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CKJ REVIEW

Inferior vena cava ultrasound and other techniques for assessment of intravascular and extravascular volume: an update

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ABSTRACT

Goals of volume management are to accurately assess intravascular and extravascular volume and predict response to volume administration, vasopressor support or volume removal.
Data are reviewed that support the following:

(i) Dynamic parameters reliably guide volume administration and may improve clinical outcomes compared with static parameters, but some are invasive or only validated with mechanical ventilation without spontaneous breathing.
(ii) Ultrasound visualization of inferior vena cava (IVC) diameter variations with respiration reliably assesses intravascular volume and predicts volume responsiveness.
(iii) Although physiology of IVC respiratory variations differs with mechanical ventilation and spontaneous breathing, the IVC collapsibility index (CI) and distensibility index are interconvertible.
(iv) Prediction of volume responsiveness by IVC CI is comparable for mechanical ventilation and spontaneous breathing patients.
(v) Respiratory variations of subclavian/proximal axillary and internal jugular veins by ultrasound are alternative sites, with comparable reliability.
(vi) Data support clinical applicability of IVC CI to predict hypotension with anesthesia, guide ultrafiltration goals, predict dry weight, predict intra-dialytic hypotension and assess acute decompensated heart failure.
(vii) IVC ultrasound may complement ultrasound of heart and lungs, and abdominal organs for venous congestion, for assessing and managing volume overload and deresuscitation, renal failure and shock.
(viii) IVC ultrasound has limitations including inadequate visualization.

Ultrasound data should always be interpreted in clinical context. Additional studies are required to further assess and validate the role of bedside ultrasonography in clinical care.

LAY SUMMARY

It is important and challenging to differentiate which unstable patients would most likely benefit from volume administration, medicines to raise the blood pressure but not additional volume administration or volume removal. Point-of-care ultrasound stands out for being a non-invasive and versatile method for doing this. Compared with some other ultrasound techniques, inferior vena cava (IVC) ultrasound is relatively simple, and can be performed rapidly. Ultrasound can be used anywhere, from office visits to intensive care units. IVC ultrasound is useful for

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answering whether patients may be intravascularly volume depleted or not, as well as whether patients may be volume overloaded or not. IVC ultrasound may be combined with other ultrasound techniques to assess intravascular as well as extravascular volume status. Limitations of point-of-care ultrasound techniques must be recognized and taken into account. Future studies are required to validate the role of bedside ultrasound to improve the quality of medical care.

Keywords: deresuscitation, inferior vena cava ultrasound, intravascular volume, volume overload, volume responsiveness

KEY MESSAGES

- The goal of volume assessment is to predict whether an unstable patient is most likely to benefit from volume administration, vasopressor support but not additional volume administration, or volume removal.
- Inferior vena cava (IVC) ultrasound assessment can potentially decrease the likelihood of either overt relative intravascular hypervolemia or hypovolemia in a given patient.
- IVC maximum diameter <2.1 cm that collapses >50% is inconsistent with intravascular volume overload while IVC collapsibility index <20% (with moderate to large IVC diameter) is inconsistent with intravascular volume depletion, with spontaneous breathing or mechanical ventilation.
- When assessed together, IVC ultrasound and lung ultrasound provide a more accurate assessment of intravascular and extravascular volume excess.

INTRODUCTION

The goals of volume management are to optimize intravascular volume, cardiac output, tissue perfusion and oxygen delivery to tissues. Insufficient volume administration may perpetuate hypoperfusion, and volume overload may result in organ congestion and dysfunction, either of which may increase morbidity and mortality, particularly in critically ill patients [1].

Only 50% of hypotensive patients are volume responsive [2]. The remainder do not respond to volume administration and may develop acute volume overload. Assessing intravascular and extravascular volume facilitates differentiation of patients most likely to benefit from volume expansion, ionotropic support but not volume therapy, or volume removal by diuresis or ultrafiltration. This is particularly difficult in critically ill patients with concurrent cardiac, hepatic or renal dysfunction.

Clinical assessment of intravascular volume in hospitalized patients, including physical examination, X-rays and laboratory findings, frequently has low sensitivity and/or specificity due the presence of non-steady state conditions, resulting in mismatch between intravascular volume and blood pressure, or between intravascular and extravascular volume (Table 1) [3]. More reliable assessments are required.

Categorize and compare different techniques to assess volume status of hospitalized or critically ill patients

Three major organizing principles can be used to categorize and compare techniques to assess volume status (Table 2).

Static parameters versus dynamic parameters versus dynamic tests versus endpoints used to assess response to dynamic tests or volume changes

Static parameters are single values of measurements such as central venous pressure (CVP), right atrial pressure (RAP), pulmonary artery occlusion pressure (PAOP) and inferior vena cava (IVC) maximum diameter (IVCmax) [4–7]. RAP and CVP values are surrogates for right ventricular preload, while PAOP is a surrogate for left ventricular preload. These may only reflect intravascular volume at the extremes [6]. Static parameters have poor sensitivity and specificity to predict volume responsiveness and are not recommended to guide volume administration [6].

Dynamic parameters reflect heart–lung interactions, and vary according to cardiac and respiratory cycles. For any dynamic parameter, cyclic variation is more pronounced in volume-responsive patients. Dynamic parameters more reliably predict volume responsiveness compared with static parameters [4–6, 8–11]. Predicting volume responsiveness accurately may help avoid volume overload and acute pulmonary edema, or inadequate volume repletion [4, 6].

Dynamic tests modify venous return to the heart by, for example, an intravenous volume bolus or passive leg raising (PLR) to predict cardiac output response to further volume administration [6]. An increase in stroke volume >10% induced by PLR predicted volume responsiveness (sensitivity 77%–100%, specificity 88%–99%) [4].

The "gold standard" positive endpoint for volume responsiveness is a 10%–15% increase in cardiac output or stroke volume by thermodilution in response to volume administration [6] or removal [12].

"Is the patient volume responsive or not?" versus "is the patient volume overloaded or not?"

Predicting whether a positive response would occur with volume administration is distinct from asking whether a positive response would occur with volume removal. Many parameters or tests are not validated in both contexts. Clarifying which question is being asked can help determine which methods of assessment would be most helpful.

Where in the cardiopulmonary circuit are parameters being measured?

Categorizing volume assessment techniques anatomically shows the conceptual continuity of contemporary methods with traditional physical examination findings (Fig. 1 and Table 2) (1, 8–11, 13–16]. Techniques that assess the same portion of the circulatory system share similar advantages and disadvantages.

Table 1: Mismatch between intravascular volume and blood pressure or extravascular volume.

States in which blood pressure is not primarily determined by intravascular volume	
Mismatch between intravascular volume and blood pressure	
Intravascular volume low	Vasoconstriction
Blood pressure high	• Stimulants (cocaine, amphetamines), catecholamines (pheochromocytoma, severe stress, delirium tremens)
	• Severe hypothyroidism
Intravascular volume high	Cardiac dysfunction
Blood pressure low	• Cardiogenic shock
	• Severe cardiomyopathy, heart failure, valvular heart disease
	Vasodilation
	• Distributive shock + excess volume resuscitation
	• Autonomic neuropathy
Mismatch between intravascular and extravascular volume	
Intravascular volume low	Vasodilation and/or "third spacing"
Extravascular volume high	• Distributive shock (sepsis, anaphylaxis)
	• Hemorrhagic pancreatitis
	• Crush injury
	Delayed re-equilibration
	• Severe renal failure + diuresis or ultrafiltration
	• Nephrotic syndrome + diuresis
	• End-stage liver disease + diuresis or large-volume paracentesis or ultrafiltration
	• Heart failure + diuresis or ultrafiltration
Intravascular volume high	• Rapid blood transfusion + anuria or severe renal failure
Extravascular volume not high	• Rapid hypertonic sodium bicarbonate or saline infusion

With permission from Kaptein and Kaptein 2017 [3].

COMPARISON OF TECHNIQUES TO ASSESS INTRAVASCULAR VOLUME OR RESPONSE TO VOLUME ADMINISTRATION OR REMOVAL

Volume responsive or not? Hypovolemia versus not-hypovolemia

Arterial dynamic parameters + dynamic tests

Three meta-analyses of randomized controlled trials (RCTs) showed that using dynamic parameters, including stroke volume variation, systolic pressure variation and pulse pressure variation, to guide volume therapy significantly improved morbidity and mortality in mechanically ventilated post-surgical and intensive care unit patients compared with standard volume management [17–19] (Fig. 2). All included studies used invasive arterial line measurement of dynamic parameters, except two that used plethysmograph waveforms. Recognizing that cyclic variation of dynamic parameters is more pronounced in volume responsive patients, many studies aimed to achieve or maintain cyclic variation of a dynamic parameter of interest below a certain threshold, usually 10%–12%. Other studies used stroke volume index or cardiac index as endpoints to assess response to volume management. Two RCTs (Prevention of Cardiac Surgery Associated Acute Kidney Injury (PrevAKI)) [20, 21] showed significantly decreased rates of moderate to severe acute kidney injury (AKI) (41% and 33%, respectively) within 72 hours after cardiac surgery with goal-directed volume therapy based on dynamic parameters or tests, compared with standard care.

Dynamic variations of stroke volume and pulse pressure by arterial pressure waveform analysis reliably predict volume responsiveness in mechanically ventilated patients who are heavily sedated without spontaneous breathing efforts, but not with arrhythmias, low tidal volumes, low lung compliance, an open chest or intra-abdominal hypertension [1, 4, 6, 14].

A systematic review and meta-analysis reports, "[Electrical cardiometry] cannot replace [thermodilution] and [transesophageal echocardiography] for the measurement of absolute cardiac output values. However, as the [mean percentage error] is comparable to clinically used minimally or non-invasive hemodynamic monitors, [electrical cardiometry] could complement monitoring in the ICU and NICU, providing continuous monitoring, relevant for goal-directed therapy and clinical decision-making" [16].

In a multicenter RCT of 83 patients who received volume therapy guided by PLR-induced stroke volume change using bioreactance, compared with 41 with usual care, who had sepsis-associated hypotension and shock, net volume balance was significantly less (–1.37 L), as was renal replacement therapy (5.1% versus 17.5%, respectively) and mechanical ventilation (17.7% versus 34.1%, respectively) [22]. A recent review citing earlier studies [6] highlights the potential of thoracic bioimpedance, while reviews citing later studies highlight limitations [1, 14].

Venous dynamic parameters

IVC diameter respiratory variation by ultrasound has been shown to predict volume responsiveness reliably and comparably to stroke volume variation by arterial pressure waveform analysis in two studies of mechanically ventilated patients [23, 24]. IVC ultrasound had low inter-operator variability and can be repeated as needed to reevaluate intravascular volume after volume administration or removal. Venous dynamic parameters do not require a dynamic test for interpretation [1].

IVC CI predicts volume responsiveness with mechanical ventilation and spontaneous or standardized breathing. Controversy exists concerning performance of IVC ultrasound to pre-

Table 2: Intravascular volume assessment and volume responsiveness classification.

	Hypovolemia vs not hypovolemia—volume responsive or not?	Hypervolemia vs not hypervolemia—volume overloaded or not?	Comments
Static parameter, venous/target organ	Single CVP/RAP	Single CVP/RAP	Low SN and SP for predicting volume responsiveness, invasive
	IVCmax diameter	IVCmax diameter VExUS	Confirmatory for suspected volume overload [15]
Static parameter, right heart/lung	Single RAP	Single RAP	Low SN and SP for predicting volume responsiveness, invasive
		E/E′ ratio by echocardiograpy, VExUS	Same SN and SP as IVCmax with IVC CI to estimate RAP [13]
		Lung ultrasound, CXR, rales	Can have mismatch between intravascular and lung
Static parameter, left heart	PAOP	PAOP	Low SN and SP for predicting volume responsiveness, invasive
		LVEDA by echo	Low SN and SP for predicting volume responsiveness
Static parameter, peripheral		Bioimpedance	Differentiates TBW distribution between ICF and ECF, but not IVV from EVV
		Total body weight, "total fluid balance"	Do not differentiate ICF from ECF or IVV from EVV
		Edema, ascites	Edema and ascites reflect EVV, not necessarily IVV
Dynamic parameter, venous, conceptually related to JVP	IVC CI	IVC CI	Good SN and SP to predict volume responsiveness
	Subclavian vein CI, IJV CI	Subclavian vein CI, IJV CI	Reasonable concordance with IVC CI
	CVP/RAP waveform	CVP/RAP waveform	Invasive
Dynamic parameter, arterial, conceptually related to "pulsus paradoxus"	Arterial pressure waveform analysis, PPV, SPV, SVV		Invasive. Only validated with MV, and no spontaneous breathing, and no cardiac arrhythmias [6]
	Plethysmograph waveform analysis		Limited data
	NICCOM/bioreactance/electrical cardiometry/thoracic bioimpedance, SVV		Non-invasive. Limited accuracy in low-flow states and arrhythmias [1, 14, 16]
Dynamic tests or volume changes	Passive leg raise		Reversible volume challenge
	End-expiratory occlusion test		Only with MV
	Isonatremic fluid bolus	Diuresis, ultrafiltration	May cause volume overload or depletion, respectively
Gold standard endpoint for dynamic tests or volume changes	CO/cardiac index ↑ by thermodilution	CO/cardiac index ↑ by thermodilution	Invasive
Surrogate endpoints for dynamic tests or volume changes	CO/cardiac index ↑ by ultrasound: LVOT VTI, carotid flow/velocity, radial artery flow/velocity[a]		Non-invasive
	Arterial pressure waveform analysis SV/SVI, CO/cardiac index		Invasive. Only validated with MV, and no spontaneous breathing, and no cardiac arrhythmias [6]
	NICCOM/bioreactance/electrical cardiometry/thoracic bioimpedance, SV/SVI, CO/cardiac index		Non-invasive. Limited accuracy and precision in low-flow states and arrhythmias [1, 14, 16]
Clinical endpoint, plasma volume	Change in HCT	Crit-line/change in HCT	
Clinical endpoint, venous	Decrease in vasopressor dose	Decrease in vasopressor dose	
Clinical endpoint, right heart/lung		Decrease in respiratory support, ↑ PO_2, ↓ FiO_2	
Clinical endpoint, arterial	Improved BP/HR	Improved BP/HR	
	Hypotension with anesthesia induction	"Tolerated" volume removal	
Clinical endpoint target organ	Improvement in serum Cr if AKI/WRF	Improvement in serum Cr if AKI/WRF	
Clinical endpoint peripheral	Capillary refill time		

[a]Changes in carotid artery blood flow after a volume bolus correlate strongly with an increase of stroke volume index or cardiac output [8, 9]. A meta-analysis showed that both corrected carotid flow time and peak velocity variations predicted volume responsiveness in pre-anesthesia as well as in critically ill patients with spontaneous breathing and mechanical ventilation [10]. Radial artery corrected blood flow and variation in blood flow peak velocity reliably predicted volume responsiveness in mechanically ventilated patients [11].

A-line = arterial-line; CO = cardiac output; Cr = creatinine; CXR = chest X-ray; ECF = extracellular fluid; E/E′ = tricuspid E/E′ ratio; EVV = extravascular volume; FiO_2 = fraction of inspired oxygen; HCT = hematocrit; ICF = intracellular fluid; IVV = intravascular volume; JVP = jugular venous pressure; LVEDA = left ventricular end-diastolic area; LVOT VTI = left ventricular outflow tract velocity time index; MV = mechanical ventilation; PO_2 = partial pressure of oxygen; PPV = pulse pressure variation; NICCOM = non-invasive continuous cardiac output monitoring; SN = sensitivity; SP = specificity; SPV = systolic pressure variation; SV = stroke volume; SVI = stroke volume index; SVV = stroke volume variation; US = ultrasound; WRF = worsening renal function.

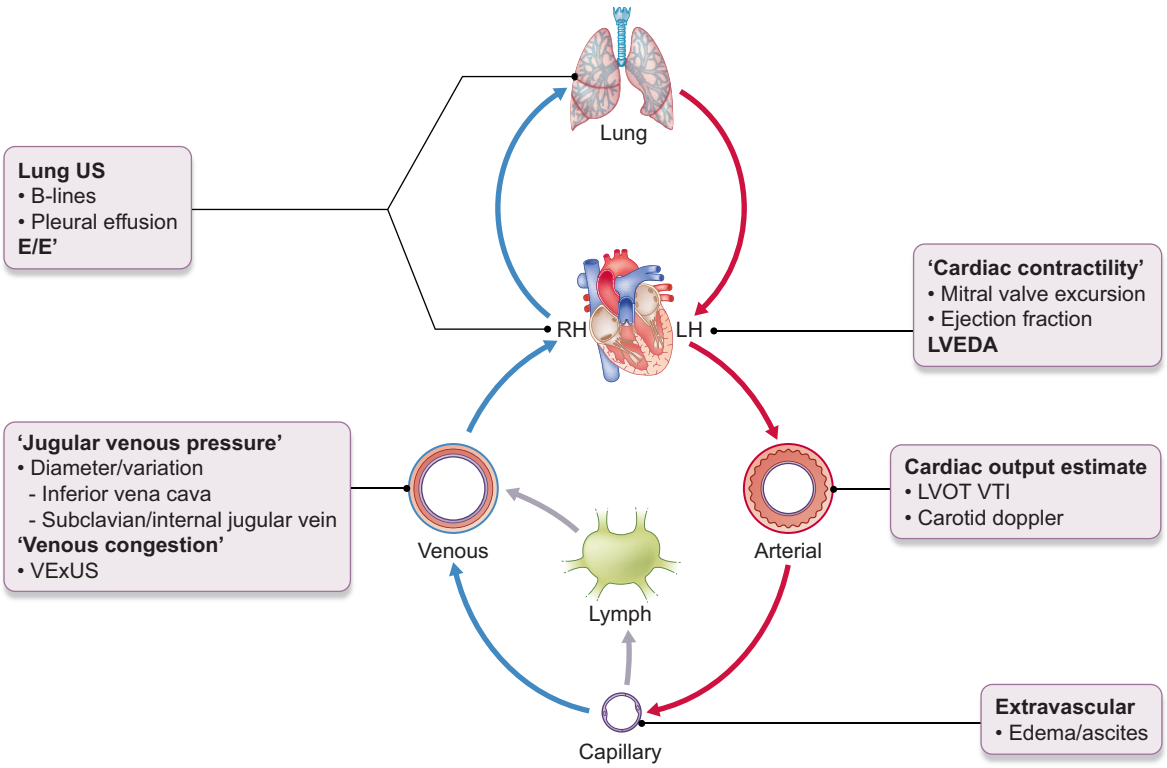


Figure 1: Venous/arterial circuit ultrasounds to assess volume status. LVEDA = left ventricular end-diastolic area; LVOT VTI = left ventricular outflow tract velocity time index; RH = right heart; LH = left heart; US = ultrasound.

dict volume responsiveness in mechanically ventilated compared with spontaneously breathing patients (Table 3) [5, 25–32]. IVC collapsibility index (CI) and distensibility index (DI) are interconvertible (Table 3 footnotes) [5]. All data in this review are presented as IVC CI to facilitate comparison.

The meta-analysis by Kim *et al.* [25] showed that IVC variation predicted volume responsiveness with similar accuracy for mechanical ventilation (sensitivity 74%, specificity 85%) as for spontaneous breathing (sensitivity 76%, specificity 81%) (Table 3); differences among prior meta-analyses are discussed [25]. We assessed individual studies from four other meta-analyses [26–29] reporting variable performance of IVC to predict volume responsiveness in mechanically ventilated compared with spontaneously breathing patients (Supplementary data, Table S2). Studies meeting specified inclusion criteria were pooled and showed similar sensitivity and specificity for mechanically ventilated and spontaneously breathing patients (Table 3). Different conclusions among meta-analyses were probably due to inclusion of dissimilar studies (Supplementary data, Table S2) [26–29]. Three additional studies of patients undergoing standardized breathing showed higher pooled sensitivity and specificity (89% and 92%, respectively) to predict volume responsiveness [30, 32, 33] than for spontaneously breathing patients (Table 3).

IVC CI predicts anesthesia-induced hypotension. IVC CI reliably predicted post-anesthesia hypotension in 10 studies (pooled sensitivity 82%, specificity 81%) (Table 4) [34–43]. In an RCT of 122 patients [37], pre-induction ultrasound-guided volume management at an IVC CI cut-off of 42% significantly lowered the incidence of hypotension by 52%, use of vasopressors by 56% and total volume administered compared with standard care. In another RCT of 160 patients [44], the IVC ultrasound-guided volume management group had a relative risk reduction for hypotension of 35% ($P < .044$), and significantly lower need for vasoactive drugs.

Hypotension risk after general anesthesia was predicted in 100 patients using pre-induction IVC CI, stroke volume variation, stroke volume, cardiac output, plethysmography variability index and perfusion index [36]. Multiple logistic regression analysis revealed IVC CI was the most significant independent factor for predicting post-induction hypotension.

Central venous collapsibility, alternatives to IVC CI. Respiratory variations of the subclavian/proximal axillary vein (SCV) and internal jugular vein (IJV) are highly position dependent, as is jugular venous distension, and should be performed at a 30–45 degree upper body incline [45]. SCV CI and IJV CI do not appear to be affected by intra-abdominal hypertension or positive end-expiratory pressure [46, 47]. The SCV is less influenced by external compression, due to the location beneath the clavicle, compared with IJV, which is located more superficially [48].

SCV CI has been shown to predict volume responsiveness (cardiac output >15%) in mechanically ventilated patients using an SCV CI cut-off of 18% (sensitivity 100%, specificity 82%) [49]. In 120 patients undergoing general anesthesia, a cut-off of 36% for SCV CI with deep breathing (presumably without head elevation) predicted post-induction hypotension in 42% of patients (sensitivity 90%, specificity 87%), similar to performance of IVC CI using a cut-off of 37% (sensitivity 94%, specificity 84%)

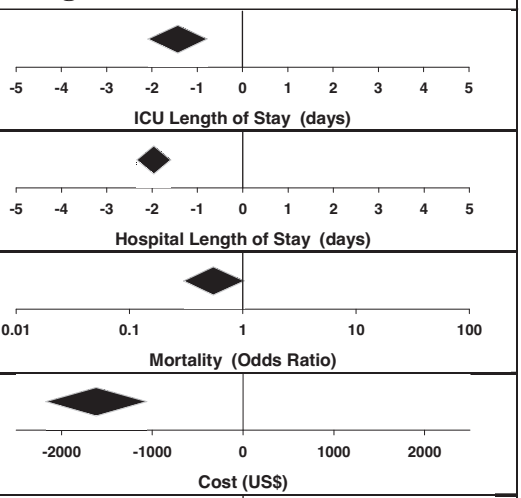
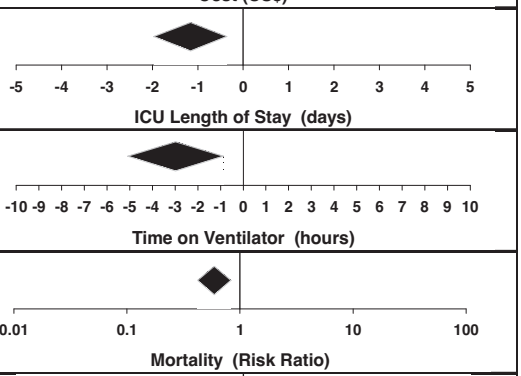
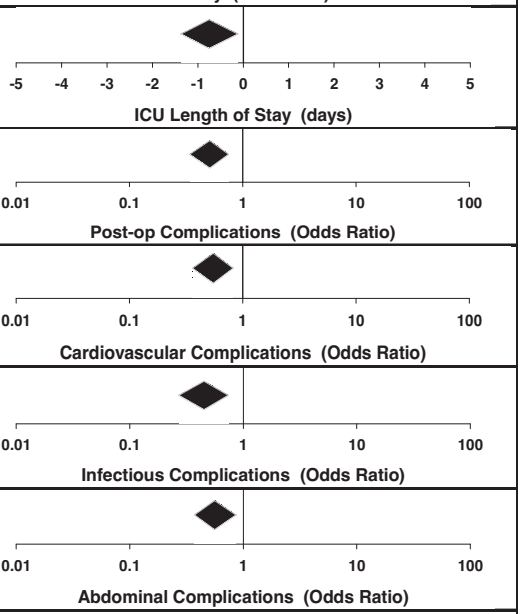
	Study characteristics	Dynamic Parameter	Outcome with goal-directed volume management versus standard care
Dave 2020 (17)	11 RCTs with 1,015 surgical patients	SVV	
Bednarczyk 2017 (18)	13 RCTs with 1,652 critically ill patients	SVV, PPV	
Benes 2014 (19)	14 RCTs with 961 post-surgical patients	SVV, PPV, SPV, PVI	

Figure 2: Clinical outcomes of goal-directed volume therapy compared with standard care from three meta-analyses. ICU = intensive care unit; PPV = pulse pressure variation; PVI = pleth variability index; SPV = systolic pressure variation; SVV = stroke volume variation. The total number of individual studies included in the three meta-analyses was 24, and the total number of patients included was 2770. Some studies were reported in multiple meta-analyses.

Table 3: Comparison of IVC CI to predict volume responsiveness in mechanically ventilated and spontaneously breathing patients.

	Reference	Level of evidence[c]	Number of studies	Number of patients	Sensitivity (%)	Specificity (%)
Mechanical ventilation	Meta-analysis Kim 2021 [25][a]	5	16	732	74	85
Mechanical ventilation	Pooled individual study data[b]		11	345	71	81
Spontaneous breathing	Meta-analysis Kim 2021 [25][a]	5	12	747	76	81
Spontaneous breathing	Pooled individual study data[b]		8	507	70	87
Standardized breathing	Preau 2017 [30]	4	3	226	89	92
	Bortolotti 2018 [31]	4				
	Caplan 2020 [32]	4				

IVC variability with respiration is calculated as (IVCmax – IVCmin)/IVC end-expiratory diameter [5]. With spontaneous breathing end-expiratory diameter is the IVCmax and CI is calculated as IVC CI = (IVCmax – IVCmin)/IVCmax while with mechanical ventilation end-expiratory diameter is the IVCmin and distensibility index (DI) is IVC DI = (IVCmax – IVCmin)/IVCmin. In hypovolemic patients, IVCmin may be very small resulting in a large IVC DI which is difficult to interpret. Since the same parameters are used to calculate IVC CI and IVC DI, these indices can be mathematically interconverted: IVC CI = IVC DI/(1 + IVC DI) and IVC DI = IVC CI/(1 – IVC CI) [5]. Interconversion to IVC CI allows comparisons of IVC variations in spontaneously breathing with mechanically ventilated patients. Some authors use the IVC mean diameter as the denominator to calculate the variability index (VI): IVC VI = (IVCmax – IVCmin)/IVCmean, and IVC CI = 2 * IVC VI/(2 + IVC VI) [5].

[a]The meta-analysis by Kim *et al.* [25] discusses reasons for the differences in their conclusions compared with meta-analyses by Orso *et al.* [26] and Si *et al.* [27] but not those of Long *et al.* [28] or Huang *et al.* [29].

[b]Individual studies from four meta-analyses by Orso *et al.* [26], Si *et al.* [27], Long *et al.* [28] and Huang *et al.* [29] chosen after excluding studies with pediatric or pregnant patients, or with non-verifiable data (true positives, true negatives, false negatives, false positives), or duplicates from the original publications, plus recent individual studies (Supplementary data, Table S2). Pooled sensitivities and specificities for included studies were then calculated.

[c]Supplementary data, Table S1.

Table 4: IVC CI to predict hypotension with anesthesia induction.

Reference	Level of evidence[a]	Type of anesthesia	Number of patients	Hypotensive (%)	IVC CI cut-off pre-induction (%)	Area under the ROC	SN (%)	SP (%)
Bhimsaria 2022 [36]	4	General	100	65	>50	0.82	71	80
Purushothaman 2020 [35]	4	General	50	30	>43	0.96	87	94
Rose 2022 [43]	4	General	120	42	>37	0.93	90	87
Zhang 2016 [24]	4	General	90	47	>43	0.90	79	92
Elbadry 2022 [42]	4	Spinal	55	47	>33	0.95	85	93
Ni 2022 [37]	2	Spinal	90	34	>42	0.83	84	76
Salama 2019 [38]	4	Spinal	100	45	>45	0.86	84	77
Saranteas 2019 [39]	4	Spinal	69	41	>30	0.77	82	61
Arican 2019 [40]	4	Conscious sedation	70	26	>45	0.85	83	83
Xu 2021 [41]	2	Conscious sedation	31	39	>37	0.68	82	61
Pooled data		10 studies	775	43	>30 to >50		82	81

[a]Supplementary data, Table S1.

ROC = receiver operator curve; SN = sensitivity; SP = specificity.

[43]. Comparison of cut-off values for the SCV CI to predict IVC CI cut-off at 30–45 degrees supine without deep breathing from four studies are shown in Table 5 [45, 48, 50, 51].

The ability of IJV CI to predict volume responsiveness at 30–45 degrees supine was similar to IVC CI in three paired studies, with good sensitivities and specificities (Table 6) [42, 52–54]. Of note, IJVmax was not significantly altered by increased intrathoracic pressure or increased intra-abdominal pressure [47].

Volume overloaded or not? Hypervolemia versus not-hypervolemia

Hemodialysis and ultrafiltration

Static peripheral parameters. Bioimpedance spectroscopy accurately estimates extracellular water and therefore volume, but cannot distinguish intravascular from extravascular volume. Multiple-frequency bioimpedance measurements for volume-based treatment of patients with end-stage renal disease was associated with significantly less hypervolemia defined by clinical findings than standard care in studies from five RCTs with 904 adults and 8 non-randomized studies with 4915 adult participants [55]. Pooled effects on systolic blood pressure, arterial stiffness and mortality were not statistically significant.

Static right heart/lung parameters. Lung ultrasound (LUS) assessment for B-lines is more sensitive than chest X-ray or auscultation [4] for assessing volume overload, and the number of B-lines increases proportionately to the degree of pulmonary edema. Lung congestion, symptomatic or asymptomatic, has a 45% prevalence in patients on maintenance hemodialysis [56]. Volume excess measured by bioimpedance spectroscopy only weakly relates to LUS findings in patients with kidney failure [56]. Severity of lung congestion correlates with death risk. In the Lung Water by Ultrasound-Guided Treat-

Table 5: Subclavian vein collapsibility index cut-offs at 30–45 degrees supine which predict IVC CI cut-offs.

	Reference	Level of evidence[a]	N pairs	SCV CI cut-off (%) for IVC CI <20%	SN/SP (%)	SCV CI cut-off (%) for IVC CI >50%	SN/SP (%)
Spontaneous breathing Heart failure	Kaptein 2022 [45]	4	36	<22	72	>33	78
Spontaneous breathing Renal failure	Kaptein 2020 [50]	4	160	<22	78	>39	79
Mechanical ventilation/ spontaneous breathing Surgical ICU patients	Kent 2013 [48]	4	94	<23	81	>40	85
Spontaneous breathing Cardiac disorders	Munir 2007 [51]	4	39	<32	81	>39	82

[a]Supplementary data, Table S1.
ICU = intensive care unit; N = number; SCV CI = subclavian vein/proximal axillary vein collapsibility index; SN = sensitivity; SP = specificity.

Table 6: Comparison of IJV CI versus IVC CI to predict volume responsiveness or post-anesthesia hypotension.

	Reference	Level of evidence[b]	N pts	Target goal	% positive[a]	Patient position	IJV CI cut-off (%)	SN (%)	SP (%)	IVC CI cut-off (%)	SN (%)	SP (%)
Mechanical ventilation	Ma 2018 [52]	4	70	Volume responsiveness	50	30°	>12	91	83	>13	86	86
Mechanical ventilation	Guarracino 2014 [53]	4	50	Volume responsiveness	60	30°	>15	80	95			
Spontaneous breathing	Haliloğlu 2017 [54]	4	44	Volume responsiveness	52	45°	>36	78	85	>35	78	85
Spontaneous breathing	Elbadry 2022 [42]	4	55	Post-spinal, hypotension	47	Supine	>36	85	83	>33	86	86

[a]Percent positive for target goal
[b]Supplementary data, Table S1.
N = number, SN = sensitivity, SP = specificity.

ment in Hemodialysis Patients (LUST) trial, an international RCT including 367 patients followed for 1.5 years, volume management guided by LUS safely relieved lung congestion and reduced the number of hypotensive episodes during dialysis, but failed to significantly reduce risk of the pre-defined endpoints including death, nonfatal myocardial infarction and decompensated heart failure [56]. As in three trials of patients with heart failure, a *post hoc* analysis of the LUST trial showed that use of LUS reduced the risk for repeated episodes of acute decompensated heart failure (ADHF) and repeated cardiovascular events [56].

Dynamic venous parameters. IVC ultrasound reflects intravascular volume but not extravascular volume. IVCmax and IVC CI were used to optimize volume removal rate during slow continuous ultrafiltration (SCUF) while avoiding hypotension, in 24 diuretic-resistant patients with acute decompensated heart failure (ADHF) [57]. IVC CI increased significantly from 12% to 24% after SCUF. Hypotension was observed only in the 2 of 24 patients whose IVC CI increased to >30%. IVC ultrasound is a rapid, simple and non-invasive means for bedside monitoring of intravascular volume during SCUF. Serial IVC measurements may be useful to optimize volume removal and avoid hypotension.

Ultrafiltration volume recommended was based upon multiple factors including pre-dialysis IVC ultrasound (Fig. 3a) [58]. In a retrospective study of 113 patients in 244 encounters receiving intermittent hemodialysis within 24 h of IVC ultrasound measurement (Fig. 3b and c) [58], IVC CI was a better predictor of ultrafiltration volume achieved (Fig. 3b) than IVCmax. Static parameters including CVP, PAOP and cardiac output were poor predictors of ultrafiltration volume achieved [58]. Net ultrafiltration volume achieved progressively decreased compared with net ultrafiltration recommended as severity of intra-dialytic hypotension increased (Fig. 3c).

Changes in cardiac output measured by thermodilution were related to IVC ultrasound-guided volume management and net volume balance in a retrospective study of 22 critically ill patients in 37 intermittent hemodialysis and 21 continuous hemodialysis encounters [12]. Net volume changes between cardiac output measurements were estimated from isonatremic volume equivalent gains and losses [12, 59, 60]. Cardiac output increased >10% in 15 of 42 encounters with IVC CI <20% after net volume removal, and in 1 of 16 encounters with IVC CI >20% after net volume administration ($P < .014$), despite intra-dialytic hypotension in all encounters (Fig. 3d).

In 30 maintenance hemodialysis patients, IVC ultrasound findings from before, during and after dialysis compared reasonably with pre-hemodialysis and post-hemodialysis bioimpedance spectroscopy measurements, and predicted intra-dialytic hypotension [mean arterial pressure (MAP) decrease >30 mmHg and/or MAP <70 mmHg] [61]. Hypovolemia, defined as IVC CI >50% with IVCmax <2.1 cm, was associated with an almost 14-fold risk of intra-dialytic hypotension, and had the advantage of repeated assessment during hemodialysis to predict and potentially prevent intra-dialytic hypotension.

Combination of ultrasound parameters: static and dynamic venous

When assessed together, IVC ultrasound and LUS provide a more accurate assessment of intravascular and extravascular volume excess.

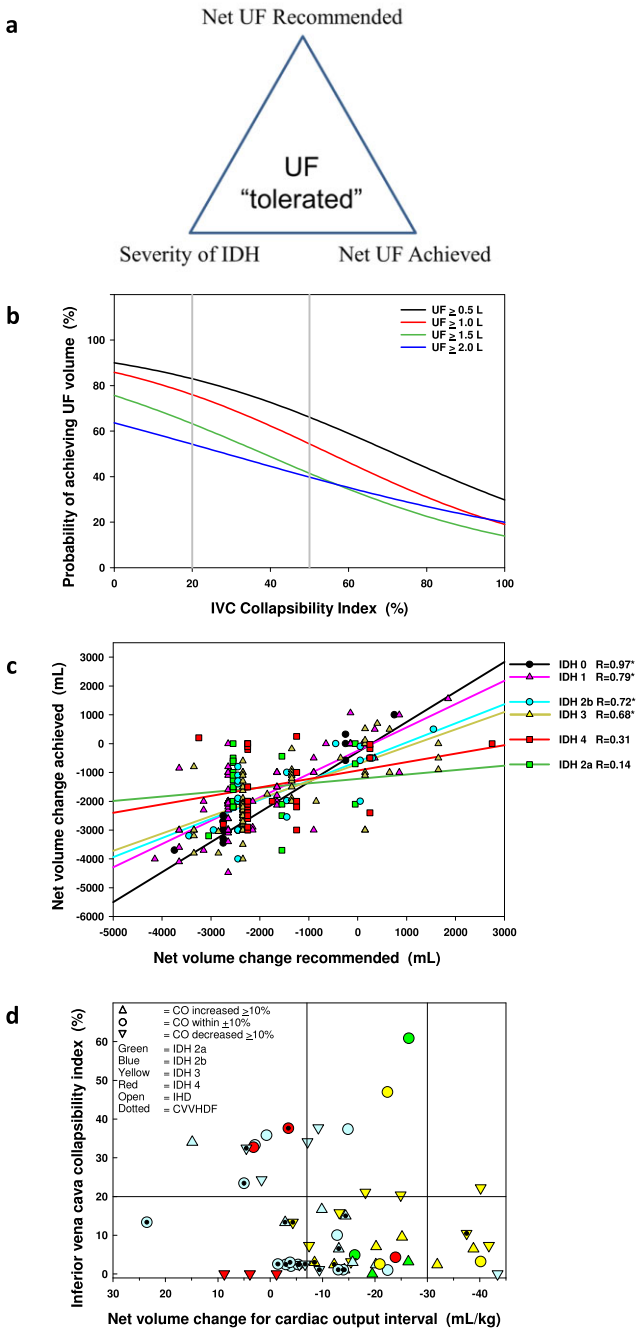


Figure 3: Relationship of IVC CI, net UF volume recommended, net UF volume achieved and IDH in critically ill hemodialysis patients. (**a**) Conceptual schematic of the relationship of UF tolerated, UF recommended, net UF achieved and IDH. Clinical and laboratory factors as well as assessment of intravascular volume by ICV CI were taken into account when recommending net UF goals. (b) Relationship of the probability of achieving UF volume to the IVC CI. Data from 244 encounters for 113 patients [58]. (**c**) Relationship of net ultrafiltration volume achieved versus recommended as limited by severity of intra-dialytic hypotension. Regressions significant at $P = .05$ level are indicated with an asterisk. Data from 244 encounters for 113 patients [58]. Classification of severity of IDH in critically ill patients from Kaptein *et al.* [58] is as follows: IDH 0: no criteria for IDH; IDH 1: saline (0.9%) >500 mL or albumin intravenously; IDH 2a: MAP <65 mmHg during hemodialysis without vasopressors; IDH 2b: pre-hemodialysis hypotension requiring a constant dose of vasopressors; IDH 3: systolic blood pressure decreased >50 mmHg or MAP decreased >20%; IDH 4a: vasopressor therapy initiated or dose increased; or IDH 4b: dialysis stopped ≤2 h due to intractable hypotension. Category assigned was the highest level of severity for each encounter. (**d**) Changes in cardiac output related to inferior vena cava collapsibility, net volume change and IDH. Data from 58 encounters for 22 patients [12]. IVC collapsibility was categorized as <20% or >20%. Net volume change was arbitrarily divided as >30 mL/kg removed, 7–30 mL/kg removed or <7 mL/kg removed or volume added. Severity of IDH categories 2a, 2b, 3 and 4, respectively, are indicated in the legend to panel (b). For IVC CI <20%, cardiac output increased >10% in 15 of 42 (36%), increased <10% or decreased <10% in 14 of 42 (33%) and decreased >10% in 13 of 42 (31%) despite IDH in all encounters [12]. CO = cardiac output; IDH = intra-dialytic hypotension; IHD = intermitent hemodialysis, CVVHDF = continuous veno-venous hemodiafiltration; UF = ultrafiltration.

Estimating "dry weight" in stable hemodialysis outpatients. Dry weight was estimated in 74 patients on biweekly hemodialysis using clinical parameters for 2 weeks followed by 2 weeks integrating IVC ultrasound and LUS performed pre-hemodialysis and 30 min after hemodialysis [62]. New dry weight, defined using IVC CI >50% and fewer than four B-lines in eight sites at 30 min post-dialysis, was achieved in 43%, with fewer symptoms related to volume overload or depletion. Combined IVC US plus LUS may facilitate dry weight estimation, and predict and minimalize intra-dialytic hypotension.

Predicting intra-dialytic hypotension in critically ill patients. In a prospective study of 248 critically ill patients receiving intermittent hemodialysis, IVC CI and LUS performed immediately before dialysis to predict intra-dialytic hypotension (MAP <65 mmHg) showed that IVC CI was >40% in 76% with intra-dialytic hypotension and in 8% without intra-dialytic hypotension ($P < .001$) [63]. No pulmonary congestion (B-line score <14) was present in 52% with intra-dialytic hypotension and in 37% without intra-dialytic hypotension ($P = .030$). Absence of hypervolemia, assessed by LUS and IVC CI, was predictive of intra-dialytic hypotension. Multiple logistic regression showed MAP, use of norepinephrine and IVC CI >40% were the only factors associated with intra-dialytic hypotension.

Acute decompensated heart failure. In 80 patients with ADHF, volume status was assessed by IVC CI and LUS upon emergency department presentation and after 3 hours of treatment [64]. IVC CI increased from 19% to 25% ($P = .001$) and number of B-lines decreased in all zones ($P = .001$). Those hospitalized had lower IVC CI and more B-lines than those discharged. Assessment of IVC CI and B-lines was better than B-type natriuretic peptide levels, ejection fraction or chest X-ray for diagnosing and assessing severity of ADHF, and guiding hospitalization and discharge decisions.

In a multi-center prospective study of 314 patients with ADHF [65], heart failure with reduced ejection fraction (HFrEF) patients had a higher initial volume overload assessed by lower IVC CI and more B-lines compared with heart failure with preserved ejection fraction (HFpEF). With diuretic treatment, HFrEF patients had a greater increase in IVC CI with a parallel decrease in B-lines compared with HFpEF patients. Serial IVC US and LUS assessment in patients with ADHF may facilitate optimizing diuretic therapy and minimizing adverse consequences of overdiuresis.

In a systematic review of 24 studies with 1900 hospitalized ADHF patients [66], a smaller IVC CI was associated with a 2.5-fold higher risk of readmission and higher mortality, and more B-lines correlated with a 1.5-fold increased risk of mortality and readmission.

Prognostic significance of IVC US and LUS was assessed in 389 elderly patients admitted with ADHF (PROFUND-IC Registry Analysis) [67]. Sixty-seven percent with IVC CI <50% had 2.3-fold increased admissions in the last year, 3.1-fold higher in-hospital mortality and 2.8-fold greater 30-day mortality ($P < .04$). Sixty-six percent with more than six B-lines per field had a 2.4-fold increased 30-day mortality ($P = .01$). Logistic regression showed IVC CI >50% best predicted survival at 30 days (odds ratio for mortality 0.359, $P = .034$).

Concurrent IVC US and LUS may be useful in assessing treatment response, guiding hospitalization and discharge decisions, and predicting readmission and mortality in ADHF patients.

Deresuscitation. In 40 critically ill patients, post-resuscitation volume removal guided by cardiac, IVC and lung ultrasound, improved efficacy of deresuscitation, compared with 45 patients with routine clinical care [68]. Volume removal began when volume expansion or large dose norepinephrine (>0.3 mg/kg/min) was not needed, with IVCmax >2.0 cm and IVC CI <12% with mechanical ventilation, or IVC CI <20% with spontaneous breathing, plus more than one B-line-positive region on LUS, and stopped with IVCmax <2.0 cm and IVC CI >13% with mechanical ventilation or IVC CI >40% with spontaneous breathing and less than one B-line-positive region on LUS. Ultrasound guided volume management resulted in significantly earlier onset and more rapid completion of volume removal, more daily volume removal and urine output, and more rapid and complete resolution of B-lines.

Venous congestion. Venous excess Doppler ultrasound (VExUS) reflects effects of increased RAP and interstitial edema within encapsulated kidneys and other abdominal organs, and may play a role in assessing and guiding volume management (Tables 2 and 7) [69].

Multi-parametic estimation of RAP including E/E′ and VExUS has not been shown to be more precise than estimates based on IVC ultrasound alone [13]. When the IVC ultrasound suggests elevated RAP (IVCmax >2.1 cm and IVC CI <50%), severe flow abnormalities in hepatic, portal and kidney parenchymal veins assessed by VExUS are associated with adverse outcomes including increased risk of AKI, since interstitial edema decreases renal blood flow [69, 70], as well as heart failure progression and cardiac death in patients with congestive heart failure or undergoing cardiac surgery [15]. In the general intensive care unit population, portal pulsatility index was associated with major adverse kidney events at 30 days, including mortality and persistent impairment of kidney function [15]. In a prospective study, resolution of AKI significantly correlated with improvement in VExUS grade, and change in the VExUS grade significantly correlated with volume balance [71]. A combined grading of ultrasound of the IVC, hepatic vein and portal vein might more reliably demonstrate venous congestion and aid in the clinical decision to perform volume removal in patients with AKI and cardiorenal syndrome [71] or acute respiratory distress syndrome [72].

Modification of VExUS to compare hepatic vein waveforms during inspiration and with apnea has been proposed as a dynamic way to guide volume management in cardiorenal syndrome and acute respiratory distress syndrome [73, 74].

CLINICAL INTERPRETATION OF IVC ULTRASOUND

The 2010 American Society of Echocardiography guidelines for interpretation of IVC ultrasound, with minor additional comments or modifications, are presented in Table 7 [15, 75]. For many sick patients who cannot sniff, including on mechanical ventilation, IVC CI <20% is considered inconsistent with intravascular hypovolemia, provided the IVC maximum diameter is not "small" [75]. When the "IVCmax is small and collapsing, this suggests hypovolemia" [75]. An IVCmax <2.1 cm with CI >50% is inconsistent with intravascular hypervolemia. Based on data from four publications each with more than 50 extractable data points (total $n = 298$) of spontaneously breathing patients, 80% with RAP <5 mmHg had an IVC CI >47%, and 90% with RAP >20 mmHg had an IVC CI <20%; these cut-offs

Table 7: Interpretation of IVC ultrasound diameters adapted from 2010 American Society of Echocardiography guidelines [75].

IVCmax diameter	IVC CI	Maneuvers	Interpretation	Approximate RAP	Comments
"Small"	"Collapsed"	Without sniff	Not hypervolemic	<10 mmHg	Rudski *et al.* [75] suggest this applies to mechanically ventilated patients, and IVCmax <1.2 is "small" in this context. We apply this concept also to patients who are not mechanically ventilated. Further investigation is warranted into the cut-off for "small" IVCmax that indicates total collapse, and is inconsistent with intravascular hypervolemia, regardless of CI
<2.1 cm	>50%[a]	With sniff or without	Not hypervolemic	0–5 mmHg 3 mmHg	In a review of 4 publications with more than 50 extractable data points of patients who did not sniff (total $n = 298$), optimal sensitivity (80%) and specificity (79%) for predicting a mean RAP <5 mmHg were obtained at a cut-off for IVC CI of >47.3% (approximately 50%) [58, 76–79]
<2.1 cm	<50%	With sniff	Intermediate	5–10 mmHg 8 mmHg	In indeterminate cases, an intermediate value may be used, or, preferably, secondary indices of elevated RAP should be integrated. These include restrictive right-sided diastolic filling pattern, tricuspid E/E′ ratio >6, and diastolic flow predominance in the hepatic veins (which can be quantified as a systolic filling fraction <55%). In indeterminate cases, if none of these secondary indices of elevated RAP are present, RAP may be downgraded to 3 mmHg
>2.1 cm	>50%	With sniff	Intermediate	5–10 mmHg 8 mmHg	
>2.1 cm	<50%	With sniff	Not hypovolemic	10–20 mmHg 15 mmHg	VExUS may be confirmatory for suspected volume overload [15]
"Not small"	<35%	With sniff	Not hypovolemic	15 mmHg	And secondary indices of elevated RAP are present
"Not small"	<20%	Without sniff	Not hypovolemic	"Elevated"	In patients who are unable to perform a sniff, IVC collapse <20% with quiet inspiration suggests an elevated mean RAP

have been applied clinically in our patients who do not sniff [58, 76–79].

CLINICAL UTILITY OF CARDIAC AND IVC ULTRASOUND

Patients with large IVCmax and small IVC CI are unlikely to have reduced cardiac filling pressures or overt hypovolemia, while those with small IVCmax or large IVC CI are unlikely to have elevated cardiac filling pressures or overt hypervolemia. A good general rule when one encounters intermediate values for respiratory variation of IVC diameters is to proceed based on other available parameters and clinical judgement.

Acute kidney injury

In patients with AKI, assessment of intravascular volume and cardiac function is required to determine the most likely cause as well as to guide volume therapy. The diagnostic performance of multi-organ point-of-care ultrasound for AKI etiological subgroups was investigated in 165 emergency department patients [80]. IVCmax >1.8 cm predicted AKI with reduced cardiac output (sensitivity 100%, specificity 70%). IVCmax <1.8 cm predicted hypovolemic AKI (sensitivity 81%, specificity 57%). IVC CI was not evaluated.

Cirrhosis and AKI

In cirrhosis with ascites and AKI, hepatorenal syndrome (HRS) is a diagnosis of exclusion. Hypovolemic AKI is less likely when AKI fails to improve after at least 2 days of intravenous albumin with diuretic withdrawal [81]. Velez *et al.* [82] reported 64% of 53 patients initially presumed to have HRS-AKI had intravascular hypovolemia or hypervolemia, or intra-abdominal hypertension by IVC ultrasound assessment, and 35% improved AKI following IVC ultrasound-guided volume management, making the diagnosis of HRS-AKI unlikely. In our study of 20 patients presumed to have HRS-AKI, 75% had intravascular hypovolemia or hypervolemia by IVC ultrasound [83]. Forty percent improved AKI with additional IVC ultrasound-guided volume management and had been misdiagnosed as HRS-AKI. Assessing intravascular volume in cirrhotic patients presumed to have HRS-AKI may improve diagnostic accuracy and guide further volume management to improve AKI.

Dysnatremias

Accurate assessment of intravascular and extravascular volume in patients with hypotonic hyponatremia is key to diagnosis and management but difficult to determine using clinical and laboratory findings [84–86]. In eight case reports of hypo-osmolar hyponatremia, clinical and laboratory volume estimates were discordant with ultrasound assessment of

Table 8: Proposed approach to volume assessment of patients with hypotonic hyponatremia.

Hypotonic hyponatremia	Hypovolemic	"Euvolemic"	Hypervolemic	Mismatch
Extravascular volume assessed by ultrasound	No B-lines on LUS No ascites, pleural or peritoneal fluid No subcutaneous edema[a]	No B-lines on LUS No ascites, pleural or peritoneal fluid No subcutaneous edema[a]	B-lines on LUS, or Ascites, pleural or peritoneal fluid, or Subcutaneous edema[a]	B-lines on LUS, or Ascites, pleural or peritoneal fluid, or Subcutaneous edema[a]
Intravascular volume assessed by ultrasound	Decreased	Normal or increased	Normal or increased	Decreased
Cause	Renal or extra-renal loss	Hypothyroid, hypoadrenal, hypopituitary, SIAD	Nephrosis, cirrhosis, heart failure	Nephrosis, cirrhosis or heart failure Plus renal or extra-renal losses

[a]Detection of subcutaneous edema is more sensitive by ultrasound than by physical examination [87].
SIAD = syndrome of inappropriate anti-diuresis.

Table 9: Ultrasound to differentiate the types of shock (RUSH protocol).

Rapid Ultrasound in Shock (RUSH) protocol: ultrasonographic findings seen with classic shock states				
RUSH evaluation	Hypovolemic shock	Cardiogenic shock	Obstructive shock	Distributive shock
Pump	Hypercontractile heart Small chamber size	Hypocontractile heart Dilated heart	Hypercontractile heart Pericardial effusion Cardiac tamponade Right ventricular strain Cardiac thrombus	Hypercontractile heart (early sepsis) Hypocontractile heart (late sepsis)
Tank	Flat IVC Flat jugular veins Peritoneal fluid (fluid loss) Pleural fluid (fluid loss)	Distended IVC Distended jugular veins Lung rockets (pulmonary edema) Pleural fluid Peritoneal fluid	Distended IVC Distended jugular veins Absent lung sliding (pneumothorax)	Normal or small IVC (early sepsis) Peritoneal fluid (sepsis source) Pleural fluid (sepsis source)
Pipes	Abdominal aneurysm Aortic dissection	Normal	Deep vein thrombosis	Normal

Perera 2010 [90], with permission.

intravascular or extravascular volume in all [86]. Multi-organ ultrasound may more accurately define intravascular and extravascular volume to enhance diagnostic accuracy and guide volume management of hypotonic hyponatremia (Table 8) [86, 87].

Shock and hypotension

The Rapid Ultrasound in SHock (RUSH) protocol enables rapid assessment and therapy of undifferentiated hypotension (Table 9) [88–90]. The RUSH examination has a sensitivity of 85% and specificity of 95% for identifying the cause of undifferentiated hypotension [88, 89, 91]. Clinical application of the RUSH protocol resulted in improved 28-day patient survival, a reduction in stage 3 AKI, and more days alive and free of renal support [92].

IVC ULTRASOUND PEARLS AND PITFALLS

Technical aspects of IVC ultrasound

The IVC is typically visualized in the long-axis from the subcostal view, but can also be viewed in the mid-axillary line [5]. Inter-rater reliability may be improved by training and experience [93]. The aorta may be mistaken for the IVC if the IVC is collapsed with intravascular volume depletion. The aorta can be identified by visualizing both vessels [5].

Factors that affect IVC diameter or collapsibility

IVC diameters and IVC CI may be altered by a number of factors, resulting in over-estimation or under-estimation of intravascular volume, which are summarized in Table 10 [5, 45, 93]. If the patient has factors that tend to cause IVC distention and yet the IVC is small or collapsing, intravascular hypervolemia is unlikely, and vice versa.

Factors that affect IVC visibility

Inadequate subcostal IVC visualization is reported in 3%–20% of studies [67, 94] due to reasons listed in Fig. 4.

Alternate views

SCV CI and IJV CI may be easier to obtain than IVC CI and useful when IVC visualization is difficult [43, 48]. Respiratory

Table 10: Factors that affect IVC diameter or collapsibility.

	IVC CI	IVCmax	Comments
Overestimate intravascular volume			
Cardiac tamponade	↓	↑	Blocks forward flow
Severe valvular stenosis	↓	↑	Blocks forward flow
Massive pulmonary embolism	↓	↑	Impairs LV filling
Right ventricular myocardial infarction	↓	↑	Impairs LV filling
Severe tricuspid regurgitation [45]	↓	↑	Impairs LV filling
High PEEP	Minimal Δ	↑	Blocks forward flow
Decreased tidal volume	↓	No Δ	Decreased pressure changes
Decreased inspiratory effort/shallow breathing	↓	No Δ?	Decreased pressure changes. Highly collapsible IVC indicates not hypervolemic
Underestimate intravascular volume			
Increased tidal volume (ventilated)	↑	No Δ?	Increased pressure changes
Increased inspiratory effort moving probe "in and out" of field (diaphragmatic breathing)	↑	No Δ	Not on center of cylinder. Can try mid-axillary or cross-sectional views
Increased inspiratory effort/deep breathing (sniff)	↑	No Δ	Negative intrathoracic pressure pulls more blood forward into heart
Valsalva maneuver	↑	↓	Increased abdominal pressure decreases flow to IVC
Intra-abdominal hypertension	No Δ	↓	Large IVCmax with no collapse indicates not hypovolemic
Late term pregnancy, supine position	↑	↓	Gravid uterus compresses IVC and decreases venous return. IVC CI decreases, and IVCmax increases in 15° left lateral decubitus position compared with supine position [95, 96]
Off-center scan (cylinder tangent effect) [93]	Minimal Δ	↓	Not on center of cylinder. Try to maximize diameter
Extracorporeal blood (hemodialysis, continuous renal replacement therapy)	↑?	↓?	Decreased IVV during procedure which increases after blood is returned

Adapted from Kaptein and Kaptein 2021 [5].
IVV = intravascular volume; LV = left ventricular; PEEP = positive end-expiratory pressure.

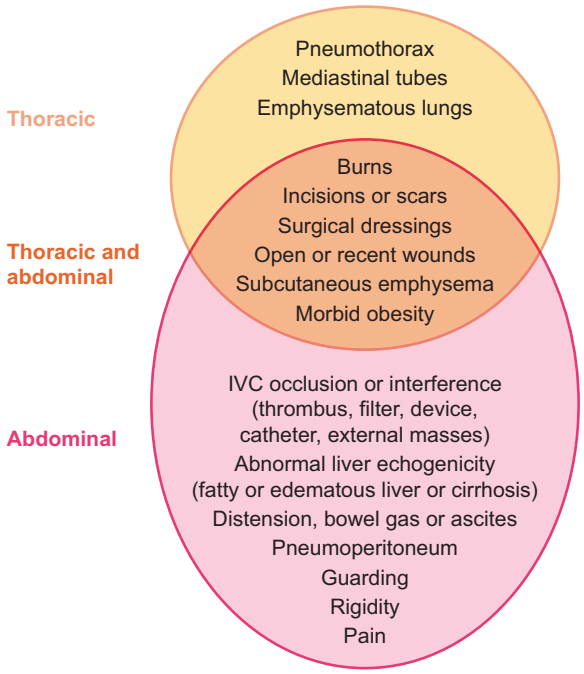


Figure 4: Factors that limit IVC visualization by ultrasound.

variations of distal SCV and IJV diameters by ultrasound have been assessed as alternative sites, and should be performed at a 30–45 degree upper body incline [45].

Limitation of the quality of the data

The quality of the data (Supplementary data, Table S1) in the cited literature varies and this has to be taken into consideration.

CONCLUSION

There is a growing body of evidence indicating improved clinical outcomes including morbidity and mortality with dynamic parameter-guided volume assessment and management compared with standard care in post-operative and critically ill patients.

Of the many methods for assessing volume status, point-of-care ultrasound stands out for being non-invasive and versatile. Ultrasound can be used in any clinical context from office visits, to hospital wards, to intensive care units. IVC ultrasound is useful for answering both whether patients may be intravascularly volume depleted or not, and whether patients may be volume overloaded or not. IVC CI has been validated in both ventilated and non-ventilated patients, in contrast to arterial pressure waveform analysis. IVC ultrasound may be combined with other ultrasound techniques to assess intravascular as well as extravascular volume status. Some ultrasound techniques are more labor intensive or technically involved than others. IVC ultrasound is relatively simple, and can be performed rapidly. Additional ultrasound techniques can be used to non-invasively estimate changes in cardiac output in response to dynamic tests or volume changes.

Limitations of IVC ultrasound and other point of care ultrasound techniques must be recognized and taken into account. Other clinical factors may impair ultrasound

visualization, which in many cases may be overcome by a wide repertoire of alternative ultrasound windows and techniques, including ultrasound assessment of the heart, lungs, central veins and organ congestion.

SUPPLEMENTARY DATA

Supplementary data are available at *ckj* online.

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AUTHORS' CONTRIBUTIONS

Both authors contributed to the research and writing of the article. Both authors read and approved the final version.

CONFLICT OF INTEREST STATEMENT

None declared.

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DATA AVAILABILITY STATEMENT

No new data were generated or analysed in support of this research.

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