

Commentary

Brachial artery flow velocity variation: another victory for hand-carried ultrasound?

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See related research by Monge Garcia *et al.*, <http://ccforum.com/content/13/5/R142>

Abstract

Dynamic predictors are clearly superior to static pressures in predicting whether a patient will respond to a fluid bolus. Hand-carried ultrasound (HCUS) can measure changes in blood flow velocity in the brachial artery that parallel arterial pulse pressure variation. The potential for HCUS to guide fluid therapy non-invasively must overcome problems of sensitivity and applicability.

In the past 5 years, hand-carried ultrasound (HCUS) devices have joined the intensivist's toolbox for guiding invasive procedures, for analyzing the pathophysiological basis for circulatory shock, and for predicting fluid responsiveness by revealing inferior vena caval collapsibility. In a recent paper, Monge Garcia and colleagues demonstrated that respiratory-induced variation in brachial artery peak flow velocity ($\Delta V_{\text{peak}_{\text{BA}}}$) could be measured with HCUS and the result predicted the cardiac output response to a fluid challenge [1].

Fluid therapy for critically ill, hemodynamically unstable patients presents clinicians with a dilemma. On the one hand, a fluid bolus may augment cardiac output, improve critical organ perfusion, and even save the patient's life. On the other hand, fluid may confer no hemodynamic benefit, while adding to pulmonary edema, amongst other ills. How often is a fluid bolus harmful, as opposed to helpful? When an intensivist judges that a fluid bolus is necessary, only one-half of patients respond with a meaningful boost in cardiac output [2]. Especially for patients most likely to be harmed (for example, those with concomitant acute lung and kidney injury), knowing whether fluids will enhance perfusion should be clinically valuable.

Historically, clinicians have relied on static hemodynamic parameters, such as the central venous pressure, to judge whether fluids are likely to aid the circulation. A multitude of

studies, accumulating for more than two decades, show that the central venous pressure and its more invasive cousin, the pulmonary artery occlusion (or wedge) pressure, are no more reliable than a coin toss in forecasting whether an individual subject will respond positively to a fluid bolus. When seen in subjects with sepsis [3], with acute respiratory failure [4], or following cardiac surgery [5], this lack of predictive accuracy was attributed to effects of surgery or positive end-expiratory pressure on, for example, ventricular compliance. How disturbing, then, to find that the central venous pressure and pulmonary artery occlusion pressure fail to correlate with ventricular volumes or fluid responsiveness even in healthy normal individuals [6]!

In contrast to the failure of static measures, a novel set of predictors that rely on perturbing the circulation accurately foretell whether fluids will augment cardiac output. These dynamic measures generally employ controlled mechanical ventilation to raise the pleural pressure (some alternatively depend on raising the legs, measuring the effect of spontaneous breathing, or altering the positive end-expiratory pressure) and quite accurately predict fluid responsiveness. In the passive patient, the stroke volume varies with ventilation to a degree that reflects whether the ventricles are operating on the rising or flat portion of the Starling function curve. Patients whose circulations can respond to fluids will therefore demonstrate substantially greater cyclical variability in stroke volume. As the stroke volume changes, so vary the systolic pressure [7], the pulse pressure [7], and the aortic blood flow velocity [8]. Similar cardiopulmonary interactions explain that changes in the diameter of the inferior vena cava also predict fluid responsiveness [9,10]. At the same time, dynamic predictors have limitations: a regular cardiac rhythm is required; most predictors rely on a fully passive patient ventilated with a tidal volume higher than that usually

$\Delta V_{\text{peak}_{\text{BA}}}$ = variation in brachial artery peak flow velocity; HCUS = hand-carried ultrasound.

accepted (8 to 10 cm³/kg); and many of the measures are invasive, requiring, for example, an arterial catheter.

We have previously shown that $\Delta V_{\text{peak}_{\text{BA}}}$ measured by HCUS mirrors the respiratory changes in arterial blood pressure transduced through a radial artery catheter [11], and suggested that this might serve as a non-invasive parameter for gauging fluid responsiveness. Monge Garcia and colleagues have confirmed and extended this work by directly correlating $\Delta V_{\text{peak}_{\text{BA}}}$ to the impact of a fluid challenge [1]. Subjects had a regular cardiac rhythm, lacked respiratory efforts (assured by examination of ventilator waveforms and, if signs of effort were seen, by neuromuscular blockade), were ventilated with tidal volumes of 8 to 10 cm³/kg ideal body weight, and were judged fluid responsive if the stroke volume index increased by at least 15% after 500 cm³ colloid. The primary finding was that $\Delta V_{\text{peak}_{\text{BA}}} > 10\%$ predicted fluid responsiveness with a sensitivity of 74% and a specificity of 95%.

Three additional findings deserve comment. First, as others have shown, radial artery pulse pressure variation quite accurately predicted the response to fluid, with a value $> 10\%$ being both sensitive and specific (95% and 95%). Second, the mean arterial blood pressure increased 13 mmHg in the nonresponders, confirming that this simple vital sign cannot serve as a surrogate for changes in perfusion. Finally, the central venous pressure performed poorly (area under the receiver operator characteristic curve only 0.64).

Dynamic predictors (especially pulse pressure variation) are clearly superior to static pressures, but the role of $\Delta V_{\text{peak}_{\text{BA}}}$ is less certain. First, a screening test demands high sensitivity (not specificity). For the clinician to withhold a fluid challenge, the predictor must identify nearly all patients capable of responding, otherwise too many patients will be denied a potentially life-saving therapy. A sensitivity of 74% does not meet this test. Second, a rapid, non-invasive monitor such as HCUS might have greatest application in the field or very early in resuscitation, before invasive lines are placed. Yet the risk–benefit dilemma posed by fluid bolus is rare in the field – renal failure is not established and the likelihood of responding to fluid is surely much higher than the 50% range typical of intensive care unit patients.

While the study of Monge Garcia and colleagues corroborates the view that fluid responsiveness is best predicted dynamically, further work is needed before $\Delta V_{\text{peak}_{\text{BA}}}$ finds a role in clinical practice.

Competing interests

The author declares that they have no competing interests.

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