of VO_2.

GASTRIC TONOMETRY—The gut represents a promising target for tissue-specific monitoring. The structure of vasculature in the bowel facilitates countercurrent flow and exchange of solutes but has the unfortunate side effect of allowing arteriovenous oxygen shunting and recirculation of CO_2. Therefore, this organ seems to be the first affected by decreased splanchnic perfusion because these tendencies are exacerbated when blood flow is low. Although the kidney has a similar physiology and could theoretically be used toward a similar purpose, the gastrointestinal (GI) tract is more practical because it is more easily accessed. Animal data also suggest that mesenteric ischemia may precede renal ischemia in shock.[90] Furthermore, the presence of relative GI ischemia is thought to be clinically relevant because of the potential for breaches in mucosal integrity and translocation of bacteria and associated vasoactive mediators. For these reasons, measurement of GI tissue perfusion can theoretically be useful in diagnosing hemodynamic insufficiency, providing goals for therapy, and predicting outcomes according to the degree of insufficiency.

Various techniques have been described for monitoring the GI mucosa, including direct measurement using pH microelectrodes and blood flow monitoring using laser Doppler techniques. Gastric tonometry was first described over 40 years ago and has become the most commonly used method for tissue-specific monitoring of perfusion. A substantial amount of animal and human experimental data has been compiled concerning its utility as a measure of gastric mucosal pH, as a useful indicator of perfusion and/or outcome, and as a meaningful variable for guiding treatment.

Tonometry is based on the principle that the pCO_2 in a small fluid- or air-filled semipermeable balloon will equilibrate with the pCO_2 of surrounding fluid if placed in the lumen of the gut. This fluid, in turn, is in equilibrium with the pCO_2 of the gastric mucosa. When mucosal perfusion decreases, decreased clearance of CO_2 and cellular hypoxia caused by decreased DO_2 will result in accumulation of CO_2 in the mucosa. The net effect of this process will increase the pCO_2 of the balloon contents. Therefore, the measured pCO_2 provides a surrogate for perfusion of the mucosa. This value can either be interpreted unmodified or used to calculate pH of the gastric mucosa [intramucosal pH (pHi)] by using arterial bicarbonate concentration to solve for the pH in the Henderson-Hasselbalch equation. More recently, the pHi has fallen out of favor because of numerous sources of error in the calculation (including the effect of systemic acid–base status), and attention has begun to focus on relative differences in measurements, particularly the mucosal–arterial pCO_2 difference, the so-called CO_2 gap. However, most of the data describing this technique in the present literature have focused on the pHi.

This concept has been validated as a measure of regional perfusion by numerous animal and human studies. Its utility as a meaningful clinical variable is somewhat more controversial. It has been shown that pHi has prognostic value in patients with shock or other risk of hemodynamic compromise. For example, in one early series of 85 patients undergoing elective cardiac surgery, Fiddian-Green and Baker found that decreased pHi was a more sensitive predictor of complications than various global measures of perfusion.[104] In 1993, Maynard and colleagues evaluated the outcomes of a series of 83 patients with acute circulatory failure and demonstrated that decreased pHi was a more accurate predictor of prognosis than a variety of standard global measures of perfusion, including lactate levels.[105] Various other studies have also supported the prognostic significance of pHi in various clinical settings, including multiple-trauma, sepsis, and liver transplantation.[106–108]

It is still unclear whether mucosal pHi or pCO_2 can be altered by therapies directed at increasing perfusion, raising doubt about the utility of using these measurements to guide treatment. Studies of protocols designed to detect and treat gastric mucosal hypoperfusion have met with mixed results, which are well summarized by a meta-analysis of several trials evaluating the effects of various vasoactive medications on gastric pHi.[109] These results suggested that pHi could be improved through use of hemodynamically active medications such as dobutamine or norepinephrine. Other attempts to improve pHi through interventions have met with mixed success.[110–112]

Similarly, it is unclear whether directing resuscitation efforts toward enhancing gastric mucosal perfusion results in improved outcomes. In the largest study of its type, Gutierrez and colleagues[113] evaluated 260 patients admitted to ICUs with Acute Physiology and Chronic Health Evaluation II (APACHE II) scores between 15 and 25. Patients were stratified by initial pHi and randomized into a control group (which received “standard care”) and a treatment group, which received intervention designed to increase DO_2 or reduce demand. This study showed a significantly higher 28-day survival in the treatment group, but only among patients whose pHi was ≥ 7.35 on admission, suggesting that this variable may be a marker of prognosis as opposed to a guide to therapy. The study has been criticized for the lack of specific treatment protocols used to guide therapy. Other studies performed along similar lines have also suffered from problems with sample size and design. Ivatury and colleagues[114] enrolled a series of 57 trauma patients and compared a strategy of guided therapy based on pHi against an alternative strategy of guiding therapy based on the global variables of DO_2 or consumption. They did not find a significant difference in overall survival, but the time required to optimize pHi was significantly longer in nonsurvivors, and an index of multi–organ system failure was higher in patients who did not have pHi optimized within 24 hours. A more recent trial evaluated a sample of 210 ICU patients with heterogenous diagnoses who were randomized to conventional therapy versus use of colloid and dobutamine therapy to increase the pHi.[115] The authors did not find significant differences in mortality, but it has been suggested that the sample size lacked sufficient statistical power to detect a difference.[116]

Although gastric mucosal monitoring has been promising in theory, reported results supporting its use are lacking. Among the problems with the studies mentioned have been small sample sizes, ill-defined treatment protocols, and inability of treatment to improve gastric pHi or pCO_2. Issues have been raised as to whether the gastric mucosa is a suitable surrogate for the rest of the splanchnic circulation[117] and whether the information obtained from this technique can be more easily obtained through a global measurement, such as base deficit.[118] Furthermore, the results of the studies done by Guttierez and Ivatury suggest that failure to improve pHi early in the course connotes a worse prognosis, indicating that gastric tonometry may be a marker for advanced shock that cannot be reversed by resuscitation. A protocol geared toward reversing gastric mucosal hypoperfusion early in the course of shock

might produce results similar to those shown by Rivers and associates in their study of early goal-directed therapy.[6] In many centers, gastric tonometry has achieved fairly routine use, and research into its ideal applications is likely to continue. Recent improvements in technique, such as the development of a continuous measurement system that utilizes air instead of fluid,[118] may decrease the cost and other barriers to usage.

SUBLINGUAL CAPNOMETRY—Attempts have been made to develop a simpler and cheaper method of monitoring mucosal perfusion. Physiological studies have suggested that esophageal pHi correlates well with gastric pHi, with both decreasing under conditions of insufficient perfusion.[119] This concept was extrapolated to the oral mucosa and was confirmed by elegant animal studies that showed sub-lingual mucosal pCO_2 ($p_{sl}CO_2$) correlates well with gastric pCO_2, CI, and lactate levels,[120] and that both gastric pCO_2 and $p_{sl}CO_2$ could be restored by reversal of shock.[121]

Although human data have been limited thus far, the technique has shown promise in initial studies. In the first trial of its type, 46 critically ill patients were evaluated with $p_{sl}CO_2$ measurements. At a $p_{sl}CO_2 > 70$ mm Hg, positive predictive value for clinical evidence of shock was 100%, and $p_{sl}CO_2 < 70$ mm Hg had a positive predictive value of 93% for survival.[122] In a study designed to validate the technique, Marik showed that $p_{sl}CO_2$ closely correlated with gastric pCO_2 in 76 critically ill patients.[123] A follow-up study compared $p_{sl}CO_2$ to the more conventional hemodynamic markers of lactate and mixed venous oxygen saturation in 54 critically ill patients. $P_{sl}CO_2$ was a better predictor of outcomes and was more responsive to interventions than the other measures.[124] Ongoing studies will focus on the value of using sublingual capnometry in guiding therapy.

Although sublingual capnometry has not yet been evaluated to the same extent as gastric tonometry, it likely provides roughly equivalent information. Because it is less cumbersome, requires less operator involvement, is less invasive, and provides instantaneous measurements, it may prove more appealing than gastric tonometry and is likely to be studied further in the near future.

OTHER METHODS—Multiple other techniques have been used for the detection of tissue hypoperfusion, and still more are in development. For example, tissue electrodes are available that can directly measure tissue O_2 and CO_2 concentrations. Because these require implantation, they are currently practical mainly for subcutaneous use and hence less helpful for monitoring the viscera. Nonetheless, they have shown some utility in clinical trials[125] and, as the technology advances, may find broader application. Another currently available method for measuring regional perfusion is laser-Doppler flowmetry. This utilizes the Doppler effect in conjuction with a laser probe to detect movement of blood and can be a useful tool for determining regional blood flow, but the technique can only be applied to directly accessible mucosal surfaces.[126] It is possible to insert probes into the stomach or colon to measure mucosal blood flow, but it is not clear that this offers a significant advantage over current tonometric techniques. The technique has been used for experimental purposes[127] and may be clinically useful.

CONCLUSIONS AND FUTURE DIRECTIONS

A minimum set of variables that we have described as “basic” monitoring techniques in shock (Table 1) represents a reasonable basis for routine practice. In addition, there is evidence that cool skin temperature provides an efficient and reasonably reliable indication of either or both inadequate cardiac output and tissue perfusion. Several invasive and noninvasive techniques allow for serial or continuous monitoring of $S_{CV}O_2$, $S_{MV}O_2$, cardiac output, and/or preload with great potential to guide resuscitative attempts in patients with

septic shock. To date, the most promising approach combines standard monitoring variables (blood pressure and urine flow) with monitoring of both CVP and $S_{CV}O_2$ to guide goal-directed therapy early in the course of shock.[6] Other trials in patients with established shock have been disappointing. The failure of goal-directed therapies later in the course suggests that organ dysfunction is due to cellular injury or "cytopathic hypoxia" that may not be reversed by increasing perfusion[94–97,113,114] and is consistent with evolving theories of the pathogenesis of shock.[8,9,128]

Beyond these basic techniques, the PAC continues to figure prominently in clinical practice despite the concerns elucidated here. Routine use in certain groups of patients[53] or resuscitation to an arbitrary set of hemodynamic parameters[10] has not produced beneficial results. Problems with procedural morbidity and accurate measurement can potentially be addressed by improving training in these areas[129] and there is encouraging evidence that PAC use is safe.[52] Clinical trials of PAC efficacy and effectiveness are needed to better define the role, if any, for monitoring PAC variables in critically ill patients. These studies are ongoing.

Overall, a major trend in the field has been toward the development of systems that provide equivalent or additional data with less invasive techniques than a PAC. Some of these, such as gastric tonometry, offer the potential to measure organ-specific indices of perfusion and have entered clinical use at some centers. Others, such as esophageal Doppler monitoring, pulse contour analysis, and sublingual capnometry, have yet to find a place in usual care practices.

In addition to an expanding role for noninvasive monitoring of hemodynamics, future research is likely to focus on more sensitive and specific measures of end-organ function. Advanced metabolic imaging techniques such as positron emission tomographic scanning or functional magnetic resonance imaging may become useful for detecting metabolic activity of tissues as a guide to relative perfusion. As our understanding of the biochemical and immunological mechanisms behind shock increases, the evolving sciences of proteomics and genomics are likely to play a greater role in clinical practice.[130] Such approaches may allow for individualized care based on anticipated environment/genotype interactions. Although it may be years away, it is now possible to envision an ICU of the future where clinicians can evaluate a patient's sequence of gene activation or the pattern of protein expression during critical illness to both assess the adequacy of resuscitation and direct new therapies.[131]

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Objectives

On completion of this article, the reader should be able to: (1) summarize the goals of monitoring in shock; (2) compare the various methods available for monitoring; and (3) select a monitoring strategy for an individual patient.

Accreditation

The University of Michigan is accredited by the Accreditation Council for Continuing Medical Education to sponsor continuing medical education for physicians.

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The University of Michigan designates this educational activity for a maximum of 1 category 1 credit toward the AMA Physician's Recognition Award.

Table 1

Basic Monitoring in Shock

Standard Monitoring techniques Employed in Shock
Conventional vital signs (heart rate, blood pressure, body temperature, oxygen saturation)
Electrocardiography
Fluid intake/output
Laboratory monitoring (complete blood count, chemistries, blood gases)

Table 2

Echocardiography and PA Catheterization Compared

	Echocardiography	**PA Catheterization**
Information	Cardiac output (Doppler flow)	Cardiac output (thermodilution)
	Preload (chamber sizes)	Preload (filling pressures)
	Regional myocardial function	Venous O_2 sampling
	Valvular disease	Calculated resistance values
Comparison	Measurements less well validated	Measurements well validated
	No procedural morbidity	Procedural morbidity
	Data intermittent	Data continuous
	High fixed cost	Incremental cost