The role of echocardiography in hemodynamic monitoring

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Introduction: do we need more than central venous pressure and central venous oxygen saturation to titrate fluids and inotropes?

One of the most challenging aspects of assessing the critically ill is incorporation of bedside information to make a decision as to whether resuscitation is adequate. This difficulty stems from dynamic changes in fluid status and cardiovascular function. Thus, two overarching goals behind hemodynamic assessment are first, to determine whether the patient’s cardiac output (CO) and oxygen delivery are adequate for normal organ function, and second, to decide whether additional fluid, vasopressors or inotropes are likely to augment this output. To help provide standard goals and diagnostic measures of success in unstable patients, an evidence-based international consensus guideline was produced in 2008 [1]. To answer the first question, is the oxygen delivery adequate, these guidelines suggest a mean arterial pressure of at least 65 mmHg, urine output of 0.5 ml/kg/min and a central venous oxygen saturation of at least 70%. Should failure to achieve these goals confirm the clinician’s impression of inadequate resuscitation, they must then decide on rational therapy.

The Surviving Sepsis Campaign guidelines further suggest that after the hematocrit has been corrected, dobutamine should be added to achieve a central venous oxygen saturation of 70% or more [1]. Again, this recommendation is based mainly on the study by Rivers et al. [2], in which it was a part of a bundle of therapies aimed at increasing oxygen delivery.

Adverse consequences of ‘over’ resuscitation

Overshooting one’s volume resuscitation goal is not innocuous, best demonstrated by an increase of 2 days in the length of mechanical ventilation, ICU stay and a trend to increased mortality in patients with acute respiratory distress syndrome who were treated with a liberal fluid strategy compared with those in the conservative arm [3]. Initial CVP measurements in both groups of

Purpose of review

Echocardiography has become more widely available to noncardiologists because of the technological advances in smaller, multipurpose ultrasound units with basic cardiac capabilities. In this review, we discuss the type of clinical information a trained intensivist can hope to obtain from bedside echocardiography and suggest the ways in which this complements traditional hemodynamic monitoring.

Recent findings

Following a 10-h hands-on course, intensivists are able to perform and interpret a goal-oriented echocardiogram in approximately 10 min with good accuracy. Bedside echocardiography can aid in determining fluid status and qualitative cardiac ejection fraction, which can then be used immediately to guide therapy.

Summary

Intensivists can safely and accurately perform goal-oriented echocardiography. Although not yet proven to influence clinical outcome, we suggest that the major utility of echocardiography is for those with distributive or mixed shock in whom target central venous pressure has been achieved without evidence of adequate tissue perfusion. In this subset of patients, echocardiography can aid in selecting those most likely to benefit from further fluid or inotropic support.

Keywords

echocardiography, hemodynamic monitoring, shock
mechanically ventilated patients in this study were 12 mmHg [3]; however, the liberal fluid strategy patients ended the study with a positive fluid balance of over 6.1 and CVP of 12 mmHg, whereas conservatively resuscitated patients ended with an even fluid balance and a CVP of 9 mmHg. Thus, although an initial CVP target as recommended in the 2008 Surviving Sepsis Campaign guidelines may aid the speed and adequacy of resuscitation in the first 6 h, the best evidence suggests that therapy aimed at maintaining this target may be detrimental and must be tempered by other clinical considerations.

Dobutamine was part of a ‘resuscitation bundle’, which resulted in a mortality advantage in those randomized to early goal-directed therapy [2]. However, in definitive randomized control trials, critically ill hemodynamically unstable patients who received inotropes targeted to normalization [4] or supra-normalization [5] of systemic oxygen delivery either realized no benefit or were harmed by this treatment.

These seemingly contradictory data can be interpreted to mean that early aggressive resuscitation improves outcome in septic shock, but the individual components of therapy can be harmful if continued beyond their useful window. We will review the current methods of determining fluid and cardiovascular status and discuss whether bedside echocardiography (ECHO) offers the promise of providing us with a real-time means to determine those most likely to benefit from additional interventions.

**Standard measures of fluid responsiveness**

Various pressure measurements are used clinically to help define whether additional fluid will augment CO. CVP can be helpful if it is low (0–4 mmHg), as fluid generally results in improved CO at this level of CVP. However, in critically ill patients enrolled into studies of fluid responsiveness (i.e. those in whom the clinician is uncertain), the mean CVP ranges from 8 to 12 mmHg [6]. In these patients, because of alterations in cardiac compliance and contractility, the static measurement of CVP in no way predicts the likelihood of a positive response to fluid challenge [6–8]. As stroke volume is proportional to pulse pressure (PP), measures of PP variation in response to respiration’s effects on cardiac preload have been developed, with greater variation correlating with a higher probability of fluid responsiveness. ‘Delta-down’ is the difference between the value of the systolic pressure during an end-expiratory pause and the lowest systolic pressure over a respiratory cycle [9]. The delta-PP is the greatest fractional difference between PPs over a respiratory cycle [10]. Both these measures rely on arterial blood pressure (BP) measurements, and neither is considered valid in the setting of arrhythmia nor have they been validated in patients not on mechanical ventilation. Furthermore, large fluctuations (most notably in diastolic BP) as a result of vasopressor use can make these techniques difficult to interpret when repeatedly assessing the same patient.

These pressure measurements, both CVP and arterial, are simply surrogates the clinician can use to estimate the ventricular filling at which CO is optimal. Rather than using pressures to estimate volumes and ventricular performance, why not directly image the great veins, ventricular size and contractility?

**Clinical information the intensivist can acquire performing a goal-directed transthoracic echocardiogram**

Although the availability of ECHO coincides with the working day of the hospital, hemodynamic compromise is not limited to these hours. Consequently, a goal-directed examination by the attending intensivist is often the best study available. Without extensive echocardiographic training, a detailed cardiac examination, including valvular function, congenital abnormalities, intracardiac shunt and estimation of pulmonary pressures, is best done by a certified echocardiographer. However, in urgent situations, a limited cardiac examination including the presence or absence of a significant pericardial effusion, left ventricular performance and volume status has been adopted as a ‘goal-directed’ approach [11]. Following 10 h of hands-on instruction, intensivists performed a goal-directed study, later reviewed by experienced echocardiographers. It was found that the studies were technically adequate in 94% of cases, whereas the diagnostic interpretation of the intensivist with respect to all three goals was considered correct in 84% of cases [11]. Given the excellent technical results in this study, with more experience, the intensivists would have likely improved their interpretative skills. Below we discuss the major reasons to add ECHO to usual hemodynamic monitoring, assessment of fluid responsiveness and left ventricular performance.

**Echocardiographic measures of fluid responsiveness**

Using two-dimensional ECHO, systolic obliteration of the left ventricular cavity signifies underfilling of the heart and hypovolemia and predicts augmentation of CO in response to fluid challenge [12]. Although this sign is an excellent marker of relative hypovolemia, it is unusual in critically ill patients, particularly those who are on positive pressure mechanical ventilation. Systolic obliteration is associated with a smaller left ventricular end-diastolic area (LVEDA), a measure that has also been proposed to identify the volume status of a patient.

Quantitation of the LVEDA is possible through tracing the endocardium in the parasternal short-axis view at the
level of the papillary muscles [13]. This approach can also be used to score ventricular size in a more quantitative manner. LVEDA provides a relatively accurate assessment of ventricular size, but the absolute area is not a reliable indicator of fluid responsiveness [14] but rather allows one to trend the changes in LVEDA (as a surrogate of \(CO\)) in response to fluid boluses. Furthermore, this technique relies on good visualization of the endocardium and is thus more reliably done using transesophageal echocardiogram (TEE) than transthoracic echocardiogram (TTE). Given the greater ease and rapidity of TTE compared with TEE, this approach can be viewed as an ancillary measure rather than our preferred technique, respiratory variation in vena cava diameter.

**Respiratory variation in vena caval diameter**

In normal, nonventilated patients, the inferior vena cava (IVC) diameter is less than 20 mm and decreases by at least 50% on inspiration [15,16] (Fig. 1). However, mechanically ventilated patients have a high rate of IVC dilation [17] due to numerous causes, including intraabdominal syndromes. Given this derangement in the physiology of the critically ill, IVC diameter alone cannot reliably distinguish fluid response (defined as an increase in \(CO\) of \(\geq 15\%\)). Both the superior vena cava (SVC) and IVC empty directly into the right atrium, thus providing a window into right atrial compliance. However, of great technical importance, the IVC can be assessed using a goal-directed TTE subxiphoid approach, whereas the SVC can only be assessed via TEE.

**Figure 1 Transthoracic view of the respiratory variation in inferior vena cava diameter**

![M-mode view of the IVC just distal to its junction with the right atrium. Positive airway pressure during insufflation results in an increase in the diameter of the IVC (dIVC). Percentage change in dIVC = (maximal dIVC – minimal dIVC)/maximal dIVC \(\times\) 100. dIVC, inferior vena cava diameter; IVC, inferior vena cava. Reproduced with permission from [16].](image)

**Transesophageal echocardiogram assessment of the superior vena cava**

Through dynamic assessment of the vena cava throughout the respiratory cycle, it appears as though those patients who will respond to additional fluids with augmented \(CO\) can be identified [16–21]. Using TEE and assessment of the SVC, Charron et al. [18] have shown that in septic ventilated patients, at least 36% distensibility of the SVC with the respiratory cycle accurately predicted an increase in \(CO\) with volume expansion [19].

**Transthoracic echocardiogram assessment of the inferior vena cava**

Two studies in 2004 [16,21] examined the clinical ability of respiratory variation in IVC diameter to predict an increase in \(CO\) of at least 15%. Both studies enrolled mechanically ventilated patients with septic shock. Feissel et al. [16] measured the percentage variation in IVC diameter (dIVC) in 39 patients prior to 8 ml/kg of 6% hydroxyethyl starch, whereas Barbier et al. [21] used the same measure in 23 patients prior to 7 ml/kg of 4% modified fluid gelatin. \(CO\) was measured using ECHO before and after volume infusion, and a ‘fluid-responsive’ \(CO\) was one with an increase of at least 15% in both studies. Feissel et al. [16] found that at least 12% variation predicted fluid responsiveness, whereas Barbier et al. [21] found that at least 18% variation was predictive in their cohort. Therefore, a 12–18% variation in IVC diameter with respiration predicts response to fluid challenge. Until further studies determine the optimal cutoff for variation in IVC diameter, we use an average value of at least 15% variation in IVC diameter to predict fluid responsiveness.

**Left ventricular performance and its relationship with dynamic changes in afterload**

Early in critical illness, and particularly in septic shock, hemodynamic instability is fuelled not only by vasoplegia but also by cardiac dysfunction. Fifty to 100% of patients in septic shock who require fluids and vasopressors have demonstrable reductions in cardiac ejection fraction [8,22–25]. Of particular importance is the role of afterload in the apparent cardiac performance. In those patients who remain hypotensive (particularly their systolic BP) despite initial resuscitation, cardiac ejection fraction may appear normal because of the reduced cardiac workload. One might dismiss the option of inotropic support in this case as cardiac function has been deemed ‘adequate’, only to discover later that upon normalization of BP, the preexisting cardiac dysfunction has been unmasked. It is only through rapid reassessment of cardiac function that the intensivist can correctly initiate and titrate inotropes.
moderate dysfunction or severe dysfunction on the basis of qualitative measures [20]. Agreement between bedside intensivists performing the bedside ECHO and expert echocardiographers who later examine the images is high [20], a necessity if one is to consider the information reliable. Thus, bedside ECHO can rapidly and accurately assess cardiac systolic function, allowing the clinician to gauge the need for inotropic support in the face of a dynamic clinical situation.

**Conclusion**

Bedside ECHO is a useful adjunct to the often challenging physical examination in the critically ill. Upon completion of a 10-h course, intensivists are able to perform and interpret a goal-directed TTE in approximately 10 min. This technique should be considered in the initial evaluation of the hemodynamically unstable patient to rule out cardiogenic causes of shock if the clinical examination is equivocal.

Bedside goal-directed TTE is also useful for hemodynamic monitoring in patients with ongoing shock, particularly in determining whether further fluids, inotropes or both are warranted. Using sepsis as a disorder capable of causing both distributive and cardiogenic shock, we advocate the following approach (Fig. 2). In those with elevated serum lactate with or without hypotension, intravenous fluids are the initial mode of resuscitation. Should fluids titrated to clinical findings prove inadequate, prompt placement of a central venous catheter whose distal tip lies in the SVC is essential. Using this line for both therapeutics and diagnostics, one then administers fluids and vasopressors to achieve a CVP of 8 mmHg in nonventilated patients and 12 mmHg in ventilated patients. Goals of therapy are a mean arterial pressure of at least 65 mmHg, urine output of 0.5 ml/kg/min and a central venous oxygen saturation of at least 70%. If the serum lactate was initially elevated, then this should be followed and should normalize as a goal of therapy.

If resuscitation targets are not met and this is associated with inadequate oxygen delivery (central venous oxygen saturation of less than 70%), the patient should be considered for transfusion to a hematocrit of more than 30%.

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**Figure 2 An approach incorporating goal-directed transthoracic echocardiogram in patients with hemodynamic compromise**

<table>
<thead>
<tr>
<th>Initial resuscitation (first 6 hrs)</th>
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</thead>
<tbody>
<tr>
<td>Begin resuscitation immediately in patients with hypotension or elevated serum lactate &gt;4 mmol/l</td>
</tr>
<tr>
<td><strong>Resuscitation goals</strong></td>
</tr>
<tr>
<td>CVP 8–12 mmHg</td>
</tr>
<tr>
<td>Mean arterial pressure &gt;65 mmHg</td>
</tr>
<tr>
<td>Urine output &gt;0.5 ml/kg/h</td>
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<tr>
<td>Central venous (superior vena cava) oxygen saturation &gt;70% or mixed venous &gt;65%</td>
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<tr>
<td><strong>If venous oxygen saturation target is not achieved</strong></td>
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<tr>
<td>* Consider further fluid</td>
</tr>
<tr>
<td>Transfuse packed red blood cells if required to hematocrit of &gt;30% and/or</td>
</tr>
<tr>
<td><strong>Start dobutamine infusion, maximum 20 ug/kg/min</strong></td>
</tr>
<tr>
<td><strong>Subcostal view, bedside ECHO.</strong></td>
</tr>
<tr>
<td>If dIVC 15% or greater, administer fluids</td>
</tr>
<tr>
<td>Monitor blood pressure, CVP and urine output</td>
</tr>
<tr>
<td><strong>2D ECHO for qualitative cardiac ejection fraction.</strong></td>
</tr>
<tr>
<td>If qualitative EF moderately or severely depressed consider dobutamine infusion</td>
</tr>
<tr>
<td>Monitor heart rate, blood pressure, CVP and urine output</td>
</tr>
<tr>
<td>Discontinue dobutamine if results in an unstable tachycardia, more hypotensive</td>
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</tbody>
</table>

We recommend an initial resuscitation as suggested in the Surviving Sepsis Campaign international guidelines. However, when considering additional fluids or inotropes in the face of inadequate resuscitation, we recommend goal-directed TTE to clarify volume status and cardiac contractility. CVP, central venous pressure; dIVC, inferior vena cava diameter; ECHO, echocardiography; EF, ejection fraction. Adapted with permission from [1](#).
It is at this stage we suggest that bedside goal-directed TTE has a role, specifically in determining whether to institute inotropic support or administer further fluids. In addition, if hemodynamic instability persists beyond the 6-h window when initial numerical targets may no longer be appropriate, then additional information provided by a goal-directed TTE might be helpful. Using the subcostal view to measure dIVC, further fluid should be strongly considered for those with a dIVC of at least 15%. Qualitatively graded cardiac ejection fraction is then obtained, with dobutamine considered for those with moderately or severely depressed systolic function.

Although the information gained from bedside goal-directed TTE should allow one to select those patients most likely to respond to additional fluids or inotropes, to date, no randomized clinical trial has shown that altering management on the basis of echocardiographic data results in improved ICU morbidity or mortality. Such a trial should clarify whether improved physiology translates into better clinical outcome, a correlation that is not always certain.

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There are no conflicts of interest.

**References and recommended reading**

Papers of particular interest, published within the annual period of review, have been highlighted as:

- of special interest
- of outstanding interest

Additional references related to this topic can also be found in the Current World Literature section in this issue (pp. 272–273).


