

From Probe to Scope: Bedside Echocardiography in the Assessment of Shock A Focused State-of-the-Art Narrative Review of Multimodal Point-of-Care Ultrasound for Haemodynamic Phenotyping, Physiology-Guided Therapy and Serial Reassessment

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ABSTRACT:

Circulatory shock is an acute, life threatening state of tissue hypoperfusion caused by hypovolemic, cardiogenic, obstructive, or distributive which requiring different treatment. Guidelines now recommended echocardiography as the first line diagnostic tool. This review aimed to synthesize evidence on point of care ultrasound (POCUS), combining cardiac, lung, venous, and abdominal imaging for phenotyping shock and guiding bedside hemodynamics management. A focused narrative review searched in the MEDLINE/PubMed, Semantic Scholar, Scopus and preprint repositories. Keywords used were shock physiology, cardiac ultrasound, fluid responsiveness and tolerance, and right ventricular assessment. Pooled analysis from 1,132 patients demonstrated POCUS identifies shock etiology with 0.78 to 0.90 sensitivity and 0.92 to 0.98 specificity. LVOT velocity time integral served as a stroke volume surrogate and 10 percent rise after passive leg raising predicted fluid responsiveness (AUC 0.91

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to 0.95). Lung ultrasound, inferior vena cava assessment, and the VExUS score identified fluid tolerance, while right ventricular size and TAPSE/PASP ratio identified high mortality phenotypes. In conclusion, bedside echocardiography should function as a repeated physiological test answering the shock phenotype, flow status, and fluid tolerance.

Keywords: Circulatory shock, Point of care ultrasound, fluid responsiveness, right ventricular function.

Introduction

Background

Circulatory shock is defined as acute circulatory failure resulting in inadequate cellular oxygen utilisation, and the defining consensus statements of the European Society of Intensive Care Medicine emphasise that arterial hypotension is not required for the diagnosis: what matters is the presence of tissue hypoperfusion, recognised clinically through altered mentation, mottled or cold skin, oliguria and a raised blood lactate. Approximately one in three patients admitted to an intensive care unit is in shock at some point during the stay, and the syndrome remains among the strongest predictors of multiorgan failure and death. The Sepsis-3 criteria formalised the most common subtype, defining septic shock as sepsis with vasopressor-dependent hypotension and a lactate above 2 mmol/L after adequate fluid resuscitation, a group in which pooled mortality approaches half of all patients.¹⁻³

The syndrome is conventionally divided into four mechanisms: hypovolaemic, cardiogenic, obstructive and distributive. However this classification is clinically useful only if the mechanism can be identified quickly, because the correct first intervention differs radically between them. A litre of crystalloid is life-saving in haemorrhagic hypovolaemia and potentially lethal in tamponade-associated obstructive shock or in decompensated left ventricular failure; a vasopressor is the correct first response to vasoplegia and may be counterproductive if it masks unrecognised preload dependence. Bedside clinical assessment, however, performs poorly at this discrimination, and reliance on static surrogates such as central venous pressure

has been explicitly discouraged for two decades.^{1,2,4}

The 2025 ESICM guidelines on circulatory shock and haemodynamic monitoring make the modern position explicit: echocardiography is suggested as the first-line imaging modality to determine the type of shock, dynamic variables are preferred over static markers of preload for predicting fluid responsiveness, and cardiac output or stroke volume should be monitored in patients who do not respond to initial therapy. In parallel, the guidelines endorse bedside markers of skin perfusion, notably capillary refill time, complemented by skin temperature and mottling. Bedside echocardiography therefore no longer sits at the periphery of resuscitation; it is embedded in the recommended diagnostic pathway.⁴

The diagnostic case is strong. A systematic review and meta-analysis of twelve studies including 1,132 patients reported pooled sensitivity and specificity of 0.82 and 0.98 for obstructive shock, 0.78 and 0.96 for cardiogenic shock, 0.90 and 0.92 for hypovolaemic shock and 0.79 and 0.96 for distributive shock, with an area under the receiver operating characteristic curve of approximately 0.95 for each subtype and a positive likelihood ratio of 40 for obstructive shock. A broader systematic review of 89 studies concluded that POCUS increases diagnostic accuracy in dyspnoea, non-traumatic hypotension and shock, narrows the differential diagnosis and shortens the time to correct diagnosis and effective treatment. In a prospective emergency department cohort, an ultrasound hypotension protocol reduced aggregate diagnostic uncertainty by 27.7 per cent and changed the use

of fluids, vasoactive agents or blood products in a quarter of patients.^{5,8,26}

The outcome case is more nuanced, and this tension is the intellectual centre of the present review. A systematic review and meta-analysis of eighteen randomised controlled trials found that POCUS-guided resuscitation probably reduces 28-day mortality (relative risk 0.88, 95% confidence interval 0.78 to 0.99), shortens the duration of vasoactive medication and improves lactate clearance, yet the largest multicentre randomised trial of a POCUS protocol in undifferentiated hypotension, the SHoC-ED trial, found no difference in survival, length of stay, imaging use or fluid administration. A prospective multicentre observational study across 178 United Kingdom critical care units found that only 54 per cent of patients admitted with shock received an echocardiogram within 72 hours, that the scan reduced diagnostic uncertainty or changed management in 54 per cent of those examined, and that only a quarter of studies complied with national governance and image-storage standards.^{6,7,9}

Two further developments have reshaped the field. First, the recognition that fluid accumulation is itself an injurious exposure: a meta-analysis of 31 observational studies and 31,076 patients found that fluid overload and positive cumulative fluid balance are

independently associated with mortality, with risk rising by roughly 19 per cent for every additional litre of positive balance, and deresuscitation strategies have been associated with improved survival in fluid-overloaded patients. Second, the emergence of fluid tolerance as a concept distinct from fluid responsiveness, a distinction with direct bedside consequences, because a multicentre proof-of-concept study found signs of venous congestion in roughly half of both fluid-responsive and fluid-unresponsive critically ill patients.^{12,13,14,78}

Finally, the resuscitation paradigm itself has moved from fixed protocolised targets towards personalised, phenotype-driven strategies applied as reversible tests within short decision cycles. ANDROMEDA-SHOCK-2 which randomised 1,467 patients in 86 centres across 19 countries, operationalised exactly this approach, capillary refill time, pulse pressure, diastolic pressure, fluid responsiveness testing and bedside echocardiography used together to tailor fluids, vasopressors and inotropes. These demonstrated superiority over usual care for a hierarchical composite outcome (win ratio 1.16, 95% confidence interval 1.02 to 1.33), driven mainly by a shorter duration of vital support. Bedside echocardiography is a structural component of that strategy rather than an optional adjunct.^{15,22}

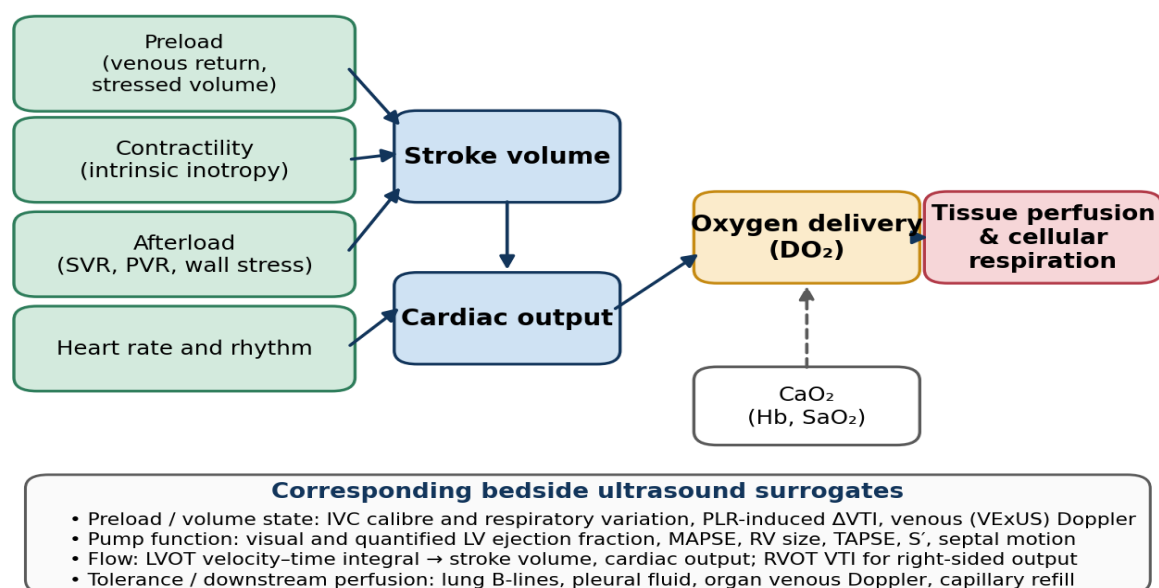


Figure 1. Physiological determinants of tissue perfusion and the bedside ultrasound variables that interrogate each step. Stroke volume is the product of preload, contractility and afterload; multiplied by heart rate it yields cardiac output, which together with arterial oxygen content determines global oxygen delivery. Each determinant has a corresponding sonographic surrogate, which is what allows a bedside examination to be read as physiology rather than as anatomy. Sources: ^{1,2,4,19}

Objectives

The general objective of this review is to synthesise contemporary guideline, consensus and clinical-trial evidence into a coherent, physiology-based framework for the use of bedside echocardiography and multimodal POCUS in the assessment and management of the patient with shock. The specific objectives are: (i) to restate the haemodynamic first principles that make sonographic variables interpretable; (ii) to define the standard views, strengths and limitations of focused cardiac ultrasound in this population; (iii) to organise the examination within the Pump Tank Pipes construct; (iv) to describe the quantification of stroke volume and cardiac output through the LVOT velocity time integral; (v) to separate fluid responsiveness from fluid tolerance and to specify how each is measured; (vi) to detail right ventricular assessment; (vii) to characterise the sonographic phenotypes of the shock subtypes; (viii) to translate findings into therapeutic decisions; (ix) to formalise serial reassessment; (x) to catalogue pitfalls; and (xi) to propose an integrated clinical decision algorithm suitable for the emergency department, the operating theatre and the intensive care unit.^{4,19,23,25}

Significance and expected benefits

For the clinician, the review provides an explicit, reproducible sequence that links each sonographic finding to a therapeutic decision and to a defined reassessment interval, which is precisely the element most often missing from published POCUS protocols. For the institution, it supplies the conceptual basis for a governance framework covering image acquisition, archiving, reporting and competence, deficiencies in which have been documented in large observational work. For the researcher, it identifies where evidence is strong (diagnostic accuracy,

prediction of preload responsiveness), where it is weak (outcome benefit of congestion-guided de-resuscitation, operator-competence thresholds) and therefore where trials are needed.^{6,9,10,84}

Hypothesis

The working hypothesis of this review is that the clinical value of bedside echocardiography in shock is determined less by its diagnostic accuracy which is already high and reproducibly demonstrated than by whether it is embedded in a closed decision loop. Specifically, we propose that a structured, serially repeated, multimodal POCUS examination that integrates flow (LVOT VTI), preload responsiveness (passive leg raising) and fluid tolerance (lung, inferior vena cava and venous Doppler), and that is coupled to explicit therapeutic rules and to a mandated re-scan after every meaningful intervention, will produce better patient-centred outcomes than either single-snapshot POCUS or non-ultrasound usual care. The positive hierarchical composite result of ANDROMEDA-SHOCK-2 and the mortality signal in the meta-analysis of POCUS-guided resuscitation are consistent with, though not definitive proof of, this hypothesis.^{6,15}

Research gap

Four gaps recur across the literature. First, an outcome and accuracy dissociation: diagnostic performance is excellent, yet randomised evidence of benefit is mixed and derives largely from small single-centre trials, while the highest-quality multicentre trial was neutral. Second, a protocol-heterogeneity gap: published protocols differ in views obtained, in the number of lung zones scanned and in whether quantitative flow measurement is mandatory, which makes pooled estimates difficult to interpret. Third, a competence gap: most trials do not report operator training level, agreement between novices and experts is only moderate for some

parameters, and systematic review evidence shows measurable decay of ultrasound competence within 1 to 12 months, with image acquisition skills deteriorating most. Fourth, a deresuscitation gap: congestion indices such as VExUS correlate well with right atrial pressure and predict adverse renal outcomes in cardiac populations, yet associations are inconsistent in general and septic ICU cohorts and no adequately powered trial has yet shown that congestion-guided de-resuscitation improves outcomes.^{6,7,9,10,54-57,83,84}

Novelty

The novelty of this review lies in three deliberate departures from the existing literature. It treats fluid responsiveness and fluid tolerance as two orthogonal axes that must both be measured before any bolus, rather than as a single 'volume status' question; it integrates the 2025 ESICM guideline recommendations, the ANDROMEDA-SHOCK-2 personalised strategy and the most recent VExUS meta-analytic evidence, which have not previously been synthesised together in a single bedside framework; and it presents a serial-reassessment loop with defined triggers and expected directional changes, converting POCUS from a diagnostic snapshot into a monitoring modality. The proposed algorithm in **Figure 3** is, to our knowledge, the first to combine obstructive-cause exclusion, quantitative flow measurement, dual responsiveness–tolerance assessment and mandated re-scanning in one operational sequence.^{4,14,15,56}

2. Methodology

This work is a focused state-of-the-art narrative review. It does not claim the protocol-registered completeness of a systematic review; rather, it follows an explicit and reproducible search and selection strategy designed to identify the highest-quality contemporary evidence bearing on each of the twelve thematic questions defined a priori by the review objectives.^{4,19}

Information sources and search strategy

Structured queries were executed in the academic search engine, which indexes MEDLINE/PubMed, Semantic Scholar, Scopus and preprint repositories, with the medical-journal filter enabled so that retrieval was restricted to high-ranking clinical journals and clinical practice guidelines. Twelve domain-specific searches were run, corresponding to the twelve sections of the narrative review: shock definition and haemodynamic monitoring; diagnostic accuracy of POCUS in undifferentiated shock; standard views and training in focused cardiac ultrasound; LVOT velocity time integral and non-invasive cardiac output; passive leg raising and dynamic predictors of fluid responsiveness; inferior vena cava assessment and its limitations; lung ultrasound, B-lines and the BLUE/FALLS protocols; venous excess ultrasound and organ congestion; right ventricular function, TAPSE and RV pulmonary arterial coupling; sepsis-induced cardiomyopathy and echocardiographic phenotypes; fluid overload, deresuscitation and the ROSE model; and competence, governance and outcome trials of POCUS-guided resuscitation.^{4,6}

Eligibility criteria

- Included: international guidelines and consensus statements, systematic reviews with or without meta-analysis, randomised controlled trials, prospective and multicentre observational cohorts, and authoritative state-of-the-art or narrative reviews written by recognised methodological groups when they defined a concept (for example the FALLS protocol or the VExUS construct).
- Excluded: exclusively paediatric or neonatal populations, except where cited explicitly for contrast, single case reports and small case series without physiological or methodological novelty, conference abstracts reporting the same cohort as a subsequent full publication, and non-shock populations unless the physiological principle under discussion was transferable.

- **Verification:** every reference retained was checked to have a resolvable public record and a valid digital object identifier, and both the record link and the DOI are reproduced in the reference list.

Priority was given to publications from 2014 onwards, corresponding to the first ESICM consensus on circulatory shock, with earlier landmark studies retained where they remain the primary source of a concept. As an example, the original description of the BLUE protocol and the foundational passive leg raising studies.^{1,35,38,48}

Synthesis

Evidence was extracted into a thematic matrix mapping each source to one or more of the twelve review domains, to its study design and to the specific quantitative estimate it contributed (for example a pooled sensitivity, an area under the curve, a diagnostic threshold or an effect estimate). Quantitative estimates are reported with their original confidence intervals. Where sources conflicted, most conspicuously for the prognostic value of VExUS in general versus cardiac surgical populations, and for the outcome benefit of POCUS-guided resuscitation, both positions are presented, and the methodological reasons for divergence are stated rather than resolved by selective citation. Figures and tables in this review are original didactic syntheses constructed from the numerical values reported in the cited primary sources; no figure has been reproduced from a copyrighted publication.^{5-7,56,57}

3. Results

Physiological Framework: From Flow to Phenotype

Shock is a disorder of flow before it is a disorder of pressure. Global oxygen delivery is the product of cardiac output and arterial oxygen content. Cardiac output is the product of stroke volume and heart rate. Stroke volume is determined by preload, myocardial contractility and afterload. Mean arterial pressure is the product of cardiac output and systemic vascular resistance, which is

the reason of a normal or even elevated pressure can coexist with profoundly inadequate flow and hypotension is neither necessary nor sufficient for the diagnosis of shock.^{1,2}

Preload is best understood as the stressed volume that drives venous return rather than as a volume of blood. This distinction explains volume-overloaded in total body terms yet preload-dependent in cardiac terms and that the inferior vena cava is an unreliable single measure of the state. The Frank-Starling curve translates preload into stroke volume, where the steep portion indicates preload dependence and the plateau defines preload independence. The exact position and slope determined by contractility and afterload.^{1,4,45}

Two key principles of this curve guide fluid resuscitation. First, only patients on the steep portion increase their stroke volume after a fluid bolus. Second, in patients on the plateau, a fluid infusion only elevates filling pressures. This pressure transmitted backwards to systemic venous circulations and forwards into the tissues, causing harmful interstitial edema. A meta-analysis of 31 observational studies with 31,076 patients found fluid overload and positive cumulative balance independently associated with mortality, with the risk of death rising by approximately 19 per cent per litre of positive balance.^{12,42,47}

Afterload is systematically under-measured: an identical arterial pressure can be produced by opposite combinations of flow and resistance, so pressure alone cannot guide the choice between vasopressor and inotrope. Ventriculo-arterial coupling has therefore been proposed as a target of critical care ultrasound protocols.^{19,20} Macrocirculatory correction also does not guarantee microcirculatory correction. Capillary refill time which responds within minutes of change in systemic flow, has become a favored resuscitation target. In ANDROMEDA-SHOCK, a capillary refill time targeted-strategy produced less organ dysfunction at 72 hours than a lactate-targeted strategy.^{15,21,22,80}

Table 1. Haemodynamic profiles of the four mechanisms of shock and their expected bedside sonographic correlates^{1,2,4,19}

Mechanism	Cardiac output	Preload	SVR	Venous pressure	Typical causes and sonographic correlate
Hypovolaemic	Low	Low	High	Low	Haemorrhage, gastrointestinal or renal losses; small hyperkinetic LV, collapsing IVC, low LVOT VTI, A-profile lungs
Cardiogenic	Low	High	High	High	Myocardial infarction, decompensated cardiomyopathy, valvular catastrophe; dilated hypokinetic LV, plump IVC, low VTI, bilateral B-profile
Obstructive	Low	Variable	High	High	Tamponade, massive pulmonary embolism, tension pneumothorax; pericardial effusion with chamber collapse, RV dilatation, absent lung sliding
Distributive	Normal or high	Low or normal	Low	Low or normal	Sepsis, anaphylaxis, neurogenic, liver failure; hyperkinetic LV, normal or high VTI despite hypotension, variable IVC
Mixed	Variable	Variable	Variable	Variable	Septic shock with sepsis-induced cardiomyopathy, cardiogenic shock with secondary infection; discordant findings requiring serial reassessment

Notes: IVC, inferior vena cava; LV, left ventricle; LVOT, left ventricular outflow tract; RV, right ventricle; SVR, systemic vascular resistance; VTI, velocity–time integral. Adapted conceptually from consensus classifications of circulatory shock and from ultrasound-based haemodynamic protocols.

Focused cardiac ultrasound (FoCUS) differs from comprehensive echocardiography in purpose rather than in equipment. It answers a small set of binary, time-critical questions, and is performed by the treating clinician in real time. A division of labour explicitly endorsed by The European Association of Cardiovascular Imaging and given graded recommendation by The

Society of Critical Care Medicine's for preload responsiveness in mechanically ventilated patients, left ventricular systolic function, acute cor pulmonale, symptomatic pulmonary embolism, pericardial effusion, and tamponade.^{16,17} The core sequence comprises the parasternal long-axis, parasternal short-axis, apical four-chamber, apical five-chamber and subcostal views, extended to the lungs, abdomen, and the deep veins. The parasternal long axis gives left ventricular size, global function, the pericardium and the outflow tract; the short axis gives septal shape and radial function; the apical four-chamber gives the right-to-left ventricular relationship and tricuspid annular motion; the

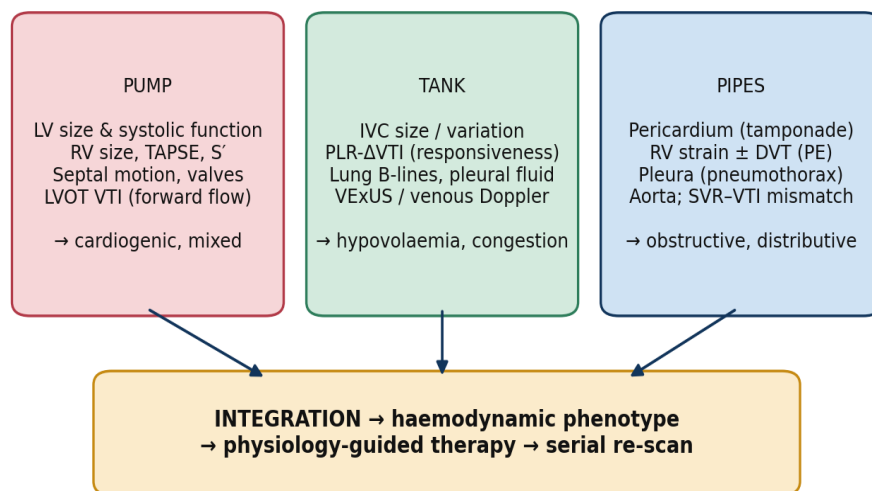
apical five-chamber allows the Doppler quantification of flow; and the subcostal view is the rescue window when parasternal and apical windows fail, as they often do in ventilated or obese patients. A visual estimate of global ejection fraction should be part of every examination.^{24,25} Structured protocols raise diagnostic accuracy from roughly 60 per cent with clinical examination alone to about 85 per cent, and alter management in up to half of patients. In cardiac arrest and peri-arrest states, focused echocardiography identifies coordinated cardiac motion in a substantial fraction of patients labelled asystolic or in pulseless electrical activity, and changed management in 78 per cent of cases in a prospective pre-hospital trial.^{5,25,75}

The limitations are equally specific. Image quality is operator- and patient-dependent; in one large multicentre echocardiographic study of ventilated patients the inferior vena cava could not be visualised in 22 per cent, and outflow-tract Doppler was obtainable in only about 78 per cent, whereas superior vena cava assessment by the transoesophageal route succeeded in almost all. Interpretation is vulnerable to the same physiological confounders that limit any single haemodynamic variable, including mechanical ventilation, arrhythmia, raised intra-abdominal pressure and isolated right or left ventricular dysfunction. And, critically, a focused study yields a set of findings rather than a diagnosis: it must be integrated with the history, examination, blood

gas, lactate and response to therapy. Where the transthoracic window fails and the patient is intubated, resuscitative transoesophageal echocardiography is a safe and high-yield alternative, changing management in 85 per cent of patients scanned for undifferentiated shock in a multicentre registry of 1,045 studies.^{42,43,74}

The Pump-Tank-Pipes approach

The Pump–Tank–Pipes construct is a mnemonic discipline rather than a physiological theory. The Pump is the myocardium: left and right ventricular size and function, septal motion, and valves. The Tank is the volume compartment: the inferior vena cava, the response to a preload challenge, and the downstream evidence of over-filling. The Pipes are the pericardium, the pulmonary vasculature, the pleura and the arterial tree.^{19,25} Reading the three compartments together, rather than sequentially, is what turns images into a phenotype (**Table 1**). Structured protocols that formalise this reading, namely RUSH, ACES, SESAME, converge on the same triage logic (**Figure 1**).^{19,25,26} A practical refinement is the Tank must be evaluated on two axes rather than one. A multicentre cross-sectional study of 90 mechanically ventilated patients, vasopressors-dependent patients found venous congestion in 53 per cent of fluid-responsive and 57 per cent of fluid-unresponsive patients. Responsiveness and tolerance are therefore two independent questions and the Tank assessment must answer both.¹⁴



The three compartments are examined in a fixed sequence, but interpreted together; no single window is diagnostic in isolation.

Figure 2. The Pump-Tank-Pipes framework for bedside interrogation of the circulation. The three compartments are examined in a fixed sequence but interpreted jointly; the integration step, not the individual image, generates the haemodynamic phenotype that determines therapy. Sources: ^{14,19,25,26}

Quantifying stroke volume and cardiac output: the LVOT velocity–time integral

The left ventricular outflow tract velocity–time integral (LVOT VTI) is the single most useful quantitative bedside measurement. It is the distance blood travels per beat and multiplying it by the outflow tract and heart rate yields stroke volume and cardiac output. Emergency physicians with focused training can obtain optimal VTI measurements in about 78% of patients, correlating at 0.82 with a certified sonographer.²⁸ Because outflow tract diameter

barely changes over hours, serial VTI alone tracks serial stroke volume, and a VTI and heart rate only formula correlates more strongly with thermodilution ($r = 0.81$) than the traditional formula ($r = 0.70$).^{30,31} As a bedside rule of thumb, VTI below about 15 cm indicates a low-flow state, 18 to 22 cm is normal range, and values above 22 to 25 cm with hypotensive patient point to vasoplegia.^{32,33,64} Averaging at least three beats in sinus rhythm and five in atrial fibrillation keeps the least significant change between studies to 11–14 per cent (**Table 2**).²⁹

Table 2. Practical framework for LVOT VTI measurement in the shocked patient^{28,29,30,31,32,33}

Element	Recommended practice	Consequence if neglected
Window	Apical five-chamber; sample volume 0.5–1.0 cm proximal to the aortic valve, in the same position on every repeat study	Sampling at the valve hinges or tips systematically over-estimates VTI and cardiac output
Doppler alignment	Keep the insonation angle below approximately 20°; do not apply angle correction	Velocity and therefore stroke volume are under-estimated as the cosine of the angle
Number of beats	≥3 consecutive beats in sinus rhythm; ≥5 in atrial fibrillation	Beat-to-beat variability is misread as a treatment effect

Absolute interpretation	<15 cm low flow; 18–22 cm typical normal; >22–25 cm with hypotension suggests vasoplegia	Low-output states are missed when only blood pressure is used
Change interpretation	Treat changes below approximately 11–14 per cent as within measurement noise	Spurious 'responsiveness' leads to unnecessary fluid
Cross-sectional area	Measure once, mid-systole, inner-edge to inner-edge; omit for pure trending	Squaring the radius doubles any diameter error in the computed stroke volume

Notes: LVOT, left ventricular outflow tract; VTI, velocity–time integral. Derived from validation and precision studies of echocardiographic cardiac output measurement.

Fluid responsiveness means the ventricles sits on the steep portion of the Frank–Starling curve, so a preload challenge will meaningfully raise stroke volume. Because roughly half of critically ill patients are not fluid responsive, guidelines recommend assessing before continuing fluid resuscitation.^{4,39} Passive leg raising is the most broadly applicable dynamic tests, reversible and valid regardless of ventilator synchrony or sinus rhythm. Across meta-analyses with over 1,000 patients, it showed pooled AUC of 0.91–0.95.^{35–37} Three methodological points determine whether the test performs as published. The effect must be measured with a flow variable, not pulse pressure (sensitivity otherwise falls to 56 to 58 per cent). It must start from a 45° semi-recumbent, not-supine (43 per cent of true responders are misclassified from supine). The haemodynamic

must be measured at its one-minute peak.^{35,36,38} Coupled to LVOT VTI in mechanically ventilated septic shock, the manoeuvre predicts responsiveness with an AUC of 0.956 (sensitivity 87.5 per cent, specificity 95 per cent), outperforming stroke volume variation and pulse pressure variation.^{34,40}

Among the comparative hierarchy of dynamic tests, the tidal volume challenge (area under the curve 0.96) and the end-expiratory occlusion test (0.95) performed best but require controlled ventilation, passive leg raising is intermediate but uniquely applicable across ventilation modes and rhythms, and inferior vena cava respiratory variation is least reliable (**Table 3**).^{36,37,47} In the FRESH randomised trial, resuscitation guided by the stroke volume change during passive leg raising produced a 1.37 L lower fluid balance at 72 hours, less renal replacement therapy (5.1 versus 17.5 per cent) and less mechanical ventilation (17.7 versus 34.1 per cent) than usual care in septic hypotension.⁴¹

Table 3. Diagnostic performance and practical constraints of tests used to predict fluid responsiveness^{35,36,37,40,44,47}

Test	Pooled AUC	Usual threshold	Principal constraints
Tidal volume challenge	0.96	$\Delta PPV \geq 3.5$ points	Requires controlled ventilation without spontaneous effort; transient increase of tidal volume to 8 mL/kg
End-expiratory occlusion	0.95	$\Delta CO \geq 5\%$	Requires 15–30 s occlusion tolerance; needs a precise, continuous flow measurement
Passive leg raising	0.91–0.95	ΔCO or $\Delta VTI \geq 10\%$	Blunted by intra-abdominal hypertension; must start semi-recumbent; measure at peak effect

Stroke volume variation	0.88–0.90	$\approx 12\%$	Invalid with spontaneous breathing, arrhythmia, low tidal volume or low lung compliance
Pulse pressure variation	0.87	$\approx 12\%$	Same constraints as SVV; performance falls at tidal volume < 8 mL/kg
Δ IVC (respiratory variation)	0.82–0.83	12–18% ventilated; $>40\%$ spontaneous	Confounded by intra-abdominal pressure, RV dysfunction, PEEP; should not be used alone
Mini-fluid challenge	0.84–0.91	Δ VTI ≥ 5 –10%	Requires a precise flow measurement able to detect a small change

Notes: AUC, area under the receiver operating characteristic curve; CO, cardiac output; IVC, inferior vena cava; PEEP, positive end-expiratory pressure; PPV, pulse pressure variation; RV, right ventricle; SVV, stroke volume variation; VTI, velocity–time integral. Values pooled from meta-analytic and umbrella-review sources.

Fluid tolerance and congestion: integrating lung ultrasound, the inferior vena cava and VExUS

Fluid tolerance asks whether the circulation and the organs can accommodate added volume without incurring congestion-mediated injury. Venous congestion signals are present in comparable proportions of fluid-responsive and fluid-unresponsive patients, a positive responsiveness test is a necessary but not sufficient condition for giving fluid.^{14,79}

Lung ultrasound

Lung ultrasound is the most direct sonographic index of the consequence of over-filling. The BLUE protocol demonstrated that predominant A-lines with lung sliding identify obstructive airway disease (89% sensitivity, 97% specificity) while diffuse anterior B-lines with sliding identify haemodynamic pulmonary oedema (97% sensitivity, 95% specificity) for an overall correct diagnosis in 90.5% of patients with acute respiratory failure. The FALLS protocol extends this to circulatory failure, once tamponade, RV enlargement and pneumothorax are excluded, an A-profile indicates that the lung is still dry enough for fluid, while the appearance of anterior B-lines

during infusion marks the end-point of fluid therapy, corresponding physiologically to a pulmonary artery pressure near 18 mmHg.⁴⁸⁻⁵⁰ B-lines are a quantitative rather than binary, their number and spatial extent allow semi-quantitative estimation of extravascular lung water, the assessment is reproducible after brief training, and an expert consensus checklist standardizes reporting given how much equipment, position, zone number, and counting method vary across studies. Prognostically, B-lines across at least half of the examined lung fields at 24 hours carry a more than fourfold increase in 30-day mortality in cardiogenic shock with reduction over the first 24 hours associated with lower mortality.⁵¹⁻⁵³

The inferior vena cava

The inferior vena cava remains the most performed and the most over-interpreted single measurement in shock. Its respiratory variation was validated in narrow populations of fully ventilated, deeply sedated septic patients with tidal volumes of 8 mL/kg or more, where a distensibility index above 18% predicted responsiveness with 90% sensitivity and specificity. Performance deteriorates sharply outside those conditions, a meta-analysis of twelve studies and 753 patients showed AUC falling from 0.88 (Tidal volume > 8 mL/kg, PEEP < 5 cmH₂O) to 0.70 outside that range and specificity dropping from 0.94 to 0.68. In spontaneously breathing patients, high collapsibility above 40% usually indicates responsiveness, but low collapsibility does not exclude it.^{44,46} A 540 ventilated patients

multicentre study found the end-expiratory unobtainable in 22%, a diameter of 13 mm or less predicting responsiveness and 25 mm or more predicting non-responsiveness with acceptable specificity, but 71% of patients fell into an uninformative intermediate zone with intra-abdominal pressure acted as a confounder. A widely cited commentary enumerated ten distinct situations in which inferior vena cava indices may mislead, including right ventricular failure, raised intra-abdominal or intrathoracic pressure, forceful spontaneous inspiration and pulmonary hypertension. The measurement is therefore best used as one input at its extremes and never as a standalone determinant of fluid therapy.^{43,45}

Venous excess ultrasound (VExUS)

The VExUS grading system formalises systemic venous congestion by combining inferior vena cava calibre with hepatic, portal and intrarenal veins Doppler wave forms into 0 (none) to 3 (severe) grade, with strong physiological validity correlated with right atrial pressure ($R^2 = 0.68$) and predicting RAP ≥ 12 mmHg with an AUC of 0.99 (versus 0.79 for IVC diameter alone). In a second cohort, VExUS grades of 2 and 3 independently associated with higher RAP, mPAP, and PCWP with an AUC of 0.90 for RAP ≥ 10 mmHg in a second.^{54,55} Its prognostic

evidence is more heterogeneous: a meta-analysis of 32 studies and 3,142 patients found moderate-to-good accuracy for elevated central venous and right atrial pressures (sensitivity 78-95%, specificity 80-90%), and in cardiac populations, a VExUS grade ≥ 2 associated with acute kidney injury (OR 4.44) and mortality (OR 3.17), yet no clear association with either outcome in general critical ill population (OR 1.45 and 1.25, respectively). A null finding echoed in a 145 patients general ICU cohort with low congestion prevalence (16% and 6% moderate-to-severe) and no link to AKI or 28-day mortality.^{56,57} The most coherent explanation is that VExUS tracks the interaction between venous return and cardiac function rather than volume itself. Patients with the highest scores often have cardiogenic physiology despite a low fluid balance, while real volume excess and preserved cardiac function may score low. The score remains useful for monitoring decongestion, where serial scores fall with successful diuresis and severe congestion has been associated with worsening renal function, blunted natriuretic response, diuretic resistance, and higher in-hospital mortality in acute heart failure. Whether a VExUS-guided decongestive strategy improves outcomes, remains the principal evidence gap.⁵⁸⁻⁶⁰

Table 4. The VExUS grading system and the evidence supporting each component^{54,55,56,57,58}

Component	Normal pattern	Mildly abnormal	Severely abnormal
Inferior vena cava	< 2 cm (gate criterion: grading applied only if ≥ 2 cm)	≥ 2 cm	≥ 2 cm
Hepatic vein Doppler	Systolic wave larger than diastolic (S > D)	S < D, both below baseline	Systolic wave reversed above baseline
Portal vein Doppler	Continuous flow, pulsatility < 30%	Pulsatility 30–49%	Pulsatility $\geq 50\%$
Intrarenal venous Doppler	Continuous flow	Biphasic (systolic and diastolic components)	Monophasic, diastolic-only flow

Composite grade	Grade 0–1: no or mild congestion	Grade 2: moderate congestion	Grade 3: severe congestion
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Grade 3 corresponds to a plump inferior vena cava plus at least two severely abnormal venous waveforms.

VExUS correlates strongly with right atrial pressure ($R^2 \approx 0.68$; AUC 0.99 for right atrial pressure ≥ 12 mmHg) and, in cardiac populations, with acute kidney injury and mortality; associations are weaker in general critical care cohorts.

Assessment of the right ventricle

Right ventricular dysfunction is common, under-recognised and disproportionately lethal in shock: present in 48% of a 393 septic shock patient cohort scanned within 24 hours and independently associated with a more than threefold increase in 28-day mortality (unlike LV dysfunction, present in 63% but not independently prognostic). A meta-analysis of 65 studies and 17,008 septic patients confirmed a peak prevalence of RV dysfunction of 47% within 48 hours and a relative risk of death of 1.62.^{61,73} Bedside assessment proceeds in three layers: Size (RV/LV end-diastolic area ratio > 0.6 dilated, > 1.0 severe which with systolic septal flattening defines acute cor pulmonale), function (TAPSE < 16 –17 mm and tissue Doppler S' < 9.5 cm/s, with TAPSE correlated best with RV EF among the standard indices ($\rho = 0.78$)), and Load since RV is exquisitely afterload-sensitive and interpretable only against the load it faces.^{61,62}

Load assessment is where practice has advanced most: the ratio TAPSE/PASP ratio as a non-invasive surrogate of RV–pulmonary arterial coupling, and independently predicted lower mortality (Hazard ratio 0.927 per unit) in 1,077 intensive care patient sub-study. A pulmonary artery acceleration time ≤ 100 ms obtained in 81% of septic patient flags raised pulmonary vascular resistance when tricuspid regurgitation velocity was unmeasurable.^{62,63} RV outflow tract VTI offers a right-sided analogue of stroke distance, independently associated with ICU and six-month mortality in pulmonary embolism.⁶⁴ Visual grading agrees well with fractional area changes for experts (kappa 0.73) but only moderate for novices (0.55), with septal motion concordance particularly low (0.28). Therefore, quantitative measurement should supplement visual estimation wherever feasible.⁶⁵ Distinguishing acute from chronic pressure overload via free-wall thickness, the 60/600 sign, McConnell's sign, and acceleration time thresholds, changes management directly with focused cardiac ultrasound is 92–100% sensitive for pulmonary embolism in patients with abnormal vital signs.^{66–68}

Table 5. Right ventricular parameters, thresholds and interpretation in the shocked patient^{61,62,63,64,65,66}

Parameter	Abnormal threshold	Interpretation and caveats	View
RV/LV end-diastolic area ratio	> 0.6 dilated; > 1.0 severe	With systolic septal flattening defines acute cor pulmonale; foreshortening inflates the ratio	A4C
TAPSE	< 16 –17 mm	Best correlate of RV ejection fraction among standard indices; angle- and translation-dependent; may be normal in radial-predominant dysfunction	A4C, M-mode

Tissue Doppler S'	< 9.5 cm/s	Load-dependent; complements TAPSE	A4C
Fractional area change	< 35%	Reference standard for visual grading concordance	A4C
TAPSE / PASP (or TAPSE/TRV)	< 0.31–0.36 mm/mmHg	Surrogate of RV–PA coupling; independently associated with ICU mortality	A4C + CW Doppler
Pulmonary acceleration time	≤ 60–100 ms	Suggests raised pulmonary vascular resistance; measurable when TR velocity is not	PSAX / RVOT
RVOT VTI	Low values (context-dependent)	Right-sided stroke distance; independently associated with mortality in critical pulmonary embolism	PSAX / RVOT
McConnell's sign	Present	Free-wall hypokinesis with apical sparing; supports acute rather than chronic overload	A4C

Notes: A4C, apical four-chamber; CW, continuous wave; LV, left ventricle; PA, pulmonary artery; PASP, pulmonary artery systolic pressure; PSAX, parasternal short axis; RV, right ventricle; RVOT, right ventricular outflow tract; TAPSE, tricuspid annular plane systolic excursion; TR/TRV, tricuspid regurgitation velocity; VTI, velocity–time integral.

Phenotyping shock with POCUS

Phenotyping is the point at which the examination becomes actionable. Pooled 12 meta-analytic studies and 1,132 patients demonstrate an AUC approximately 0.95 and a negative likelihood ratio of about 0.2 across subtypes. Obstructive shock shows the strongest positive likelihood ratio of 40, fitting since it is the subtype in which delay is least forgivable.⁵

Hypovolaemic shock

Hypovolemic shock presents a small hyperkinetic left ventricle with collapsible IVC, a low LVOT VTI, and an A-profile (sensitivity 0.90, specificity 0.92). This is the one configuration in which fluid is both responsive and tolerated, though it should be confirmed with a preload test rather than assumed.^{5,25}

Cardiogenic shock

The signature is a dilated, hypokinetic left ventricle, with reduced EF, a low VTI, plethoric IVC, and bilateral B-lines (sensitivity 0.78, specificity 0.96). B-lines burden at 24 hours independently predicts 30-day mortality (hazard ratio 4.7 in the 185 patient Altshock 2 registry). Contemporary expert recommendations places echocardiographic phenotyping and standardised staging at the centre of management.^{5,53,76,77}

Obstructive shock

Obstructive shock covers three distinct lesions: Tamponade, Massive pulmonary embolism (LVOT VTI below 15 cm identifies a pre-cardiopulmonary-failure), and Tension pneumothorax. Pooled sensitivity and specificity for obstructive shock are 0.82 and 0.98, respectively.^{5,33,66,69}

Distributive shock

Distributive shock is largely a diagnosis of exclusion, a hyperkinetic left ventricle with normal-to-high VTI despite hypotension (sensitivity 0.79, specificity 0.96), often complicated by the coexistence of vasoplegia and myocardial depression.^{5,27}

Mixed shock and sepsis-induced cardiomyopathy

Sepsis-induced cardiomyopathy is now understood as a spectrum encompassing LV systolic dysfunction (reported in 5-32%), LV diastolic dysfunction (20-57%), hyperdynamic systolic states and RV injury (18-79%), occurring alone or in combination with a U-shaped mortality relationship in which both EF below 25% (OR 2.75) and an EF of 70% or more (OR 1.70) carry higher in-hospital mortality than mid-range reference. Meanwhile, hyperdynamic function alone, present in about 18% of septic patients, carried more than double the odds of death.⁷⁰⁻⁷² The practical consequence is that a 'normal-looking' or vigorous left ventricle should never close the assessment, since it may reflect profound vasoplegia, occult preload dependence or unrecognised RV failure. Only the combination of chamber sizes, flow, tolerance, and re-scanning distinguishes between these possibilities.^{70,73}

POCUS-guided treatment: from image to prescription

The therapeutic value of the examination lies in the rules that connect a finding to a prescription spanning four categories of decision: whether to give fluid, whether and which vasoactive agent to start, whether to remove fluid, and whether a causal intervention is required. The 2025 ESICM guidelines provide the scaffolding: assess fluid responsiveness before continuing fluid resuscitation, prefer dynamic over static preload variables, monitor cardiac output or stroke volume in non-responders, and use echocardiography as the first-line modality to determine the type of shock.⁴ Fluid decisions follow the two-axis rule: fluid is given, in 250-500 mL aliquots with the test repeated after each, only when the patient is both responsive and tolerant. Fluid is withheld when unresponsive and withheld or given only in minimally with early

vasopressor support when the patient is responsive but intolerant. Non-resuscitation fluid is equally important, since maintenance solutions and drug diluents, not boluses, account for the largest share of cumulative positive balance in critically ill patients.^{14,78,79} Vasoactive decisions follow the flow measurement: (1) hypotension with a normal or high VTI and no pump or obstructive pathology indicates vasoplegia and calls for early vasopressor initiation to avoid futile fluid accumulation, (2) hypotension with a low VTI and a dilated hypokinetic ventricle indicates pump failure and raises the question of an inotrope once preload and afterload are optimized, (3) hypotension with a low VTI and a small hyperkinetic ventricle indicates preload dependence, (4) in septic shock with a high-normal EF and low VTI should raise suspicion of dynamic LVOT obstruction which inotropes and further vasodilatation are harmful.^{19,20,25}

De-resuscitation decisions follow the tolerance axis in reverse. In an 400 observational study across ten ICUs, day-3 fluid balance was an independent risk factor for 30-day mortality (OR 1.26 per litre) while active deresuscitation to a negative balance was associated with lower mortality. Serial VExUS grades falling with successful decongestion offer a rational, though not yet outcome-validated, monitoring signal for the evacuation phase of the ROSE model.^{13,60,78} Causal decisions are the most immediately life-saving and the least contested, including pericardiocentesis for tamponade, reperfusion for massive pulmonary embolism, decompression for tension pneumothorax, revascularisation for infarct-related cardiogenic shock, and source control for sepsis. These are also the decisions in which POCUS most clearly changes practice, repeatedly uncovered unsuspected life-threatening diagnoses and altered management in one quarter to one half of patients with undifferentiated hypotension.^{8,9,25}

Table 6. From sonographic finding to therapeutic decision^{4,13,14,19,25,78}

Dominant finding	Inferred physiology	First-line action and the variable used to verify it
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Small hyperkinetic LV, collapsing IVC, VTI < 15 cm, A-profile	Preload dependence	Fluid in 250–500 mL aliquots; verify with $\Delta VTI \geq 10\%$ after each aliquot
Dilated hypokinetic LV, plump IVC, VTI < 15 cm, B-profile	Pump failure with congestion	Inotrope $\pm$ vasopressor, avoid fluid, consider decongestion; verify with ΔVTI and B-line reduction
Hyperkinetic LV, VTI normal or high, persistent hypotension	Vasoplegia	Vasopressor early; verify with MAP and capillary refill rather than with more fluid
Circumferential effusion with chamber collapse	Tamponade	Pericardiocentesis; verify with re-expansion of chambers and rise in VTI
Dilated RV, septal flattening, McConnell, low RVOT VTI	Acute cor pulmonale	Reperfusion pathway; cautious fluid, noradrenaline, avoid over-filling the RV
Absent lung sliding with lung point	Tension pneumothorax	Immediate decompression; verify with return of sliding
Responsive but VExUS ≥ 2 or new B-lines	Congested yet preload-dependent	Minimal or no fluid, early vasopressor, reassess; consider decongestion once perfusion is stable
Unresponsive, VExUS ≥ 2 , positive balance	Fluid accumulation	Active de-resuscitation with diuretics or ultrafiltration; verify with serial VExUS and perfusion markers

Notes: IVC, inferior vena cava; LV, left ventricle; MAP, mean arterial pressure; RV, right ventricle; RVOT, right ventricular outflow tract; VExUS, venous excess ultrasound; VTI, velocity–time integral.

Serial reassessment

Shock is a moving target, and a single examination describes only the instant in which it was performed. This is the most plausible explanation for the discrepancy between the diagnostic and the outcome literature: trials that used POCUS once, at presentation, largely failed to demonstrate benefit, while incorporating ultrasound into routine daily rounds in sepsis has been associated with lower cumulative fluid balance, shorter mechanical ventilation, and shorter ICU stay, and the personalised strategy tested in ANDROMEDA-SHOCK-2 mandated repeated assessment within short decision

cycles.^{6,7,15} Reassessment should be triggered by events rather than by the clock alone, including after every fluid bolus, after any change in vasopressor or inotrope dose, after intubation or a change in positive end-expiratory pressure, after any procedure intended to relieve an obstructive cause, whenever the clinical trajectory changes, and at least once daily thereafter. The variables re-measured should be the same ones on which the original decision was based, since interpreting a change requires an identical measurement technique, the same window, the same sample position, the same number of averaged beats.²⁹

Interpretation must respect measurement precision. A change in VTI below approximately 11% for a single operator, or 14% between operators, lies within measurement noise and should not be treated as a therapeutic effect. Similarly, capillary refill time has a least significant

change of about 23 to 25% but responds quickly, with measurable improvement 6-8 minutes after the start of a fluid infusion and maximal effect at 10 to 12 minutes, making it a useful rapid companion signal to the sonographic variables. Discordance between an improving flow variable and a static or worsening perfusion marker suggests macro-to-microcirculatory uncoupling and identifies a group with higher mortality in whom further macrocirculatory escalation is unlikely to help.^{22,29,80}

Pitfalls and limitations

Operator dependency is the dominant limitation. International training standards distinguish general critical care ultrasonography, basic and advanced critical care echocardiography, and the ESICM has defined a core competency set of fifteen echocardiographic, five thoracic, and four abdominal skills. Focused training works: a three-hour course with five hours practice brought non-cardiologist residents good-to-excellent agreement against experts on binary questions, and residents averaging 33 transthoracic studies achieved kappa values above 0.80 against experienced operators for fluid, inotrope, and vasopressor decision, though only moderate agreement pursuing a negative fluid balance.^{81-83,85} Competence decays quickly, however, a review of 31 studies and 1,638 participants documented deterioration across all competency domains within 1-12 months, worst for image

acquisition (median -11.8%) and steepest after courses of four hours or less, consistent with only a quarter of echocardiograms performed in a large multicentre cohort meeting national governance and image-storage standards.^{9,84}

Four physiological confounders recur, on top of frequent acoustic failure. Mechanical ventilation degrades respirophasic indices outside narrow tidal-volume/PEEP ranges. Arrhythmia, particularly Afib, invalidates them altogether and requires five-beats for VTI. Raised intra-abdominal pressure blunts both the IVC and the passive leg raising signals, and isolated ventricular dysfunction can render IVC distensibility falsely reassuring.⁴³⁻⁴⁷ Acoustic window fail often and are under-reported. The IVC was not obtainable in 22 per cent of ventilated patients in a large multicentre study, and outflow-tract Doppler were each unobtainable in about 22% of ventilated patients in a large multicenter study, versus 99.6% success for the transoesophageal SVC assessment. These make resuscitative transoesophageal echocardiography is safe and high yield fallback when the transthoracic window fails in an intubated patient. Above all, no sonographic finding should be read outside the clinical context: a plethoric IVC on a high PEEP, a hyperdynamic ventricle on high-dose vasopressors, or portal pulsatility in cirrhosis each mean something the image alone cannot disclose.^{42,43,45,74}

Table 7. Principal pitfalls, their mechanism and mitigation^{43,44,45,46,47,81,84}

Pitfall	Mechanism	Mitigation
Operator dependency and skill decay	Acquisition skill deteriorates within months, most after short courses	Structured longitudinal training, logbooks, image archiving, periodic re-assessment of competence
Poor acoustic window	Obesity, ventilation, dressings, subcutaneous emphysema	Use subcostal rescue view; consider resuscitative transoesophageal echocardiography in intubated patients

Over-reliance on the IVC	Confounded by ventilation, PEEP, intra-abdominal pressure, RV failure	Use only at extremes; combine with flow measurement and venous Doppler; never as a sole determinant
Invalid respirophasic indices	Spontaneous effort, low tidal volume, arrhythmia, low compliance	Prefer passive leg raising, which is valid across ventilation modes and rhythms
Measurement noise misread as effect	Least significant change of VTI is 11–14%	Average ≥ 3 beats (≥ 5 in AF); keep operator, window and sample position constant
Single-snapshot interpretation	Shock physiology evolves within hours	Mandate re-scan after every meaningful intervention
Isolated abnormal finding taken as diagnosis	Sonographic signs are load- and context-dependent	Integrate with history, examination, lactate, perfusion markers and treatment response

Notes: AF, atrial fibrillation; IVC, inferior vena cava; PEEP, positive end-expiratory pressure; RV, right ventricle; VTI, velocity–time integral.

An integrated clinical decision algorithm

The algorithm proposed in Figure 3 sequences the preceding sections into one operational sequence. Step 1 excludes the immediately lethal, immediately reversible obstructive causes, where POCUS has the highest positive likelihood ratio and delay is least recoverable. Step 2 quantifies flow with the LVOT VTI, turning 'which type of shock?' into 'how much forward flow is there?', and separating low-flow from high-flow hypotension. Step 3 asks responsiveness and tolerance together, generating three actionable configurations. Step 4 closes the loop by re-scanning after every meaningful intervention.^{4,5,19,25} Two features set this apart from earlier protocols: (1) flow measurement is

mandatory, since the vasopressor versus inotrope choice cannot be made from blood pressure and chamber appearance alone, (2) tolerance carries equal status to responsiveness, so a positive preload test alone never authorises an infusion. Both derived directly from VTI's prognostic value and the demonstrated coexistence of responsiveness with congestion over half of patients.^{14,32,33}

Where the loop fails to restore perfusion across two or three cycles, escalation to advanced haemodynamic monitoring, transoesophageal imaging, mechanical circulatory support and multidisciplinary shock-team review is warranted. Consistent with contemporary cardiogenic shock recommendations emphasizing structured teams and standardised staging protocols. Persistent hypoperfusion despite adequate macrocirculatory targets should itself be treated as a distinct high-risk, not simply a trigger for further indiscriminate escalation.^{22,74,76,77}

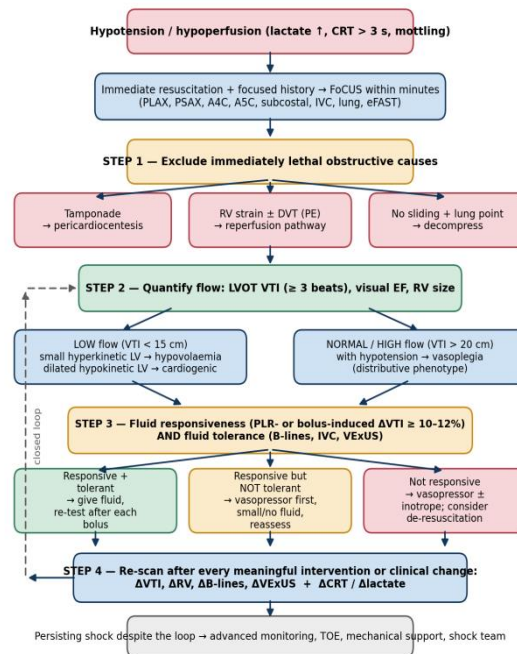


Figure 3. Proposed POCUS-guided clinical decision algorithm for the patient with undifferentiated shock. The sequence is deliberately ordered so that lethal obstructive causes are excluded before flow is quantified, and so that fluid is authorised only when responsiveness and tolerance are both satisfied. The dashed line indicates the closed loop of serial reassessment. Sources: 4,5,14,19,2

4. Discussion

The central tension in this field is that diagnostic performance and demonstrated outcome benefit have not advanced in step. The diagnostic case is settled: pooled sensitivities of 0.78 to 0.90 and specificities of 0.92 to 0.98 across shock subtypes, areas under the curve of approximately 0.95, a positive likelihood ratio of 40 for obstructive shock, a 27.7 per cent reduction in aggregate diagnostic uncertainty, and changes in management in a quarter to a half of patients examined. The outcome case is not: a meta-analysis of eighteen randomised trials found a probable reduction in 28-day mortality (relative risk 0.88, 95% confidence interval 0.78 to 0.99), shorter vasoactive therapy and better lactate clearance with low to moderate certainty, while the largest and methodologically strongest single trial found no benefit at all.⁵⁻⁸

Four explanations for this dissociation are supported by the literature and each generates a testable prediction. First, dose and timing: most trials applied POCUS once, at presentation,

whereas the physiology being interrogated evolves over hours; incorporating ultrasound into daily rounds has been associated with lower cumulative fluid balance and shorter ventilation and ICU stay. Second, the missing link between image and action: several protocols specified what to scan but not what to do with the answer, so the intervention could not plausibly change the exposure that determines outcome, precisely the gap closed by the FRESH trial, in which acting on the passive leg raising result reduced fluid balance by 1.37 L and halved the need for renal replacement therapy and mechanical ventilation. Third, operator heterogeneity: trials rarely report training level, expert-novice agreement is only moderate for several parameters, and competence decays measurably within months. Fourth, outcome dilution: mortality in unselected hypotensive populations is driven by underlying disease, and diagnostic tools rarely move such endpoints in isolation.^{6,9,41,65,84}

A second theme is the reconceptualisation of the volume question. The field has moved from a one-dimensional notion of volume status to a

two-dimensional assessment of responsiveness and tolerance. The empirical basis is the observation that congestion signals are present in 53 per cent of fluid-responsive and 57 per cent of fluid-unresponsive critically ill patients, without any association with fluid balance or diagnostic category. This finding invalidates the common clinical shortcut of inferring tolerance from responsiveness and provides the physiological rationale for the parallel-assessment structure adopted in the algorithm presented here.¹⁴

Third, the interpretation of venous congestion indices requires recalibration. The VExUS score correlates strongly with right atrial pressure and with pulmonary pressures, and in cardiac populations it predicts acute kidney injury and mortality, but in general critical care cohorts these associations largely disappear. The most coherent reading, and the one advanced in a recent critical appraisal, is that VExUS reflects the interaction between venous return and cardiac function rather than volume itself: the highest scores are frequently found in cardiogenic physiology with low fluid balance. Used as a measure of the pressure consequences of filling and as a monitoring signal during decongestion, the score remains valuable; used as a proxy for total body water, it will disappoint.⁵⁴⁻⁵⁸

Fourth, the right ventricle deserves the prominence it has recently acquired. Right ventricular dysfunction was independently associated with a threefold increase in 28-day mortality in early sepsis while left ventricular systolic dysfunction was not; meta-analytic data confirm a relative risk of 1.62; and coupling indices such as TAPSE/PASP carry independent prognostic information in general intensive care populations. Because right ventricular failure

inverts the usual fluid logic, additional volume worsens rather than improves output through ventricular interdependence, its detection changes management immediately, and its assessment should be mandatory rather than discretionary in the shock examination.^{61-63,73}

Fifth, macrocirculatory and microcirculatory targets must be pursued together. ANDROMEDA-SHOCK showed less organ dysfunction at 72 hours with a capillary refill time-targeted strategy, its Bayesian reanalysis found a high posterior probability of mortality benefit, and ANDROMEDA-SHOCK-2 demonstrated superiority of a personalised protocol incorporating bedside echocardiography for a hierarchical composite outcome. The lesson for sonographic practice is that flow variables should be interpreted alongside a rapidly responsive perfusion signal, and that discordance between them is itself clinically informative.^{15,21,22,80}

This review has limitations. It is a narrative rather than a systematic synthesis, and although the search was structured and reproducible, selection of sources within each domain involved judgement and is therefore susceptible to reviewer bias. Publication in English-language indexed journals was a practical requirement, which may under-represent work from low- and middle-income settings where POCUS is arguably most valuable given constrained access to computed tomography and formal echocardiography. Several key sources are recent and some are in press, so their conclusions may be revised. Finally, the algorithm proposed here is a synthesis of evidence rather than a prospectively validated instrument; it should be tested, not assumed.^{10,26}

Table 8. Summary of the principal outcome evidence for POCUS-guided resuscitation in shock^{5,6,7,8,9,15,41}

Study or synthesis	Design	Population	Principal finding
Basmaji et al., 2024	SR/MA of 18 RCTs	Adults with shock	28-day mortality RR 0.88 (0.78–0.99); shorter vasoactive duration; better lactate clearance; low–moderate certainty

Atkinson et al., 2018 (SHoC-ED)	Multicentre RCT (n = 273)	Undifferentiated ED hypotension	No difference in survival, length of stay, CT use, inotrope use or fluid volume
Shokoohi et al., 2015	Prospective observational (n = 118)	ED hypotension after initial fluid	Diagnostic uncertainty reduced 27.7%; management changed in 24.6%
Douglas et al., 2020 (FRESH)	Multicentre RCT (n = 124 analysed)	Sepsis-associated hypotension	Fluid balance 1.37 L lower; less renal replacement (5.1% vs 17.5%) and ventilation (17.7% vs 34.1%)
Hernández et al., 2025 (ANDROMEDA-SHOCK-2)	Multicentre RCT (n = 1,467)	Early septic shock	Win ratio 1.16 (1.02–1.33) for hierarchical composite, driven by shorter vital support
Flower et al., 2024	Prospective multicentre observational (n = 1,015)	Critical care admissions with shock	Echocardiography in 54%; reduced uncertainty or changed management in 54% of those scanned; 25% governance compliance
Yoshida et al., 2023	SR/MA of 12 studies (n = 1,132)	Shock of any type	Sensitivity 0.78–0.90, specificity 0.92–0.98, AUC $\approx$ 0.95 per subtype

Notes: AUC, area under the receiver operating characteristic curve; CT, computed tomography; ED, emergency department; MA, meta-analysis; RCT, randomised controlled trial; RR, relative risk; SR, systematic review.

Table 9. Research agenda arising from this review^{6,15,56,84}

Gap	Question to be answered	Suggested design
Care bundle versus single scan	Does a mandated serial POCUS loop with explicit therapeutic rules improve patient-centred outcomes?	Multicentre RCT of a complete bundle with protocolised re-scan intervals and hierarchical composite endpoints
Congestion-guided de-resuscitation	Does VExUS- or lung-ultrasound-guided decongestion reduce AKI, ventilation days or mortality?	Randomised trial with pre-specified de-resuscitation triggers and safety stopping rules for perfusion
Competence thresholds	What operator training level is required for POCUS-guided decisions to be safe and effective?	Trials reporting and stratifying by operator competence; competence-decay prevention studies
Right ventricular phenotyping	Does explicit RV phenotyping change	Prospective phenotype-stratified interventional studies using coupling indices

	therapy and outcome in septic and mixed shock?	
Automation and artificial intelligence	Can AI-assisted acquisition make VTI measurement reliable in the hands of minimally trained operators?	Randomised interchangeability studies of AI-guided versus expert acquisition
Low-resource settings	What is the incremental value of POCUS where CT and formal echocardiography are unavailable?	Pragmatic multicentre trials in low- and middle-income settings

Notes: AI, artificial intelligence; AKI, acute kidney injury; CT, computed tomography; RCT, randomised controlled trial; RV, right ventricle; VExUS, venous excess ultrasound; VTI, velocity–time integral.

Conclusion

Bedside echocardiography, embedded within a multimodal point-of-care ultrasound examination, is now the first-line imaging modality for determining the mechanism of shock, and its diagnostic performance across the four canonical phenotypes is high and reproducible. Its greater contribution, however, is physiological rather than diagnostic: through the LVOT velocity time integral it quantifies forward flow, through passive leg raising it converts an untested assumption about preload into a measured prediction, and through lung, inferior vena cava and venous Doppler assessment it defines whether the circulation can tolerate what is being given. Right ventricular size, longitudinal function and coupling identify the phenotype that carries the highest mortality and that inverts the usual fluid logic.

The evidence supports a specific conclusion about how the tool should be used. Fluid responsiveness and fluid tolerance are independent questions and must both be answered before any bolus; the choice between vasopressor and inotrope requires a flow measurement and cannot be made from pressure alone; and the examination must be repeated after every meaningful intervention, since a single snapshot describes a physiology that will have changed within the hour. Where POCUS has

been used in this way, as a repeated, rule-linked physiological test rather than as a diagnostic photograph, it has reduced fluid exposure, shortened organ support and, in meta-analysis, probably reduced mortality.

Recommendations for practice

1. Perform a focused cardiac and lung ultrasound examination within minutes of recognising shock, ordered so that tamponade, acute cor pulmonale and pneumothorax are excluded first.
2. Measure the LVOT velocity time integral in every patient in whom the window permits, averaging at least three beats in sinus rhythm and five in atrial fibrillation, and record the value so that later studies can be compared with it.
3. Before every fluid bolus, establish both responsiveness (passive leg raising from a semi-recumbent position with a flow variable, threshold ≥ 10 per cent) and tolerance (lung profile, inferior vena cava, venous Doppler).
4. Assess the right ventricle explicitly in every examination: size, TAPSE and, where obtainable, a coupling index and interpret right ventricular function against its load rather than in isolation.⁶¹⁻⁶³
5. Interpret changes against measurement precision: treat variations in VTI below approximately 11 to 14 per cent as noise, and

keep the operator, window and sampling position constant across serial studies.

6. Re-scan after every meaningful intervention and at least daily, and treat discordance between improving flow and static perfusion markers as a signal of macro-to-microcirculatory uncoupling rather than as a reason for further escalation of the same therapy.

7. Escalate to transoesophageal imaging when transthoracic windows fail in an intubated patient, and to advanced monitoring and shock-team review when two or three decision cycles fail to restore perfusion.

8. Support practice with governance: structured longitudinal training with logbooks, image archiving and reporting, and periodic re-assessment of competence, recognising that acquisition skill decays within months.

Recommendations for research

Future trials should evaluate complete POCUS-guided care bundles, mandated serial reassessment, explicit therapeutic rules, standardised and reported operator competence, against usual care, using hierarchical composite or organ-support-free-day endpoints rather than diagnostic accuracy or mortality alone. The two most pressing specific questions are whether congestion-guided de-resuscitation improves renal and respiratory outcomes, and what threshold of operator competence is required before POCUS-guided decisions are safe. Artificial-intelligence-assisted acquisition, currently under randomised evaluation for velocity–time integral measurement by minimally trained operators, may prove decisive in making the quantitative components of this framework generalisable beyond expert centres.

6. References

- Cecconi, M., De Backer, D., Antonelli, M., Beale, R., Bakker, J., Hofer, C., ... Rhodes, A. (2014). Consensus on circulatory shock and hemodynamic monitoring. Task force of the European Society of Intensive Care Medicine. *Intensive Care Medicine*, 40(12), 1795–1815. <https://doi.org/10.1007/s00134-014-3525-z>
- Antonelli, M., Levy, M., Andrews, P. J. D., Chastre, J., Hudson, L. D., Manthous, C., ... Torres, A. (2007). Hemodynamic monitoring in shock and implications for management. International Consensus Conference, Paris, France, 27–28 april 2006. *Intensive Care Medicine*, 33(4), 575–590. <https://doi.org/10.1007/s00134-007-0531-4>
- Shankar-Hari, M., Phillips, G. S., Levy, M. L., Seymour, C. W., Liu, V. X., Deutschman, C. S., ... Singer, M. (2016). Developing a new definition and assessing new clinical criteria for septic shock. *JAMA*, 315(8), 775. <https://doi.org/10.1001/jama.2016.0289>
- Monnet, X., Messina, A., Greco, M., Bakker, J., Aissaoui, N., Cecconi, M., ... Chew, M. S. (2025). ESICM guidelines on circulatory shock and hemodynamic monitoring 2025. *Intensive Care Medicine*, 51(11), 1971–2012. <https://doi.org/10.1007/s00134-025-08137-z>
- Yoshida, T., Yoshida, T., Noma, H., Nomura, T., Suzuki, A., & Mihara, T. (2023). Diagnostic accuracy of point-of-care ultrasound for shock: A systematic review and meta-analysis. *Critical Care*, 27(1). <https://doi.org/10.1186/s13054-023-04495-6>
- Basmaji, J., Arntfield, R., Desai, K., Lau, V. I., Lewis, K., Rochwerf, B., ... Guyatt, G. (2024). The impact of point-of-care ultrasound-guided resuscitation on clinical outcomes in patients with shock: A systematic review and meta-analysis. *Critical Care Medicine*, 52(11), 1661–1673. <https://doi.org/10.1097/CCM.0000000000006399>
- Atkinson, P. R., Milne, J., Diegelmann, L., Lamprecht, H., Stander, M., Lussier, D., ... Lewis, D. A. (2018). Does point-of-care ultrasonography improve clinical outcomes in emergency department patients with undifferentiated hypotension? An international randomized controlled trial from the SHoC-ED investigators. *Annals of Emergency Medicine*, 72(4), 478–489. <https://doi.org/10.1016/j.annemergmed.2018.04.002>
- Shokoohi, H., Boniface, K. S., Pourmand, A., Liu, Y. T., Davison, D. L., Hawkins, K. D., ... Yadav, K. (2015). Bedside ultrasound reduces diagnostic uncertainty and guides resuscitation in patients with undifferentiated hypotension*. *Critical Care Medicine*, 43(12), 2562–2569. <https://doi.org/10.1097/ccm.0000000000001285>

- Flower, L., Waite, A., Boulton, A., Peck, M., Akhtar, W., Boyle, A. J., ... Munro, A. (2024). The use of echocardiography in the management of shock in critical care: A prospective, multi-centre, observational study. *Intensive Care Medicine*, 50(10), 1668–1680. <https://doi.org/10.1007/s00134-024-07590-6>
- Ceriani, E., Schiavon, R., La Cava, L., Ruscitti, C., & Cogliati, C. (2025). Point of care ultrasound: Focus on evidence for a critical appraisal. *European Journal of Internal Medicine*. <https://doi.org/10.1016/j.ejim.2025.06.005>
- Vieillard-Baron, A., Millington, S. J., Sanfilippo, F., Chew, M., Diaz-Gomez, J., McLean, A., ... Fletcher, N. (2019). A decade of progress in critical care echocardiography: A narrative review. *Intensive Care Medicine*, 45(6), 770–788. <https://doi.org/10.1007/s00134-019-05604-2>
- Messmer, A. S., Zingg, C., Müller, M., Gerber, J. L., Schefold, J. C., & Pfortmueller, C. A. (2020). Fluid overload and mortality in adult critical care patients—A systematic review and meta-analysis of observational studies*. *Critical Care Medicine*, 48(12), 1862–1870. <https://doi.org/10.1097/ccm.0000000000004617>
- Malbrain, M. L. N. G., Langer, T., Annane, D., Gattinoni, L., Elbers, P., Hahn, R. G., ... Van Regenmortel, N. (2020). Intravenous fluid therapy in the perioperative and critical care setting: Executive summary of the International Fluid Academy (IFA). *Annals of Intensive Care*, 10(1). <https://doi.org/10.1186/s13613-020-00679-3>
- Muñoz, F., Born, P., Bruna, M., Ulloa, R., González, C., Philp, V., ... Kattan, E. (2024). Coexistence of a fluid responsive state and venous congestion signals in critically ill patients: A multicenter observational proof-of-concept study. *Critical Care*, 28(1). <https://doi.org/10.1186/s13054-024-04834-1>
- Pálizas, F., Lattanzio, B., Durandal, N., Alonso, M. E., Duque, J., Falcon, N., ... Cavalcanti, A. B. (2025). Personalized hemodynamic resuscitation targeting capillary refill time in early septic shock. *JAMA*. <https://doi.org/10.1001/jama.2025.20402>
- Levitov, A., Frankel, H. L., Blaivas, M., Kirkpatrick, A. W., Su, E., Evans, D., ... Elbarbary, M. (2016). Guidelines for the appropriate use of bedside general and cardiac ultrasonography in the evaluation of critically ill patients—Part II. *Critical Care Medicine*, 44(6), 1206–1227. <https://doi.org/10.1097/ccm.0000000000001847>
- Neskovic, A. N., Edvardsen, T., Galderisi, M., Garbi, M., Gullace, G., Jurcut, R., ... Stefanidis, A. (2014). Focus Cardiac Ultrasound: The European Association of Cardiovascular Imaging viewpoint. *European Heart Journal. Cardiovascular Imaging*, 15(9), 956–960. <https://doi.org/10.1093/ehjci/jeu081>
- Kirkpatrick, J. N., Grimm, R., Johri, A. M., Kimura, B. J., Kort, S., Labovitz, A. J., ... Turner, J. (2020). Recommendations for echocardiography laboratories participating in cardiac point of care cardiac ultrasound (POCUS) and critical care echocardiography training: Report from the American Society of Echocardiography. *Journal of the American Society of Echocardiography*. <https://doi.org/10.1016/j.echo.2020.01.008>
- Camilo Pérez, Diaz-Caicedo, D., Fernando, D., Moreno-Araque, L., Yepes, A. F., & Armando, J. (2024). Critical care ultrasound in shock: A comprehensive review of ultrasound protocol for hemodynamic assessment in the intensive care unit. *Journal of Clinical Medicine*, 13(18), 5344–5344. <https://doi.org/10.3390/jcm13185344>
- De Backer, D., Cecconi, M., Chew, M. S., Hajjar, L., Monnet, X., Ospina-Tascón, G. A., ... Vincent, J.-L. (2022). A plea for personalization of the hemodynamic management of septic shock. *Critical Care*, 26(372), 372. <https://doi.org/10.1186/s13054-022-04255-y>
- Hernández, G., Ospina-Tascón, G. A., Damiani, L. P., Estenssoro, E., Dubin, A., Hurtado, J., ... Bakker, J. (2019). Effect of a resuscitation strategy targeting peripheral perfusion status vs serum lactate levels on 28-Day mortality among patients with septic shock. *JAMA*, 321(7), 654. <https://doi.org/10.1001/jama.2019.0071>
- Hernandez, G., Hunsicker, O., de Backer, D., Angus, D. C., Bakker, J., Basmaji, J., ... Kattan, E. (2026). Twenty-five years of septic shock hemodynamic resuscitation trials: A conceptual perspective. *Critical Care*, 30(1). <https://doi.org/10.1186/s13054-026-06201-8>
- McLean, A. S. (2016). Echocardiography in shock management. *Critical Care*, 20(1). <https://doi.org/10.1186/s13054-016-1401-7>
- Haskings, E. M., Eissa, M., Allard, R. V., MirGhassemi, A., McFaul, C. M., & Miller, E. C. (2022). Point-of-care ultrasound use in emergencies: What every anaesthetist should know. *Anaesthesia*, 78(1), 105–118. <https://doi.org/10.1111/anae.15910>

- Rowe, M., & Ferrada, P. (2025). Ultrasound to guide critical decisions: All that you need to know. *Journal of Trauma and Acute Care Surgery*. <https://doi.org/10.1097/ta.0000000000004815>
- Kok, B., Wolthuis, D., Bosch, F., van der Hoeven, H., & Blans, M. (2022). POCUS in dyspnea, nontraumatic hypotension, and shock; a systematic review of existing evidence. *European Journal of Internal Medicine*, 106, 9–38. <https://doi.org/10.1016/j.ejim.2022.07.017>
- Kaselitz, T. B., & Seymour, C. W. (2025). Point-of-care ultrasound in sepsis and septic shock. *JAMA*. <https://doi.org/10.1001/jama.2025.1983>
- Vi Am Dinh, Ko, H., Rao, R., Bansal, R., Smith, D. B., Tae Eung Kim, & H. Bryant Nguyen. (2012). Measuring cardiac index with a focused cardiac ultrasound examination in the ED. *American Journal of Emergency Medicine*, 30(9), 1845–1851. <https://doi.org/10.1016/j.ajem.2012.03.025>
- Jozwiak, M., Mercado, P., Jean-Louis Teboul, Anouar Benmalek, Giménez, J., François Dépret, ... Monnet, X. (2019). What is the lowest change in cardiac output that transthoracic echocardiography can detect? *Critical Care*, 23(1). <https://doi.org/10.1186/s13054-019-2413-x>
- Gaspardone, C., Romagnolo, D., Baldetti, L., Fasolino, A., Peveri, B., Calvo, F., ... Scandroglio, A. M. (2023). A simplified echocardiographic formula to estimate cardiac index in the intensive care unit. *International Journal of Cardiology*, 372, 76–79. <https://doi.org/10.1016/j.ijcard.2022.12.010>
- Olivieri, P. P., Patel, R., Kolb, S., Fatima, S., Galvagno, S. M., Haase, D. J., ... Murthi, S. B. (2019). Echo is a good, not perfect, measure of cardiac output in critically ill surgical patients. *Journal of Trauma and Acute Care Surgery*, 87(2), 379–385. <https://doi.org/10.1097/ta.0000000000002304>
- Chinen, D., Fujino, M., Anzai, T., M. Kitakaze, Goto, Y., Ishihara, M., ... Ogawa, H. (2013). Left ventricular outflow tract velocity time integral correlates with low cardiac output syndrome in patients with acute decompensated heart failure. *European Heart Journal*, 34(suppl 1), P4249–P4249. <https://doi.org/10.1093/eurheartj/ehs309.p4249>
- Zhang, R. S., Yuriditsky, E., Nayar, A. C., Elbaum, L., Greco, A. A., Rhee, A. J., ... Bangalore, S. (2024). Low left ventricular outflow tract velocity time integral predicts normotensive shock in patients with acute pulmonary embolism. *American Heart Journal*, 272, 106–108. <https://doi.org/10.1016/j.ahj.2024.03.006>
- Wang, Jinzhong, Zhou, D., Gao, Y., Wu, Z., Wang, X., & Lv, C. (2020). Effect of VTILVOT variation rate on the assessment of fluid responsiveness in septic shock patients. *Medicine*, 99(47), e22702. <https://doi.org/10.1097/MD.00000000000022702>
- Monnet, X., Marik, P., & Teboul, J.-L. (2016). Passive leg raising for predicting fluid responsiveness: A systematic review and meta-analysis. *Intensive Care Medicine*, 42(12), 1935–1947. <https://doi.org/10.1007/s00134-015-4134-1>
- Cherpanath, T. G. V., Hirsch, A., Geerts, B. F., Lagrand, W. K., Leeftang, M. M., Schultz, M. J., & Groeneveld, A. B. J. (2016). Predicting fluid responsiveness by passive leg raising. *Critical Care Medicine*, 44(5), 981–991. <https://doi.org/10.1097/ccm.0000000000001556>
- Meco, M., Giustiniano, E., Nisi, F., Zulli, P., & Agosteo, E. (2026). Predicting fluid responsiveness in mechanically ventilated adults: An umbrella review of diagnostic accuracy meta-analyses. *Anesthesia & Analgesia*. <https://doi.org/10.1213/ane.0000000000008215>
- Jabot, J., Teboul, J.-L., Richard, C., & Monnet, X. (2008). Passive leg raising for predicting fluid responsiveness: Importance of the postural change. *Intensive Care Medicine*, 35(1). <https://doi.org/10.1007/s00134-008-1293-3>
- Monnet, X., Shi, R., & Teboul, J.-L. (2022). Prediction of fluid responsiveness. What's new? *Annals of Intensive Care*, 12(1), 46. <https://doi.org/10.1186/s13613-022-01022-8>
- Iván, J., Ruiz, J., José, J., Eduardo, L., Carreño, L., Andrés, C., & Gustavo Adolfo Ospina-Tascón. (2023). Variables influencing the prediction of fluid responsiveness: A systematic review and meta-analysis. *Critical Care*, 27(1). <https://doi.org/10.1186/s13054-023-04629-w>
- Douglas, I. S., Alapat, P. M., Corl, K. A., Exline, M. C., Forni, L. G., Holder, A. L., ... Hansell, D. M. (2020). Fluid response evaluation in sepsis hypotension and shock: A randomized clinical trial. *Chest*, 158(4). <https://doi.org/10.1016/j.chest.2020.04.025>

- Philippe Vignon, Repessé, X., Emmanuelle Begot, Léger, J., Jacob, C., Koceila Bouferrache, ... Vieillard-Baron, A. (2017). *Comparison of echocardiographic indices used to predict fluid responsiveness in ventilated patients*. 195(8), 1022–1032. <https://doi.org/10.1164/rccm.201604-0844oc>
- Vieillard-Baron, A., Evrard, B., Repessé, X., Maizel, J., Jacob, C., Goudelin, M., ... Vignon, P. (2018). Limited value of end-expiratory inferior vena cava diameter to predict fluid responsiveness impact of intra-abdominal pressure. *Intensive Care Medicine*, 44(2), 197–203. <https://doi.org/10.1007/s00134-018-5067-2>
- Si, X., Xu, H., Liu, Z., Wu, J., Cao, D., Chen, J., ... Guan, X. (2018). Does respiratory variation in inferior vena cava diameter predict fluid responsiveness in mechanically ventilated patients? A systematic review and meta-analysis. *Anesthesia & Analgesia*, 127(5), 1157–1164. <https://doi.org/10.1213/ane.0000000000003459>
- Via, G., Tavazzi, G., & Price, S. (2016). Ten situations where inferior vena cava ultrasound may fail to accurately predict fluid responsiveness: A physiologically based point of view. *Intensive Care Medicine*, 42(7), 1164–1167. <https://doi.org/10.1007/s00134-016-4357-9>
- Muller, L., Bobbia, X., Toumi, M., Louart, G., Molinari, N., Ragonnet, B., ... Lefrant, J. (2012). Respiratory variations of inferior vena cava diameter to predict fluid responsiveness in spontaneously breathing patients with acute circulatory failure: Need for a cautious use. *Critical Care*, 16(5), R188. <https://doi.org/10.1186/cc11672>
- Carneiro, R., Silvia, C., Neves, V., Ary Serpa Neto, Rodrigo Octavio Deliberato, Adriano José Pereira, ... Thiago Domingos Corrêa. (2024). Assessment of fluid responsiveness using pulse pressure variation, stroke volume variation, plethysmographic variability index, central venous pressure, and inferior vena cava variation in patients undergoing mechanical ventilation: A systematic review and meta-analysis. *Critical Care*, 28(1). <https://doi.org/10.1186/s13054-024-05078-9>
- Lichtenstein, D. A., & Mezière, G. A. (2008). Relevance of lung ultrasound in the diagnosis of acute respiratory failure*: The BLUE protocol. *Chest*, 134(1), 117–125. <https://doi.org/10.1378/chest.07-2800>
- Lichtenstein, D. A. (2015). BLUE-Protocol and FALLS-Protocol. *Chest*, 147(6), 1659–1670. <https://doi.org/10.1378/chest.14-1313>
- Lichtenstein, D. A., & Bar, S. (2024). Lung ultrasound for causal diagnosis of shock (FALLS-protocol), a tool helping to guide fluid therapy while approaching fluid tolerance. Some comments on its accuracy. *Annals of Intensive Care*, 14(1). <https://doi.org/10.1186/s13613-024-01329-8>
- Picano, E., & Pellikka, P. A. (2016). Ultrasound of extravascular lung water: A new standard for pulmonary congestion. *European Heart Journal*, 37(27), 2097–2104. <https://doi.org/10.1093/eurheartj/ehw164>
- Platz, E., Jhund, P. S., Girerd, N., Pivetta, E., McMurray, J. J. V., Peacock, W. F., ... Study Group on Acute Heart Failure of the Acute Cardiovascular Care Association and the Heart Failure Association of the European Society of Cardiology. (2019). Expert consensus document: Reporting checklist for quantification of pulmonary congestion by lung ultrasound in heart failure. *European Journal of Heart Failure*, 21(7), 844–851. <https://doi.org/10.1002/ehhf.1499>
- Guido Tavazzi, Julia, N., Matteo Pagnesi, Maurizio Bertaina, Montisci, A., Frea, S., ... Sacco, A. (2025). Lung ultrasound and mortality in a cardiogenic shock population: A prospective registry-based analysis. *European Journal of Heart Failure*. <https://doi.org/10.1002/ehhf.3692>
- Longino, A., Martin, K. R., Leyba, K., Siegel, G., Thai, T., Riscinti, M., Douglas, I. S., Gill, E. A., & Burke, J. (2023). Prospective evaluation of venous excess ultrasound (VExUS) for estimation of venous congestion. *Chest*. <https://doi.org/10.1016/j.chest.2023.09.029>
- Longino, A., Martin, K., Leyba, K., Siegel, G., Gill, E. A., Douglas, I. S., & Burke, J. (2023). Correlation between the VExUS score and right atrial pressure: A pilot prospective observational study. *Critical Care*, 27(1). <https://doi.org/10.1186/s13054-023-04471-0>
- Klompaker, P., Opschoor, K., Dalen, J., de Vries, R., Balan, C., Veelo, D. P., ... Tuinman, P. R. (2026). Diagnostic and prognostic value of the venous excess ultrasound grading system (VExUS): A systematic review and meta-analysis. *Critical Care Medicine*, 54(9), 2419–2429. <https://doi.org/10.1097/ccm.0000000000007219>
- Andrei, S., Bahr, P.-A., Nguyen, M., Bouhemad, B., & Guinot, P.-G. (2023). Prevalence of systemic venous congestion assessed by venous excess ultrasound grading system (VExUS) and association with acute kidney injury in a

general ICU cohort: A prospective multicentric study. *Critical Care*, 27(1). <https://doi.org/10.1186/s13054-023-04524-4>

- Guinot, P.-G. (2025). VExUS score: Optimizing its use in perioperative and critical care management. *Critical Care*, 29(1). <https://doi.org/10.1186/s13054-025-05687-y>
- Beaubien-Souligny, W., Galarza, L., Buchanan, B., Lau, V. I., Adhikari, N. K. J., Deschamps, J., ... Wald, R. (2023). Prospective study of ultrasound markers of organ congestion in critically ill patients with acute kidney injury. *Kidney International Reports*, 9(3), 694–702. <https://doi.org/10.1016/j.ekir.2023.12.018>
- Sovetova, S., Charaya, K., Erdniev, T., Shchekochikhin, D., Bogdanova, A., Panov, S., ... Andreev, D. (2024). Venous excess ultrasound score is associated with worsening renal function and reduced natriuretic response in patients with acute heart failure. *Journal of Clinical Medicine*, 13(20), 6272. <https://doi.org/10.3390/jcm13206272>
- Lanspa, M. J., Cirulis, M. M., Wiley, B. M., Olsen, T. D., Wilson, E. L., Beesley, S. J., ... Grissom, C. K. (2021). Right ventricular dysfunction in early sepsis and septic shock. *Chest*, 159(3), 1055–1063. <https://doi.org/10.1016/j.chest.2020.09.274>
- Bowcock, E., Huang, S., Yeo, R., Walisundara, D., Duncan, C. F., Pathan, F., ... Orde, S. (2024). The value of right ventricular to pulmonary arterial coupling in the critically ill: A national echocardiography database of Australia (NEDA) substudy. *Annals of Intensive Care*, 14(1), 10. <https://doi.org/10.1186/s13613-024-01242-0>
- Bowcock, E. M., Gerhardy, B., Huang, S., & Orde, S. (2022). Right ventricular outflow tract doppler flow analysis and pulmonary arterial coupling by transthoracic echocardiography in sepsis: A retrospective exploratory study. *Critical Care*, 26(1). <https://doi.org/10.1186/s13054-022-04160-4>
- Ruiz-Bailén, M., Hidalgo-Martín, J., Ramos Cuadra, J. Á., Manetsberger, J., Pola Gallego de Guzmán, M. D., Díaz-Castellanos, M. Á., ... Lavilla-Lerma, M. L. (2025). Right ventricular strain and outflow tract velocity time integral are associated with mortality in critically ill patients with pulmonary embolism. *American Journal of Respiratory and Critical Care Medicine*, 211(8), 1427–1441. <https://doi.org/10.1164/rccm.202407-1433oc>
- Levy, D., Boutrot, L., Petit, M., Hekimian, G., Charron, C., Kikoine, J., ... Saura, O. (2026). Visual right ventricular assessment in ICU: A multicenter international study. *Critical Care Medicine*, 54(8), 1990–1999. <https://doi.org/10.1097/ccm.00000000000007180>
- Alerhand, S., & Robert James Adrian. (2023). What echocardiographic findings differentiate acute pulmonary embolism and chronic right pulmonary hypertension? *American Journal of Emergency Medicine*, 72, 72–84. <https://doi.org/10.1016/j.ajem.2023.07.011>
- Daley, J. I., Dwyer, K. H., Grunwald, Z., Shaw, D. L., Stone, M. B., Schick, A., ... Moore, C. L. (2019). Increased sensitivity of focused cardiac ultrasound for pulmonary embolism in emergency department patients with abnormal vital signs. *Academic Emergency Medicine*, 26(11), 1211–1220. <https://doi.org/10.1111/acem.13774>
- Melo, R. H., Gioli-Pereira, L., Lourenço, I. D., Da Hora Passos, R., Bernardo, A. T., & Volpicelli, G. (2025). Diagnostic accuracy of multi-organ point-of-care ultrasound for pulmonary embolism in critically ill patients: A systematic review and meta-analysis. *Critical Care*, 29(1). <https://doi.org/10.1186/s13054-025-05359-x>
- Hunsicker, O., Daum, N., Hernandez, G., Kattan, E., Göpfert, M., Chew, M., ... Dünser, M. W. (2026). Acute pulmonary embolism: Clinical and echocardiographic recognition of normotensive shock. *Critical Care*, 30(1). <https://doi.org/10.1186/s13054-026-06214-3>
- Sato, R., Sanfilippo, F., Lanspa, M., Duggal, A., & Dugar, S. (2025). Sepsis-induced cardiomyopathy: Mechanism, prevalence, assessment, prognosis, and management. *Chest*, 168(6), 1383–1394. <https://doi.org/10.1016/j.chest.2025.08.013>
- Dugar, S., Sato, R., Chawla, S., Young, J., Wang, X., Grimm, R., ... Duggal, A. (2023). Is left ventricular systolic dysfunction associated with increased mortality among patients with sepsis and septic shock? *Chest*. <https://doi.org/10.1016/j.chest.2023.01.010>
- Sato, R., Sanfilippo, F., Hasegawa, D., Narut Prasitlumkum, Duggal, A., & Dugar, S. (2024). Prevalence and prognosis of hyperdynamic left ventricular systolic function in septic patients: A systematic review and meta-analysis. *Annals of Intensive Care*, 14(1). <https://doi.org/10.1186/s13613-024-01255-9>

- Wang, Jie, Tong, Z., Wang, X., & Wang, G. (2025). Time course of morbidity and mortality across echocardiographic phenotypes in patients with sepsis: A systematic review and meta-analysis. *Critical Care Medicine*, 53(11), e2294–e2304. <https://doi.org/10.1097/ccm.0000000000006844>
- Teran, F., Owyang, C. G., Wray, T. C., Hipskind, J. E., Lessard, J., Bédard Michel, W., ... Resuscitative TEE Collaborative Registry (rTEECorE) Investigators. (2025). Development and implementation of a multicenter registry for resuscitation-focused transesophageal echocardiography. *Annals of Emergency Medicine*, 85(2), 147–162. <https://doi.org/10.1016/j.annemergmed.2024.08.004>
- Breitkreutz, R., Price, S., Steiger, H. V., Seeger, F. H., Ilper, H., Ackermann, H., ... Walcher, F. (2010). Focused echocardiographic evaluation in life support and peri-resuscitation of emergency patients: A prospective trial. *Resuscitation*, 81(11), 1527–1533. <https://doi.org/10.1016/j.resuscitation.2010.07.013>
- Aissaoui, N., Delmas, C., Merdji, H., Schurtz, G., Baudry, G., Beurton, A., ... Kuteifan, K. (2026). Experts' recommendations for the management of adult patients with cardiogenic shock. *Annals of Intensive Care*, 16, 100038. <https://doi.org/10.1016/j.aicoj.2026.100038>
- Hoong Sern Lim, José González-Costello, Belohlavek, J., Zweck, E., Blumer, V., Schrage, B., & Hanff, T. C. (2024). Hemodynamic management of cardiogenic shock in the intensive care unit. *The Journal of Heart and Lung Transplantation*. <https://doi.org/10.1016/j.healun.2024.03.009>
- Silversides, J. A., Fitzgerald, E., Manickavasagam, U. S., Lapinsky, S. E., Nisenbaum, R., Hemmings, N., ... Marshall, J. C. (2018). Deresuscitation of patients with iatrogenic fluid overload is associated with reduced mortality in critical illness*. *Critical Care Medicine*, 46(10), 1600–1607. <https://doi.org/10.1097/ccm.0000000000003276>
- Vanden Eede, M., & Van Regenmortel, N. (2026). Unnecessary fluid therapy in patients with shock: Five classic fluid pitfalls and five ways to do it better. *Annals of Intensive Care*, 16, 100074. <https://doi.org/10.1016/j.aicoj.2026.100074>
- Jacquet-Lagréze, M., Bouhamri, N., Portran, P., Schweizer, R., Baudin, F., Lilot, M., ... Fellahi, J.-L. (2019). Capillary refill time variation induced by passive leg raising predicts capillary refill time response to volume expansion. *Critical Care*, 23(1). <https://doi.org/10.1186/s13054-019-2560-0>
- Expert Round Table on Ultrasound in ICU. (2011). International expert statement on training standards for critical care ultrasonography. *Intensive Care Medicine*, 37(7), 1077–1083. <https://doi.org/10.1007/s00134-011-2246-9>
- Wong, A., Galarza, L., Forni, L., De Backer, D., Slama, M., Cholley, B., ... Duška, F. (2020). Recommendations for core critical care ultrasound competencies as a part of specialist training in multidisciplinary intensive care: A framework proposed by the European Society of Intensive Care Medicine (ESICM). *Critical Care*, 24(1). <https://doi.org/10.1186/s13054-020-03099-8>
- Goudelin, M., Evrard, B., Donisanu, R., Gonzalez, C., Christophe Truffy, Orabona, M., ... Philippe Vignon. (2024). Therapeutic impact of basic critical care echocardiography performed by residents after limited training. *Annals of Intensive Care*, 14(1). <https://doi.org/10.1186/s13613-024-01354-7>
- Wang, Liang Wei, Liu, C. H., Li, W. Y., Chiang, W. C., Chiu, Y. L., Ma, M. H. M., ... Yang, C. W. (2025). Is ultrasound training sustainable? A systematic review of competency retention in healthcare trainees. *Medical Education*. <https://doi.org/10.1111/medu.15751>
- Vignon, P., Dugard, A., Abraham, J., Belcour, D., Gondran, G., Pepino, F., ... Gastinne, H. (2007). Focused training for goal-oriented hand-held echocardiography performed by noncardiologist residents in the intensive care unit. *Intensive Care Medicine*, 33(10), 1795–1799. <https://doi.org/10.1007/s00134-007-0742-8>

8. Author Declaration

The authors state no conflict of interest.