Assessing Volume Status and Fluid Responsiveness in Resuscitation

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Fluid loading is the first step in the resuscitation of hemodynamically unstable patients. Adequate tissue or organ perfusion is the goal because under-resuscitation results in inadequate organ perfusion, while over-resuscitation increases the morbidity and mortality in critically ill patients. The traditional measurements used to assess resuscitation were based on cardiac filling pressures. They were static measurements such as central venous pressure (CVP) and pulmonary artery occlusion pressure. However, these measures are very invasive and have not been shown to predict fluid responsiveness. There are newer, dynamic tests of volume responsiveness based on the principles of the Frank-Starling curve. Each test uses a maneuver that changes the venous return (preload) and then measures the corresponding change in stroke volume as a marker of fluid responsiveness. Tests that assess changes in pulse pressure, stroke volume, and oximetric waveform are used in mechanically ventilated patients, while passive leg raise maneuvers are done in the spontaneously breathing patient. The role of ultrasound in determining fluid responsiveness has also been studied. In the end, the ultimate goal is to assess whether a critically ill patient's stroke volume will be increased by the administration of a fluid bolus.


Central venous pressure measurements are utilized in many intensive care units (ICUs), emergency departments, and operating rooms throughout the world. It is used to assess the fluid status of patients and to aid in decisions regarding administration of fluids or diuretics. This review addressed CVP monitoring and its ability to predict fluid responsiveness. Clinical trials that reported either of the following were included in the study: (1) the correlation between CVP and measured blood volume, or (2) the correlation between CVP or change in CVP and change in stroke index/cardiac output following a fluid challenge. Five of the studies compared CVP with measured blood volume and 19 of the studies assessed changes in CVP and cardiac performance following a fluid challenge. Of the five trials that addressed CVP and its relation to the measured blood volume, the pooled correlation coefficient was 0.16. The pooled correlation coefficient between change in CVP and change in cardiac index (CI) following a fluid challenge was 0.11.

Based on the findings above, Marik et al. concluded that there was no association between CVP and blood volume, and that CVP did not predict fluid responsiveness.


Clinical studies demonstrated that only 50% of hemodynamically unstable critically ill patients are volume-responsive. So, how do we determine which patients should receive fluids? Using the Frank-Starling Principle, as the preload increases, left ventricular stroke volume increases until the optimal preload is achieved at which point the stroke volume remains relatively constant.

In the mechanically ventilated patients, the pulse pressure variation (PPV) using the arterial waveform, the stroke volume variation (SVV) from pulse contour analysis, and the variation of the amplitude of the pulse oximeter plethysmographic waveform have been shown to be HIGHLY PREDICTIVE of fluid responsiveness. These techniques are based on the simple physiology of positive pressure ventilations. Insufflation decreases RV preload, increases RV afterload and these leads to a decreased RV stroke volume, which are at the minimum at the end inspiration. The LV stroke volume is at the minimum at end expiration. The cyclic changes in RV and LV stroke volume are greater...
when the ventricles are on the sloped portion of the Frank-Starling curve. A variation of greater than 12% is highly predictive of volume responsiveness.

The “Pleth Variability Index” (PVI) is an automated measurement of the “Perfusion Index” (PI) which reflects the amplitude of the pulse oximeter waveform. The PVI correlates with the respiratory induced variation and can be used to predict fluid responsiveness noninvasively in mechanically ventilated patients. There are limitations on PVV and PVI, such that tidal volume needs to be at least 8ml/kg (which is the higher end of standard lung protective tidal volumes of 6-8ml/kg) and it cannot be accurately used in patients with arrhythmias or spontaneously breathing.

In ventilated patients, mechanical insufflation with end inspiratory occlusion for 15 seconds can increase arterial pulse pressure and pulse contour cardiac output. An increase of more than 5% accurately predicts fluid responsiveness.

In spontaneously breathing patients, passive leg raise (PLR) is the technique of choice. Blood transferred to the heart during PLR increases cardiac preload and is considered a reversible “autotransfusion,” it serves as a test of preload responsiveness. The response of aortic blood flow measured by esophageal Doppler, velocity time integral measured by transthoracic echocardiography, and femoral artery flow measured by arterial Doppler to PLR, are helpful in predicting the response to volume administration.


Bolus thermodilution has historically been the gold standard for determining cardiac output; however, it is invasive as it requires a pulmonary artery catheter. A technique for estimating cardiac output is the esophageal Doppler technique. A Doppler transducer probe is placed in the esophagus and the cardiac output is calculated based on the diameter of the descending aorta and the aortic blood velocity signal. However, there are several limitations: there is a significant learning curve, poor positioning of the probe tends to underestimate the cardiac output, and it does not allow for continuous monitoring.

Cardiac output can also be estimated using the concept of pulse contour analysis. This method can calculate stroke volume from the arterial pressure waveform as long as both arterial compliance and systemic vascular resistance are known. It does not, however, correct for discrepancies between central and peripheral blood pressures, which can be large in conditions of intense vasoconstriction and can therefore result in a falsely low cardiac output value. Examples of two pulse contour analysis systems are the LiDCO and the PICCO Systems. Both of these systems have been shown to be at least as reliable as intermittent pulmonary artery catheter thermodilution. The FloTrac System is an example of a pulse contour analysis that does not require calibration, but instead estimates a patient’s vascular compliance based on variables such as sex, age, height, and weight. The system is operator independent and only requires a peripheral arterial catheter. Unfortunately, it has shown poor agreement when compared to intermittent thermodilution and has not been shown to accurately trend changes in stroke volume after a volume challenge or with vasopressors.

Thoracic Electrical Bioimpedance (TEB) has also been used to calculate stroke volume. A high-frequency electric current of known amplitude and frequency is applied across the thorax and the voltage of the returning signal is measured and compared to that of the injected signal. The ratio between voltage and current amplitudes is a measure of transthoracic impedance, which varies in proportion to the amount of fluid in the thorax. These bioimpedance values have shown poor agreement and are sensitive to placement of electrodes, lung edema, and body size, so are not ready for prime-time use.

Another noninvasive example is the NICOM device, which estimates stroke volume by measuring the bioreactance across the thorax. Bioreactance refers to a phase shift in voltage, which can only occur from pulsatile flow. In the thorax, the overwhelming majority of pulsatile flow is from the aorta. Therefore, the NICOM device can estimate CO by measuring bioreactance. Multiple studies have shown a high correlation between CO measured by bioreactance to that measured by thermodilution and pulse contour analysis. In fact, one study by Raval et al. showed the NICOM system to be more precise than thermodilution in accurately tracking changes in CO.

Conclusion

Fluid administration has been a key component in the resuscitation of critically ill, hemodynamically unstable patients. Various methods for assessing volume status and fluid responsiveness have developed over time.

Static measures to predict fluid responsiveness include CVP, PAOP, RVEDVI, LVEDA, and GEDV; which have had poor to moderate accuracy in determining fluid responsiveness.

In contrast to static preload measures, which rely on hemodynamic values at a given point in time, dynamic predictors have increasingly gained favor for predicting fluid responsiveness. PPV and SPV have been found to be superior to several static measures of fluid responsiveness such as LVEDA and PAOP.

Other dynamic measures include peak aortic blood flow velocity variation (which is measured by TEE), measurement of respiratory variation in IVC diameter, and PLR, which have all shown to be good predictors of fluid responsiveness.

Cardiac output can be estimated using the concept of pulse contour analysis. These devices include the LiDCO system, the PICCO system, the FloTrac System, and the MostCare system.

The NICOM device is noninvasive and estimates the SV by measuring the bioreactance across the thorax. In the thorax, the majority of pulsatile flow is from the aorta which allows the device to estimate the CO.

Additional References: