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WHAT'S NEW IN INTENSIVE CARE

Ten situations where inferior vena cava ultrasound may fail to accurately predict fluid responsiveness: a physiologically based point of view

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Introduction

Assessment of the size of the inferior vena cava (IVC) and its change in diameter in response to respiration have been investigated as a tool to screen for severe hypovolaemia [1], predict fluid responsiveness (FR) [2, 3] and assess potential intolerance to fluid loading. IVC size, collapsibility (IVCc) [2] and distensibility (IVCd) [3] have gained acceptance by emergency and intensive care unit (ICU) clinicians as FR predictors in patients with shock [4]. The ease of acquisition, reproducibility of measurements and increasing availability of ultrasound devices have supported the expansion of its use. Conflicting results have also been published [5, 6]. Injudicious application in clinical contexts where these indices have not been specifically tested may, however, mislead. On the basis of physiological principles and available, although limited, scientific evidence, it can be hypothesized that in a number of clinical conditions IVC size and/or respiratory variability may not depend on volume status and may not predict FR accurately. Although not specifically investigated yet, these conditions can be described and grouped on the basis of their main physiological determinant, as follows (Table 1) (pictorial samples are also presented as electronic supplementary material, ESM):

Ventilator settings

High PEEP and/or low tidal volumes IVCd has only been validated for the assessment of FR in mechanically ventilated patients with low levels of PEEP [3, 5]. High PEEP has been demonstrated to raise right atrial pressure (RAP), IVC pressure, and either increase or leave unaltered IVC size [7], while simultaneously reducing venous return [7], introducing a bias factor in the relationship between IVC size and FR. Furthermore, where tidal volumes less than 8 ml/kg are used, causing smaller variations in intrathoracic blood volume and pressure, IVC dimensions in response to ventilation are theoretically expected to be smaller, irrespective of volume status: available evidence suggests that IVCd shows here lower sensitivity [5] and overall inaccuracy [8] in predicting FR (Clip 1-ESM).

Patient's inspiratory efforts

Assisted ventilation modalities/non-invasive ventilation/CPAP Inspiratory variations of IVC dimension have only been validated either in spontaneous respiration or entirely passive ventilation [2–4, 6]. These two situations share a concordant increase in abdominal pressure, and opposite (but predictable) changes in intrathoracic pressure, during inspiration. By contrast, assisted ventilation modalities/non-invasive ventilation involves a variable patient contribution to inspiration. As a result of the unpredictable interplay of ventilator-generated positive pressure and patient-generated negative pressure, IVCc loses a univocal correlation with preload [9] (Clip 2-ESM). No studies have systematically validated the IVC in determining FR in patients supported with CPAP.

Varying respiratory pattern in spontaneous breathing There are no data regarding the breathing pattern of patients with spontaneous respiration evaluated with

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Table 1 Ten conditions potentially affecting inferior vena cava (IVC) ultrasound reliability in predicting fluid responsiveness (FR)

Physiological determinant	Condition affecting IVC ultrasound reliability for FR	Cause of inaccuracy for FR	Type of inaccuracy for FR
Ventilator settings	1. Mechanical ventilation with high PEEP and/or low tidal volumes	Larger IVC size, potentially with systemic venous congestion and low respiratory variations, but coexisting with FR	FN
Patient's inspiratory efforts	2. Assisted ventilation modalities, NIV, CPAP	Spontaneous breathing activity makes IVC variation unpredictable	FP and FN
	3. Varying respiratory pattern in spontaneous breathing	Significant inspiratory effort, producing markedly negative intrathoracic pressures may induce IVCc in absence of FR	FP
		Shallow breathing, with small intrathoracic pressure changes, may induce absence of IVCc in presence of FR	FN
Lung hyperinflation	4. Asthma/COPD exacerbation	Lung hyperinflation and auto-PEEP simultaneously reduce venous return and induce IVC distension: this may mimic absence of FR	FN
		Forced expiration ("abdominal breathing" causing expiratory collapse) may mimic IVCc	FP
Cardiac conditions impeding venous return	5. Chronic RV dysfunction, severe TR	Chronic enlargement of IVC and reduced IVCc may erroneously rule out FR	FN
	6. RV myocardial infarction	RV dilatation and systemic venous congestion (large IVC) may be associated with FR	FN
	7. Cardiac tamponade	Marked venous return hindrance: fluid challenge may be a beneficial haemodynamic intervention despite IVC plethora	FN
Increased abdominal pressure	8. Intra-abdominal hypertension	Smaller IVC size, IVCd or IVCc abolition (depending on type respiration/ventilation mode)	FP and FN
Other factors	9. Local mechanical factors	Venous return hindrance, IVC dilatation (stenosis, thrombosis)	FN
		IVC compression (masses)	FP
		Hindrance to IVC size change (ECMO cannulae, cava filters)	FN
	10. Patients with pronounced IVC inspiratory lateral displacement	Migration of IVC imaging plane, false inspiratory size reduction	FP

IVC inferior vena cava, *RV* right ventricle, *PEEP* positive end-expiratory pressure, *NIV* non-invasive ventilation, *CPAP* continuous positive airway pressure, *IVCc* IVC collapsibility, *IVCd* IVC distensibility, *COPD* chronic obstructive pulmonary disease, *TR* tricuspid regurgitation, *ECMO* extracorporeal membrane oxygenation, *FN* false negative, *FP* false positive

IVCc for FR [2, 4, 6]. The amplitude of intrathoracic pressure swings and size of tidal volumes are hard to quantify in spontaneous breathing. Evidence in healthy volunteers shows that the deeper the breathing is, the larger the diaphragmatic excursions and IVCc are, irrespective of volume status [10]. This implies that shallow breaths may reduce the sensitivity of IVCc for determining FR [6], while marked inspiratory efforts seen in respiratory distress may magnify IVCc and therefore reduce its specificity (Clip 3-ESM).

Lung hyperinflation

Asthma/COPD exacerbation is associated with lung hyperinflation, development of auto-PEEP and increased intrathoracic pressure. In spontaneously breathing patients in this condition the IVC at end-expiration has been described as dilated [11]. At the same time, large negative inspiratory intrathoracic pressure swings induce IVC collapse [10]. Although not extensively investigated, these phenomena introduce a variable that affects IVC size and IVCc irrespective of central volaemia and

FR. Expiratory effort (and the associated increases in abdominal pressure) may also affect IVC dimensions in an opposite manner, leading to IVC expiratory collapsibility, rather than the expected inspiratory collapse (Clip 4-ESM).

Cardiac conditions impeding venous return

Chronic RV dysfunction/tricuspid regurgitation Chronic pulmonary hypertension leads to right ventricular (RV) remodelling and decreased compliance, RAP elevation, and chronic IVC enlargement with reduced inspiratory collapse. Here, an IVC size above the validated diagnostic cut-offs for severe hypovolaemia may coexist with FR (Clip 5-ESM); data on IVC respiratory variations and FR in this population are lacking. Furthermore, patients with severe tricuspid regurgitation exhibit a dilated IVC per se, which shows no correlation with their fluid status [12].

RV myocardial infarction induces acute systolic and diastolic dysfunction and a reduction in RV compliance, partly as a result of the constrictive effect of the pericardium secondary to RV enlargement. This leads to a disproportionate increase in right side filling pressures and systemic venous congestion. In extreme conditions the right heart behaves as a passive conduit between the venous system and the left heart. The LV becomes hyperdynamic, and the patient may be volume-responsive, despite the IVC being significantly dilated [13] (Clip 6-ESM).

Cardiac tamponade Here impeded cardiac filing ensues, and there may be consequent systemic venous congestion. IVC plethora is the associated ultrasound finding (Clip 7-ESM): the finding of a fixed, dilated IVC does not equate to an absence of FR and should not preclude volume resuscitation pending definitive intervention (if required).

Increased abdominal pressure

Intra-abdominal hypertension and abdominal compartment syndrome IVC width and size variation in response to respiration are determined by its transmural pressure, under the influence of the pressure gradient between abdominal and intrathoracic compartments. Experimental evidence suggests that increased intra-abdominal pressure reduces IVC size regardless of volume status; indeed, it compresses and deforms the IVC [14], blunting the effect of mechanical inspirations on its size. Therefore, abdominal hypertension is likely to affect the reliability of IVC-based FR indices.

Other factors

Local mechanical factors: IVC restriction/compression/thrombosis/vena cava filters/ECMO cannulae Several mechanical factors have the potential to affect IVC size and its respiratory size changes, invalidating the applicability of IVC ultrasound to estimate FR. The use of VV-ECMO and VA-ECMO always entails presence of semi-rigid central venous cannula(s) occupying the IVC to a variable extent, thereby limiting its collapsibility (Clip 8-ESM). Further, negative venous pressure interferes with IVC size and respiratory dynamics, possibly to the extent of complete collapse around the cannulae. Masses compressing/occupying the vessel, vena cava filters or IVC thrombosis can equally affect physiological IVC patency and size.

Marked IVC respiratory translational motion Some patients may exhibit a pronounced lateral IVC displacement during inspiration. This can result in misalignment of the ultrasound scanning plane and thus overestimation of IVCc [15]. Where suspected, interrogation of the IVC in its short axis may assist (Clip 9-ESM).

Conclusion

Using ultrasound to evaluate the IVC as part of the haemodynamic assessment of critically ill patients appears superficially simple. However, in order to correctly guide clinical decisions it is key to understand the basis of the evidence supporting its use. Measurements must be interpreted in the context of the patient's underlying pathophysiological status including the mode of ventilation/respiration and ventilator settings/respiratory pattern. It must always be integrated with other findings from either a systematic focused cardiac ultrasound or a comprehensive echocardiogram. Although still very useful in the patient populations where it has been validated, IVC-based assessment of FR may not be applicable to a large proportion of ICU/emergency patients. Further studies should test its accuracy in the clinical conditions described.

Electronic supplementary material
The online version of this article (doi:10.1007/s00134-016-4357-9) contains supplementary material, which is available to authorized users.

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Compliance with ethical standards

Conflicts of interest
None of the authors has any conflict to declare.

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