Authors and Disclosures

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Hemodynamic Parameters to Guide Fluid Therapy

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Abstract and Introduction

Abstract

The clinical determination of the intravascular volume can be extremely difficult in critically ill and injured patients as well as those undergoing major surgery. This is problematic as fluid loading is considered the first step in the resuscitation of hemodynamically unstable patients. Yet, multiple studies have demonstrated that only about 50% of hemodynamically unstable patients in the ICU and operating room respond to a fluid challenge. Cardiac filling pressures including the central venous pressure and pulmonary artery occlusion pressure have traditionally been used to guide fluid management. However, studies performed over the last 30 years have demonstrated that cardiac filling pressures are unable to predict fluid responsiveness. Over the last decade a number of studies have been reported that have used heart–lung interactions during mechanical ventilation to assess fluid responsiveness. Specifically, the pulse pressure variation derived from analysis of the arterial waveform, the stroke volume variation derived from pulse contour analysis and the variation of the amplitude of the pulse oximeter plethysmographic waveform have been shown to be highly predictive of fluid responsiveness. While the left ventricular end-diastolic area as determined by transesophageal echocardiography is a more accurate measure of preload than either the central venous pressure or pulmonary artery occlusion pressure, it does not predict fluid responsiveness as well as the dynamic indices. This paper reviews the evolution and accuracy of methods for assessing fluid responsiveness in patients in the ICU and operating room.

Introduction

Despite improvements in resuscitation and supportive care, progressive organ dysfunction occurs in a large proportion of patients with acute, life-threatening illnesses and those undergoing major surgery. It has been proposed that the multi-organ dysfunction syndrome of the critically ill is a consequence of tissue hypoxia due to inadequate oxygen delivery, often exacerbated by a microcirculatory injury and increased tissue metabolic demands.[1,2] This may be further compounded by cytopathic hypoxia due to mitochondrial dysfunction.[3,4] Emerging data suggest that early aggressive resuscitation of critically ill patients may limit and/or reverse tissue hypoxia, progression to organ failure and improve outcome.[5] In a landmark study, Rivers *et al*. demonstrated that a protocol of early goal-directed therapy reduces organ failure and improves survival in patients with severe sepsis and septic shock.[6] Similarly, optimization of cardiac output in patients undergoing major surgery may reduce postoperative complications and length of stay.[7] Fluid therapy is considered the first step in the resuscitation of most patients with hypotension and shock. Uncorrected hypovolemia, leading to inappropriate infusions of vasopressor agents, may increase organ hypoperfusion and ischemia.[8] However, overzealous fluid resuscitation has been associated with increased complications, increased length of ICU and hospital stay and increased mortality.[9,10] The first step in the hemodynamic management of critically ill patients is to determine the adequacy of tissue/organ perfusion. While the signs of shock may be obvious those of subclinical hypoperfusion may be more subtle (see Table 1). It should however be noted, that increasing cardiac output/oxygen delivery in patients with adequate organ perfusion serves no useful purpose. Indeed, studies of yesteryear have demonstrated that targeting 'supra-normal' hemodynamic parameters may be harmful.[11,12] In patients with indices of inadequate tissue perfusion fluid resuscitation is generally regarded as the first step in resuscitation. However, clinical studies have consistently demonstrated that only about 50% of hemodynamically unstable critically ill patients are volume responsive.[13] The resuscitation of the critically ill patient therefore requires an accurate assessment of the patients' intravascular volume status (cardiac preload) and the ability to predict the hemodynamic response following a fluid challenge (volume responsiveness).

Table 1. Clinical indices of the adequacy of tissue/organ perfusion

- Mean arterial pressure
  - Cerebral and abdominal perfusion pressures
- Urine output
- Mentation
- Capillary refill
- Skin perfusion/mottling
- Cold extremities (and cold knees)
- Blood lactate
- Arterial pH, BE and HCO_3
- Mixed venous oxygen saturation $SmvO_2$ (or $ScvO_2$)
- Mixed venous pCO_2
- Tissue pCO_2
- Skeletal muscle tissue oxygenation (StO_2)

A reduction in intravascular volume results in a fall in stroke volume, which is initially compensated for by an increase in heart rate thereby maintaining cardiac output. However, with further volume depletion cardiac output and then blood pressure falls. This is associated with a reduction in organ perfusion. At the organ level local autoregulatory mechanism comes into play in an attempt to maintain tissue perfusion. A reduction in renal perfusion normally results in dilatation of the glomerular afferent arteriole and constriction of the glomerular efferent arteriole so that glomerular capillary hydrostatic pressure and glomerular filtration rate remains constant. However, a decrease in renal perfusion pressure below the autoregulatory range (mean arterial pressure < 70 mmHg) leads to an abrupt fall in glomerular filtration rate and urine output (oliguria). In the elderly and in patients with diseases affecting the integrity of the afferent arterioles, lesser degrees of hypotension may cause a decline in renal function and oliguria. While primary renal diseases and urinary tract obstruction may lead to oliguria, intravascular volume depletion with renal hypoperfusion is the commonest cause of oliguria in clinical practice. Other features of intravascular volume depletion include a concentrated urine, postural hypotension, tachycardia and cold extremities (see Table 1).

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Preload is best defined as left ventricular end-diastolic volume (LVEDV). According to the Frank–Starling principle as the preload increases left ventricular stroke volume increases until the optimal preload is achieved at which point the stroke volume remains relatively constant (see Figure 1). This optimal preload is related to the maximal overlap of the actin-myosin myofibrils. It is important to note that in an intact heart the actin-myosin links cannot be disengaged and hence there is *no descending limb* of the Frank–Starling curve. 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 hypoxia. In normal physiologic conditions, both ventricles operate on the ascending portion of the Frank–Starling curve.[14] This mechanism provides a functional reserve to the heart in situations of acute stress. In normal individuals, an increase in preload (with volume challenge) results in a significant

increase in stroke volume.[15] In contrast, only about 50% of patients with circulatory failure will respond to a fluid challenge.[13,16] Furthermore, as a result of altered left ventricular compliance and function, the position of an acutely ill patient on his/her Frank–Starling curve cannot be predicted from their preload (LVEDV) alone. In critically ill patients it is therefore important not only to determine the patients' preload (LVEDV) but their fluid responsiveness, i.e. to whether the patient will increase his/her stroke volume or cardiac output with fluid loading (i.e. have recruitable cardiac output). Simultaneously, it is important to determine the patients' overall fluid balance and more specifically the interstitial fluid volume (third space volume). In patients with increased interstitial fluid volume it maybe more appropriate to increase cardiac output by using a vasoactive agent rather than with fluid boluses alone (see Measures of volume overload).

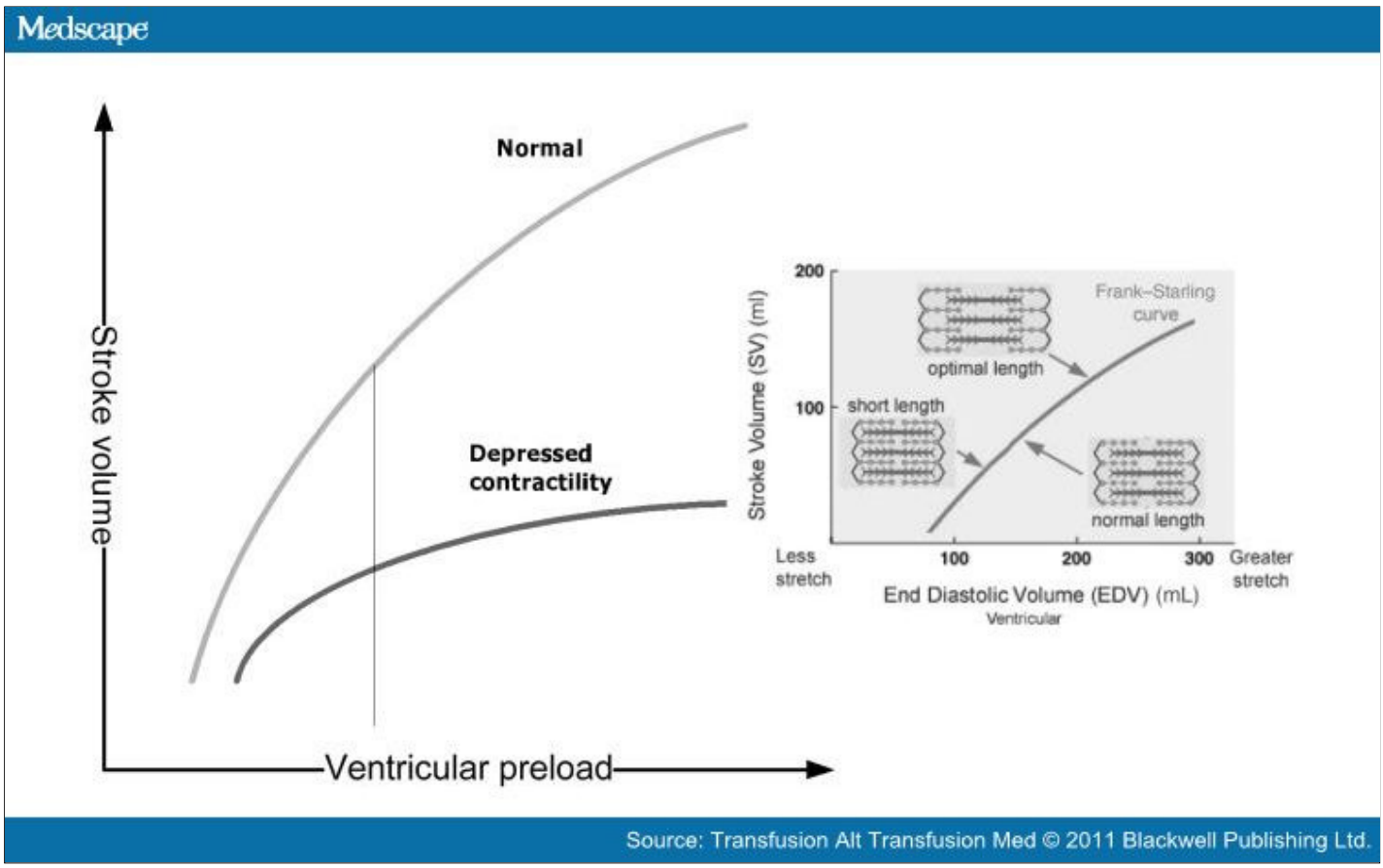

Figure 1. The relationship between preload and stroke volume (Frank–Starling principle).

'Static' Measures of Intravascular Volume

The Central Venous Pressure

The central venous pressure (CVP) is frequently used to guide fluid management. Indeed, two surveys of European intensivists/anesthesiologists reported that over 90% used the CVP to guide fluid management.[17,18] A recent Canadian survey reported that 90% of intensivists use the CVP to monitor fluid resuscitation in patients with septic shock.[19] The basis for using the CVP to guide fluid management comes from the dogma that the CVP reflects intravascular volume; specifically it is widely believed that patients with a low CVP are volume depleted while patients with a high CVP are volume overloaded. Furthermore, the '5-2' rule that was popularized in the 1970's is still widely used today for guiding fluid therapy.[20] According to this rule, the change in CVP following a fluid challenge is used to guide subsequent fluid management decisions.

The CVP describes the pressure of blood in the thoracic vena cava near the right atrium of the heart. The CVP is a good approximation of right atrial pressure, which is a major determinant of right ventricular filling. It has therefore been assumed that the CVP is a good indicator of right ventricular preload. Furthermore, as right ventricular stroke volume determines left ventricular filling, the CVP is assumed to be an indirect measure of left ventricular preload. However, because of the changes in venous tone, intrathoracic pressures (positive end expiratory pressure, etc.), left and right ventricular compliance and geometry that occur in critically ill patients, there is a poor relationship between the CVP and right ventricular end-diastolic volume. Furthermore, the right ventricular end-diastolic volume may not reflect the patients' position on the Frank–Starling curve and therefore his/her preload

reserve.

We performed a systematic review to assess the value of the CVP in directing fluid management.[21] We identified five studies that compared the CVP with the measured circulating blood volume while 19 studies determined the relationship between the CVP/delta-CVP and the change in cardiac performance following a fluid challenge. The pooled correlation coefficient between the CVP and the measured blood volume was 0.16 (95% CI 0.03–0.28). The pooled correlation coefficient between the baseline CVP and change in stroke index/cardiac index was 0.18 (95% CI 0.08–0.28). The pooled area under the receiver operator characteristic (ROC) curve was 0.56 (95% CI 0.51–0.61). The pooled correlation between the delta-CVP and the change in stroke index/cardiac index was 0.11 (95% CI 0.015–0.21). The results of this systematic review clearly demonstrate that there is no association between the CVP and circulating blood volume, that the CVP is a poor indicator of left and right ventricular preload and that the CVP does not predict fluid responsiveness. Based on these results we believe that the CVP should no longer be routinely measured in the ICU, operating room or emergency room.

Pulmonary Artery Occlusion Pressure

Since the introduction of the pulmonary artery catheter almost 30 years ago the pulmonary artery occlusion pressure (PAOP) was assumed to be a reliable and valid indicator of left ventricular preload. However, it was not long after the introduction of the pulmonary artery catheter that studies began to appear demonstrating that the PAOP was a poor reflection of preload. Recent studies have clearly demonstrated that the PAOP is a poor predictor of preload and volume responsiveness.[16,21–23] The PAOP suffers many of the limitations of the CVP. The PAOP is a measure of left ventricular end-diastolic pressure and not LVEDV or LV preload. The use of the PAOP to measure left ventricular preload assumes a direct relationship between the left ventricular end-diastolic pressure and LVEDV. This pressure–volume curve, which describes left ventricular compliance, is normally curvilinear. Furthermore, alterations in left ventricular compliance shift the pressure–volume curve. Factors that alter left ventricular compliance include left ventricular preload, left ventricular afterload, left ventricular mass and ventricular fiber stiffness. Myocardial ischemia, sepsis, diabetes, obesity, aging, sustained tachycardia, dialysis, cardioplegia as well as other factors alter myocardial fiber stiffness. In addition, the left ventricular pressure–volume curve is affected by the degree of right ventricular filling. As the two ventricles are physically coupled by the interventricular septum and by the constraining effects of the pericardium, the end-diastolic pressure–volume curve of either ventricle is dependent upon the diastolic volume of the other. Furthermore, the PAOP is influenced by the juxtacardiac pressure, particularly if positive end expiratory pressure is used.

Left Ventricular End-diastolic Area

Transesophageal echocardiography (transgastric, mid-papillary short axis view) has been used to assess left ventricular dimensions in patients undergoing mechanical ventilation. The left ventricular end-diastolic area (LVEDA) has been shown to correlate well with the intrathoracic blood volume (ITBV) and global end-diastolic volume (GEDV),[24,25] as well as with LVEDV as measured by scintography.[26–28] An end-diastolic diameter of < 25 mm and a LVEDA of < 55 cm^2 have been used to diagnose hypovolemia.[29] While a number of studies have found the LVEDA to be a good predictor of fluid responsiveness[30–34] other studies have failed to replicate this finding.[35–40] It should be recognized that a small LVEDA does not always reflect decreased intravascular volume. Small LV volumes can be seen with restriction to filling because of decreased ventricular compliance (hypertrophy, ischemia), acute cor pulmonale [acute right ventricle (RV) dysfunction] and pericardial disease. Therefore, while the LVEDA may be an accurate measure of preload, preload does not necessarily translate into preload responsiveness. In addition to a decreased LVEDA, systolic obliteration of the LV cavity has been used as a sign of deceased preload.[29] However, LV end-systolic cavity obliteration does not necessarily imply decreased left ventricular filling.[41] A major limitation of echocardiography is that it provides a snapshot of ventricular function at a single period in time. Recently, a disposable transesophageal echocardiography probe that allows continuous monitoring of LV function has been developed (ClariTEE, ImaCor, Uniondale, NY, USA). Such technology allows monitoring of LV volumes and function over time, allowing the clinician to determine the response to various therapeutic interventions.

Inferior Vena Caval Diameter

The diameter of the inferior vena cava (IVC) as it enters the right atrium can be measured by subcostal echocardiography. A collapsed IVC is assumed to be indicative of volume depletion while a distended IVC is reflective of a high right atrial pressure. A number of authors have demonstrated that the mean end-diastolic IVC dimension correlates with mean right atrial pressure in both spontaneously breathing and mechanically ventilated patients.[42,43] Measurement of the IVC diameter is therefore an indirect indicator of the CVP and is associated with all the limitations of CVP measurement.

ITBV Index and GEDV Index

Transpulmonary thermodilution using a single-indicator (cold bolus) is a minimally invasive technique that allows for the computation of the cardiac output (PiCCO monitoring system, Pulsion Medical Systems, Munich, Germany).[44] Transpulmonary thermodilution requires the use of a specific thermodilution tipped arterial catheter (usually placed in the femoral artery), which measure the change in temperature following the injection of a bolus of cold saline through a central vein (in the neck). Mathematical analysis of the transpulmonary thermodilution curve allows the calculation of the ITBV index as well as the volume of blood contained in the four chambers of the heart, called the GEDV index (GEDVI). While the GEDVI provides a good estimate of intravascular volume and preload; it has the same limitations as the LVEDA (as measured by transesophageal echocardiography) in predicting volume responsiveness.[32,38,45]

Dynamic Indices of Intravascular Volume

Over the last decade a number of studies have been reported that have used heart–lung interactions during mechanical ventilation to assess fluid responsiveness. Specifically, the pulse pressure variation (PPV) derived from analysis of the arterial waveform, the stroke volume variation (SVV) derived from pulse contour analysis and the variation of the amplitude of the pulse oximeter plethysmographic waveform have been shown to be highly predictive of fluid responsiveness.

SVV and PPV

The principles underling the PPV (and SVV) are based on simple physiology (see Figure 2). Intermittent positive pressure ventilation induces cyclic changes in the loading conditions of the left and right ventricles. Mechanical insufflation decreases preload and increases afterload of the RV. The RV preload reduction is due to the decrease in the venous return pressure gradient that is related in the inspiratory increase in pleural pressure.[46] The increase in RV afterload is related to the inspiratory increase in transpulmonary pressure. The reduction in RV preload and increase in RV afterload both lead to a decrease in RV stroke volume, which is at a minimum at the end of the inspiratory period.[47] The inspiratory reduction in RV ejection leads to a decrease in LV filling after a phase lag of two or three heart beats because of the long blood pulmonary transit time. Thus the LV preload reduction may induce a decrease in LV stroke volume, which is at its minimum during the expiratory period. The cyclic changes in RV and LV stroke volume are greater when the ventricles operate on the steep rather than the flat portion of the Frank–Starling curve (see Figures 1 and 3). Therefore, the magnitude of the respiratory changes in LV stroke volume is an indicator of biventricular preload dependence.[46]

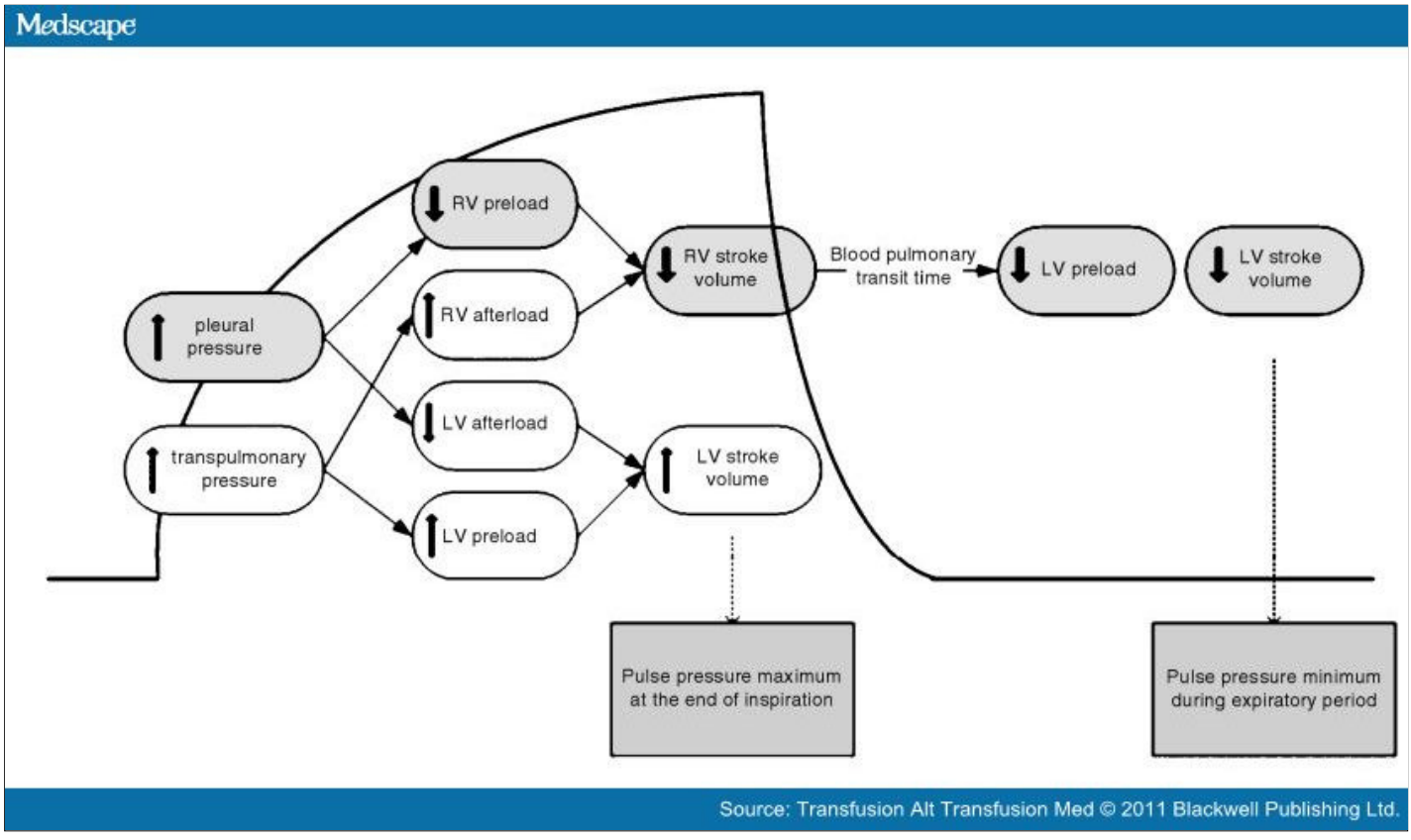

Figure 2. Hemodynamic effects of mechanical ventilation. The cyclic changes in left ventricle (LV) stroke volume are mainly

related to the expiratory decrease in LV preload because of the inspiratory decrease in right ventricle (RV) filling. Reproduced with permission from Critical Care/Current Science Ltd.[46]

We performed a systematic review to determine the accuracy of the PPV and SVV to predict volume responsiveness.[48] Twenty-nine clinical studies were included in this meta-analysis. We demonstrated that the PPV and SVV measured during volume controlled mechanical ventilation predicted with a high degree of accuracy (ROC of 0.94 and 0.84, respectively) those patients likely to respond to a fluid challenge as well the degree to which the stoke volume is likely to increase. The sensitivity, specificity and diagnostic odds ratio were 0.89, 0.88 and 59.86 for the PPV and 0.82, 0.86 and 27.34 for the SVV, respectively. The predictive value was maintained in patients with poor LV function. Furthermore, with remarkable consistency these studies reported a threshold PPV/SVV of 12%–13%. In this study the area under the ROC curves were 0.55 for the CVP, 0.56 for the GEDVI and 0.64 for the LVEDA index (see Table 2). The enormous appeal of using the PPV/SVV as a marker of volume responsiveness is that it dynamically predicts an individual patients' position on their Starling curve and this is independent of ventricular function and compliance as well as pulmonary pressures and mechanics (see Figure 3). Furthermore, this technology is relatively simple both in concept and in execution and is conducive to monitoring both in the operating room and ICU. In a pilot study Lopes and colleagues demonstrated that optimization of cardiac output based on the PPV during high risk surgery significantly reduced the number of postoperative complications as well as ICU and hospital length of stay.[7]

Table 2. Predictive value of techniques used to determine fluid responsiveness[13]

Method	Technology	AUC*
Pulse pressure variation	Arterial waveform	0.94 (0.93–0.95)
Systolic pressure variation	Arterial waveform	0.86 (0.82–0.90)
Stroke volume variation	Pulse contour analysis	0.84 (0.78–0.88)
Left ventricular end-diastolic area	Echocardiography	0.64 (0.53–0.74)
Global end-diastolic volume	Transpulmonary thermodilution	0.56 (0.37–0.67)
Central venous pressure	Central venous catheter	0.55 (0.18–0.62)

*AUC = area under the curve with 95% confidence intervals.

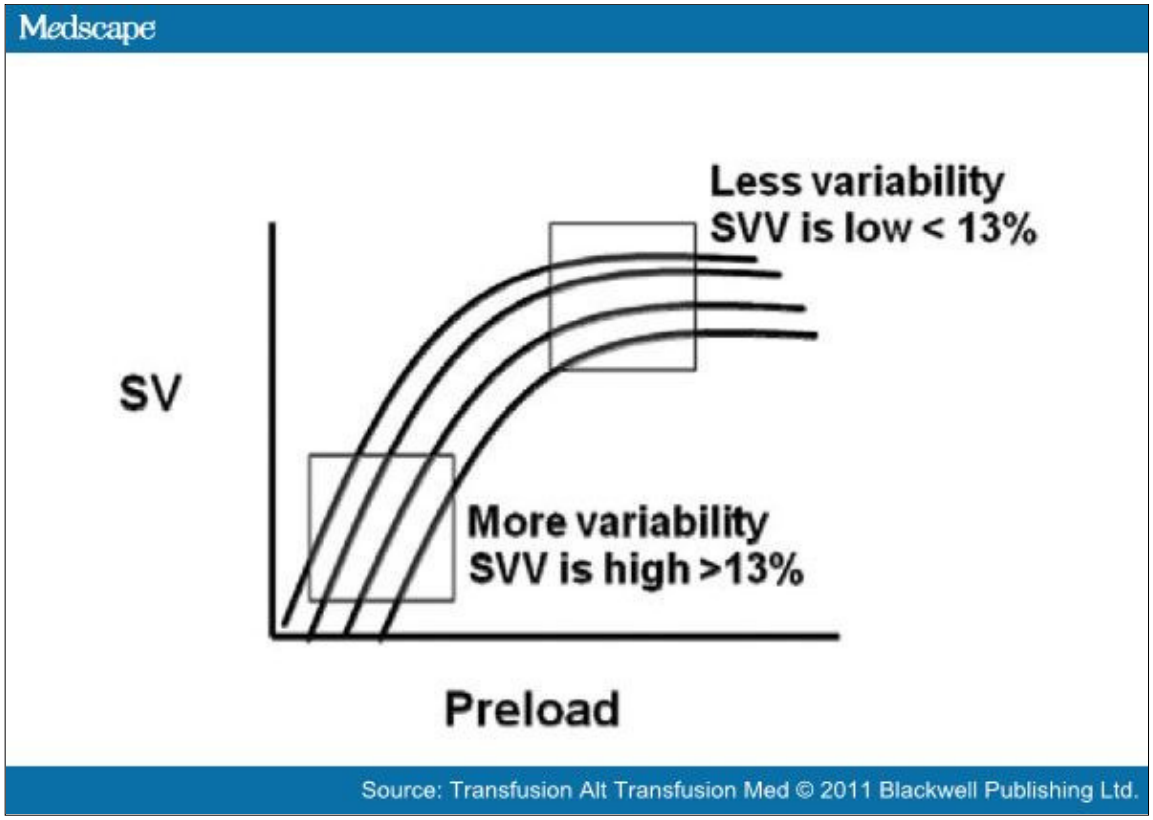

Figure 3. Arterial waveform analysis during positive pressure ventilation predicts an individual patients' position on their Starling curve and allows optimization of cardiac performance. SVV, stroke volume variation.

It should be appreciated that both arrhythmias and spontaneous breathing activity will lead to misinterpretations of the respiratory variations in pulse pressure/stroke volume. Furthermore, for any specific preload condition the PPV/SVV will vary according to the tidal volume. Reuter and colleagues demonstrated a linear relationship between tidal volume and SVV.[49] De Backer and colleagues evaluated the influence of tidal volume on the ability of the PPV to predict fluid responsiveness.[50] These authors reported that the PPV was a reliable predictor of fluid responsiveness only when the tidal volume was at least 8 mL/kg. For accuracy, reproducibility and consistency we suggest that the tidal volume be increased to 8–10 mL/kg ideal body weight prior to and after a fluid challenge.

The pulse oximeter plethysmographic waveform differs from the arterial pressure waveform by measuring volume rather than pressure changes in both arterial and venous vessels. As an extension of pulse pressure analysis during mechanical ventilation, dynamic changes in both the peak and amplitude of the pulse oximeter plethysmographic waveform have been used to predict fluid responsiveness.[51] The dynamic changes of the plethysmographic waveform with positive pressure ventilation have shown a significant correlation and good agreement with the PPV, and has accurately predicted fluid responsiveness in both the operating room and ICU setting.[52–54] The 'pleth variability index' (PVI) is an automated measure of the dynamic change in the perfusion index that occurs during a respiratory cycle (Masimo Corporation, Irvine, CA, USA). The perfusion index is the infrared pulsatile signal indexed against the non-pulsatile signal and reflects the amplitude of the pulse oximeter waveform. The PVI correlates closely with the respiratory induced variation in the plethysmographic and arterial pressure waveforms and can predict fluid responsiveness non-invasively in mechanically ventilated patients.[55,56] These oximetry techniques may be valuable for monitoring fluid responsiveness in ICU and surgical patients who do not have an arterial catheter *in situ*.

Dynamic Changes in Aortic Flow Velocity/Stoke Volume Assessed by Echocardiography

The respiratory changes in aortic flow velocity and stroke volume can be assessed by Doppler echocardiography. Assuming that the aortic annulus diameter is constant over the respiratory cycle, the changes in aortic blood flow should reflect changes in LV stroke volume. Feissel and colleagues demonstrated that the respiratory changes in aortic blood velocity predicted fluid responsiveness in mechanically ventilated patients.[35] In this study the LVEDA index was unable to predict fluid responsiveness. Wiesenack and colleagues were however unable to replicate these findings.[45]

Positive Pressure Ventilation Induced Changes in Vena Caval Diameter

Cyclic changes in superior (SVC) and inferior vena caval (IVC) diameter as measured by echocardiography have been used to predict fluid responsiveness. Barbier and colleagues and Feissel and coworkers demonstrated that the distensibility index of the IVC, which reflects the increase in the IVC diameter on inspiration (mechanical ventilation) was able to predict fluid responsiveness.[57,58] This technique has a number of limitations, including the fact that subcostal echocardiography may be difficult in obese patients and those that have undergone laparotomy. Furthermore, changes in IVC diameter are affected by intra-abdominal pressure (IAP) making this technique unreliable in patients with high IAP. Vieillard-Baron and colleagues have demonstrated that the collapsibility index of the SVC is highly predictive of volume responsiveness.[59,60] The SVC collapsibility index is considered to be more reliable than the IVC distensibility index in predicting fluid responsiveness.[61] The major drawback of this technique is that the SVC can only be adequately visualized by transesophageal echocardiography and this technique is not conducive to continuous monitoring.

The dynamic indices of volume responsiveness reviewed above are dependent on the cyclic changes in intrathoracic pressure induced by positive pressure ventilation and are not applicable to spontaneously breathing patients. However, changes in aortic flow velocity and stroke volume induced by passive leg raising in non-ventilated patients have been demonstrated to be predictive of volume responsiveness.[62,63] Similarly, Delerme and colleagues demonstrated changes in the amplitude of the pulse oximeter plethysmographic waveform during passive leg raising in spontaneously breathing individuals.[64] This technique may prove useful in assessing fluid responsiveness in spontaneously breathing patients.

Measures of Volume Overload

Recent data strongly suggest that in critically ill and injured patients excessive fluid resuscitation with an accumulating positive fluid balance is associated with worse clinical outcomes.[9] Data from the ARDSNet study group indicate that the cumulative fluid balance by the fourth day of study was highly predictive of hospital mortality as well as ventilator- and ICU-free days.[65] In a cohort of patients with acute lung injury secondary to septic shock Murphy and colleagues demonstrated that a fluid management strategy that included adequate initial fluid resuscitation (within the first 6 hours) followed by a conservative late fluid management strategy (even-to negative fluid balance on at least 2 days during the first 7 days of treatment) was associated with improved patients outcomes.[66]

Dynamic changes in pulse pressure, stroke volume or perfusion index (PVI) together with echocardiographic assessment of LV function and size provide useful information for assessing preload and preload responsiveness; however, these techniques do not allow for the detection and quantification of fluid excess. Similarly, the CVP and PAOP are poor measures of volume status and cannot be used reliably to detect volume overload.[21] Some have suggested that patients receive volume resuscitation until they develop pulmonary edema (indicating that the 'tank is full'); this is clearly a dangerous approach.[67] Furthermore, radiographic and clinical signs of pulmonary edema and clinical evidence of anasarca are late signs of volume overload and poor end points for fluid resuscitation. Extravascular lung water (EVLW) as determined by transpulmonary thermodilution and IAP monitoring are two techniques that 'measure' interstitial fluid volume (tissue edema) and may aid in the assessment of volume overload.

EVLW

EVLW may be calculated from the descending limb (indicator dissipation) of the transpulmonary thermodilution curve.[68] This technique has been shown to compare favorably with the double indicator dilution technique and the ex-vivo gravimetric method.[69–72] Furthermore, this technique can detect small (10%–20%) increases in lung water.[73] The 'normal' value for EVLW is reported to be 5–7 mL/kg with values as high as 30 mL/kg during severe pulmonary edema. In an intriguing study, Sakka *et al*. found that the mortality was approximately 65% in ICU patients with an EVLW > 15 mL/kg whereas the mortality was 33% in patients with an EVLW < 10 mL/kg.[74] EVLW has been demonstrated to be an accurate indicator of the severity of lung injury and a reliable prognostic indicator in patients with sepsis-induced acute lung injury.[75,76] EVLW should be indexed to ideal body weight rather than actual body weight.[77] It is likely that clinicians using EVLW measurements to guide fluid therapy may reduce the cumulative positive fluid balance, the duration of mechanical ventilation and ultimately patient outcome. Furthermore, by dynamically following a patient's preload and preload responsiveness together with his/her EVLW, will allow the clinician to determine the patient's optimal preload (see Figure 4).

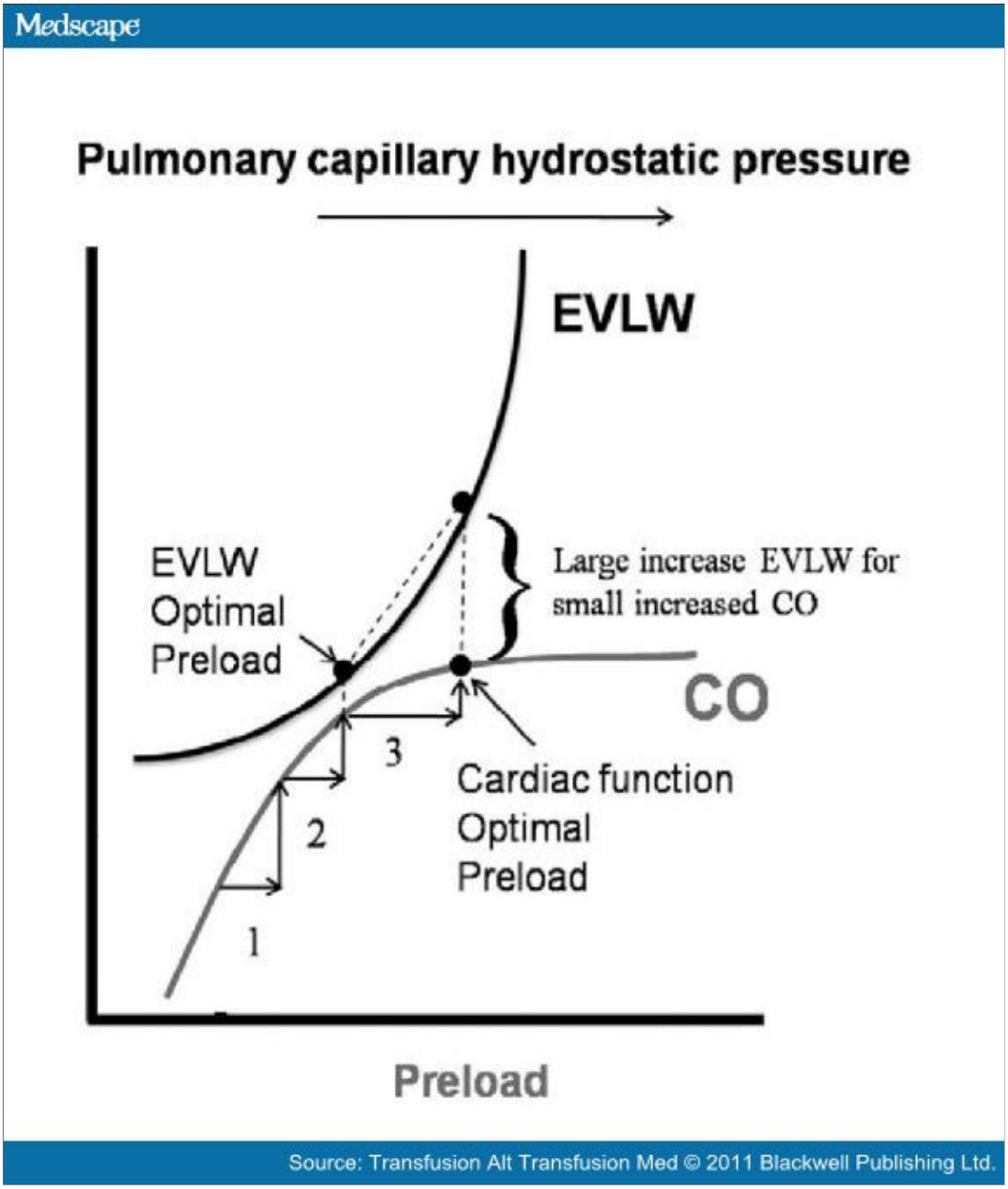

Figure 4. Optimization of preload based on dynamic changes in preload responsiveness and extravascular lung water (reproduced with permission from Dr Charles Phillips). EVLW, extravascular lung water.

IAP Monitoring

IAP is the pressure concealed within the abdominal cavity.[78] The World Society of the Abdominal Compartment Syndrome (http://www.wsacs.org) has recently developed consensus definitions outlining standards for IAP measurement as well as diagnostic criteria for intra-abdominal hypertension (IAH). According to the consensus guidelines IAH is defined as an IAP ≥ 12 mmHg and abdominal compartment syndrome as an IAP above 20 mmHg with evidence of organ dysfunction/failure.[79] The abdominal perfusion pressure is a more accurate predictor of visceral perfusion (MAP-IAP) with a target above 60 mmHg correlating with improved survival.[78] Major risk factors for IAH include abdominal surgery/trauma, high volume fluid resuscitation (> 3500 mL/24 hours), massive blood transfusion (> 10 units/24 hours), large burns, ileus, damage control laparotomy, liver failure with ascites, severe pancreatitis and liver transplantation. The IAP should be measured in all 'at risk patients' with repeated measures in those with IAH and following clinical deterioration. In patients with an increasing IAP volume resuscitation should be limited with attempts to achieve a negative fluid balance.

Conclusion

By virtue of its simplicity, accuracy and availability as a continuous monitoring tool, dynamic monitoring of the pulse pressure, stroke volume and pulse oximeter plethysmographic waveform would appear to be the ideal methods for the titration of fluid resuscitation in critically ill patients undergoing mechanical ventilation. Echocardiographic methods of assessing ventricular function and size complement the information obtained by these dynamic indices of fluid responsiveness. Measurement of EVLW and IAP may be useful in preventing volume overload. These data should be interpreted in the context of the patients' clinical condition as well as other parameters including the chest radiograph, PaO_2/FiO_2, urine output, renal function and cumulative fluid balance. The CVP and PAOP no longer have a place in modern hemodynamic monitoring.

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