

COMMENTARY

Defining the boundaries of bedside pulse contour analysis: dynamic arterial elastance

Michael R Pinsky

See related research by García *et al.*, <http://ccforum.com/content/15/1/R15>

Abstract

Assessment of vasomotor tone is essential in defining appropriate resuscitation strategies for the hypotensive patient. Although changes in mean arterial pressure to cardiac output define arterial resistance, resistance is only one component of vasomotor tone. Compliance is the other component. The reciprocal of compliance is arterial elastance (E_a). Importantly, dynamic E_a can be estimated by the pulse pressure variation to stroke volume variation relation. Dynamic E_a is only one component of vasomotor tone, however, and increases in pulse pressure may not be proportional to increases in mean arterial pressure. Also, devices that use the arterial pressure pulse to calculate the stroke volume have an inherent bias that is different amongst devices based on their transfer function algorithms. The use of dynamic E_a for clinical decision-making thus needs to be validated separately for different devices and types of patients.

Resuscitation from hypotensive circulatory shock is often more complex than just giving intravascular fluids. Even in fluid-responsive hypotensive patients, vital organ perfusion may remain compromised despite fluid infusion if the mean arterial pressure (MAP) does not also increase with increasing cardiac output (CO). García and colleagues suggested an approach that would predict whether the hypotensive patient would increase their MAP in response to intravascular fluid loading [1].

There is a tight correlation between positive-pressure ventilation-induced changes in arterial pulse pressure (PP), called pulse pressure variation (PPV), and fluid responsiveness [2]. PPV is calculated as the ratio of difference between maximum and minimum PP to their

mean as assessed over about 5 breaths or 20 seconds. The greater the PPV, the greater the CO increase. Not surprisingly, subsequent studies showed that similarly calculated stroke volume variation (SVV), when measured independently, also predicted volume responsiveness [3]. Importantly, PP is created by stroke volume (SV) into the central arterial compartment as quantified by a transfer function [4]. If arterial elastance (E_a) and compliance remain constant, then aortic PP will vary directly with SV. If the bedside physician wants to know whether their hypotensive patient will increase their MAP in response to fluid loading, then they need to know two things. First, is the patient volume responsive? If the patient is not responsive, then volume loading will not increase CO. Second, the physician needs to know the patient's vasomotor tone. If the patient has marked vasodilation, as commonly occurs in septic shock, then MAP may not increase in response to fluid loading even if CO does.

To know whether a fluid-responsive patient is also pressure responsive, one needs to assess vasomotor tone. The relation between dynamic changes in MAP to CO defines arterial resistance. But resistance is only one aspect of arterial tone. Vasomotor tone is defined by both the resistance and compliance. The reciprocal of compliance is E_a , which defines the PP/SV relation [5]. Increasing vasomotor tone increases both MAP and PP relative to CO and SV. If one knew both PPV and SVV, their ratio would define a dynamic E_a ($E_{a_{dyn}}$) [6]. Theoretically, MAP and PP should co-vary with changes in CO if the heart rate remains constant. We previously predicted that $PPV/SVV > 0.8$ would define pressure-responsive subjects if CO increased [7].

Relative to this construct, García and colleagues examined the ability of the PPV/SVV ratio, deferred to as $E_{a_{dyn}}$, to predict changes in MAP in 25 hypotensive patients with preserved volume responsiveness (defined as MAP <65 mmHg or systolic blood pressure <90 mmHg and SVV >10%) [1]. Using a standard 500 ml colloid fluid bolus, the authors defined MAP responders as those with a >15% increase in MAP. They found that a baseline $E_{a_{dyn}}$ value >0.89 predicted >15% MAP increase after fluid administration with a sensitivity of 94% (95% confidence

Correspondence: pinskymr@upmc.edu
Department of Critical Care Medicine, University of Pittsburgh, 606 Scaife Hall,
3550 Terrace Street, Pittsburgh, PA 15261, USA

interval = 69.8 to 99.8%) and a specificity of 100% (95% confidence interval = 66.4 to 100%). Their clinically derived Ea_{dyn} threshold of >0.89 is remarkable similar to our >0.8 value based on vascular modeling. Importantly, as long as the PPV and SVV values are great enough to define a slope, this relationship will remain constant and predictive even during spontaneous ventilation and with cardiac arrhythmias because Ea_{dyn} is independent of volume responsiveness. Since all of the commercially available arterial pressure-derived CO monitoring devices report PPV and SVV, this added Ea_{dyn} parameter to define those patients needing vasopressors earlier in their management should have a significant impact on resuscitation efficacy.

Before embracing this approach and these findings totally, caution needs to be used in its routine bedside application. Importantly, Ea_{dyn} is not a direct measure of Ea . Ea_{dyn} is a measure of arterial stiffness, which is itself partially determined by vasomotor tone. Baseline Ea is not constant. Ea is lower in premenopausal women than in age-matched men. Ea increases with age, with the expression of atherosclerosis and with aortic cross-clamping during aortic vascular procedures. For all these conditions, however, if SV increases then PP will also increase. Regrettably, MAP may not increase as much as PP because diastolic pressure may remain constant. Furthermore, PP can be independently increased by increasing left ventricular ejection velocity [6]. To the extent that inotropic agents are being used, one may presume an increase in MAP not realized by increasing CO even if $Ea_{dyn} > 0.89$.

If PPV and SVV are independently measured, then the clinical utility becomes manifest. The above study, however, used an arterial pulse contour estimating method to derive CO and SVV [6], and herein rests a potential problem. Much attention has focused on using arterial pulse contour analysis devices at the bedside to measure CO and SVV from the arterial pulse. Unfortunately, most indwelling arterial catheters sample a more peripheral arterial pressure signal, whose waveform may be altered in unexpected ways as the arterial tone, pulse wave velocity and left ventricular contractility vary. Although this variability in CO estimates can be readily improved by calibration, it will not improve the accuracy of the PPV/SVV relation (that is, Ea_{dyn}). The Ea_{dyn} accuracy is intrinsic to the assumptions used by each device to estimate SVV. This is because PPV can be measured directly and is accurate when compared with PPV measured manually [8].

All arterial pressure-sensing devices that estimate CO do so by assuming a constant Ea . How, then, is it possible for an algorithm that uses arterial pressure to calculate CO to show differing changes in PP relative to SV over time? This is the Achilles' heel of all these pulse contour

monitoring devices. The three major commercially available monitoring devices, however, do not share the same shoe size – the variance amongst these devices to estimate changing CO is significant [9].

García and colleagues used the FloTrac® device to make their Ea_{dyn} measures. The FloTrac® device estimates SVV from the standard deviation of the individual arterial pressure values over single beats averaged over 20 seconds. Assuming the SV variance has a normal distribution, this assumption is valid for calculating SVV. Importantly, SVV as a time-series function may not be normally distributed during atrial fibrillation and with vigorous spontaneous inspiratory efforts. How much error such non-normal distribution would introduce into the SVV calculation is not known, but on theoretical modeling the degree of non-normal distribution would need to be great for it to affect SVV by the standard deviation method.

The other two devices are the PiCCO and the LiDCO. The PiCCO uses a proprietary algorithm based on the ventriculo-arterial coupling transfer function [3] to calculate CO. This CO estimate is averaged over 20 to 30 seconds and is quite accurate [9]. However, since the PiCCO no longer reports individual SV values on a beat-to-beat basis, it is unclear how it derives SVV. The LiDCO uses a simple power transfer function to estimate SV on a beat-to-beat basis and calculates CO from the mean SV values. To the extent that this power transfer function is accurate over time, the SVV estimates should also be accurate. Since this device does not use the pulse contour, the LiDCO remains accurate with dampened arterial pressure signals.

Accordingly, although each device reports PPV and SVV values, the cross-correlation amongst devices based on their different algorithms is poor [9]. Validation of Ea_{dyn} thus needs to be done independently both for each device and for different types of patients before this new bedside parameter is used for clinical decision-making.

Abbreviations

CO, cardiac output; Ea , arterial elastance; Ea_{dyn} , dynamic arterial elastance; MAP, mean arterial pressure; PP, pulse pressure; PPV, pulse pressure variation; SV, stroke volume; SVV, stroke volume variation.

Conflicts of interest

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