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Is Brachial Artery Peak Velocity Variation Ready for Prime Time?

Atul Malhotra, MD, Matthias Eikermann, MD, and Sheldon Magder, MD

Dr. Malhotra is Assistant Professor of Medicine, and Dr. Eikermann is Visiting Assistant Professor of Medicine, Divisions of Pulmonary/Critical Care and Sleep Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA. Dr. Magder is Professor of Medicine and Physiology, Division of Critical Care, McGill University, McGill University Health Centre, Montreal, QC, Canada

The assessment of intravascular volume status is important in the ICU, since adequate resuscitation is critical for optimizing tissue energy supply, whereas excessive fluid may worsen outcome.^{1,2} Rather than a single variable, detailed knowledge of a patient's underlying physiology and careful consideration of concomitant organ function must be put together into the whole clinical picture to determine if a patient needs fluid.

One variable that is crucial for determining who needs fluid is whether or not the patient can respond with an increased cardiac output to fluid infusion. While traditionally central venous pressure (CVP) and pulmonary artery occlusion pressure (wedge) have been used by clinicians to determine whether or not to administer fluid, numerous studies^{3,4} have shown that cardiac filling pressures in isolation are often misleading when used to predict the effects of volume expansion on cardiac output.

There is increasing interest in the use of respiratory variations in vascular pressures (CVP, arterial and wedge pressure) to predict the cardiac output response to a fluid challenge. In patients receiving mechanical ventilation with *no* spontaneous efforts, continuous monitoring consistently demonstrates an early inspiratory rise in arterial pressure⁵ ("reversed pulsus paradoxus"⁶), which is followed during late inspiration and expiration by a decrease in systolic pressure. Perel and Segal⁵ defined the difference between maximum and minimum arterial systolic pressure during a respiratory cycle as *systolic pressure variation*. The inspiratory increase in pressure relative to the value at end-expiration was called *dUp*, and the fall in pressure relative to the end-expiratory value was called *dDown*. While *dUp* reflects a direct mechanical effect of positive pressure on the ventricle, *dDown* is an index of preload reserve. The larger the *dDown* and pressure variation, the greater the predicted increase in cardiac output with volume loading. Based on similar considerations on the interplay between respiration and stroke volume, other variables such as stroke volume variation (SVV),⁴ which can be measured by the commercially available PiCCO system (PiCCO *plus*; Pulsion Medical Systems; Munich, Germany), or aortic blood velocity variation (ABVV)³ have been shown to predict the hemodynamic effects of volume expansion on cardiac output as well. Basically, all of these new techniques (pulse pressure variation [PPV], SVV, ABVV) are predicated on the observation that the magnitude of the cyclic variation in stroke volume depends on whether the patient is operating on the steep or the flat portion of the Frank-Starling curve; that is, whether the heart is preload responsive or not.

Correspondence to: Matthias Eikermann, MD, Visiting Assistant Professor of Medicine, Divisions of Pulmonary/Critical Care and Sleep Medicine, 75 Francis St, Boston MA, 02115; e-mail: meikermann@rics.bwh.harvard.edu.

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While the superiority of respiratory variations of dynamic hemodynamic variables over static indicators in predicting fluid responsiveness has been demonstrated, several physiologic aspects must be considered to avoid misinterpretations. Breathing affects left ventricular (LV) stroke volume by changing independently LV filling and emptying.⁷⁻⁹ Positive pressure in the thorax decreases preload and increases afterload on the right ventricle (RV).¹⁰ The accompanied inspiratory decrease in RV stroke volume during positive pressure ventilation decreases subsequent LV preload and stroke volume, usually reaching its nadir during expiration. Conversely, during rapid intrathoracic pressure changes, LV stroke volume usually increases with pleural pressure.⁹ The early inspiratory increase in LV stroke volume observed during PPV may be the consequence of enhanced pulmonary venous return, increased LV compliance because of decreased RV dimensions, decreased LV afterload, and/or external pressure on the LV.

Aortic elastance is an important variable that directly determines aortic pressure and flow. Elastance of the aorta is curvilinear, so that change in pressure for a change in volume positively correlates with initial aortic volume. Thus, blood that enters at a high initial aortic volume will produce a larger pulse pressure than if it enters at a lower initial volume. Aortic elastance varies with age and disease as does the relationship of stroke volume to pulse pressure.¹¹ For a given cardiac output, stroke volume also varies with heart rate and therefore so will pulse pressure.¹²

Thus, the relationship of stroke volume to pulse pressure differs widely between and within patients, and this greatly limits quantitative predictions. The variability of pulse pressure responses to a given stimulus should be particularly high in the population of critically ill patients, since several variables that affect stroke volume and aortic elastance (heart rate, chest wall compliance, or treatment with vaso-active drugs) vary considerably between critically ill patients. Notably, the predictive value of variables derived from stroke volume on volume responsiveness is valid only when patients have regular rhythm, do not have any spontaneous inspiratory or expiratory efforts,¹³⁻¹⁵ and are receiving ventilation with same parameters as used in original studies. Deep sedation and/or neuromuscular blockade are therefore required to avoid spontaneous ventilation-related artifacts.

Nevertheless, data indicate that PPV and aortic blood flow variation are sensitive parameters for qualitative prediction of the response to volume infusion in patients receiving mechanical ventilation.¹¹ In this issue of *CHEST* (see page 1301), Brennan and coworkers¹⁶ compared the correlation between brachial artery velocity variation (BAVV) and radial artery PPV and found that these variables correlate quite well. This observation is of interest because assessment of BAVV is a noninvasive approach, and the authors report that minimal training is required to accomplish reliable BAVV measurements. If it would be possible to develop a device that allows continuous measurement of respiratory changes in brachial artery blood flow, resuscitation with fluid infusions could potentially be guided by using noninvasive techniques.

However, the study of Brennan et al¹⁶ has some limitations. Firstly, the authors did not compare BAVV, their proposed measure of volume responsiveness, to a clinically relevant end point such as cardiac output or end-organ perfusion, and did not assess actual response of BAVV to volume challenge. Thus, although the correlation between brachial artery peak velocity variation and PPV is interesting, one could argue that the authors are assessing one surrogate marker for another surrogate. Secondly, the authors took their cutoff point used for comparison (PPV of 13%) from a study of patients with sepsis. However, because pulmonary and vascular compliance affect PPV, the 13% cutoff may not be widely generalizable. Thirdly, Bland-Altman analysis revealed that clinically meaningful variation does exist between BAVV and PPV for individual patients. Thus, whether specific patients would be mismanaged based

on BAVV is unclear. Finally, the premise that noninvasive measures are preferable to invasive ones is certainly true; however, patients who require heavy sedation and/or paralysis should probably receive an arterial line. Therefore, the applicability of this noninvasive technique could be questioned.

We should use lessons from the past and not waste the clinical value of “new” parameters by improper use. The pulmonary artery catheter has been used for cardiac output measurement for > 30 years, and a study¹⁷ suggests that physicians’ knowledge in this area is still inadequate. In addition, the end point volume responsiveness *per se* is problematic, since such studies fail to identify patients who need fluid as opposed to those who may respond to fluid.

To evaluate if BAVV as a viable method for managing ICU patients, effects of BAVV-guided volume challenges on clinical outcome must be rigorously studied. Brennan et al¹⁶ are commended on taking the first step.

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