Use of Ultrasound to Assess Fluid Responsiveness in the Intensive Care Unit

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Abstract: Determining the appropriate amount of fluid resuscitation to administer to a critically ill patient is a complex decision. Traditional tools for the assessment of preload sensitivity such as central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP) are inaccurate in predicting whether a patient requires volume resuscitation [1]. Diagnostic ultrasonography in the form of echocardiography offers an alternative means of determining whether a patient is preload sensitive.

Keywords: Shock, inferior vena cava, fluid therapy, stroke volume variation, fluid responsiveness, venous return, ultrasound.

While the determination of the cause of hypotension in a trauma patient or one who is bleeding from a gastrointestinal source is straight forward, most patients in the medical ICU have multiple etiologies of shock. This requires careful assessment prior to institution of fluid therapy. Patients in the ICU who are hypotensive may be preload sensitive; however, in some circumstances, volume resuscitation may be detrimental [2, 3]. For example, a patient in shock with acute cor-pulmonale and a dilated right ventricle should not receive volume resuscitation. Bedside echocardiography is a non-invasive tool that may be used to rapidly assess whether a patient with shock is fluid responsive.

PATHOPHYSIOLOGY OF HYPOVOLEMIA

Hypovolemia in critically ill patients is either absolute or relative. Absolute hypovolemia is characterized by a reduction in circulating volume. It is often easily identifiable on clinical grounds; as it may derive from major blood loss, third spacing, or dehydration. Relative hypovolemia is due to a maldistribution of circulating blood volume between the central and peripheral compartments as seen in distributive shock. Hypovolemia leads to compensatory physiological responses designed to defend against tissue hypoperfusion.

If hypovolemia is of sufficient severity or if the cardiovascular response is inadequate, shock may result. Hypovolemia leads to a decrease in venous return to the heart, which by Frank-Starling mechanism leads to a decrease in stroke volume (SV) and cardiac output (CO). Infusion of volume increases SV, if the heart is on the steep part of the Frank-Starling curve; and, within this range, the patient will be preload sensitive. However, if the heart is on the flat part of the Frank-Starling curve, volume resuscitation will not improve SV. Bedside echocardiography allows the clinician to identify whether volume resuscitation will improve cardiac output in the patient with shock i.e. whether the patient is operating on the steep part of the Frank-Starling curve.

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STANDARD METHODS OF EVALUATING PRELOAD SENSITIVITY

Clinical Examination

The history and physical examination may allow the clinician to identify hypovolemia in a patient in shock state. For example, the hypotensive patient with major gastro-intestinal hemorrhage who is in shock is highly likely to be preload sensitive. In this case, the intensivist should not hesitate to initiate immediate volume resuscitation on the basis of clinical assessment.

Central Venous Pressure and Pulmonary Artery Occlusion Pressure

The CVP is commonly used to identify the patient who is preload sensitive despite the fact that it is known to be an inaccurate predictor [1]. The use of PAOP as a means of guiding volume replacement has been questioned [4]. The PAOP does not predict preload sensitivity in a shock state unless it is below 5mmHg. It is very uncommon for the PAOP to be this low in the critically ill.

Pulse Pressure Variation (ΔPP)

Aortic pulse pressure is known to be directly proportional to the SV. Respiratory changes in pulse pressure (PP) in passive mechanically ventilated patients reflect changes in the SV.

Michard *et al*. [5] demonstrated that ΔPP (%) can be used to assess preload sensitivity in mechanically ventilated patients with septic shock. Maximal PP (PPmax) and minimum PP are determined in a single respiratory cycle using simultaneous recording of the airway and arterial pressures. ΔPP (%) is then calculated as:

100x {(PPmax-PPmin)/ ([PPmax +PPmin] /2)}. Huang *et al*. [6] also demonstrated that a ΔPP >12% in patients with acute respiratory distress syndrome on low tidal volume ventilation strategy and high PEEP, predicted preload sensitivity. The ΔPP is superior to CVP and PAOP in determining preload sensitivity [5]. The ΔPP requires insertion of an arterial catheter with associated delay and potential complications. A further limitation of ΔPP measurement is that it requires the patient be completely

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passive in their interaction with the ventilator and in a regular heart rhythm, limiting the utility of ΔPP.

Ultrasound Methods of Evaluating Preload Sensitivity

Ultrasound modalities allow the use of dynamic parameters to determine whether the patient is situated on the ascending portion of the Frank-Starling curve (i.e. preload dependent) or on the plateau portion (i.e. preload independent). Ultrasound modalities to evaluate preload sensitivity include transthoracic echocardiography (TTE), transesophageal echocardiography (TEE), esophageal Doppler (ED) and ultrasound of the inferior vena cava (IVC). Inferior vena cava assessment requires only basic level training, enables quick evaluation, and can be easily integrated into routine procedures like Focused Assessment with Sonography for Trauma (FAST).

The heart is a pressure chamber within a pressure chamber. Changes in intrathoracic pressure affect the pressure gradients for venous return to both the RV and the LV filling. During mechanical ventilation, inflation increases intrathoracic pressure which in turn reduces RV preload, increases RV afterload, and consequently reduces RV stroke volume if the RV is on the steep portion of the Frank-Starling curve. A change in RV stroke volume, after a phase delay of about 2 to 3 beats; leads to a decrease in left ventricular (LV) preload and LV stroke volume. This pass through effect on the LV stroke volume will be observed during the exhalation phase on the ventilator, if the LV is on the steep portion on the Frank-Starling curve. During mechanical ventilation, an increase in intrathoracic pressure increases LV filling by compression of the pulmonary veins. This compression effect increases venous return to the left atrium and the LV during inspiration. If the LV is on the steep portion of the Frank-Starling curve, SV will increase as a result of this augmentation of LV filling. These changes in SV from the RV and the LV are exaggerated in the setting of hypovolemia and are the basis for the dynamic methods of evaluating preload sensitivity.

Inferior Vena Cava Diameter Variation

The IVC is a retroperitoneal structure lying in longitudinal orientation to the spinal column and to the right of the abdominal aorta. It passes posterior to the liver and drains into the right atrium. The hepatic veins drain into the IVC just caudad to the right atrium.

The IVC is a highly compliant vessel with no valves and can be easily distended. Anatomical anomalies of the IVC are quite rare.

The IVC can be identified using either a standard cardiac or abdominal 2-D transducer by a subcostal approach (Fig. **1**). Measurements should be made at the hepatic segment of the IVC just cephalad to the origin of the hepatic vein. The IVC is identified with two-dimensional (2-D) imaging. The respiratory variation in the diameter of the IVC is measured from a frozen M-mode image using the electronic calipers of the machine coincident with ventilator cycling (Fig. **2**). Respiratory variation of IVC size in a passive patient on mechanical ventilation in regular heart rhythm has been described as a method of determining volume responsiveness that is easily mastered by the critical care physician (Video **1** and **2**).

Barbier *et al.* [7] in a prospective study of 23 patients with septic shock, showed that a variation of 18% in IVC diameter (dIVC) was strongly correlated with preload sensitivity. The dIVC calculated as [(maximum diameter on inspiration-minimum diameter on expiration)/ minimum diameter on expiration], identified fluid responders with a 90% sensitivity and 90% specificity. Feissel *et al.* [8]

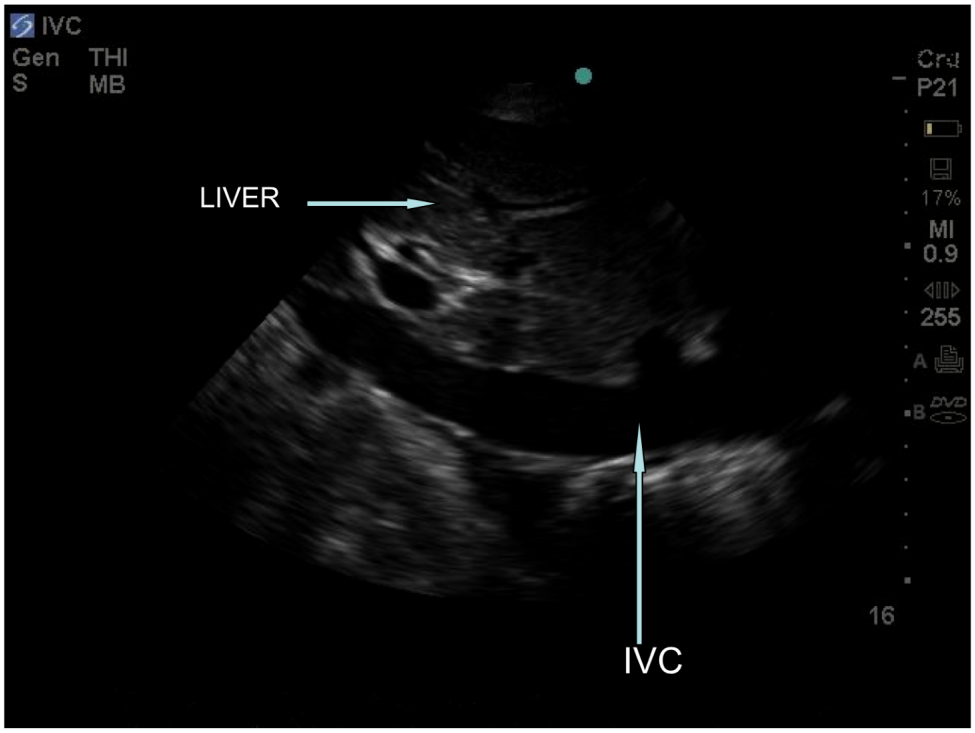

Fig. (1). Longitudinal image of the IVC using a subcostal view.

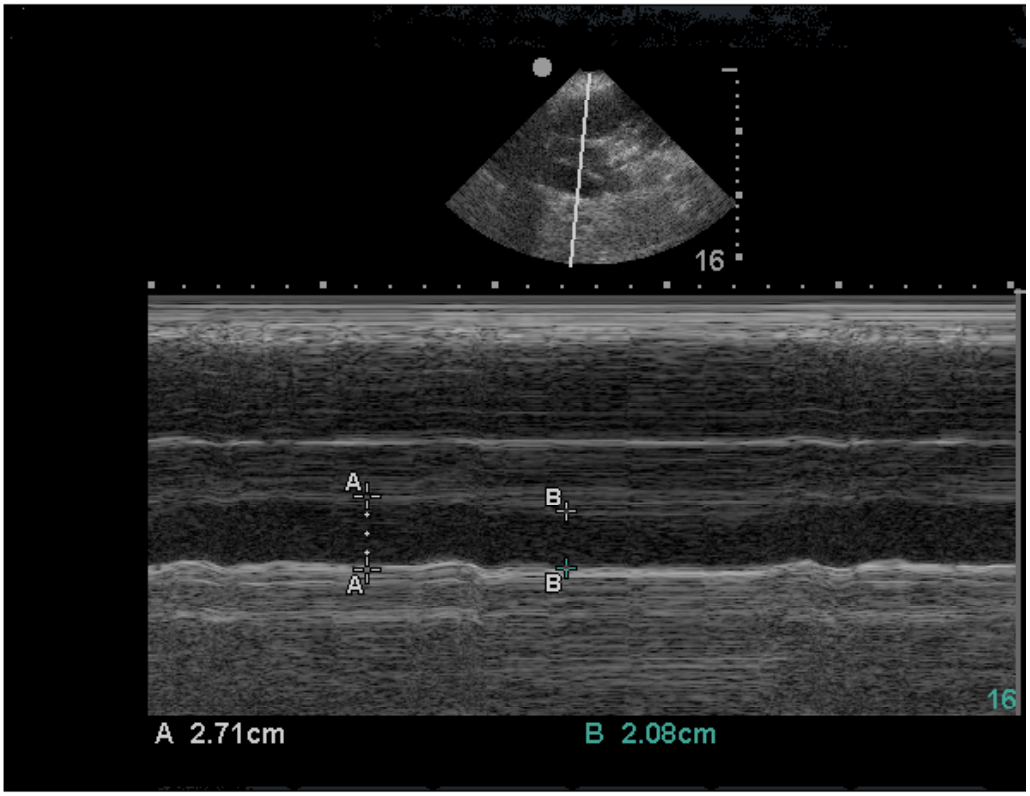

Fig. (2). Measurement of respiratory variation in the diameter of the IVC from a frozen M-mode image. A=Maximum diameter, B= Minimum diameter. dIVC is calculated as [(A-B)/B].

confirmed the utility of dIVC using a different method of measuring variation (max-min/mean value).

A limitation of IVC variation use in determining fluid responsiveness is that the patient must be passive in their interaction with the ventilator. Intraabdominal hypertension may invalidate this method. Tidal volume and PEEP, due to their effect on intrathoracic pressure, may also limit its utility. It should also be noted that IVC variation has not been studied in patients with pre-existing heart disease.

Because of these limitations in many patients, preload sensitivity may not always be determined by dIVC. Nevertheless, even in these cases, IVC size alone may have clinical utility. Anecdotally Patients with an IVC diameter of ≥3cm are unlikely to respond to volume, and Patients with IVC diameter less than 1cm or an IVC that is so small that it cannot be seen ("virtual IVC") have a high probability of being preload sensitive (Video **3**).

Superior Vena Cava Diameter Variation

Unlike IVC measurement, SVC measurement requires transesophageal echocardiography which requires specialized training and is not routine in many ICUs. Viellard-Baron *et al.* [9] showed that in passive mechanically ventilated patients, a variation of 36 % [(maximum on expiration-minimum diameter on inspiration)/maximum diameter] allowed discrimination between preload sensitive and non-sensitive patients with a sensitivity of 90% and specificity of 100%.

Cardiac Ultrasonography

For long the lone dominion of the cardiologist, cardiac ultrasonography has major applications for the intensivist responsible for bedside management of the patient with hemodynamic failure. The intensivist with skill at critical care echocardiography is able to rapidly identify major causes of shock that require prompt intervention such as pericardial tamponade, severe valve failure, acute cor pulmonale pattern related to pulmonary embolism, and segmental wall motion abnormalities related to myocardial infarction or ischemia [10-12]. Another major application of bedside ultrasonography is the determination of preload sensitivity.

Ventricular Size

In the patient with shock, the finding of a small hyperdynamic LV with effacement of the end systolic cavity ("kissing LV walls") strongly suggests major preload sensitivity and is a simple visual clue for immediate volume resuscitation of a hypotensive patient. This pattern can be easily identified during goal directed cardiac ultrasound from a parasternal long, short, or subcostal view (Video **4**).

Cannesson *et al.* [13] measured changes in LV diastolic area with TEE in the short axis view finding that a 16% variation of LV diastolic area between inspiration and expiration was predictive of preload sensitivity.

Respiratory Variation of Stroke Volume

LV stroke volume can be measured using pulse wave Doppler both with TTE and TEE. The velocity time interval (VTI) of LV systolic outflow is measured in the LV out flow tract (LVOT). The LVOT diameter is then measured at the same point. The product of VTI and LVOT diameter is equal to SV. This allows the cardiac ultrasonographer to measure real time LV SV on a stroke by stroke basis while the patient is on mechanical ventilatory support. The effect of ventilator cycling on stroke volume may be used to identify the patient who is preload sensitive. Conceptually, this is identical to the use of Δ PP. The diameter of the LVOT is constant throughout the cardiac cycle, therefore variation of VTI relates directly to variation of SV. To simplify measurement, peak velocity through the LVOT may be used as a surrogate for variation in VTI. An alternative method of looking for SV variation that may occur during ventilator cycling is to use Doppler flow measurement from the descending thoracic aorta.

Feissel *et al*. [14] used TEE to measure both peak aortic velocity and VTI to calculate respiratory variation of these values in patients on mechanical ventilatory support. A ΔVpeak of > 12% and ΔVTI of >20 % were highly predictive of preload sensitivity in patients with septic shock and normal LV function. Slama *et al*. [15] showed in an animal study that progressive blood loss was closely related to increase respiratory variation of aortic blood flow. Although these results have not been validated using TTE, it is very likely that they would be the same.

Measurements of VTI and velocity require advanced training in cardiac ultrasonography. The operator must be skilled in Doppler applications including knowledge of angle and position effect on Doppler measurements. Cardiac translation movement artifact must be recognized. Another limitation is that the patient must be passive in their interaction with the ventilator and in regular rhythm. Image acquisition quality may be suboptimal for TTE measurements in the critically ill, and the deep gastric TEE view may be difficult to obtain in some patients. Using the descending thoracic aorta Doppler signal has a specific limitation, as the aortic diameter may change in size depending on volume status of the patient. The presence of RV failure may invalidate measurement of respiratory variation of SV using Doppler for determination of preload sensitivity. The operator may use echocardiography to identify this class of patient.

Passive Leg Rising

Raising the legs of a supine patient, result in the immediate distribution of about 300 cc of blood to the intrathoracic cavity. Effectively, the clinician can observe the effects of a volume challenge on hemodynamics that is immediate and fully reversible.

Lamia *et al*. [16] used TTE to measure VTI, SV, and CO in spontaneously breathing patients before and after passive leg raising (PLR) finding that an increase in SV of 12.5% or more, predicted preload sensitivity. Miazel *et al*. [19] confirmed these findings using TTE in spontaneously breathing patients. Other groups have reported data that support the value of PLR in identifying preload sensitivity in patients in shock [13, 17, 18]. A major advantage of PLR is that it can be performed at the bedside in patients who are breathing spontaneously or on mechanical ventilation and in those with irregular heart rhythm. It requires a straight forward Doppler measurement through the LVOT using TTE before and after elevation of the patient's legs to a 45° angle for a minute or two. The effect of a major volume but reversible volume bolus is measured directly with Doppler echocardiography. If there is a significant change in SV following PLR, the patient in shock is volume responsive..

An important proviso for the clinician is to consider that all measurements of preload sensitivity must be placed in clinical context. Healthy individuals are preload sensitive i.e. they will increase SV in response to fluid administration because the normal LV is on the steep slope of the Frank-Starling curve. Identification of preload sensitivity is relevant only if the patient is in shock. Patients with normal hemodynamic function do not require volume resuscitation.

CONCLUSION

Skill in basic critical care ultrasonography permits the intensivist to identify patients who require volume resuscitation by measuring IVC dynamics. The advanced echocardiographer can use respiratory variation of SV and PLR technique to identify preload sensitivity. When combined with other aspects of cardiac and body ultrasonography, these methodologies allow the intensivist to confidently make important bedside management decisions.

SUPPLEMENTARY MATERIAL

This article contain 4 video files and it can be viewed at www.bentham.org/open/toccmj

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