

Fluid responsiveness in critically ill patients

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The first therapeutic approach to patients affected by shock is fluids infusion. In particular, patients affected by sepsis usually require a great amount of fluids in the first phase of resuscitation.

Fluids must be considered as other drugs with beneficial but also adverse effects especially in patients with a limited cardiac reserve. For this reason, it is helpful to know, if the patient will respond to fluids. Several studies have shown that hemodynamic parameters classically use to evaluate vascular volumes such as central venous pressure (CVP) and pulmonary artery occlusion pressure (PAOP), are not able to predict the response to fluids administration.^[1] Volumetric parameters such as global end diastolic volume (GEDV) and left ventricular end diastolic volume (LVEDV), are better related to volume status but are not able to accurately predict fluid responsiveness.^[2]

Therefore, several dynamic parameters have been developed during the last years to assess fluid responsiveness. The easiest approach is a fluid challenge. It consists to give a small amount of fluid (250–500 ml of crystalloid in few minutes) and verify patient response in term of increase in cardiac output (CO).^[3] However, also this small amount of fluid could be deleterious in patients with a limited cardiac reserve.

In mechanically ventilated patients, the clinician can use cardiopulmonary interaction to predict patient response to the fluid.^[4] Dynamic parameters such as pulse pressure variation, stroke volume variation, and systolic pressure variation are a better predictor of fluid responsiveness than static pressometric and volumetric parameters such as CVP, PAOP, GEDV, and LVEDV.^[1] Furthermore, several miniinvasive monitoring systems are able to calculate CO and

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stroke volume continuously showing dynamic parameters. ^[5,6] However, these parameters have several limitations and cannot be used in every patient. In fact, a correct interpretation of dynamic parameters requires controlled ventilation with a tidal volume at least of 8 ml/kg, absence of arrhythmias, ventricular dysfunction, intra-abdominal hypertension, and a ratio between heart rate and respiratory rate 3 to 6. All these criteria are difficult to meet in the intensive care setting where we usually apply protective lung ventilation and patients are frequently in spontaneous ventilation.

In these situations, an alternative approach could be the assessment of CO variation after a passive leg raising maneuver, responsible for a shift of small amount of blood from legs to the heart.

Considering these approaches to hemodynamic evaluation, which could be the role for CVP? Do we still need to measure this parameter?

The actual value of CVP is not related to volume status because it is determined by interaction between cardiac

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If blood pressure is low, and CO is normal or elevated, low systemic vascular resistance is responsible for low blood pressure. If the CO is decreased, this can be due to a decrease in cardiac function or a decrease in the venous return. CVP helps to define whether a decrease in cardiac function or a decrease in return function is the primary problem. If the CVP is high, the problem is primarily decreased cardiac function. On the other hand, if the CVP is low, the primary problem is the venous return and providing more volume will probably solve the problem.

CVP is also helpful to evaluate the effect of fluid challenge and to determine the amount of fluid need to perform this test. Sufficient fluid is given when the CVP will be raised by 2 mmHg or more. A concomitant increase in CO indicates that the patient is fluid responsive whereas an increase in CVP without an increase in CO shows that further fluids are not indicated.

During spontaneous ventilation, CVP assessment during an inspiratory fall in pleural pressure is very helpful. According to Guyton's approach, a decrease in pleural pressure makes the pressures in the heart more negative. When the heart functions on the ascending part of the cardiac function curve, this results in a fall in CVP and an increase in the gradient for venous return and, an increase in right heart output. Under this condition, a volume infusion should increase CO. However, when the heart is functioning on the flat part of the cardiac function curve, the fall in pleural pressure does not produce a change in CVP and therefore the gradient for venous return and consequently CO do not change.

Finally, we know that response to fluids may be different if we consider macro-hemodynamic parameters or if we look at the micro-circulatory level. Macro- and micro-hemodynamic are not always coupled, and patients may improve hemodynamic parameters without a concomitant improvement of micro-vascular flow.^[8,9]

Because the capillary network is the site of oxygen delivery to tissue, every therapeutic intervention should aim to improve micro-vascular flow. The evaluation of sublingual micro-circulation is able to predict which patients are really fluid responsive.^[10]

In conclusion, the assessment of fluid responsiveness is very important in the management of critically ill patients. Dynamic parameters derived from heart-lungs interaction are very helpful in this setting, but the intensivist should not forget the important information that classical hemodynamic parameters such as CVP, can give us. The evaluation of sublingual micro-circulation may add useful information in decision making about the fluid administration.

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Conflicts of interest

There are no conflicts of interest.

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