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REVIEW

Inferior vena cava guided fluid resuscitation – Fact or fiction?

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SUMMARY

Bedside ultrasound assessment of critically ill patients is gaining popularity and importance. Ultrasound assessment of the inferior vena cava has been suggested to be helpful in guiding fluid resuscitation. Although numerous studies have been performed a consensus has not been reached and expert opinions are divided. This review aims to further the reader's understanding and ability to navigate the controversies surrounding this modality in the care of the critically ill.

1. Introduction

The care of the critically ill patient involves rapid diagnosis, targeted treatment of underlying pathology while supporting organ function and avoiding iatrogenic harm.[1] One of the tenets of organ support is the optimization of cardiovascular function. This has led to extensive critical care research in the field of hemodynamics and fluid intervention. One of the most influential scientific works in hemodynamic manipulation is attributed to Frank, Starling[2] and several others.[3] The fundamentals of the cardiovascular function can be simplified into three components of pre-load, contractility and after-load of the cardiac ventricles. Assuming constant ventricular compliance, heart rate and after-load, an increase in pre-load by fluid administration results in an increase in ventricular end diastolic volume (EDV). This, in turn, increases ventricular contractility and stroke volume, thus, augmenting cardiac output. Patients whose cardiac output responds in this fashion are described as being "fluid responsive". However, this relationship between contractility and EDV is not linear and decreases with increasing EDV, resulting in "fluid unresponsive or intolerant" states (Fig. 1). Mounting evidence suggests that excessive fluid administration in both paediatric[4,5] and adult[6,7] critically ill patients results in poorer outcomes and increased mortality. There is impetus to find reliable means to avoid this.

Technological advances and greater affordability have resulted in increased enthusiasm for the use of portable ultrasound (US) machines in the Intensive Care Unit (ICU) to guide therapy. It is regarded as a safe, rapid, non-invasive and repeatable modality. US assessment of the inferior vena cava (IVC) to estimate the right ventricle (RV) function was first described in the field of cardiology in 1981.[8] As the IVC is compliant and distends with increased intra-luminal pressure, investigators discovered that patients with elevated Right Atrial Pressures (RAP) had correspondingly dilated IVC. Kircher et al.[9] subsequently demonstrated a positive correlation between IVC US parameters and the RAP measured via central catheters. Although methodological criticism of delayed RAP measurement in that study exists, it is often cited as evidence to support the use of IVC measurements as a surrogate for RAP and thus, to determine clinical decisions and fluid management.

Many investigators have attempted to validate the use of IVC US parameters in critically ill patients, but conclusions are conflicting and medical opinions are not unanimous in endorsement of its use to guide fluid resuscitation.[10–12] This review aims to assist the reader's understanding of the US derived IVC parameters and help navigate controversies of this modality in the care of the critically ill.

2. Commonly measured IVC parameters

The IVC can be assessed at a depth of 6–15 cm via subcostal or transhepatic US windows using either linear or phase array US probes in the majority of patients.

Depending on patient characteristics, respiratory cycle and US technique, the diameter of normal adult IVCs varies between being totally collapsed to about 2.1 cm. Patients in obvious fluid overload states, such as in cardiac failure,[13] have been described to have IVCs

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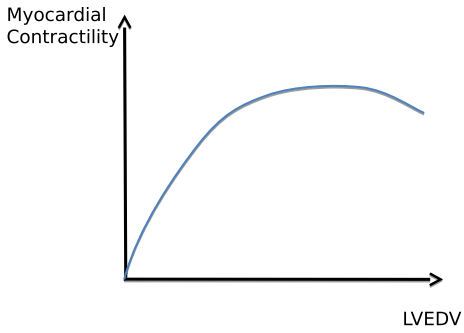

Fig. 1. The relationship between myocardial contractility and left ventricular end diastolic volume.

measuring 2.5 cm or more. Studies in hypovolemic trauma patients,[14,15] on the other hand, show maximum IVC diameters of less than about 0.9 cm.

Inter-individual variability of IVC sizes limits predictive value of specific cut-offs. As such, IVC collapsibility index (IVC–CI) is a percentage of the maximum IVC diameter. An IVC-CI of more than 40–50%[9,17,29] in spontaneously ventilated patients or more than 12–18%[18,19] in mechanically ventilated patients have been described as cutoffs indicating hypovolemia.

Another described IVC parameter is the IVC-CI during a "sniff test". The "sniff test" involves the patient taking a short and sharp inspiration while clinician assesses the IVC collapsibility.[20] Normally, exaggerated collapse of the IVC is observed but patients with high intravascular volume are observed to have reduced IVC collapse during this maneuver. This purportedly allows estimation of RAP and identification of fluid overloaded states (Table 1).[29]

Validation studies in children are currently limited but it was recently suggested that IVC parameters might be indexed to body surface area (BSA) to allow comparison within the paediatric population.[21]

3. Factors affecting IVC parameters

Changes in IVC parameters result from interaction between intra- and extra-luminal pressures. Conceptually, it can also be considered to result from interactions between intra-thoracic and intra-abdominal pressures (IAP). Collapse of the IVC can result from a reduction of RAP or CVP, an increase in IAP, or a combination of both. Although empiric observations suggest changes in IVC parameters reflect intravascular blood volume, there are important caveats, which can be considered in four categories:

3.1. Factors affecting intra-luminal pressure

3.1.1. Right ventricular compliance

The understanding of the RV lacks behind that of the left ventricle (LV), as it is difficult to study. While LV diastolic dysfunction is progressively well characterized, less is determined of RV diastolic dysfunction. RV diastolic dysfunction impedes venous return and results in elevated pressures, possibly translating into greater IVC distension despite lower intravascular volumes.

Yu et al.[22] found a close correlation between individual RV and LV diastolic parameters, suggesting that both often co-exist. More than one third of critically ill patients were reported to have LV diastolic dysfunction on admission,[23] with an additional one third developing the reversible condition at least once during the ICU stay.[24] This may mean high prevalence of reversible RV diastolic dysfunction, making US derived IVC parameters less reliable.

3.1.2. Tricuspid valve disease

Normally, the RV generates 15–30 mm Hg of pressure in systole while the pressure in the RA remains between 0 and 5 mm Hg. The tricuspid valve (TV) protects the RA against the RV pressures. If the TV becomes incompetent due to valve leaflet abnormalities, found in infective endocarditis or connective tissue disease, or due to annular dilatation, secondary to dilated or septic cardiomyopathy, tricuspid regurgitation (TR) occurs. RAP consequently increases and may result in a dilated IVC. Tricuspid stenosis is less common but may also elevate RAP independent of volume status. In both instances of TV disease, IVC parameters may not accurately reflect intravascular volume status.

3.1.3. Obstruction distal to the right atrium

Distal blood flow obstruction may occur within the heart or in the lungs. Intra-cardiac causes may include rare primary intracardiac tumors, congenital heart diseases, such as Tetralogy of Fallot, or primary pulmonary valvular disease. Tumor extension into the RA via the IVC may also occur in cases of advanced renal cell carcinoma.[25] Pulmonary hypertension can result from lung parenchymal disease, such as chronic obstructive lung disease and interstitial lung disease, or from acute or chronic embolism of deep vein thrombosis. Again, the IVC parameters may overestimate volume status.

3.1.4. Blood flow diversion

The majority of blood from the lower extremity returns to the right atrium (RA) via the IVC, while a smaller portion returns via the azygous system. However, pathological states can result in portosystemic shunts providing alternative pathways of venous return, for example, in patients with advanced liver cirrhosis or Budd-Chari Syndrome. The relationship between IVC parameters and volume status in such situations has not been well validated.

3.2. Extra-luminal factors

3.2.1. Intra-thoracic pressure

Tension pneumothorax results in increased intra-thoracic pressure and prevents venous return. This confounds the situation by manifesting as a falsely distended IVC. Respiratory mechanics differ between spontaneously breathing and mechanically ventilated patients and need to be considered separately.

3.2.1.1. Spontaneous ventilation. IVC parameters are altered by respiratory efforts during spontaneous ventilation as illustrated by the sniff test. Increased respiratory effort by the critically ill patient due to distress from underlying cardiopulmonary pathology, such as asthma or chronic obstructive lung disease, metabolic acidosis or systemic inflammatory response syndrome (SIRS) will result in spurious IVC measurements.[10]

Besides respiratory effort, the respiratory pattern has been shown to alter IVC parameters as well. Kimura et al.[26] studied the effects of abdominal and thoracic breathing patterns on IVC parameters within the same study subjects and found significant differences. This is possibly due to compression of the IVC during diaphragmatic excursion. As it is near impossible to standardize patient breathing effort or pattern, the validity of the relationship of US IVC parameters to intravascular volume status of spontaneously breathing patients is naturally tenuous.

3.2.1.2. Mechanical ventilation. During mechanical ventilation, positive intra-thoracic pressure is applied for inspiration, while