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What every intensivist should know about the IVC

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ABSTRACT

Assessment of the IVC by point-of-care ultrasound in the context of resuscitation has been a controversial topic in the last decades. Most of the focus had been on its use as a surrogate marker for fluid responsiveness, with results being equivocal. We review its important anatomical aspects as well as the physiological rationale behind ultrasound assessment and propose a new way to do so, as well as explain its central role in the concept of fluid tolerance.

1. Introduction

With the gradual adoption of Point-of-Care Ultrasound (POCUS) over the last two decades, the inferior vena cava has maintained its place as a controversial tool for the assessment of volume status in the critically ill. Initially prompted by promising studies suggesting a role in assessing volume responsiveness using the superior vena cava, a rash of studies using respiratory variation were done, with equivocal results and less clinician enthusiasm for its use [1,2]. More recently, the focus has begun to shift towards assessing venous congestion and the important concept of fluid tolerance, prompting a resurgence of interest in the IVC.

It is important to understand that the appearance of the IVC, by its anatomical position as the last transit of (lower body) venous return to the right atrium, reflects the interplay between external compressive pressure of the surrounding abdomen that will tend to collapse the IVC, and the internal distending pressures inside the IVC that keeps it open. While the external compressive forces are relatively static, the internal pressure of the IVC is very dynamic and is the result of the combination of factors promoting venous return to the heart, right heart function, and intrathoracic pressures relative to the intra-abdominal pressures. Venous return to the heart is driven by the mean systemic filling pressure (Pmsf), which is generated by the elastic recoil, of the venous vessels and is impeded by the central venous pressure (CVP), which rises as the right ventricle becomes overloaded. It is also key to note that this reflection also includes interplay over time, as venous structures are adaptive to chronic alterations in flow, pressure, or both.

2. The IVC and “Volume Status”

These factors make the goal of using the IVC as a simple fuel gauge to measure the fullness of the tank of highly dubious value. For instance, a patient with a normal intravascular volume who suffers a vasodilatory insult may have a very small IVC, as venodilation will drop Pmsf, decreasing venous return, and arterial vasodilation will decrease afterload and may prompt a drop in CVP. Both changes (decreased Pmsf and CVP) will decrease the size of the IVC – with no change in blood volume. Conversely, a patient suffering from an obstructive pathology raising CVP will increase the IVC size, again without a change in total blood volume.

3. The IVC and Fluid Responsiveness

Increasing cardiac output is the parameter that gave initial appeal to IVC POCUS and reflects the predominant focus of the past decades on forward flow among resuscitators, who largely used a fluid responsiveness strategy, consisting of seeking to identify the patients whose cardiac output would increase with fluids, and administer it until hemodynamics corrected or responsiveness stopped. Not surprisingly, the IVC was no better than the CVP at predicting volume responsiveness, and once this became clear, following a number of studies, the verdict on the IVC as a resuscitation tool was that it was not reliable [2]. This, unfortunately, is somewhat akin to throwing the baby out with the bathwater, particularly since it had been clearly established in the 80's

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and 90's that maximizing cardiac output as a way to provide supra-normal systemic oxygen delivery is generally a harmful strategy [3]. Indeed, the physiology found on the flat part of the Frank-Starling curve is exclusively a pathological one.

4. The IVC and Fluid Tolerance

Recently a position statement was put forward on the concept of fluid tolerance, in an attempt to bring attention to what could be a more personalised and physiological approach to resuscitation and de-resuscitation [6]. Fluid tolerance refers to the patient's capacity to receive volume without suffering from its adverse effects. This is a distinct concept and often overlaps with fluid responsiveness rather than intolerance only beginning where responsiveness ends. (Fig. 1).

Hence, given the physiological relationships of the IVC, systemic congestion begins when venous pressures exceed maximally tolerated tissue afterload. This may also vary with local capillary permeability. While this can happen to inflamed tissue beds with almost any venous pressure, normal tissue will also start to suffer from a decreased effective perfusion as CVP rises beyond a certain point. It should be noted that driving pressure for flow across the capillary is not the MAP minus the CVP, but rather the precapillary pressure minus the postcapillary pressure. The precapillary pressure is much lower than the MAP as there is a large pressure drop across the precapillary resistors. This makes the reduction to flow contributed by the postcapillary venous pressures much more important than is generally recognized. Furthermore, because the precapillary arterial resistance is so much higher than the venous resistance, a rise in venous pressure affects intracapillary pressure much more than a rise in arterial pressure. A rise in intracapillary

pressure promotes capillary hyperfiltration and the development of interstitial edema—particularly when CVP is high as it is the “after-loading” force that impedes lymphatic return. Vellinga et al. showed using microcirculatory parameters that a CVP above 12 cm/H₂O generally worsened microvascular perfusion [8]. We can thus see how splanchnic congestion begins with distension of the IVC. This was elegantly illustrated with the development of the venous excess ultrasound score (VExUS) by the group of Beaubien-Souligny, where there is a gradual increase in organ dysfunction when Doppler envelope abnormalities progress beyond the finding of a lone plethoric IVC. In the absence of this plethoric IVC, there seems to be much less risk of congestive dysfunction as measured by acute kidney injury [3]. So, while macrocirculation may improve with fluids, a worsening in microcirculatory parameters indicates a lack of hemodynamic coherence, suggesting an inappropriate therapeutic strategy.

5. The IVC in Specific Clinical Scenarios

It is important to highlight that in certain scenarios, the pathophysiology will cause a plethoric IVC, and the absence of such, in fact, can essentially rule these out as the principal mechanism of hemodynamic instability. Two of these three scenarios are essentially similar, causing obstructive shock: tamponade and tension pneumothorax. In both of these cases, shock results when intrapericardial and intrathoracic pressures, respectively, exceed right atrial pressure and restrict venous return. These are the only clinical cases where - at least temporarily until definitive decompression is achieved - fluids are indicated in presence of a full IVC. One should differentiate a general tamponade due to a circumferential effusion from a localised tamponade

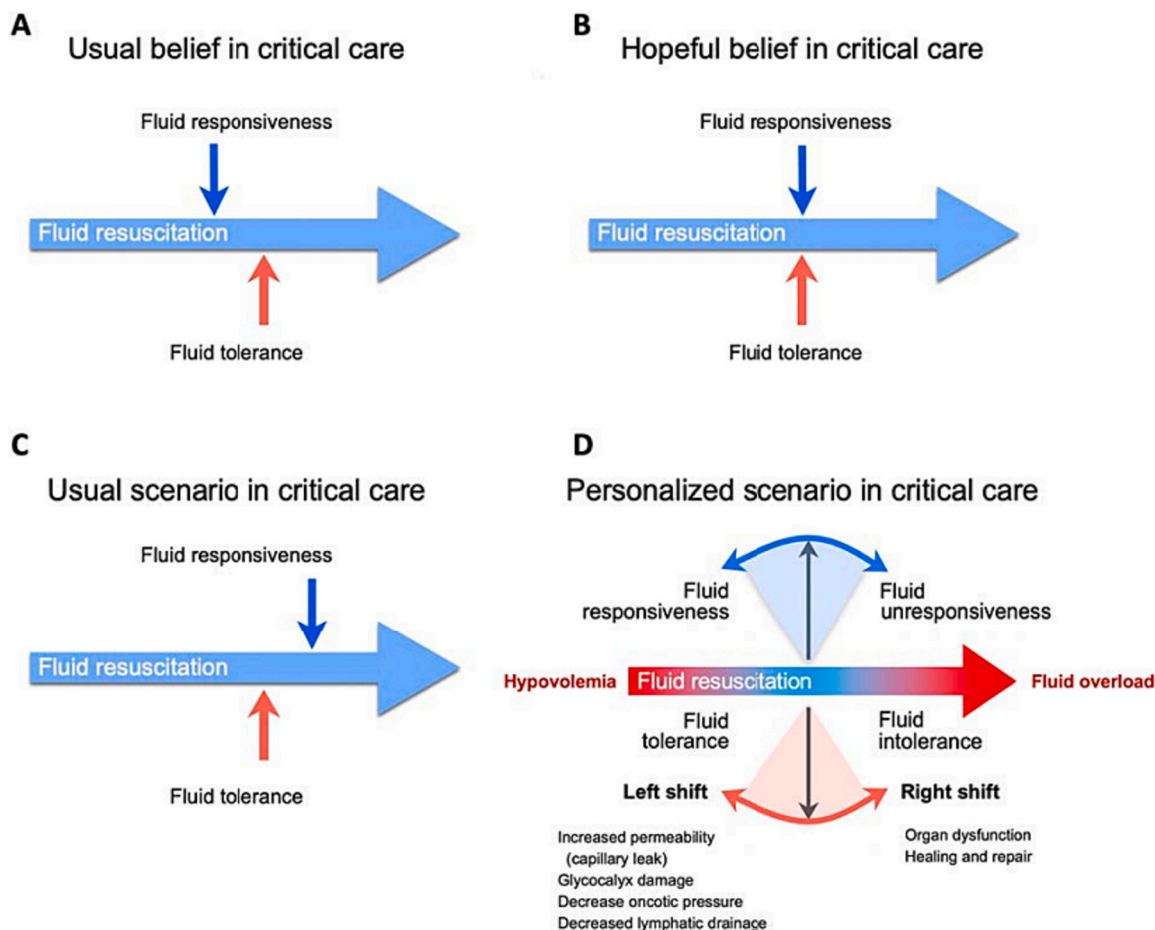


Fig. 1. Relationship between fluid tolerance and fluid responsiveness during resuscitation. *Reproduced with permission from [6].*

as may occur post-cardiac surgery and may not result in right atrial compression and hence may not cause a plethoric IVC (e.g. clot compressing the left atrium).

The third scenario is that of massive pulmonary embolism as the sole cause of shock. While the lack of a plethoric IVC in no way rules out that diagnosis, its absence points to another mechanism also contributing to shock such as sepsis or volume depletion.

Hence, the finding of a plethoric IVC should prompt the clinician to rule out these life-threatening conditions early in the clinical assessment, especially since they have specific and efficacious therapies.

6. Anatomy of the IVC & the Physiology of IVC Variation

When interpreting the IVC, the savvy clinician will have to take several anatomical, physiological and even mathematical factors into account.

- Respiratory variation:** A key measurement parameter worth reviewing. With spontaneous breathing, inspiration decreases the intrathoracic pressure and CVP, thus increasing the pressure difference between the downstream right atrium and the upstream intra-abdominal pressure and Pmsf. This will drive an increase in venous flow from the IVC into the heart and the IVC will collapse as internal pressures of the IVC recedes relative to the external intra-abdominal pressure. This process is reversed with spontaneous expiration and the IVC then distends. In mechanically ventilated patients (without spontaneous respiratory effort) delivery of a breath increases the intrathoracic pressure and decreases the pressure gradient between the thorax and the abdomen and Pmsf, thus distending the IVC as venous return decreases and pressures inside the IVC rise relative to the external pressure. Release of the delivered breath restores the baseline difference in abdominal and thoracic pressures, the flow to the heart increases, and the IVC collapses. Of course, the patient may also increase or decrease the intra-abdominal pressures by various means, and this will also affect the collapse or dilation of the IVC. Finally, it must be appreciated that either the magnitude of respiratory effort or the pressure delivered by the ventilator as well as patient-ventilator interactions will affect venous return and collapsibility of the IVC. Therefore dogmatic “cut off” indices of respiratory collapse of the IVC to predict the response to a volume challenge are dubious at best. Essentially then, the collective force of multiple factors, influenced by their relative effect on venous return, which comes from the IVC “reservoir” must be considered with interpretation of the IVC.
- Short axis vs Long axis:** one must realise that, in most cases, the IVC does not behave as a cylinder through most of its CVP range. Instead, if looked at in its short axis, it is in fact elliptoid, except at elevated CVP levels where it becomes more circular with a short diameter to long diameter ratio approaching 1, as illustrated in Fig. 2 [4]. Assessing it only in the long axis, therefore, presents several limitations, without having any advantages except for the reproducibility and practicality, which are not patient care factors. It is also important to note that, in the more elliptoid IVCs, the respiratory variation usually occurs in the short diameter. As will be explained below, this can lead to a significant source of misinterpretation.
- Elliptical tilt:** Additionally, if we look at the IVC in the transverse plane, the angle or “tilt” of the ellipse, noticeable at normal/low CVPs, varies greatly, but is generally oblique. Hence the use of an antero-posterior diameter measurement as is traditionally done, generally represents a variable-angle diagonal cut across the ellipse neither measuring the long or the short diameters. Presumably, when the long-axis diameter measurement approach began, the hope was that IVCs were in fact cylinders, making the measurement of any diameter a somewhat reliable surrogate for volume. This tilt, if the long diameter of the ellipse nears an anterior-posterior axis, can potentially be quite misleading to the clinician using a subxiphoid

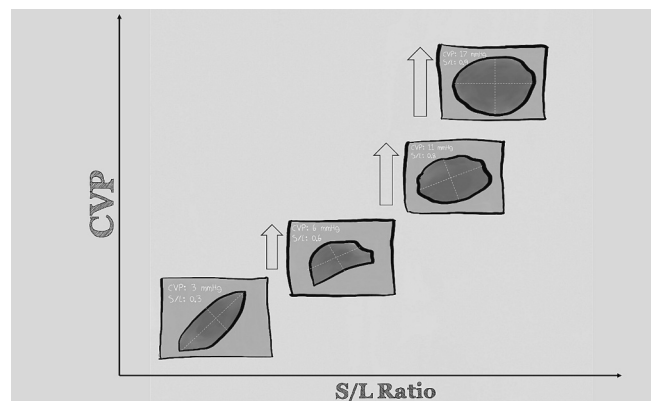


Fig. 2. Relationship between central venous pressure and the short to long diameter ratio. Note the elliptoid shape observed in most IVCs at low CVP, making single plan LAX an unreliable measure of IVC distension.

long axis view alone, presenting a “distended” IVC with little variation, when in fact it may be quite “small” or representative of a lower CVP.

- Long axis course:** the cephalad portion of the IVC, nearest to the diaphragm, remains “open” in some cases whereas the distal IVC becomes quite small, giving a wedge appearance in the long axis. The traditional method of measuring about 2 cm below the diaphragm may thus be misleading as it may display a “large” diameter with little variation, equating it to an evenly distended IVC while those two IVCs are not truly equivalent in terms of venous pressure/congestion. (Fig. 3)

Understanding all the above immediately puts into question the mathematical validity of using a single point diameter of a non-cylindrical structure - on top of that not including the magnitude of ITP changes - for a volumetric variation assessment. It would appear that the most mathematically and physiologically appropriate measurement of the IVC would be a short axis scan across the entire intrahepatic segment, somewhat akin to “eyeballing” the left ventricle to determine the ejection fraction, while also taking into account the swings of intrapleural pressure (referred to as the effort-variation index - Fig. 4) and an assessment of the intra-abdominal pressure [5].

7. The IVC in patients with chronic RV dysfunction

When faced with chronically elevated right atrial pressures, the IVC will dilate over time, such that the “usual” or baseline size will be significantly increased. We have noted several IVCs to be over 30 mm in diameter. Naturally, in these cases, an IVC measuring 25 mm in a single axis may in fact represent a lower RAP than the patient’s baseline, but if the IVC is examined in SAX, it will likely not be truly plethoric, and its short to long diameter ratio will likely not be near 1. This could cause a VExUS score - if measured - to be falsely elevated, because by LAX criteria, it would be a VExUS 1, whereas physiologically would more be akin to a VExUS 0. This is being validated in ongoing studies, but suggests that clinicians should be assessing for plethoric index, particularly in states of chronic RAP elevation - as explained above - rather than a single diameter. Hence, IVCs should be interpreted in parallel with its “surroundings,” particularly the RA/RV.

8. The IVC in elite athletes

A similar adaptation is seen in high level athletes where the venous system adapts to chronically elevated flow as well as likely some elevation of RAP [10,11]. We have seen this as well in clinical practice. While this may be obvious in a young patient, it may be necessary to add this to the clinical history-taking as the hemodynamic findings may

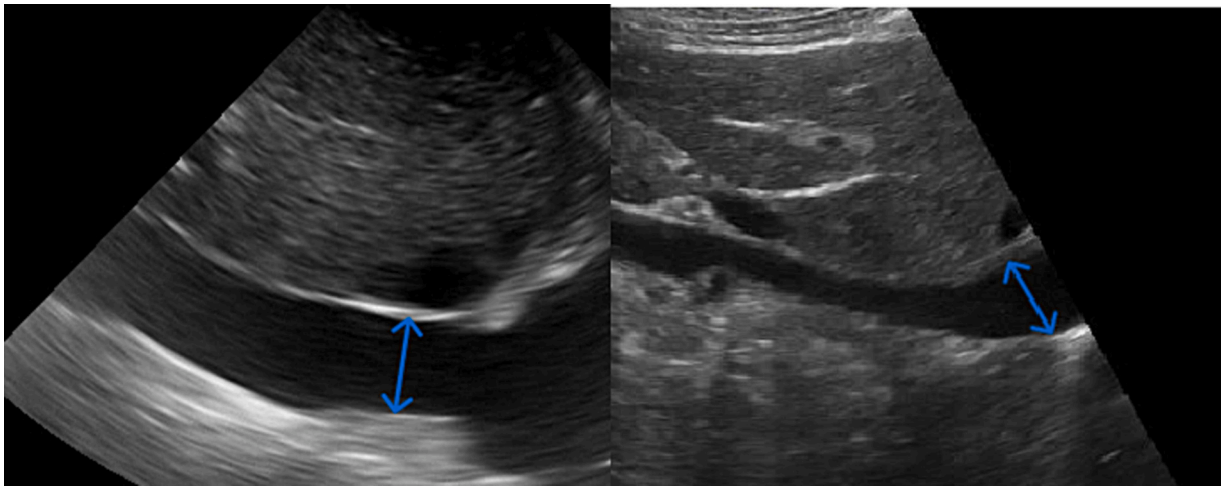


Fig. 3. Both IVCs measure approximately 2 cm when measured in LAX just below the diaphragm. However, the IVC on the right panel is clearly less distended across the intrahepatic segment than the one on the left.

The Effort-Variation Index (EVI)

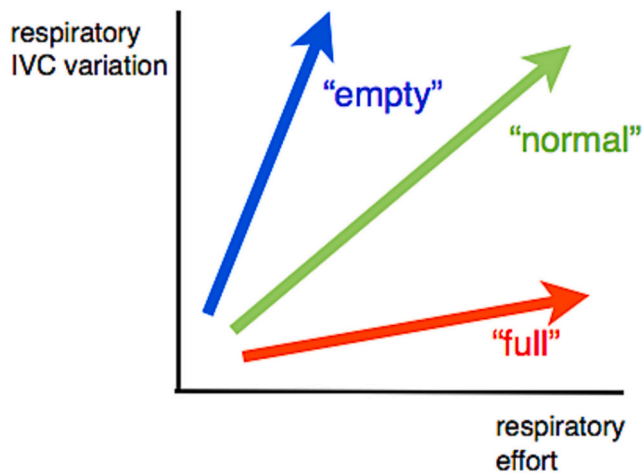


Fig. 4. A theoretical construct to understand the relationship between IVC variation and respiratory effort. On any horizontal line, the variation is identical, and may occur with different filling states, given sufficient swings in intrapleural pressure [7].

persist long after the individual has stopped actively practicing her or his sport with intensity and regularity.

9. Conclusion

It does appear that it may be worth rescuing the baby and only throwing out the bathwater-like errors associated with IVC assessment such as (a) attempting to fill it to the brim (fluid-responsiveness strategy), (b) measuring it by single point long axis diameter and (c) assessing its respiratory variation without taking into account intra-abdominal pressure and intrathoracic pressure variations.

At this point, available data and physiological principles point us towards using the IVC as a gauge of venous congestion and as a fluid stop point, as well as a prompt to do further Doppler assessment to grade the severity of congestion. While it may hold to some degree at the extremes – similarly to the CVP – it should probably not be used as a fluid responsiveness tool.

In assessing critically ill patients, clinicians should be trying to put

the puzzle together rather than seeking to find and use a single metric to assess fluid issues. Hence, assessing upstream, downstream and surrounding characteristics should be done as opposed to looking at the IVC in isolation.

Hopefully, future studies will include proper IVC assessment and provide more data to fine tune our resuscitation further. Currently, several studies are underway such as ANDROMEDA-2, which has an observational venous congestion cohort and should shed more light onto congestion in sepsis resuscitation [9]. The IVC never lies, and will always represent the interplay of all multiple factors, and as such represents a cornerstone of fluid management. The physiologically-minded resuscitator's goal, in the words of Dr. Glenn Hernandez, should be to preserve hemodynamic coherence, that is, optimise perfusion without congestion. It remains up to the clinician to navigate the available data points to achieve this, and, properly assessed in context, the IVC provides one of them.

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Declaration of Competing Interest

The authors have no conflicts of interest to declare.

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