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Dynamic concepts of volume responsiveness

Volume expansion is a first line therapy for patients with circulatory failure. However, in only half of such patients is cardiac output increased after a fluid challenge and thus only these can be considered as responders to fluid therapy.[1] Therefore, physicians need reliable criteria to distinguish responder patients who can benefit from fluid administration from non-responder patients for whom fluid therapy can even be harmful.

Markers of preload have been used to help in deciding to which patients to administer fluid. Unfortunately, cardiac filling pressures and cardiac end-diastolic dimensions have been demonstrated to be of poor value in detecting volume responsiveness in critically ill patients.[1] The first explanation is that these parameters are not reliable markers of cardiac preload. For example, ventricular filling pressures measured by invasive catheters can be high, despite low preload in the case of reduced ventricular compliance. The right ventricular end-diastolic volume is poorly assessed with a fast-response thermodilution pulmonary artery catheter, in case of tricuspid regurgitation, a frequent event in critically ill patients with pulmonary hypertension. The left ventricular end-diastolic area measured using echocardiography cannot reflect end-diastolic volume (and thus preload) in the presence of regional wall motion abnormalities, a frequent condition in patients with coronary artery disease.

The second important factor in explaining why the static markers of preload are poor indicators of volume responsiveness is related to the basic physiology. The slope of the Frank-Starling curve (ventricular preload *vs.* stroke volume) depends on systolic cardiac function. In this regard, for the same baseline ventricular preload, the increase in ventricular preload induced by fluid will result in an increase in ventricular stroke volume significantly higher in patients with normal ventricular systolic function than in those with reduced ventricular systolic function. Therefore, even if an accurate measure of preload is available, it will not be possible to use it to predict volume responsiveness reliably.

Dynamic parameters such as systolic pressure variation (SPV) and its delta down component (ΔDown), pulse pressure variation (PPV) and stroke volume variation (SVV) have been recently demonstrated to be good predictors of volume responsiveness in critically ill patients receiving mechanical ventilation.[2–5] In their article, Parry-Jones and Pittman[6] have given a nice description of the rationale and the limitations of using such indices. The rationale for their use is based on the assumption that mechanical insufflation which results in a decrease in cardiac preload will also result in a decrease in stroke volume only when the heart is preload-dependent, according to the Frank-Starling relationship. Therefore, appreciable changes in left ventricular stroke volume will be seen in the case of biventricular preload dependence, while no change in left ventricular stroke volume should occur if at least one of the two ventricles is preload independent. Because a significant response to fluid (in terms of increase in cardiac output) should occur only under biventricular preload-dependent conditions, it has been logically postulated that the magnitude of cyclic changes of stroke volume would correlate with the degree of response to fluid. In patients receiving controlled ventilation, this hypothesis has been confirmed by taking cyclic changes of arterial pulse pressure or the area under the systolic part of a peripheral artery pressure curve as surrogates of cyclic changes of left ventricular stroke volume.[3–5]

The bedside use of dynamic indices using heart-lung interaction may help in the decision-making process concerning volume loading. In a patient with circulatory failure, the presence of a large respiratory variation of surrogates of stroke volume would encourage the physician to decide to give fluid as a first choice rather than cardiovascular drugs. In contrast, for a patient without evidence of respiratory variation of haemodynamic signals, the priority option might be early administration of drugs, thus avoiding the deleterious consequences of useless fluid loading.

While the usefulness of heart-lung interaction to detect preload sensitivity is indisputable, a number of limitations must be remembered. As underlined by Parry-Jones and Pittman,[6] dynamic indices using heart-lung interaction cannot be used in a patient with spontaneous breathing activity and/or with arrhythmias. The influence of tidal volume is also a matter of debate. The magnitude of SVV has been demonstrated to depend on the tidal volume delivered by the ventilator.[7] However, this does not represent a true limitation of the interpretation of large SVV as an indicator of volume responsiveness since increasing the tidal volume should also further decrease venous return and cardiac preload[8] making the response to fluid greater. Theoretically, a very low tidal volume can induce very small changes in intrathoracic pressure and hence low SVV even in the case of low cardiac filling conditions. However, low tidal volumes are generally used in patients with acute respiratory distress syndrome (ARDS) who exhibit major changes in alveolar pressure, such that changes in intrathoracic pressure over the respiratory cycle are not necessarily small in the case of low tidal volumes. Furthermore, it is not definitely established that using low tidal volume (6 ml/lg) ventilation in patients with ARDS is better than using normal tidal volume (8–10 ml/kg) ventilation.[9]

Even if the detection of fluid responsiveness is found to be of use in the decision-making process concerning volume expansion in patients with circulatory shock, two important points must be kept in mind. First, since both ventricles of healthy subjects operate on the steep portion of the preload/stroke volume relationship, volume responsiveness is a physiological phenomenon related to a normal preload reserve. Therefore, detecting volume responsiveness must not systematically lead to a decision of infusing fluid. Such a decision must be based on the presence of signs of cardiovascular compromise and must be balanced with the potential

risk of pulmonary oedema formation and/or worsening gas exchange. Second, it is reasonable to postulate that volume loading should be more beneficial in a hypotensive patient with low cardiac output and volume responsiveness than in a hypotensive patient with a relatively high cardiac output and some degree of volume responsiveness for whom early administration of a vasopressive agent should be more logical. This emphasises the great interest in new commercially available devices that monitor and display both cardiac output and indices of volume responsiveness (PPV, SVV) from beat-to-beat analysis of arterial pressure waveform.

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