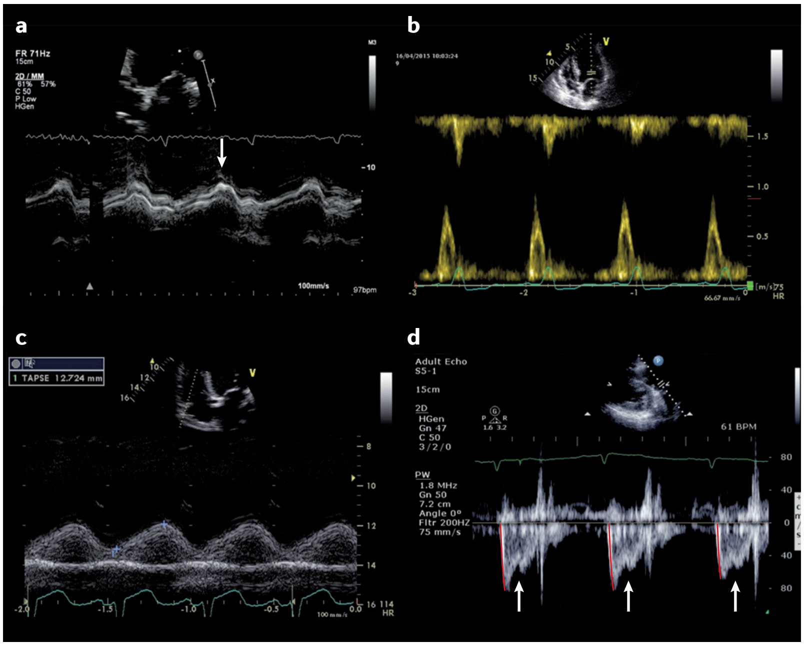

Figure 6 | **The haemodynamic effects of thrombosis (coronary and pulmonary) as demonstrated by echocardiography. a** | Early features of myocardial ischaemia can be demonstrated by the presence of prolonged long-axis shortening, measured by M-mode echocardiography across the base of the left ventricle (post-ejection shortening; arrow). **b** | Prolonged left ventricular wall tension suppresses early transmitral filling, resulting in an isolated late-diastolic transmitral A wave. **c** | Increased right ventricular afterload leads to a reduction in right ventricular systolic function, as demonstrated by tricuspid annular plane systolic excursion on M-mode echocardiography across the tricuspid annulus. **d** | A substantial increase in pulmonary vascular resistance might be associated with a midsystolic notch (arrows) on pulmonary valve pulsed-wave Doppler ejection wave and a short pulmonary valve acceleration time (78 ms; red lines).

50% of unselected patients with acute pulmonary embolism, and has a sensitivity of 50–60% and specificity of 80–90%[77]. Therefore, other investigations are used to confirm the diagnosis, with echocardiography used as a complementary imaging technique[19]. The principal indirect echocardiographic findings are nonspecific, and include right heart dilatation, right ventricular (RV) hypokinesis (with or without apical sparing), abnormal septal motion, and inferior vena cava dilatation[78] (FIG. 3a). Secondary tricuspid regurgitation might be present, allowing estimation of pulmonary arterial systolic pressure using the simplified Bernoulli equation[79] (FIG. 3b). Given that the right ventricle can generate a pulmonary artery systolic pressure of only ≤60 mmHg acutely, a higher pressure suggests a more chronic process (either multiple repeated episodes or chronic pulmonary parenchymal disease, with or without pulmonary embolism)[80]. Although the peak tricuspid regurgitation gradient is the most commonly used parameter to assess pulmonary artery systolic pressure in clinical practice, difficulties in the detection of good tricuspid regurgitation envelope might occur. Pulsed Doppler recordings of pulmonary valve flow acceleration time, pre-ejection period, and ejection time at the RV outflow tract can also be used to estimate pulmonary artery pressure and resistance[81,82].

Pericardial collection and tamponade

Echocardiography is pivotal for recognition of the haemodynamic consequences of a pericardial collection (FIG. 3c), allowing demonstration of features of tamponade including right atrial and/or RV diastolic collapse, in addition to guiding pericardiocentesis[83]. A number of potential pitfalls exist when interpreting the echocardiographic features of tamponade in the acute setting. These pitfalls include the effects of positive pressure ventilation (reversal of changes in transvalvular flows) and localized collections, in particular after cardiac surgery when substantial haemodynamic compromise might be present, even in the absence of echocardiographic features of tamponade[84].

Monitoring of therapy

Echocardiography is not recommended for the monitoring of therapy in patients with AHF in the absence of cardiogenic shock[4,9,11], given the complexity of LAP estimation using echocardiography, its lack of association with pulmonary congestion and symptoms, and superiority of natriuretic peptide levels in monitoring response to therapy. An emerging area in which echocardiography might be of use is in risk stratification before discharge from hospital. In patients with AHF with dyspnoea, persistent pulmonary congestion before discharge (demonstrated on LUS) has been shown to be an independent predictor of rehospitalization for AHF at 6 months after discharge[36].

Cardiogenic shock

Cardiogenic shock is the most severe manifestation of AHF. Although relatively uncommon, the published prevalence (5% of patients with AHF) varies according to the point of initial contact and management (1–2% of patients with AHF in the prehospital or emergency setting versus 29% in intensive care)[4,9,10,16]. Precise definitions of cardiogenic shock can vary; however, the syndrome generally results from inadequate cardiac output for peripheral organ requirements[85,86]. Cardiogenic shock can manifest as hypotension despite adequate filling (with or without vasopressors), altered mentation, cool peripheries, oliguria, hyperlactataemia, metabolic acidaemia, and low mixed venous oxygen saturation[86]. In addition to standard evaluation of critically ill patients in parallel with resuscitation, echocardiography is mandated immediately in patients with cardiogenic shock, because without identification and treatment of the underlying cause, the outcome is usually fatal[9,85] (FIG. 3d). Additional information that should be obtained from echocardiography includes estimation of stroke volume and cardiac output levels, because these data can provide guidance on how to maximize the cardiac output at the lowest filling pressures (see Supplementary information S2 (table)). These measurements should be taken during the echocardiogram, and should be performed repeatedly to monitor the response to therapeutic interventions and minimize potentially injurious treatment. Every study must be interpreted in the context of the level of inotropic and ventilatory support, as well as metabolic and arterial blood gas status, because these variables might have profound effects on echocardiographic findings.

Assessment of volume status. The physiological basis of providing 'optimal' filling in cardiogenic shock is that a critical decrease in intravascular-stressed volume reduces the difference between mean systemic venous and right atrial pressure, thereby limiting stroke volume. Although frequently used, invasive static pressure monitoring is not helpful for determining whether an individual patient is volume-responsive[87,88]. Static echocardiographic parameters are widely used to predict volume responsiveness in critically ill patients (FIG. 4); however, their use requires that a number of strict criteria (relating to the patient, their underlying pathology, and medical interventions) are met, otherwise the investigation becomes invalid (see Supplementary information S3 (table)). Similarly, although thought to be superior, dynamic echocardiographic parameters to predict volume responsiveness are valid only in fully mechanically ventilated patients in sinus rhythm and without chronic heart disease[89]. In the presence of cardiac disease (either left-sided and/or right-sided), these measurements can be misleading and should not be used. Conversely, tolerance to volume loading among different patients is variable. The conventional teaching to increase volume in RV failure has not been upheld by

Table 2 | **Echocardiography for acute mechanical circulatory support**

Type of mechanical support	Indications	Contraindications	Role of echo
VA ECMO	• Cardiogenic shock • Inability to wean from cardiopulmonary bypass after cardiac surgery • Arrhythmic storm • Pulmonary embolism • Isolated cardiac trauma • Acute anaphylaxis • Periprocedural support for high risk percutaneous intervention	• Nonrecoverable disease and not suitable for transplantation or VAD • Severe neurologic injury or intracerebral bleeding • Unrepaired aortic dissection • Severe aortic regurgitation	• Validation of the underlying cause • Biventricular function assessment • Guidewire position during cannulation • Optimal cannula positioning Postinsertion: • Effective LV offloading during ECMO (LV size, LVEDV monitoring if aortic regurgitation is present, aortic valve opening during systole, mitral or aortic regurgitation worsening, biphasic backflow across MV during diastole, retrograde systolic pulmonary flow) • Detection of complications (thrombosis, cannula migration, tamponade, intraventricular gradient as per excessive offloading) • Weaning from ECMO: assessment of dynamic changes during reduction of ECMO flow (LV and RV systolic function, RV and LV TDI of S′, LV size, LV VTI on aortic valve, mitral and aortic regurgitation, LAP assessment)
Impella (Abiomed, USA)	• Additional support for VA ECMO for inadequate offload • High-risk PCI and acute MI • AMI complicated by cardiogenic shock • Acute decompensated ischaemic cardiomyopathy • Myocarditis with cardiogenic shock • Acute RV dysfunction • Bridge to VAD or transplantation • Acute ablation of VT (where otherwise nontolerated haemodynamically) • Support for BAV (experimental)	• Nonrecoverable disease and not suitable for transplantation or VAD • Severe neurologic injury or intracerebral bleeding • LV thrombus present • Ventricular septal defect, or interatrial defect, severe aortic stenosis, and severe aortic regurgitation • Mechanical aortic valve • Sepsis • Bleeding diathesis • Severe peripheral vascular disease (left-sided device)	• Validation of underlying cause • Biventricular function assessment • Adequate device position • Positioning of inlet and outlet of device Postinsertion: • Exclusion of right-to-left atrial shunting • Optimization of biventricular filling • Detection of complication (cannula thrombus, displacement, inadequate cardiac output, inadequate offloading, failure of the nonsupported ventricle in face of increased forward flow from the supported ventricle)
Tandem Heart (Cardiac Assist, USA)	• High-risk PCI and acute MI • AMI complicated by cardiogenic shock	• Bleeding diathesis • Nonrecoverable disease and not suitable for transplantation or VAD • Severe peripheral vascular disease	• Validation of underlying cause • Biventricular function assessment • Transeptal puncture • Adequate cannula position Postinsertion: • Detection of complications (cannula thrombus, displacement, inadequate cardiac output, failure of the nonsupported ventricle in the face of increased forward flow from the supported ventricle)
IABP	• Mechanical complication and cardiogenic shock complicating AMI • Additional offloading of LV during peripheral VA ECMO • Severe MR	• Severe peripheral vascular disease • Aortic regurgitation	• Optimal positioning (TOE, when fluoroscopy not available)

BAV, balloon aortic valvuloplasty; Echo, echocardiography; IABP, intra-aortic balloon pump; LAP, left atrial pressure; LV, left ventricular; LVEDV, left ventricular end-diastolic volume; MI, myocardial infarction; MR, mitral regurgitation; MV, mitral valve; PCI, percutaneous coronary intervention; RV, right ventricular; S′, peak systolic annular velocity; TDI, tissue Doppler imaging; TOE, transoesophageal echocardiography; VAD, ventricular assist device; VT, ventricular tachycardia; VTI, velocity time integral; VA ECMO, venoarterial extracorporeal membrane oxygenation.

findings published in the past 3 years[90,91]. Physiological models suggest that in some patients, progressive fluid loading leads to a plateauing of cardiac output, with a progressive increase in pulmonary artery occlusion pressure. In addition, higher volume is associated with worse outcome in critically ill patients[92–94].

Inotropes and vasoactive agents. Although inotropes and vasopressors are commonly used to improve cardiac output and blood pressure in patients with cardiogenic shock, there is currently insufficient evidence to support the use of any particular agent in this context[9,95,96]. Dobutamine is generally the first-line inotrope of choice in the clinic[9,95,96]. The detrimental effects of positive inotropic agents have been extensively described in the literature[97,98], and their use should, therefore, be restricted to the shortest possible duration and the lowest dose, both individualized to the patient. Although little guidance exists on how inotrope treatment should be individualized, echocardiography might be helpful in certain scenarios.

First, not all patients with cardiac disease respond to escalating doses of dobutamine by increasing their stroke volume; in some patients, dobutamine can result in an increase in the total isovolumic time (tIVT)[99]. Echocardiographic identification of an abnormally prolonged tIVT with dobutamine use, or an increase in tIVT in response to escalating inotropic support might indicate that inotropes are directly impairing myocardial performance, thereby prompting a reduction in dose or a change in treatment strategy[99–101] (FIG. 5). Second, the combination of LV end-diastolic pressure (LVEDP) and low aortic root pressure might result in a mismatch of coronary perfusion and myocardial oxygen demand. If untreated, this mismatch can result in type 2 myocardial infarction[102] (FIG. 3d). Echocardiographic demonstration of a dominant or isolated A wave on transmitral Doppler in combination with postejection shortening can also be diagnostic (FIG. 6a,b), and indicates that aortic root pressure should be increased and/or LVEDP reduced[103,104]. Third, physiological studies have demonstrated that the combination of RV ischaemia and increased RV afterload is particularly injurious to RV performance, resulting in a fall in systemic blood pressure and cardiac output levels[105]. Echocardiography can be used to estimate pulmonary artery systolic pressure and pulmonary vascular resistance, as well as measure RV dimensions and performance[106]. Echocardiographic identification of high pulmonary vascular resistance with or without pulmonary hypertension in combination with RV dysfunction in cardiogenic shock might necessitate the introduction of a pressor agent plus treatment to reduce RV afterload[90,107] (FIG. 6c,d). Finally, in a patient with falling cardiac output levels despite escalating inotropic support, echocardiography can help to diagnose LV outflow tract obstruction (with or without associated mitral regurgitation)[27,108]. Treatment in this context involves reduction or cessation of positive inotropic agents, in combination with volume and pressor support.

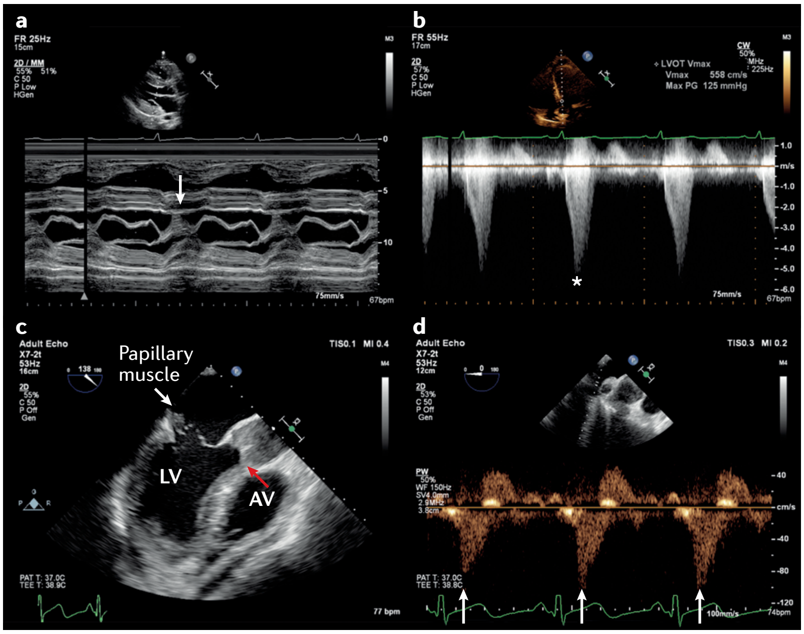

Figure 7 | **Echocardiographic features in patients receiving extracorporeal support.** Transthoracic echocardiography in a patient with severe respiratory failure receiving venovenous extracorporeal membrane oxygenation (ECMO). **a** | Parasternal long axis M-mode echocardiography across the mitral valve showing systolic anterior motion of the mitral valve leaflets (arrow). **b** | This motion was associated with substantial left ventricular intracavity gradient of 125 mmHg (asterisk). **c** | A complication of ST-segment elevation myocardial infarction requiring peripheral ECMO is revealed on M-mode echocardiography; papillary muscle rupture had resulted in a flail anterior mitral valve leaflet (white arrow) with associated torrential mitral regurgitation. The increase in left ventricular afterload from ECMO has resulted in failure of the left ventricle (LV) to eject, with a persistently closed aortic valve (AV; red arrow) and stasis of blood in the aortic root. **d** | Reversal of systolic pulmonary venous flow (arrows) in a patient receiving peripheral venovenous ECMO, suggesting inadequate offloading of the LV.

Cardiac arrest. The most extreme presentation of cardiogenic shock is cardiac arrest. International evidence-based guidelines recommend the use of echocardiography to diagnose or exclude some of the causes of arrest[109]. However, echocardiography should not affect the delivery of high-quality cardiopulmonary resuscitation, and specific training in advanced cardiovascular life support is required, even for experienced practitioners. As images are obtained and recorded only during the pulse/rhythm check, studies performed during cardiac arrest are strictly time-limited, and therefore are dissimilar to comprehensive studies that use only focused 2D imaging aimed at diagnosis or exclusion of potentially reversible causes in a simple, binary manner. The pathology leading to arrest is likely to be extreme (tamponade, massive pulmonary embolism, severe LV and/or RV dysfunction, myocardial infarction/ischaemia, hypovolaemia, or tension pneumothorax) and fairly easy to diagnose without more sophisticated echocardiographic techniques. Whether the use of echocardiography in cardiac arrest (and as part of care after resuscitation) can improve outcomes is unknown, but its application in the prehospital setting has been found to change management strategies in up to 60% of patients[110,111].

Table 3 | **Proposed initial focused cardiac and lung ultrasonography assessment for patients with suspected AHF in acute care setting**

Clinical question	Structural and functional assessment	Views (2D imaging)	Comments	Evidence
Focused echocardiography[131,132]				
Alternative diagnoses for patient's signs and symptoms?	• Pericardial effusion • RV dilatation/ systolic function	Subxiphoid, parasternal long-axis and short-axis views, apical four-chamber view	Absence of RV dilatation/dysfunction cannot exclude the presence of pulmonary emboli	• Pericardial effusion: sensitivity up to 100%, specificity 95% for detection of pericardial effusion[133,134] • RV dysfunction (various criteria): sensitivity 74%, specificity 54% for diagnosis of acute PE[19]
Evidence of impaired systolic function?	Global LV systolic function	Subxiphoid, parasternal long-axis and short-axis views, apical four-chamber view	Might be useful in new-onset HF for identification of reduced EF	Sensitivity and specificity for diagnosis of AHF depending on prevalence of HFrEF[38,135]
Is there (additional) evidence of volume overload?	IVC assessment	IVC (subxiphoid)	IVC collapsibility <50%	Sensitivity 83%, specificity 81% for diagnosis of AHF in patients with dyspnoea in the ED[135]
Gross structural abnormality as AHF aetiology?	• Gross valvular abnormality* • Intracardiac mass‡	Subxiphoid, parasternal long-axis and short-axis views, apical four-chamber view	AHF aetiology might be identified in rare cases	NA
Lung and pleural ultrasonography[37,38]				
Alternative diagnoses for patient's signs and symptoms?	Pneumothorax assessment	Anterior, upper chest on each hemithorax	Presence of lung sliding along pleural line rules out pneumothorax in the scanned chest zones	Sensitivity 91%, specificity 98% for detection of pneumothorax[136]
Evidence of pulmonary oedema?	Pulmonary oedema detection	Three or four anterior/ lateral chest zones on each hemithorax	Three or more B-lines in two or more zones on each hemithorax considered diagnostic for AHF	Sensitivity 94%, specificity 92% for diagnosis of AHF in patients with dyspnoea in the ED[33,38]
Evidence of pleural effusions?	Pleural effusion detection	Posterior axillary line on both hemithoraces	Echo-free space above the diaphragm	Sensitivity 79–84%, specificity 83–98% for diagnosis of AHF in patients with dyspnoea in the ED[44,45]

*Valvular abnormalities recognizable with focused echocardiography (without the use of Doppler-based techniques) entail leaflet or cusp massive disruption or marked thickening, flail, or anatomical gaps. ‡Refers to large valve vegetations or visible intracardiac or IVC thrombi. AHF, acute heart failure; Echo, echocardiography; ED, emergency department; EF, ejection fraction; HF, heart failure; HFrEF, heart failure with reduced ejection fraction; IVC, inferior vena cava; LV, left ventricular; NA, not available; PE, pulmonary embolism; RV, right ventricular.

Acute mechanical circulatory support. The indications for mechanical circulatory support (MCS) in the acute setting are constantly changing[112,113]. Intra-aortic balloon pumps are no longer routinely recommended for cardiogenic shock[114]. A range of new percutaneous ventricular assist devices are available, in addition to extracorporeal membrane oxygenation (ECMO). These techniques can be used as a bridge to recovery or for longer-term support, and differ not only in terms of their technical aspects, but the degree and type of support provided (LV and/or RV support, with or without the addition of respiratory support)[115–120]. Echocardiography is critical for successful implementation of acute MCS[121,122] (TABLE 2). MCS is not a treatment *per se*, but instead a supportive therapy for patients awaiting treatment or resolution of the underlying pathological process. As in all cases of AHF, the most important role of echocardiography is to diagnose the underlying cardiac cause. When the decision to institute MCS is made, echocardiography is then used to corroborate the decision regarding the type and level of support required. Although clear echocardiography parameters have been used to guide longer-term MCS for both the left and right heart[123,124], these parameters are not yet available for devices designed for short-term use. Furthermore, clear contraindications to MCS exist that can be diagnosed only using echocardiography. Echocardiography is used in the initiation of MCS, including the use of vascular ultrasonography to guide safe vessel cannulation and steer device or cannula placement. Echocardiography is subsequently used to monitor MCS by ensuring the goals of support are met, and for detecting complications and assessing tolerance to assistance[121]. Unfortunately, peripheral ECMO can paradoxically worsen cardiac function by increasing LV afterload. Although a number of echocardiographic parameters exist that might indicate this complication (including lack of aortic valve opening, biphasic retrograde flow across the mitral valve in diastole, and retrograde systolic pulmonary venous flow; FIG. 7), the inherent limitations of echocardiography in estimating LAP and LVEDP, especially when the heart is partially bypassed, makes this strategy particularly challenging[122]. Echocardiography can be used, however, to guide interventions to ensure that the heart is adequately offloaded.

Finally, a number of echocardiographic parameters are used in conjunction with clinical and haemodynamic assessment to predict which patients might be successfully weaned off MCS[125,126].

Other indications

Transoesophageal echocardiograpy can also be used in the acute setting in patients with dynamic mitral regurgitation (see Supplementary information S4 (figure)). Furthermore, features of infective endocarditis caused by aortic prostheses or a device can be demonstrated using transoesophageal echocardiography (see Supplementary information S5 (figure)).

Quality assurance

A detailed overview of the necessary organizational structure and processes for use of ultrasonography and echocardiography in the acute setting is beyond the scope of this Review, and has been published previously[26,127–130]. However, when used in routine clinical care, training, education, protocols, and ongoing certification of practitioners are required, which should all be performed within existing governance structures.

Conclusions

Echocardiography and LUS can assist in the rapid assessment of patients with acute dyspnoea and hypotension, and have the potential to transform the way in which clinicians assess and manage critically ill patients with AHF and cardiogenic shock (TABLE 3). The current AHF guidelines are cautious in recommendations for the widespread use of advanced echocardiography techniques in the acute care setting because robust applicability data are lacking, interpretation of findings requires highly specialized, in-depth knowledge of cardiac pathophysiology, and there is potential for harm by injudicious application in this patient population. The opportunities to improve diagnostic accuracy, reduce delays in treatment, and improve outcomes through the use of advanced echocardiography need to be further explored.

1. Yancy, C. W. *et al.* 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. *J. Am. Coll. Cardiol.* **62**, e147–e239 (2013).
2. Roger, V. L. Epidemiology of heart failure. *Circ. Res.* **113**, 646–659 (2013).
3. Dworzynski, K., Roberts, E., Ludman, A., Mant, J. & Guideline Development Group of the National Institute for Health and Care Excellence. Diagnosing and managing acute heart failure in adults: summary of NICE guidance. *BMJ* **349**, g5695 (2014).
4. Ponikowski, P. *et al.* 2016 ESC guidelines for the diagnosis and treatment of acute and chronic heart failure: the task force for the diagnosis and treatment of acute and chronic heart failure of the European Society of Cardiology (ESC). Developed with the special contribution of the Heart Failure Association (HFA) of the ESC. *Eur. Heart J.* **37**, 2129–2200 (2016).
5. Braunwald, E. Heart failure. *JACC Heart Fail.* **1**, 1–20 (2013).
6. Ambrosy, A. P. *et al.* The global health and economic burden of hospitalizations for heart failure: lessons learned from hospitalized heart failure registries. *J. Am. Coll. Cardiol.* **63**, 1123–1133 (2014).
7. Cook, C., Cole, G., Asaria, P., Jabbour, R. & Francis, D. P. The annual global economic burden of heart failure. *Int. J. Cardiol.* **171**, 368–376 (2014).
8. Ray, P. *et al.* Acute respiratory failure in the elderly: etiology, emergency diagnosis and prognosis. *Crit. Care* **10**, R82 (2006).
9. Mebazaa, A. *et al.* Acute heart failure and cardiogenic shock: a multidisciplinary practical guidance. *Intensive Care Med.* **42**, 147–163 (2016).
10. Mebazaa, A. *et al.* Recommendations on pre-hospital and early hospital management of acute heart failure: a consensus paper from the Heart Failure Association of the European Society of Cardiology, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine. *Eur. J. Heart Fail.* **17**, 544–558 (2015).
11. Yancy, C. W. *et al.* 2013 ACCF/AHA guideline for the management of heart failure: a report of the American College of Cardiology Foundation/American Heart Association task force on practice guidelines. *Circulation* **128**, e240–e327 (2013).
12. Maisel, A. S. *et al.* Timing of immunoreactive B-type natriuretic peptide levels and treatment delay in acute decompensated heart failure: an ADHERE (Acute Decompensated Heart Failure National Registry) analysis. *J. Am. Coll. Cardiol.* **52**, 534–540 (2008).
13. Januzzi, J. L. *et al.* NT-proBNP testing for diagnosis and short-term prognosis in acute destabilized heart failure: an international pooled analysis of 1256 patients: the International Collaborative of NT-proBNP Study. *Eur. Heart J.* **27**, 330–337 (2006).
14. Miro, O. *et al.* European Society of Cardiology — Acute Cardiovascular Care Association position paper on safe discharge of acute heart failure patients from the emergency department. *Eur. Heart J. Acute Cardiovasc. Care* http://dx.doi.org/10.1177/2048872616633853 (2016).
15. Mueller, C. *et al.* European Society of Cardiology — Acute Cardiovascular Care Association position paper on acute heart failure: a call for interdisciplinary care. *Eur. Heart J. Acute Cardiovasc. Care* **6**, 81–86 (2017).
16. Mebazaa, A. *et al.* Recommendations on pre-hospital and early hospital management of acute heart failure: a consensus paper from the Heart Failure Association of the European Society of Cardiology, the European Society of Emergency Medicine and the Society of Academic Emergency Medicine — short version. *Eur. Heart J.* **36**, 1958–1966 (2015).
17. Weintraub, N. L. *et al.* Acute heart failure syndromes: emergency department presentation, treatment, and disposition: current approaches and future aims: a scientific statement from the American Heart Association. *Circulation* **122**, 1975–1996 (2010).
18. Wang, C. S., FitzGerald, J. M., Schulzer, M., Mak, E. & Ayas, N. T. Does this dyspneic patient in the emergency department have congestive heart failure? *JAMA* **294**, 1944–1956 (2005).
19. Konstantinides, S. V. *et al.* 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. *Eur. Heart J.* **35**, 3033–3069 (2014).
20. Mueller, C. *et al.* Use of B-type natriuretic peptide in the evaluation and management of acute dyspnea. *N. Engl. J. Med.* **350**, 647–654 (2004).
21. Budweiser, S. *et al.* NT-proBNP in chronic hypercapnic respiratory failure: a marker of disease severity, treatment effect and prognosis. *Respir. Med.* **101**, 2003–2010 (2007).
22. Medina, A. M. *et al.* Prognostic utility of NT-proBNP in acute exacerbations of chronic pulmonary diseases. *Eur. J. Intern. Med.* **22**, 167–171 (2011).
23. Campo, G. *et al.* Cardiovascular history and adverse events in patients with acute exacerbation of COPD. *COPD* **12**, 560–567 (2015).
24. Gaggin, H. K. & Januzzi, J. L. Jr. Biomarkers and diagnostics in heart failure. *Biochim. Biophys. Acta* **1832**, 2442–2450 (2013).
25. Peacock, W. F., Cannon, C. M., Singer, A. J. & Hiestand, B. C. Considerations for initial therapy in the treatment of acute heart failure. *Crit. Care* **19**, 399 (2015).
26. Lancellotti, P. *et al.* The use of echocardiography in acute cardiovascular care: recommendations of the European Association of Cardiovascular Imaging and the Acute Cardiovascular Care Association. *Eur. Heart J. Acute Cardiovasc. Care* **4**, 3–5 (2015).
27. Price, S., Nicol, E., Gibson, D. G. & Evans, T. W. Echocardiography in the critically ill: current and potential roles. *Intensive Care Med.* **32**, 48–59 (2006).
28. Porter, T. R. *et al.* Guidelines for the use of echocardiography as a monitor for therapeutic intervention in adults: a report from the American Society of Echocardiography. *J. Am. Soc. Echocardiogr.* **28**, 40–56 (2015).
29. Volpicelli, G. *et al.* International evidence-based recommendations for point-of-care lung ultrasound. *Intensive Care Med.* **38**, 577–591 (2012).
30. Platz, E. *et al.* Utility of lung ultrasound in predicting pulmonary and cardiac pressures. *Eur. J. Heart Fail.* **14**, 1276–1284 (2012).
31. Liteplo, A. S. *et al.* Emergency thoracic ultrasound in the differentiation of the etiology of shortness of breath (ETUDES): sonographic B-lines and N-terminal pro-brain-type natriuretic peptide in diagnosing congestive heart failure. *Acad. Emerg. Med.* **16**, 201–210 (2009).
32. Anderson, K. L. *et al.* Inter-rater reliability of quantifying pleural B-lines using multiple counting methods. *J. Ultrasound Med.* **32**, 115–120 (2013).
33. Pivetta, E. *et al.* Lung ultrasound-implemented diagnosis of acute decompensated heart failure in the ED: a SIMEU multicenter study. *Chest* **148**, 202–210 (2015).
34. Platz, E., Jhund, P. S., Campbell, R. T. & McMurray, J. J. Assessment and prevalence of pulmonary oedema in contemporary acute heart failure trials: a systematic review. *Eur. J. Heart Fail.* **17**, 906–916 (2015).
35. Volpicelli, G. *et al.* Bedside ultrasound of the lung for the monitoring of acute decompensated heart failure. *Am. J. Emerg. Med.* **26**, 585–591 (2008).
36. Gargani, L. *et al.* Persistent pulmonary congestion before discharge predicts rehospitalization in heart failure: a lung ultrasound study. *Cardiovasc. Ultrasound* **13**, 40 (2015).
37. Al Deeb, M., Barbic, S., Featherstone, R., Dankoff, J. & Barbic, D. Point-of-care ultrasonography for the diagnosis of acute cardiogenic pulmonary edema in patients presenting with acute dyspnea: a systematic review and meta-analysis. *Acad. Emerg. Med.* **21**, 843–852 (2014).
38. Martindale, J. L. *et al.* Diagnosing acute heart failure in the emergency department: a systematic review and meta-analysis. *Acad. Emerg. Med.* **23**, 223–242 (2016).
39. Agricola, E. *et al.* "Ultrasound comet-tail images": a marker of pulmonary edema: a comparative study with wedge pressure and extravascular lung water. *Chest* **127**, 1690–1695 (2005).
40. Miglioranza, M. H. *et al.* Lung ultrasound for the evaluation of pulmonary congestion in outpatients: a comparison with clinical assessment, natriuretic peptides, and echocardiography. *JACC Cardiovasc. Imaging* **6**, 1141–1151 (2013).
41. Volpicelli, G. *et al.* Lung ultrasound predicts well extravascular lung water but is of limited usefulness in the prediction of wedge pressure. *Anesthesiology* **121**, 320–327 (2014).

42. Gargani, L. *et al.* Ultrasound lung comets for the differential diagnosis of acute cardiogenic dyspnoea: a comparison with natriuretic peptides. *Eur. J. Heart Fail.* **10**, 70–77 (2008).
43. Frasure, S. E. *et al.* Impact of patient positioning on lung ultrasound findings in acute heart failure. *Eur. Heart J. Acute Cardiovasc. Care* **4**, 326–332 (2014).
44. Cibinel, G. A. *et al.* Diagnostic accuracy and reproducibility of pleural and lung ultrasound in discriminating cardiogenic causes of acute dyspnea in the emergency department. *Intern. Emerg. Med.* **7**, 65–70 (2011).
45. Russell, F. M. *et al.* Diagnosing acute heart failure in patients with undifferentiated dyspnea: a lung and cardiac ultrasound (LuCUS) protocol. *Acad. Emerg. Med.* **22**, 182–191 (2015).
46. Volpicelli, G. Sonographic diagnosis of pneumothorax. *Intensive Care Med.* **37**, 224–232 (2011).
47. Lichtenstein, D., Meziere, G., Biderman, P. & Gepner, A. The "lung point": an ultrasound sign specific to pneumothorax. *Intensive Care Med.* **26**, 1434–1440 (2000).
48. Zoccali, C. *et al.* Pulmonary congestion predicts cardiac events and mortality in ESRD. *J. Am. Soc. Nephrol.* **24**, 639–646 (2013).
49. Gargani, L. Lung ultrasound: a new tool for the cardiologist. *Cardiovasc. Ultrasound* **9**, 6 (2011).
50. Bataille, B. *et al.* Integrated use of bedside lung ultrasound and echocardiography in acute respiratory failure: a prospective observational study in ICU. *Chest* **146**, 1586–1593 (2014).
51. Neskovic, A. N. *et al.* Emergency echocardiography: the European Association of Cardiovascular Imaging recommendations. *Eur. Heart J. Cardiovasc. Imaging* **14**, 1–11 (2013).
52. Cholley, B. P., Vieillard-Baron, A. & Mebazaa, A. Echocardiography in the ICU: time for widespread use! *Intensive Care Med.* **32**, 9–10 (2006).
53. Sicari, R. *et al.* The use of pocket-size imaging devices: a position statement of the European Association of Echocardiography. *Eur. J. Echocardiogr.* **12**, 85–87 (2011).
54. Flachskampf, F. A. *et al.* Recommendations for transoesophageal echocardiography: EACVI update 2014. *Eur. Heart J. Cardiovasc. Imaging* **15**, 353–365 (2014).
55. Silvestry, F. E. *et al.* Echocardiography-guided interventions. *J. Am. Soc. Echocardiogr.* **22**, 213–231 (2009).
56. Zamorano, J. L. *et al.* EAE/ASE recommendations for the use of echocardiography in new transcatheter interventions for valvular heart disease. *Eur. Heart J.* **32**, 2189–2214 (2011).
57. Reeves, S. T. *et al.* Basic perioperative transesophageal echocardiography examination: a consensus statement of the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists. *J. Am. Soc. Echocardiogr.* **26**, 443–456 (2013).
58. Hawkins, N. M. *et al.* Heart failure and chronic obstructive pulmonary disease: diagnostic pitfalls and epidemiology. *Eur. J. Heart Fail.* **11**, 130–139 (2009).
59. Ferguson, N. D. *et al.* The Berlin definition of ARDS: an expanded rationale, justification, and supplementary material. *Intensive Care Med.* **38**, 1573–1582 (2012).
60. Pinsky, M. R. Clinical significance of pulmonary artery occlusion pressure. *Intensive Care Med.* **29**, 175–178 (2003).
61. Luchsinger, P. C., Seipp, H. W. Jr & Patel, D. J. Relationship of pulmonary artery-wedge pressure to left atrial pressure in man. *Circ. Res.* **11**, 315–318 (1962).
62. Nagy, A. I. *et al.* The pulmonary capillary wedge pressure accurately reflects both normal and elevated left atrial pressure. *Am. Heart J.* **167**, 876–883 (2014).
63. Chatterjee, K. The Swan–Ganz catheters: past, present, and future. A viewpoint. *Circulation* **119**, 147–152 (2009).
64. Oh, J. K., Park, S. J. & Nagueh, S. F. Established and novel clinical applications of diastolic function assessment by echocardiography. *Circ. Cardiovasc. Imaging* **4**, 444–455 (2011).
65. Diwan, A., McCulloch, M., Lawrie, G. M., Reardon, M. J. & Nagueh, S. F. Doppler estimation of left ventricular filling pressures in patients with mitral valve disease. *Circulation* **111**, 3281–3289 (2005).
66. Oh, J. K. Echocardiography as a noninvasive Swan–Ganz catheter. *Circulation* **111**, 3192–3194 (2005).
67. Temporelli, P. L., Scapellato, F., Eleuteri, E., Imparato, A. & Giannuzzi, P. Doppler echocardiography in advanced systolic heart failure: a noninvasive alternative to Swan–Ganz catheter. *Circ. Heart Fail.* **3**, 387–394 (2010).
68. Ritzema, J. L. *et al.* Serial Doppler echocardiography and tissue Doppler imaging in the detection of elevated directly measured left atrial pressure in ambulant subjects with chronic heart failure. *JACC Cardiovasc. Imaging* **4**, 927–934 (2011).
69. Lester, S. J. *et al.* Unlocking the mysteries of diastolic function: deciphering the Rosetta Stone 10 years later. *J. Am. Coll. Cardiol.* **51**, 679–689 (2008).
70. Vignon, P. *et al.* Echocardiographic assessment of pulmonary artery occlusion pressure in ventilated patients: a transoesophageal study. *Crit. Care* **12**, R18 (2008).
71. Vignon, P. Hemodynamic assessment of critically ill patients using echocardiography Doppler. *Curr. Opin. Crit. Care* **11**, 227–234 (2005).
72. Cikes, M. & Solomon, S. D. Beyond ejection fraction: an integrative approach for assessment of cardiac structure and function in heart failure. *Eur. Heart J.* **37**, 1642–1650 (2016).
73. Blyakhman, F. A. *et al.* Validity of ejection fraction as a measure of myocardial functional state: impact of asynchrony. *Eur. J. Echocardiogr.* **10**, 613–618 (2009).
74. Jones, C. J., Raposo, L. & Gibson, D. G. Functional importance of the long axis dynamics of the human left ventricle. *Br. Heart J.* **63**, 215–220 (1990).
75. Henein, M. Y. & Gibson, D. G. Long axis function in disease. *Heart* **81**, 229–231 (1999).
76. Tavazzi, G., Via, G., Braschi, A. & Price, S. An 82-year-old woman with ongoing dyspnea. *Chest* **150**, e9–e11 (2016).
77. Perrier, A., Tamm, C., Unger, P. F., Lerch, R. & Sztajzel, J. Diagnostic accuracy of Doppler-echocardiography in unselected patients with suspected pulmonary embolism. *Int. J. Cardiol.* **65**, 101–109 (1998).
78. Casazza, F., Bongarzoni, A., Capozi, A. & Agostoni, O. Regional right ventricular dysfunction in acute pulmonary embolism and right ventricular infarction. *Eur. J. Echocardiogr.* **6**, 11–14 (2005).
79. Amsallem, M. *et al.* Addressing the controversy of estimating pulmonary arterial pressure by echocardiography. *J. Am. Soc. Echocardiogr.* **29**, 93–102 (2016).
80. Champion, H. C., Michelakis, E. D. & Hassoun, P. M. Comprehensive invasive and noninvasive approach to the right ventricle-pulmonary circulation unit: state of the art and clinical and research implications. *Circulation* **120**, 992–1007 (2009).
81. Lindqvist, P., Calcutteea, A. & Henein, M. Echocardiography in the assessment of right heart function. *Eur. J. Echocardiogr.* **9**, 225–234 (2008).
82. Bossone, E. *et al.* Echocardiography in pulmonary arterial hypertension: from diagnosis to prognosis. *J. Am. Soc. Echocardiogr.* **26**, 1–14 (2013).
83. Imazio, M. & Adler, Y. Management of pericardial effusion. *Eur. Heart J.* **34**, 1186–1197 (2013).
84. Adler, Y. *et al.* 2015 ESC guidelines for the diagnosis and management of pericardial diseases: the task force for the diagnosis and management of pericardial diseases of the European Society of Cardiology (ESC). Endorsed by: the European Association for Cardio-Thoracic Surgery (EACTS). *Eur. Heart J.* **36**, 2921–2964 (2015).
85. Thiele, H., Ohman, E. M., Desch, S., Eitel, I. & de Waha, S. Management of cardiogenic shock. *Eur. Heart J.* **36**, 1223–1230 (2015).
86. Reynolds, H. R. & Hochman, J. S. Cardiogenic shock: current concepts and improving outcomes. *Circulation* **117**, 686–697 (2008).
87. Monnet, X. & Teboul, J. L. Assessment of volume responsiveness during mechanical ventilation: recent advances. *Crit. Care* **17**, 217 (2013).
88. Di Somma, S. *et al.* The emerging role of biomarkers and bio-impedance in evaluating hydration status in patients with acute heart failure. *Clin. Chem. Lab. Med.* **50**, 2093–2105 (2012).
89. Charron, C., Caille, V., Jardin, F. & Vieillard-Baron, A. Echocardiographic measurement of fluid responsiveness. *Curr. Opin. Crit. Care* **12**, 249–254 (2006).
90. Mebazaa, A., Karpati, P., Renaud, E. & Algotsson, L. Acute right ventricular failure — from pathophysiology to new treatments. *Intensive Care Med.* **30**, 185–196 (2004).
91. Inohara, T., Kohsaka, S., Fukuda, K. & Menon, V. The challenges in the management of right ventricular infarction. *Eur. Heart J. Acute Cardiovasc. Care* **2**, 226–234 (2013).
92. Bendjelid, K. & Romand, J. A. Fluid responsiveness in mechanically ventilated patients: a review of indices used in intensive care. *Intensive Care Med.* **29**, 352–360 (2003).
93. Pinsky, M. R. My paper 20 years later: effect of positive end-expiratory pressure on right ventricular function in humans. *Intensive Care Med.* **40**, 935–941 (2014).
94. Reuse, C., Vincent, J. L. & Pinsky, M. R. Measurements of right ventricular volumes during fluid challenge. *Chest* **98**, 1450–1454 (1990).
95. Francis, G. S., Bartos, J. A. & Adatya, S. Inotropes. *J. Am. Coll. Cardiol.* **63**, 2069–2078 (2014).
96. Unverzagt, S. *et al.* Inotropic agents and vasodilator strategies for acute myocardial infarction complicated by cardiogenic shock or low cardiac output syndrome. *Cochrane Database Syst. Rev.* **1**, CD009669 (2014).
97. Bangash, M. N., Kong, M. L. & Pearse, R. M. Use of inotropes and vasopressor agents in critically ill patients. *Br. J. Pharmacol.* **165**, 2015–2033 (2012).
98. Singer, M. Catecholamine treatment for shock — equally good or bad? *Lancet* **370**, 636–637 (2007).
99. Duncan, A. M., Francis, D. P., Gibson, D. G. & Henein, M. Y. Limitation of exercise tolerance in chronic heart failure: distinct effects of left bundle-branch block and coronary artery disease. *J. Am. Coll. Cardiol.* **43**, 1524–1531 (2004).
100. Duncan, A. M., O'Sullivan, C. A., Gibson, D. G. & Henein, M. Y. Electromechanical interrelations during dobutamine stress in normal subjects and patients with coronary artery disease: comparison of changes in activation and inotropic state. *Heart* **85**, 411–416 (2001).
101. Duncan, A. M., Francis, D. P., Henein, M. Y. & Gibson, D. G. Limitation of cardiac output by total isovolumic time during pharmacologic stress in patients with dilated cardiomyopathy: activation-mediated effects of left bundle branch block and coronary artery disease. *J. Am. Coll. Cardiol.* **41**, 121–128 (2003).
102. Thygesen, K. *et al.* Third universal definition of myocardial infarction. *Eur. Heart J.* **33**, 2551–2567 (2012).
103. Gibson, D. G. & Francis, D. P. Clinical assessment of left ventricular diastolic function. *Heart* **89**, 231–238 (2003).
104. Henein, M. Y. & Gibson, D. G. Suppression of left ventricular early diastolic filling by long axis asynchrony. *Br. Heart J.* **73**, 151–157 (1995).
105. Brooks, H., Kirk, E. S., Vokonas, P. S., Urschel, C. W. & Sonnenblick, E. H. Performance of the right ventricle under stress: relation to right coronary flow. *J. Clin. Invest.* **50**, 2176–2183 (1971).
106. Rudski, L. G. *et al.* Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography endorsed by the European Association of Echocardiography, a registered branch of the European Society of Cardiology, and the Canadian Society of Echocardiography. *J. Am. Soc. Echocardiogr.* **23**, 685–713 (2010).
107. Hoeper, M. M. & Granton, J. Intensive care unit management of patients with severe pulmonary hypertension and right heart failure. *Am. J. Respir. Crit. Care Med.* **184**, 1114–1124 (2011).
108. Chockalingam, A., Tejwani, L., Aggarwal, K. & Dellsperger, K. C. Dynamic left ventricular outflow tract obstruction in acute myocardial infarction with shock: cause, effect, and coincidence. *Circulation* **116**, e110–e113 (2007).
109. Soar, J. *et al.* European Resuscitation Council Guidelines for Resuscitation 2015: Section 3. Adult advanced life support. *Resuscitation* **95**, 100–147 (2015).
110. Monsieurs, K. G. *et al.* European Resuscitation Council Guidelines for Resuscitation 2015: Section 1. Executive summary. *Resuscitation* **95**, 1–80 (2015).
111. Beaulieu, Y. Bedside echocardiography in the assessment of the critically ill. *Crit. Care Med.* **35**, S235–S249 (2007).
112. Shah, K. B. *et al.* Mechanical circulatory support devices in the ICU. *Chest* **146**, 848–857 (2014).
113. Stewart, G. C. & Givertz, M. M. Mechanical circulatory support for advanced heart failure: patients and technology in evolution. *Circulation* **125**, 1304–1315 (2012).

114. Thiele, H. *et al.* Intra-aortic balloon counterpulsation in acute myocardial infarction complicated by cardiogenic shock (IABP-SHOCK II): final 12 month results of a randomised, open-label trial. *Lancet* **382**, 1638–1645 (2013).
115. Werdan, K., Gielen, S., Ebelt, H. & Hochman, J. S. Mechanical circulatory support in cardiogenic shock. *Eur. Heart J.* **35**, 156–167 (2014).
116. Drakos, S. G. & Uriel, N. Spotlight on cardiogenic shock therapies in the era of mechanical circulatory support. *Curr. Opin. Cardiol.* **29**, 241–243 (2014).
117. Peura, J. L. *et al.* Recommendations for the use of mechanical circulatory support: device strategies and patient selection: a scientific statement from the American Heart Association. *Circulation* **126**, 2648–2667 (2012).
118. Kapur, N. K. *et al.* Mechanical circulatory support for right ventricular failure. *JACC Heart Fail.* **1**, 127–134 (2013).
119. Cheung, A. W., White, C. W., Davis, M. K. & Freed, D. H. Short-term mechanical circulatory support for recovery from acute right ventricular failure: clinical outcomes. *J. Heart Lung Transplant.* **33**, 794–799 (2014).
120. Kirklin, J. K. *et al.* Seventh INTERMACS annual report: 15,000 patients and counting. *J. Heart Lung Transplant.* **34**, 1495–1504 (2015).
121. Platts, D. G., Sedgwick, J. F., Burstow, D. J., Mullany, D. V. & Fraser, J. F. The role of echocardiography in the management of patients supported by extracorporeal membrane oxygenation. *J. Am. Soc. Echocardiogr.* **25**, 131–141 (2012).
122. Doufle, G., Roscoe, A., Billia, F. & Fan, E. Echocardiography for adult patients supported with extracorporeal membrane oxygenation. *Crit. Care* **19**, 326 (2015).
123. Ammar, K. A. *et al.* The ABCs of left ventricular assist device echocardiography: a systematic approach. *Eur. Heart J. Cardiovasc. Imaging* **13**, 885–899 (2012).
124. Stainback, R. F. *et al.* Echocardiography in the management of patients with left ventricular assist devices: recommendations from the American Society of Echocardiography. *J. Am. Soc. Echocardiogr.* **28**, 853–909 (2015).
125. Aissaoui, N. *et al.* Predictors of successful extracorporeal membrane oxygenation (ECMO) weaning after assistance for refractory cardiogenic shock. *Intensive Care Med.* **37**, 1738–1745 (2011).
126. Aissaoui, N. *et al.* Two-dimensional strain rate and Doppler tissue myocardial velocities: analysis by echocardiography of hemodynamic and functional changes of the failed left ventricle during different degrees of extracorporeal life support. *J. Am. Soc. Echocardiogr.* **25**, 632–640 (2012).
127. Evangelista, A. *et al.* European Association of Echocardiography recommendations for standardization of performance, digital storage and reporting of echocardiographic studies. *Eur. J. Echocardiogr.* **9**, 438–448 (2008).
128. Price, S. *et al.* Echocardiography practice, training and accreditation in the intensive care: document for the World Interactive Network Focused on Critical Ultrasound (WINFOCUS). *Cardiovasc. Ultrasound* **6**, 49 (2008).
129. Spencer, K. T. *et al.* Focused cardiac ultrasound: recommendations from the American Society of Echocardiography. *J. Am. Soc. Echocardiogr.* **26**, 567–581 (2013).
130. Levitov, A. *et al.* Guidelines for the appropriate use of bedside general and cardiac ultrasonography in the evaluation of critically ill patients — part II: cardiac ultrasonography. *Crit. Care Med.* **44**, 1206–1227 (2016).
131. Labovitz, A. J. *et al.* Focused cardiac ultrasound in the emergent setting: a consensus statement of the American Society of Echocardiography and American College of Emergency Physicians. *J. Am. Soc. Echocardiogr.* **23**, 1225–1230 (2010).
132. Via, G. *et al.* International evidence-based recommendations for focused cardiac ultrasound. *J. Am. Soc. Echocardiogr.* **27**, e1–e33 (2014).
133. Jones, A. E., Tayal, V. S. & Kline, J. A. Focused training of emergency medicine residents in goal-directed echocardiography: a prospective study. *Acad. Emerg. Med.* **10**, 1054–1058 (2003).
134. Lucas, B. P. *et al.* Diagnostic accuracy of hospitalist-performed hand-carried ultrasound echocardiography after a brief training program. *J. Hosp. Med.* **6**, 340–349 (2009).
135. Kajimoto, K. *et al.* Rapid evaluation by lung-cardiac-inferior vena cava (LCI) integrated ultrasound for differentiating heart failure from pulmonary disease as the cause of acute dyspnea in the emergency setting. *Cardiovasc. Ultrasound* **10**, 49 (2012).
136. Alrajhi, K., Woo, M. Y. & Vaillancourt, C. Test characteristics of ultrasonography for the detection of pneumothorax: a systematic review and meta-analysis. *Chest* **141**, 703–708 (2012).

Author contributions

S.P., L.C., and G.T. researched data for the article. S.P., E.P., and G.T. wrote the manuscript. S.P., L.C., E.P., J.M., and W.F.P. substantially contributed to the discussion of content. All the authors reviewed and edited the manuscript before submission.

Competing interests statement

The authors declare no competing interests.

Publisher's note

SUPPLEMENTARY INFORMATION

S1 (video) | S2 (table) | S3 (table) | S4 (figure) | S5 (figure)

ALL LINKS ARE ACTIVE IN THE ONLINE PDF