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Case Report

# Utilizing left ventricular outflow tract velocity changes to predict fluid responsiveness in septic patients: a case report

**Abstract**

Toxin-mediated vasodilation in the sepsis syndrome can lead to end-organ dysfunction and shock. Assessing for fluid responsiveness and preload optimization with intravenous fluids is a central tenet in the management of sepsis. Aggressive fluid administration can lead to pulmonary edema and heart failure, whereas premature inotropic or vasopressor support can worsen organ perfusion. Inferior vena cava ultrasonography is commonly used to assess for fluid responsiveness but has multiple limitations.

We present a case of a 79-year-old female patient with a history of congestive heart failure who presented with urosepsis. Our alternative method for predicting fluid responsiveness via assessment of velocity changes through the left ventricular outflow tract (LVOT) allowed for successful preload optimization and avoidance of vasopressors and fluid overload.

This is a report of the use of LVOT Doppler measurements in assessing fluid responsiveness in septic patients. This technique can be used when patients present with comorbidities that limit inferior vena cava ultrasonography value. In addition, the reported technique simplifies LVOT velocity measurement without the need to measure cross-sectional area or heart rate.

A 79-year-old woman with a history of congestive heart failure presented to the emergency department with altered mental status. The patient had a temperature of 37.8°C; blood pressure of 100/60; pulse, 102; respiratory rate, 22; and $O_2$ saturation, 98% on room air. Her physical examination revealed clear lung sounds, tachycardia but regular rhythm, and a benign abdomen. Mental status examination revealed deficits of attention and recall, but neurologically, she was nonfocal. A Foley catheterization revealed cloudy urine.

Ultrasonography of her inferior vena cava (IVC) revealed respiratory variation less than 50%, and her left ventricular systolic function exhibited moderate depression. Although the patient had severe sepsis, based on traditional ultrasonographic measurements, she was not fluid responsive [1]. This leads the clinician to a treatment dilemma. A trial of intravenous fluids may precipitate acute decompensation of her heart failure. Conversely, starting vasoactive support when patients are not preload optimized may raise indirect signs of perfusion—such as blood pressure and mean arterial pressure—but via vasoconstriction and increased cardiac contractility and possibly at the expense of worsening end organ perfusion, leading to worsening mental status, coronary ischemia, gut ischemia, or acute kidney injury [2].

We used an alternative method to assess for fluid responsiveness. A phased array probe was placed under her left nipple with the plane pointed toward her right shoulder to obtain an apical 5-chamber view. Pulsed wave Doppler of the left ventricular outflow tract (LVOT) was obtained and showed a change in maximal velocity (ΔVmax) of 25%, indicating that she would likely be fluid responsive (Fig. 1). One liter of normal saline was infused, which normalized her pulse to 85. A repeat ΔVmax of 10% was obtained, indicating that she had been preload optimized. Via this method, we were able to more accurately assess for fluid responsiveness in a patient with comorbidities that limit the usefulness of IVC ultrasound. We were also able to monitor response to therapy and avoid empiric use of fluids or vasopressors.

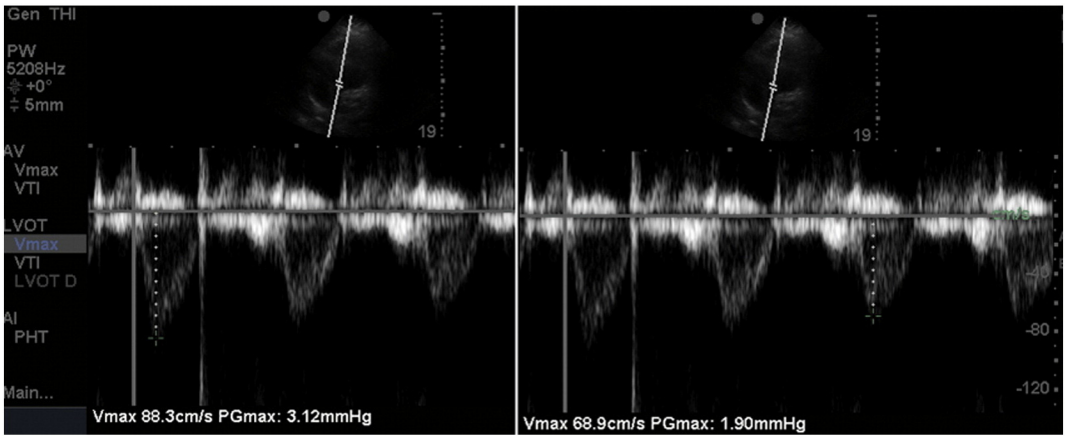


**Fig. 1.** Change in maximal velocity measurement. Maximal velocity is obtained from the greatest and smallest peaks, as seen in the images on the left and right, respectively. Note that the ΔVmax is greater than 12%, and therefore, this patient is fluid responsive.

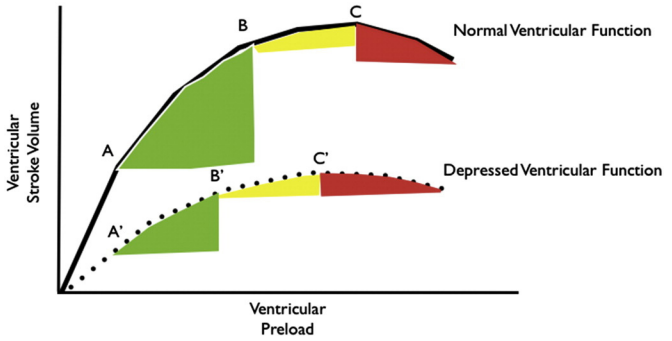


**Fig. 2.** Frank-Starling curve for patients with normal and depressed left ventricular function. The green area under the curves represents a steep part of the Frank-Starling curve where an administration of intravenous fluids leads to a concomitant rise in stroke volume. The yellow area represents a much less rise in stroke volume with administration of fluids, whereas the red area represents a decompensation of ventricular function and development of pulmonary edema associated with fluid administration. Note that, in patients with depressed ventricular function, the green area is much smaller and the red area is much larger than in patients with normal ventricular function.

The sum of all velocities in one cardiac cycle is called the velocity time integral (VTI). The product of the VTI and the cross-sectional area of the LVOT—which is measured on a parasternal long-axis view—yields the stroke volume. A stroke volume variation with respiration of greater than 10% to 15% predicts fluid responsiveness (Fig. 2) [3,4]. Because the cross-sectional area does not change, one can focus solely on the change in velocities over several cardiac cycles. A change in the VTI (ΔVTI) of greater than 20% or a change in maximal velocity (ΔVmax) of greater than 12% is predictive of fluid responsiveness [5]. Alternatively, an eyeball method can be used to grossly discern fluid responsiveness (Fig. 3). Because this method looks directly at the differential velocities of blood leaving the heart, it bypasses many of the limitations associated with both IVC and central venous pressure evaluation.

The initial step is to acquire a 5-chamber apical view of the heart while in cardiac software mode. One reliable method is to obtain a parasternal short-axis view first, then slide the probe to the apex. The apex is apparent on ultrasonography as an obscuration of the left ventricular chamber with only surrounding muscle remaining visible. Once visualized, slowly fan toward the patient's right shoulder. An apical 4-chamber view will initially be seen. By further flattening the sonographic plane to image the base of the heart, the fifth chamber (the LVOT) is imaged (Fig. 4).

The second step is to identify the LVOT. Enter pulse wave Doppler mode and place the sampling gate just proximal to the aortic valve cusps within this area (Fig. 5). Obtain the spectral Doppler tracing by pressing the pulse wave Doppler key a second time, which results in negatively inflected waveforms that indicate flow away from the probe.

After adjustment of the baseline, sweep speed, and scale to be able to visualize many cycles on one screen, one can either visually estimate a qualitative change in the peaks (ie, eyeball) or calculate ΔVTI or ΔVmax. To measure, first identify the largest and smallest peaks over one to two respiratory cycles.

To obtain the ΔVTI, a manual or automatic tracing of the largest and smallest peaks should be performed (Fig. 6). To obtain the ΔVmax, the highest point of the biggest and smallest peaks should be measured. The ΔVTI is the change between the maximum and minimum VTI, divided by the average VTI. The ΔVmax is calculated in similar fashion. A ΔVTI greater than 15% to 20% or a ΔVmax greater than 12% predicts fluid responsiveness.

We have discussed an efficient, alternative technique for assessing fluid responsiveness that emergency physicians can use when IVC ultrasound is not possible or is equivocal in septic patients. Further clinical investigation should be initiated comparing the utility of this method to IVC ultrasound as well as other methods of noninvasive cardiac output monitoring.

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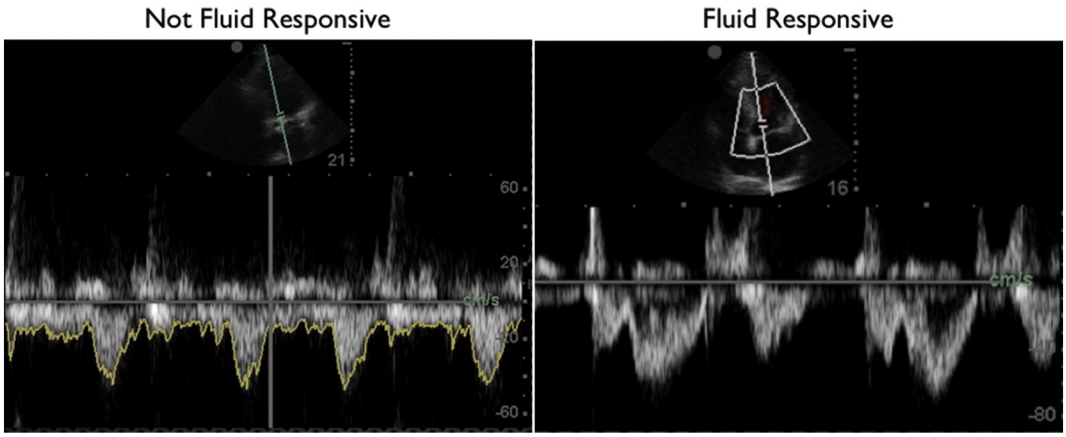


**Fig. 3.** Spectral pulsed-wave Doppler tracings in nonfluid and fluid responsive states. Spectral pulsed-wave Doppler tracings of blood velocities through the LVOT in patients that are not fluid responsive (left) and fluid responsiveness (right). Note that each peak represents a set of velocities for 1 systolic cycle. Nonfluid responsiveness peaks do not vary in velocities with respiratory variation, whereas fluid responsiveness peaks do vary in velocities with respiratory variation.