Volume Responsiveness in Critically Ill Patients

Use of Sonography to Guide Management

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Invited paper

The Sound Judgment Series consists of invited articles highlighting the clinical value of using ultrasound first in specific clinical diagnoses where ultrasound has shown comparative or superior value. The series is meant to serve as an educational tool for medical and sonography students and clinical practitioners and may help integrate ultrasound into clinical practice.

Received August 28, 2013, from the Department of Emergency Medicine, Virginia Commonwealth University School of Medicine, Richmond, Virginia USA (D.E.); Ultrasound Unit, Department of Infectious Diseases, Fondazione Istituto Di Ricovero e Cura a Carattere Scientifico Policlinico San Matteo, Medical School, University of Pavia, Pavia, Italy (G.F.); and Division of Pulmonary and Critical Care Medicine, Department of Internal Medicine, Eastern Virginia Medical School, Norfolk, Virginia USA (J.S., A.L.). Revision requested September 23, 2013. Revised manuscript accepted for publication October 28, 2013.

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Abbreviations

CCA, common carotid artery; IVC, inferior vena cava; LVOT, left ventricular outflow tract

doi:10.7863/ultra.33.1.3

Modern resuscitation has changed since the advent of goal-directed therapy. Today, practitioners providing fluid resuscitation are cognizant of the danger associated with volume depletion while being aware of the morbidity of volume overload.[1] Thus, fluid resuscitation must be rapid, precise, and individually tailored to each patient based on reliable data.[2] Critically ill patients have a mixture of intravascular volume depletion, low systemic vascular resistance, and decreased cardiac output, which makes responses to attempts at volume resuscitation difficult to predict. As a result, despite initial attempts at fluid resuscitation, persistent hypotension is common and poses the dilemma of whether the patient should receive additional fluid boluses, a vasopressor, or a positive inotropic agent. Traditionally, resuscitation was guided by static measurements, such as central venous pressure, which was thought to determine the patient's "intravascular volume."[1] These static measurements have been shown to be unreliable predictors of a patient's ability to positively respond to volume expansion.[3]

Clinicians have increasingly relied on fluid responsiveness, defined as an increase in cardiac output by 15% following a 500-mL fluid bolus given over 10 minutes, to guide the resuscitation process. Several minimally invasive methods have been used to determine whether a patient is fluid responsive, including pulse counter analysis,[4] transpulmonary thermodilution,[5] and reactance.[6] All of these methods have shown promise in evaluation of the volume status of septic patients; however, bedside sonography has also emerged as a useful tool for evaluating cardiac function in critically ill patients.[7] The echocardiographic methods described below are entirely noninvasive, provide real-time data, can be taught reasonably quickly, and can be repeated frequently until desirable clinical outcomes are achieved.

This article will review 3 methods aimed at predicting volume responsiveness in critical ill patients: (1) measurement of the caval index; (2) measurement of cardiac output with passive leg raising; and (3) measurement of common carotid artery (CCA) blood flow with passive leg raising.[8–10] These latter techniques enable bedside clinicians to determine changes in blood flow in the left ventricular outflow tract (LVOT) and aorta in response to manipulations of right ventricular preload by passive leg raising, thus predicting the response to a fluid bolus without exposing the patient to potentially harmful hypervolemia.[11]

Technique

Inferior Vena Cava Collapsibility

The inferior vena cava (IVC) diameter should be measured in a supine patient in the sagittal (long-axis) subxyphoid window, making sure to angle the transducer to the patient's right. The technique is performed using a 2–5-MHz phased array transducer. The IVC should be visualized in a longitudinal view at the level of the caval–right atrial junction. The IVC diameter is measured on inspiration and expiration at approximately 1 cm distal to the IVC–hepatic vein junction (Figure 1).[8–10] Muller et al[12] showed that caval collapse of greater than 40% indicated fluid responsiveness with 70% sensitivity and 80% specificity. However, they also showed that collapse of less than 40% had no bearing on the patient's ability to respond to fluid.

Cardiac Output

To date, it appears that the response to passive leg raising seems to hold the most promise for assessing volume responsiveness in a variety of critically ill patients. This maneuver rapidly mobilizes about 300 to 500 mL of blood from the lower limbs to the intrathoracic compartment, thereby increasing right ventricular preload. This increase in right ventricular preload essentially mimics the effects of intravenous fluid boluses but with the patient's own whole blood, without the untoward effects of crystalloid overload. The test consists of first putting the patient's head and upper torso at 45° upright, followed by a flat supine position, and then raising both legs of the supine patient to an angle of 45° in relation to the bed, while measuring the stroke volume and cardiac output before and directly after the maneuver (Figure 2). It should be noted that it sometimes takes up to 3 minutes to observe an increased stroke volume in patients with decreased cardiac output and high venous pressures. Beyond that exception, the increase in cardiac output is nearly instantaneous.

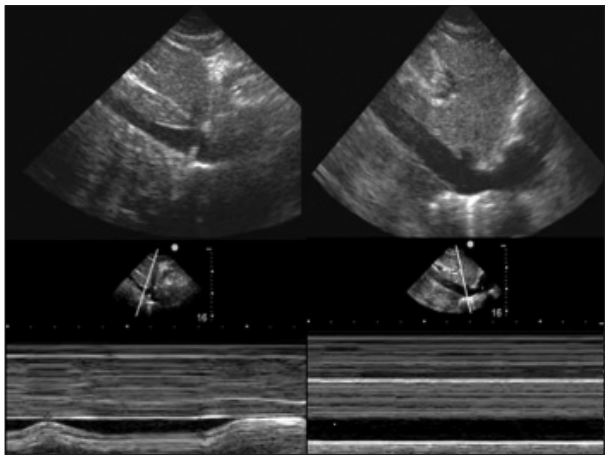
Cardiac output during passive leg raising is measured using pulsed Doppler imaging. The sample volume, with a gate of 3 to 5 mm, is positioned just proximal to the aortic valve (Figures 3 and 4). An optimal signal shows a smooth velocity curve with a narrow velocity range at each time point. The velocity time integral is measured by tracing the modal velocity as shown in Figure 5.

Stroke volume is calculated as the velocity time integral multiplied by $(\text{LVOT diameter}/2)^2 \times \pi$. Cardiac output is stroke volume × heart rate. Percent change is [(cardiac output after passive leg raising – cardiac output before passive leg raising)/cardiac output after passive leg raising] × 100%. A greater than 10% increase in cardiac output would predict volume responsiveness and constitute an indication for a 500-mL fluid bolus. Measurements can be repeated as needed, and fluid resuscitation continues until no further response to passive leg raising is noted. If a patient is still in shock at this point, vasoactive agents or inotropes are usually initiated.

Carotid Artery Flow

Carotid flow is measured during the passive leg raising maneuver by using a linear array transducer positioned in the long axis over the CCA. The CCA diameter is measured from opposing points of the vessel's intimal wall, with the velocity time integral determined automatically using spectral Doppler envelopes and the sample obtained from the center of the artery (Figure 6). Common carotid artery blood flow per minute is calculated by the equation $\pi \times (\text{CCA diameter})^2/4 \times \text{CCA velocity time integral} \times \text{heart rate}$. This parameter is measured both before and after the passive leg raising to determine the percent change in CCA blood flow. An increase in CCA flow with passive leg raising only occurs in patients with shock, and an increase of greater than 65% is highly predictive of volume responsiveness; a disproportionate increase in carotid flow after passive leg raising, when compared to cardiac output, as well as no response to passive leg raising in healthy volunteers can be explained by further redistribution of cardiac output into low-resistance vascular beds in shock states.

Figure 1. Two-dimensional and M-mode images of the IVC. Note the position of the M-mode curser. The image on the left depicts substantial respiratory variations in IVC diameter suggestive of volume responsiveness. The patient on the right is unlikely to positively respond to volume resuscitation.



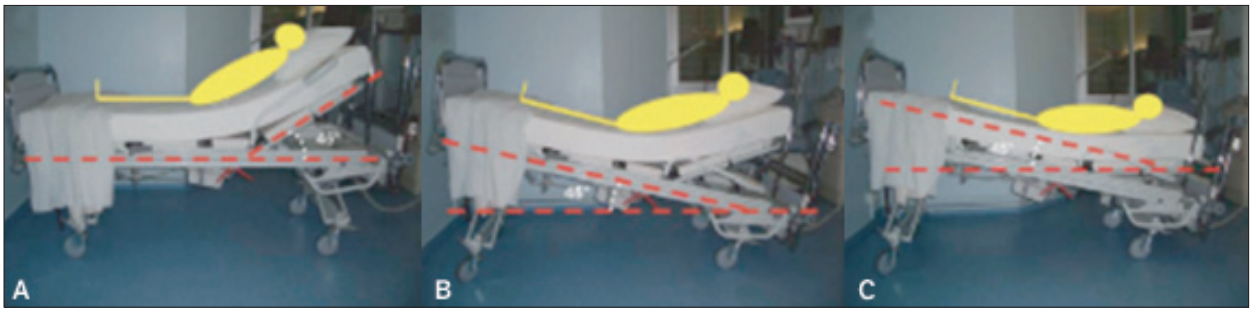


Figure 2. Realization of a passive leg raising maneuver in 3 steps: **A**, at baseline, the patient is lying in a semirecumbent position with the trunk at 45° up from the horizontal position; **B**, the entire bed is pivoted to obtain a head-down tilt at 45°; **C**, the head of the bed is adjusted to obtain a strictly horizontal position. Reproduced with permission from Levitov A, Mayo P, Slonim A. *Critical Care Ultrasonography*. New York, NY: McGraw-Hill Professional; 2012.

Discussion

Inferior vena cava collapsibility can be a useful tool when assessing for fluid responsiveness; however, it is subject to error from extrinsic pathologic conditions such as tamponade, pulmonary hypertension, and tricuspid insufficiency. Using the aortic stroke volume change method, Monnet et al[13] demonstrated that when passive leg raising induced an increase in aortic flow of greater than 10%, it was predictive of an increase in aortic flow of greater than 15% in response to volume expansion (sensitivity, 97%; specificity, 94%). Volume expansion was performed with 500 mL of isotonic saline over 10 minutes; 37 of the 71 patients (52%) included in this study responded to volume expansion. Maizel et al[14] studied 34 spontaneously breathing patients; an increase in cardiac output or stroke volume by greater than 12% during passive leg raising was highly predictive of volume responsiveness. Sensitivity and specificity values were 63% and 89%, respectively. In addition, the study demonstrated that passive leg raising may be used to predict volume responsiveness in patients with atrial fibrillation. Increased intra-abdominal pressure, however, strongly interferes with the ability of passive leg raising to predict fluid responsiveness.[8] Similarly, Guinot et al[15] showed that in patients with acute respiratory distress syndrome receiving extracorporeal membrane oxygenation, those whose stroke volume increased by 15% were shown to be volume responsive with 62% sensitivity and 92% specificity.

The main limitation to the cardiac output method is that it requires advanced skills in echocardiography and can be directly affected by the patient's body habitus. Although it remains the gold standard, CCA flow measurement is a much simpler technique, which can be pre-

Figure 3. The LVOT diameter (D) is measured in the parasternal long-axis view with the calipers at the aortic valve annulus during systole.

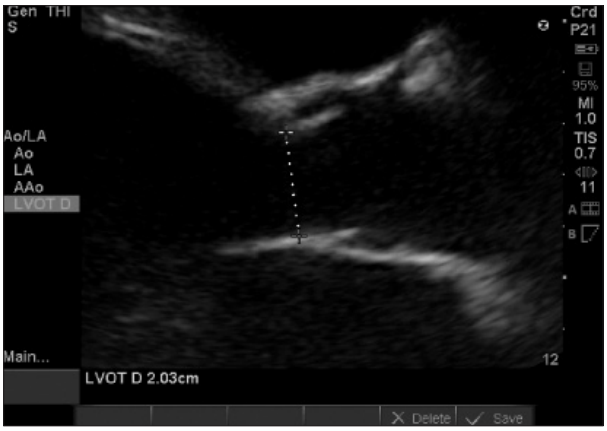
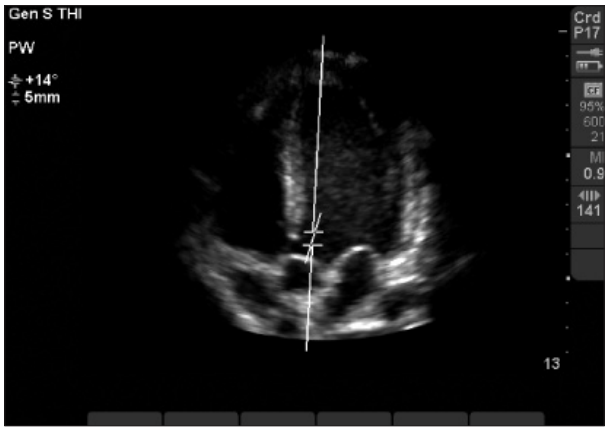


Figure 4. Apical 5-chamber view with spectral Doppler gates set at 5 mm within the LVOT in measure the LVOT velocity time integral.



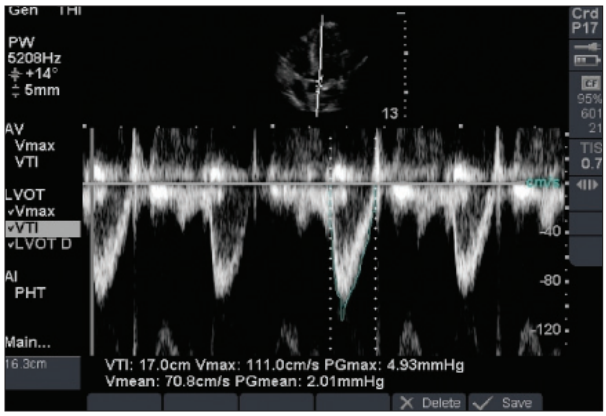
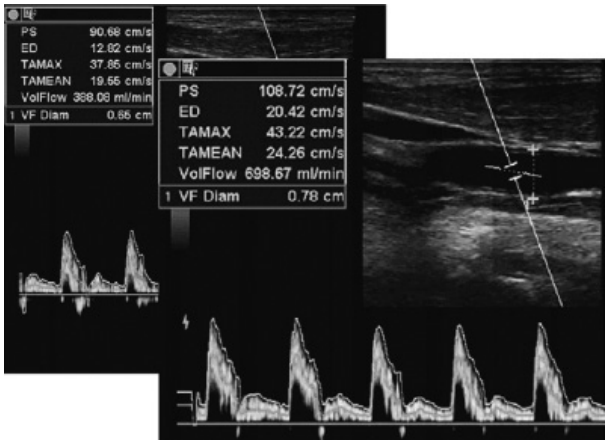


Figure 5. Spectral Doppler tracing of the LVOT velocity time integral (VTI). PGmax indicates maximum pressure gradient; PGmean, mean pressure gradient; Vmax, maximum velocity; and Vmean, mean velocity.

formed with less difficulty than the previously described method. Marik et al[16] recently described this technique, showing a 60% increase in carotid blood flow following passive leg raising, predicting volume responsiveness with 94% sensitivity and 86% specificity.

In conclusion, we believe that according to present evidence, an optimal strategy for assessing volume responsiveness involves first attempting to measure cardiac output via the apical approach; however, if this method proves unsuccessful, the CCA flow method should be attempted.

Figure 6. Carotid artery Doppler flow imaging at baseline (background) and after a passive leg raising maneuver (foreground) in a fluid responder. Carotid blood flow increased by 80%, and the vascular diameter increased by 20%. Note the increase in both systolic and diastolic flow. ED indicates end-diastolic velocity; PS, peak systolic velocity; TAMAX, time-averaged maximum velocity; TAMEAN, time-averaged mean velocity; and VF, volume flow.



Failing this, the provider should attempt to evaluate the IVC for collapsibility. A rigorous, data-driven approach to fluid therapy will undoubtedly continue to be important in the treatment of critically ill patients. Through the use of bedside echocardiography, clinicians can scientifically approach the care of the critically ill patient in shock, bringing confidence to the provider and a better outcome to the patient.

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