The Use of Stroke Volume Variation and Arterial Pressure Based Cardiac Output to Guide Intra-operative Volume Resuscitation in Cardiovascular Surgery

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Hemodynamic monitoring is a central aspect in cardiovascular diagnosis and care. Adequate volume replacement to achieve optimal cardiac performance is a primary goal of hemodynamic management in patients undergoing cardiovascular surgery. However, in only half of such patients, the cardiac output (CO) increases after a fluid challenge and thus only these can be considered as responders to fluid therapy. Therefore, physicians need reliable criteria to distinguish between responders and non-responders to avoid any deleterious consequences of fluid overload.

It has been demonstrated that neither the standard preload indices such as central venous pressure (CVP) or pulmonary capillary wedge pressure (PCWP) nor their changes in response to fluid challenges reflect ventricular end-diastolic volumes in patients receiving a fluid challenge for hemodynamic instability, and are not capable of predicting cardiac response to fluid therapy (1). As an alternative to these static variables, assessment of stroke volume variation (SVV) has been used as a dynamic monitoring modality for guiding fluid therapy in patients receiving mechanical ventilation. Cardiac preload is highly susceptible to changes in intrathoracic pressure induced by mechanical ventilation. As the stroke volume (SV) varies, the systolic arterial pressure and pulse pressure variation (PPV) can be observed. The SVV and PPV are more pronounced during hypovolemia, and it has been shown to reliably predict changes in CO (2).

The assessment of a patient’s CO typically involves the placement of a catheter in the pulmonary artery and performing thermodilution measurement. Although this method is accurate under most clinical conditions and has been well accepted by most clinicians, it is invasive and associated with many complications. Most importantly, it is a measurement of the right heart. Bouchard et al compared right and left ventricular stroke work index with echocardiography-derived indices of left ventricular performance in cardiac surgery patients. Correlations between right and left ventricular stroke work index changes were poor. Thus, there is a significant discrepancy and limited relation between the preload and the left ventricular performance (3). The arterial pressure based cardiac output (APCO) is a CO monitoring system based on the analysis of the systemic arterial pressure wave that does not require pulmonary artery catheterization and most importantly it provides a measurement of the left heart function.

The use of arterial pulse wave to determine the CO has intrigued both scientists and clinicians for decades. But only 1% of physicians in a survey consider the “swings” in blood pressure during respiration as part of their decision-making process regarding volume expansion (4). Only recently this method has been used for commercial development of clinically applied equipment to measure the CO. These devices include the LiDCO system (LiDCO Limited, UK), which requires calibration using lithium dilution; the PICCO system (Pulsion Medical System, Munich, Germany), requiring transpulmonary thermodilution; the Finapres Modelflow system (Finapres Medical Systems, The Netherlands), with which calibration with another means of CO measurement is advisable to achieve acceptable accuracy; and, most recently, Edwards Vigleovo/FloTrac system (Edwards Lifesciences, LLC, USA), which needs no calibration and provides continuous CO, and SVV measurements from the arterial pressure wave (5). Here we use Vigleovo/FloTrac system as an example. The Vigleovo/FloTrac system calculates the arterial pressure using arterial pulsatility, resistance and compliance. It also takes gender, age, height and weight, as well as waveform characteristics, into consideration (6). The calibration constant is recalibrated every 10 minutes, compared to the other methods in which the constant is recalibrated every few hours.

Morgan et al. first reported that mechanical ventilation induces cyclic changes in vena cava blood flow, pulmonary blood flow and aortic blood flow. During the inspiratory period of mechanical ventilation, the vena cava blood flow decreases first, followed by a decrease in pulmonary artery flow and then in aortic blood flow. The decrease in vena cava blood flow has been related both to an increase in right atrial pressure and to the compression of the vena cava due to the inspiratory increase in pleural pressure during mechanical ventilation (7). According to the Frank-Starling mechanism, the inspiratory decrease in right ventricular preload results in a decrease in right ventricular output and pulmonary artery blood flow that finally leads to a decrease in left ventricular filling and output (2).

The left ventricular end-diastolic area (LVEDA) assessed by echocardiography has been considered a better indicator of left ventricular preload than the PCWP and a parameter very sensitive to changes in blood volume (8, 9). Coriat et al. found a significant correlation between the left ventricular diastolic area and the magnitude of systolic pressure variation in aortic surgery patients (10). Hofer et al reported the use of SVV and PPV for prediction of fluid responsiveness in patients undergoing off-pump coronary artery bypass grafting (OPCABG). The authors concluded that SVV and PPV are closely related. SVV and PVV assessment showed comparable good performance in predicting fluid responsiveness in patients with preserved left ventricular function undergoing OPCABG (11). In postoperative patients who have undergone coronary artery bypass grafting, other authors have demonstrated that PPV can be used to predict whether or not volume expansion will increase CO and that PPV is far superior to CVP and PCWP (12,13).

While the usefulness of using SVV and APCO to detect preload sensitivity is indisputable, a number of limitations must be remembered. The dynamic indices cannot be used in a patient who is spontaneously breathing and/or has arrhythmias. Even if the detection of fluid responsiveness is found to be of use in the decision-making process regarding volume expansion, two important points must be kept in mind. First, since both ventricles of a healthy subject operate on the steep portion of the preload/SV relationship, volume responsiveness is a physiological phenomenon related to a normal preload reserve. Therefore, detecting volume responsiveness must not automatically lead to a decision of infusing fluid. Second, it is reasonable to postulate that volume loading should be more beneficial in a hypotensive patient with low CO and volume responsiveness than in a hypotensive patient with a high CO and less degree of volume responsiveness, for whom administration of vasopressors should be more logical. This emphasizes the great interest in new commercially available devices that monitor and display both CO and SVV/PVV together from beat-to-beat analysis of arterial pressure waveform.
In summary, the dynamic indices, such as SVV/PVV and APCO, are superior to static indicators of cardiac preload in predicting the response to fluid administration, but there are some limitations. When used within their limitations, they are sensitive tools that can be used to guide the appropriate management of the patient’s preload to achieve optimal CO.

References:

7. Morgan BC, Martin WE, Hornbein TF, Crawford EW, Guntheroth WG. Hemodynamic effects of intermittent positive pressure ventilation. Anesthesiology 1966; 27: 584-590