Heart failure is the primary cause of hospital admission in >1 million patients per year in the USA, with 25% of patients being readmitted within 1 month, and 10–20% mortality at 6 months after discharge. Acute heart failure (AHF) — either a new diagnosis in patients with no history of cardiac disease, or as a result of acute decompensation in patients with known heart failure — is the leading cause of hospital admission in individuals aged >65 years in the UK. According to data from Europe, approximately 50% of these patients will be readmitted within 12 months, and 30% will be deceased at the 1-year follow-up. Despite numerous clinical trials to assess optimal treatment and management strategies for patients with AHF, little improvement has been made in AHF outcomes in the past 30 years, with management decisions largely based on expert consensus rather than robust evidence. The burden of AHF is therefore substantial, both to individual patients and to society. The successful management of patients with any acute condition involves early diagnosis, the identification of underlying reversible causes, and the implementation of effective therapies in a timely manner, all while avoiding harm; all these factors are associated with better in-hospital and short-term prognosis. This Consensus Statement, prepared by the Acute Heart Failure Study Group of the ESC Acute Cardiovascular Care Association, reviews the existing and potential roles of echocardiography and lung ultrasonography in the assessment and management of patients with acute heart failure, highlighting the differences from established practice where relevant.

Heart failure is a syndrome rather than a diagnosis per se, caused by a wide array of pathologies that result in a spectrum of disease severity ranging from breathlessness to cardiogenic shock or cardiac arrest. AHF is a highly lethal condition, and studies have shown that minimizing the ‘time to appropriate therapy’ — the initiation of treatment as soon as possible, including in the prehospital setting — is potentially beneficial in improving outcomes. AHF is variably defined as the rapid onset or acute
Key points

- Over-reliance of traditional clinical findings and symptoms can potentially delay diagnosis of acute heart failure (AHF), prolonging the time to appropriate therapy
- The use of echocardiography and lung ultrasonography can help to improve diagnostic accuracy and monitor responses to interventions in patients with AHF
- Lung ultrasonography allows for rapid assessment of numerous conditions, including pulmonary oedema, pleural effusion, and pneumothorax
- Use of echocardiography has extended beyond the traditional application in stable patients to become widespread in the acute and emergency settings
- In the setting of AHF, echocardiography can be used to assess pericardial effusion, right ventricular dilatation, left ventricular systolic function, gross valvular abnormality, and potentially the presence of intracardiac masses
- Echocardiography can also be used to monitor treatment in patients with cardiogenic shock

delay diagnosis and implementation of appropriate therapy, or contribute to a missed diagnosis in up to 20% of patients. Furthermore, patients’ clinical features might vary according to the site of initial medical contact and the management strategies employed.

The majority of patients with AHF present to emergency departments; however, many patients are also assessed and managed in other acute care settings such as in intensive care and inpatient cardiology units. Patients with AHF usually present with symptoms of congestion and breathlessness rather than cardiac arrest or shock. Symptoms of breathlessness account for 3–5% of emergency department attendances in Europe and the USA, and the major causes of breathlessness and their prevalence include AHF (50%), pneumonia or bronchitis (20%), exacerbation of chronic obstructive pulmonary disease or asthma (20%), and pulmonary embolism (5–10%). Current guidelines recommend that clinical examination and investigations should be integrated to form the diagnosis, including the use of electrocardiogram (ECG), chest radiograph, and biomarkers such as natriuretic peptides, troponin, and D-dimer as indicated. Unfortunately, these data can be challenging to interpret, in particular in the 10–15% of patients in whom two concomitant diagnoses exist. Specifically, although included in the current definition of AHF, levels of natriuretic peptides can be elevated in respiratory disease and other acute conditions such as pulmonary embolism, sepsis, and anaemia.

Any acute condition can be further complicated by the external factors present in emergency settings, such as high ambient noise and restrictive space, limiting a clinician’s ability to position the patient optimally for examination. Furthermore, the frequently atypical features of very severe pathology (in particular valvular disease), and the time pressures imposed by an acutely deteriorating patient can contribute to poor outcomes. These factors are further confounded by the presence of concomitant pathologies in the increasingly ageing patient population.

Echocardiography and LUS are readily available and widely validated techniques that can be used to reveal anatomical and physiological abnormalities in patients with AHF, which when correctly applied in the acute setting, can improve patient assessment, management, and outcomes. Unlike other biomarkers used in AHF, echocardiography and LUS can be used to identify not only inadequate cardiac output and/or the presence of congestion, but also the underlying cause, allowing the most appropriate, individualized interventions to be delivered immediately to the patient. Furthermore, these imaging modalities can be used to monitor the effects of treatment (either beneficial or detrimental), as well as to guide patient disposition and interventions as indicated. Pocket-sized echocardiography devices are practical for screening, and provide information to clinicians in addition to that gathered from auscultation by a stethoscope alone. When AHF is suspected, an integrative approach is recommended, including determination of cardiopulmonary
CONSENSUS STATEMENT

instability and evaluation of congestion (pulmonary and peripheral) using a combination of techniques\(^1\). When image quality is inadequate, either transoesophageal echocardiography or the use of contrast should be considered.

**Lung ultrasonography**

Based on the interpretation of a number of artefacts, specific ultrasonography appearances, and their distribution (FIG. 1), LUS allows for a rapid point-of-care evaluation of a number of conditions, including pulmonary oedema, lung consolidation, pleural effusion, and pneumothorax\(^29\). High intra-rater and inter-rater reproducibility, ease of learning, short exam duration (<5 min), and the noninvasive nature of this technique makes it an advantageous point-of-care tool\(^30,31\). LUS is increasingly used in the acute care setting, and has improved diagnostic accuracy compared with clinical assessment and chest radiography for the identification of a cardiac aetiology in patients presenting to the emergency department with undifferentiated dyspnoea\(^31\).

**Interstitial fluid and pulmonary oedema**

Quantification of B-lines (vertical artefacts that result from an increase in interstitial density; FIG. 1b) has been shown to be useful for the diagnosis, monitoring, and risk assessment of patients with known or suspected AHF\(^34-38\). Either curvilinear or phased array transducers can be used, typically at an imaging depth of 18 cm. Although the assessment of eight or more anterior and lateral thoracic zones (four on each hemithorax) has been recommended in a consensus statement\(^39\), a subsequent study demonstrated high diagnostic accuracy with examination of only six thoracic regions\(^40\). The visualization of three or more B-lines in two or more intercostal spaces bilaterally should be considered diagnostic for pulmonary oedema, with sensitivity of 94% (95% CI 81–98%) and specificity of 92% (95% CI 84–96%)\(^31,37\). By contrast, physical examination and chest radiography have a sensitivity of only 62% (95% CI 61–64%) and 57% (95% CI 55–59%), and a specificity of 68% (95% CI 67–69%) and 89% (95% CI 88–90%) for a diagnosis of pulmonary oedema, respectively\(^31\).

The presence of multiple bilateral B-lines in AHF has been well-correlated with natriuretic peptide levels, and only variably correlated with pulmonary capillary wedge pressure and measures of extravascular lung water\(^30,33,35,39-41\). Given that studies to assess the incremental diagnostic value of LUS compared with natriuretic peptides for the identification of AHF in patients with dyspnoea reported variable results in different cohorts, this topic warrants further investigation\(^31,33,42\). The number of B-lines is thought to decrease with treatment for AHF and, therefore, this technique is potentially useful in the monitoring of pulmonary oedema in response to therapy\(^31,36\). For serial assessments, patient positioning (sitting versus supine) should be kept consistent\(^43\). Importantly, a higher number of B-lines on LUS at the time of discharge from hospital might help to identify patients with heart failure who have a worse prognosis\(^36\).

**Echocardiographic methods to estimate left atrial pressure**

The upper panels show the echocardiographic scan of a patient aged 45 years admitted to hospital with dyspnoea owing to severe acute respiratory failure. a | Transthoracic echocardiogram (TTE) of the mitral inflow pattern showing a normal early (E) and late (A) transmitral flow pattern. b | Tissue Doppler imaging (TDI) of the lateral mitral valve annulus from the same patient; S is systolic annular velocity, E’ is early annular diastolic velocity, and A’ is late annular diastolic velocity (related to atrial contraction). c | Pulmonary venous Doppler (transoesophageal echocardiography) demonstrating a dominant systolic wave (S) and smaller diastolic wave (D), with a normal deceleration time. The E/A ratio is >1 and the E/E’ is <8 cm/s with a dominant S wave on pulmonary vein, consistent with a normal left atrial pressure. The lower panels show the echocardiographic scan of a female patient aged 59 years admitted with dyspnoea owing to severe left ventricular dysfunction with pulmonary oedema. d | TDI of the septal mitral valve annulus with a very low early diastolic velocity (E’), and e | pulmonary venous Doppler (transoesophageal echocardiography) showing a blunted systolic wave (S) and dominant diastolic wave (D). The E/E’ is 16.3 cm/s, and dominant D wave on pulmonary venous Doppler with D deceleration time <150 ms are consistent with an elevated left atrial pressure.

**Lung and pleural ultrasonography.** a | Normal lung with pleural line, and ribs (*) with shadowing. b | Pulmonary oedema with multiple vertical B-lines (arrows) arising from the pleural line. c | Diaphragmatic view with spine ending at the level of the diaphragm, with no pleural effusion. d | Pleural effusion seen as anechoic (echo-free) space above the diaphragm with atelectatic lung. Spine can be visualized beyond the diaphragm owing to the effusion.

**Figure 1** | **Echocardiographic methods to estimate left atrial pressure.** The upper panels show the echocardiographic scan of a patient aged 45 years admitted to hospital with dyspnoea owing to severe acute respiratory failure. a | Transthoracic echocardiogram (TTE) of the mitral inflow pattern showing a normal early (E) and late (A) transmitral flow pattern. b | Tissue Doppler imaging (TDI) of the lateral mitral valve annulus from the same patient; S is systolic annular velocity, E’ is early annular diastolic velocity, and A’ is late annular diastolic velocity (related to atrial contraction). c | Pulmonary venous Doppler (transoesophageal echocardiography) demonstrating a dominant systolic wave (S) and smaller diastolic wave (D), with a normal deceleration time. The E/A ratio is >1 and the E/E’ is <8 cm/s with a dominant S wave on pulmonary vein, consistent with a normal left atrial pressure. The lower panels show the echocardiographic scan of a female patient aged 59 years admitted with dyspnoea owing to severe left ventricular dysfunction with pulmonary oedema. d | TDI of the septal mitral valve annulus with a very low early diastolic velocity (E’), and e | pulmonary venous Doppler (transoesophageal echocardiography) showing a blunted systolic wave (S) and dominant diastolic wave (D). The E/E’ is 16.3 cm/s, and dominant D wave on pulmonary venous Doppler with D deceleration time <150 ms are consistent with an elevated left atrial pressure.

**Figure 2** | **Lung and pleural ultrasonography.** a | Normal lung with pleural line, and ribs (*) with shadowing. b | Pulmonary oedema with multiple vertical B-lines (arrows) arising from the pleural line. c | Diaphragmatic view with spine ending at the level of the diaphragm, with no pleural effusion. d | Pleural effusion seen as anechoic (echo-free) space above the diaphragm with atelectatic lung. Spine can be visualized beyond the diaphragm owing to the effusion.

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### Table 1: Challenges in using echocardiography to determine the underlying cause of AHF

| Underlying cause | AHF-related clinical presentation | Echo findings | Notes and potential pitfalls |
|------------------|-----------------------------------|---------------|------------------------------|
| **ACS and ischaemic heart disease** | Dyspnoea, as atypical presentation of ACS | • Standard RWMA  
• Abnormalities on transmural Doppler imaging | • Transient ischaemia: echo might be normal  
• RWMA not specific for coronary disease  
• Contrast might improve diagnostic accuracy in critically ill patients |
| **Shock** | | • LV dysfunction | • EF influenced by volume, loading, and inotropic status  
• Normal or hyperdynamic left ventricle in unstable AMI implies potential mechanical complication |
| **Severe MR:** | | • Ventricular wall rupture: only evidence is pericardial collection (30% of patients)  
• Primary (papillary muscle rupture and dysfunction)  
• Secondary (leaflets normal, but associated with RWMA) | • Easy to underestimate degree of LV dysfunction  
• In very severe MR, colour Doppler might underestimate severity  
• Complete or partial papillary muscle rupture  
• Secondary MR can be dynamic |
| **Ventricular septal rupture:** | | • RV infarct: features of inferior MI ± RV dysynergy and paradoxical septal motion  
• ZD defect in area of infarction with corresponding colour Doppler  
• Can be multiple | • Suspected if TR is low velocity, but PR has steep pressure half-time  
• Assessment of LV function can be challenging, owing to reduced preload  
• Extent of LV dysfunction might be revealed if RV MCS is used |
| **Myocarditis** | Widely variable, might be within AHF spectrum | • Nonspecific: LV systolic and diastolic dysfunction, resting RWMA, and nonspecific changes in image texture | • Additional features: thrombi, secondary MR/TR, pericardial involvement  
• More fulminant: thickening of myocardial walls (oedema)  
• Speckle tracking: reduction in GLS correlates with myocardial inflammation (but nonspecific for the disease)  
• Real-time low-mechanical index MCE might be helpful |
| **Takotsubo syndrome** | Widely variable, might be within AHF spectrum | • Reversible LV dysfunction with RWMA extending beyond coronary territory distribution | Echocardiographically more heterogeneous than originally described  
• Biventricular involvement in 25%  
• Midsegment involvement in 40% |
| **Dissection** | Shock | • Dissection flap, varying degrees of AR, and RWMA from coronary involvement | Normal TTE does not exclude dissection  
• AR might be overestimated if dissection flap prolapses through aortic valve |
| **Cardiomyopathy** | Full spectrum of AHF | • Doppler evidence of elevated filling pressures  
• LUS might show pulmonary oedema | • EF influenced by volume, loading, and inotropic status  
• RWMA might occur in absence of coronary disease  
• GLS potentially useful (≤10% indicates severe reduction)  
• GLS and STE not well-validated in acute settings and in the context of positive inotropic agents  
• HCM: standard echo features, including estimation of PASP and LAF, plus degree of LVOTO | Severity of LVOTO might be dynamic and worsen with positive inotropic agents and/or hypervolaemia  
• Worsening MR might be dynamic |
| **Pulmonary embolism** | Full spectrum of AHF | • Dilatation of right heart, RV hypokinesia, abnormal interventricular septal motion  
• Diagnostic: mobile serpentine thrombus in right heart/pulmonary artery | • Findings nonspecific for pulmonary embolism  
• Expect to see high PVR  
• In shock, normal right heart virtually excludes pulmonary embolism as the cause  
• Very severe RV dysfunction might underestimate degree of pulmonary obstruction  
• Very severe TR might underestimate degree of pulmonary hypertension |
| **Pneumothorax** | From dyspnoea to cardiac arrest | • Absence of pleural sliding  
• Demonstration of lung point is diagnostic | If tension pneumothorax suspected in cardiac arrest, treatment should not be delayed for LUS  
• In right mainstem intubation, expect absent lung sliding on left hemithorax |
| Underlying cause | AHF-related clinical presentation | Echo findings | Notes and potential pitfalls |
|------------------|----------------------------------|---------------|-----------------------------|
| Valve disease    | Mitral regurgitation; from dyspnoea to shock | • Severity assessed according to standard echo parameters (integrated approach)  
• Underlying causes: ischaemia, endocarditis, trauma, heart failure | • Must include cardiorespiratory support: PPV and pharmacological agents can reduce severity significantly  
• Almost always severe in context of papillary muscle rupture  
• Colour Doppler might underestimate severity if valve disease is very severe owing to rapid equalization of pressures  
• Early truncation of MR velocities is a useful sign  
• Suspect in patients with hyperdynamic left ventricle and pulmonary oedema  
• Premature closure of MV (with diastolic MR) implies catastrophic regurgitation  
• If endocarditis suspected, and TTE is nondiagnostic, TOE should be performed |
| Aortic regurgitation; from dyspnoea to shock | • Severity assessed according to standard echo parameters (integrated approach)  
• Underlying causes: dissection, endocarditis | • Short PHT (<200 ms)  
• Diastolic flow reversal in descending aorta (EDV >20 cm/s)  
• Premature diastolic opening of aortic valve implies catastrophic regurgitation  
• Care in evaluation if considering ECMO; even mild degrees of AR might be important (and preclude peripheral ECMO). No aortic valve opening with use of ECMO suggests further LV decompression might be indicated |
| Mitral stenosis; might mimic ARDS | • Severity assessed according to standard echo parameters (integrated approach) | • Acute deterioration might be caused by physiological (pregnancy) or pathological (arrhythmia) precipitant  
• Might see pulmonary infiltrates even in not very severe disease if in combination with lung injury |
| Aortic stenosis; from dyspnoea to shock to cardiac arrest | • Severity assessed according to standard echo parameters (integrated approach) | • Care in evaluation in presence of peripheral ECMO, because increase in afterload might reduce aortic valve opening  
• Contraindication to Impella (Abiomed, USA) |
| Valve prosthesis dysfunction; from dyspnoea to shock | • Echo features of valve dysfunction  
• Underlying causes: thrombus, pannus, endocarditis, dehiscence, degeneration | • Normalization of septal motion should raise suspicion  
• Consider if pulmonary infiltrates and ‘good’ or hyperdynamic left ventricle in patient with previous AV/MV replacement  
• Indication for expert TOE  
• Increased transvalvular velocities must be interpreted in context of CO |
| Sepsis | Clinically septic, but inadequate CO | • Frequently hyperkinetic  
• Pulmonary hypertension: degree of RV dysfunction not uncommon (30%)  
• LV/biventricular dysfunction might occur | • If sepsis accompanies pneumonia and venovenous ECMO anticipated, take care to assess right ventricle as it might not tolerate volume load  
• Intracardiac source of sepsis might be present (related to line, device, or valve)  
• Speckle tracking proposed (not validated in adults) to identify early sepsis-related dysfunction |
| Tamponade | Dyspnoea to shock to cardiac arrest | • Demonstration of accumulation of fluid in pericardial space with or without features of tamponade | • Small collections occurring rapidly can result in tamponade  
• Localized collections/presence of cardiac or pulmonary disease might suppress features of tamponade  
• Results of postcardiac surgery TTE are frequently negative |

ACS, acute coronary syndrome; AHF, acute heart failure; AR, aortic regurgitation; ARDS, acute respiratory distress syndrome; AV, aortic valve; CO, cardiac output; Echo, echocardiography; ECMO, extracorporeal membrane oxygenation; EDV, end-diastolic velocity; EF, ejection fraction; GLS, global longitudinal strain; HCM, hypertrophic cardiomyopathy; LAP, left atrial pressure; LUS, lung ultrasonography; LV, left ventricular; LVOTO, left ventricular outflow tract obstruction; MCE, myocardial contrast echocardiography; MCS, mechanical circulatory support; MI, myocardial infarction; MR, mitral regurgitation; MV, mitral valve; PASP, pulmonary artery systolic pressure; PHT, pressure half-time; PPV, positive pressure ventilation; PR, pulmonary regurgitation; PVR, pulmonary vascular resistance; RV, right ventricular; RWMA, regional wall motion abnormality; STE, speckle-tracking echocardiography; TOE, transoesophageal echocardiography.

**Pleural effusion**

Similarly to B-lines, the presence of pleural effusions can be assessed using curvilinear or phased array transducers in the posterior–axillary line\(^44\) (FIG. 1d). Current data regarding the diagnostic utility of pleural effusions identified on ultrasonography in patients with AHF are less robust, but have been reported with sensitivities of 79–84% and specificities of 83–98% in small studies of patients with dyspnoea\(^44,45\).

**Pneumothorax**

LUS can be used to exclude pneumothorax in the area scanned with higher sensitivity than supine chest radiography by recognizing lung sliding, a slight horizontal movement of the pleural line with respiration; see Supplementary information S1 (video)\(^46\). In the setting of a pneumothorax, lung sliding is absent in the affected area of the chest. At the border of a pneumothorax, a transition point between normal lung surface
might indicate that two pathologies coexist, or that the B-lines are an expression of pathology other than AHF (for example, acute respiratory distress syndrome, or pulmonary oedema in patients receiving haemodialysis). Third, large pleural effusions might interfere with B-line quantification in the affected thoracic zones and induce lung consolidation (Fig. 1d). Together, these considerations outline why LUS should not be used in isolation, but rather integrated into clinical and laboratory assessment.

Echocardiography in AHF
Driven by progressive advances in ultrasonography technology and an expanding evidence base, the use of echocardiography has extended beyond the traditional application in stable patients to become widespread in the acute and emergency settings. Mirroring the concept of critical care, echocardiography is increasingly used as a tool to guide management of the most acutely unwell patients wherever they present along the management pathway. Pocket-sized devices have been recommended in the emergency department, intensive care unit, and coronary units for fast initial qualitative screening of ventricular and valvular function, pericardial and pleural effusion, or extravascular lung water. However, owing to the known limitations of this technique, they are not intended as a substitute for comprehensive echocardiography. Remote expert review of images is now a possibility, and in the future, telemedicine will probably have an important role in guiding the assessment and management of these acutely unwell patients.

Echocardiography is used in AHF to help to confirm diagnosis, delineate potential underlying causes, identify associated pathophysiology, and monitor the response to therapy. Echocardiography can also be used to guide specialist interventions in the catheter laboratory or operating room. Furthermore, echocardiography can address several major questions, including whether a patient has a cardiac cause for their symptoms and signs, the severity of the cardiac impairment and its physiological effect, whether there is an underlying reversible cause, what the most appropriate initial treatment is, and how the patient responds to treatment.

Guidelines recommend immediate echocardiographic assessment for patients with suspected AHF with haemodynamic instability; however, interpretation of echocardiographic data in these acutely unwell patients can be extremely complex (Table 1). First, the finding of a structurally or functionally abnormal heart does not necessarily mean the cause of dyspnoea is cardiac-related. Second, patients might be misdiagnosed as having primary respiratory disease, even in the presence of very severe cardiac pathology. Third, substantial cardiac and respiratory disease might coexist, and determining the degree of cardiac contribution is frequently challenging in this setting. These considerations are further compounded by the relative paucity of high-quality evidence to support the use of echocardiography techniques in the acute arena, as they have been predominantly validated in the outpatient clinic.
resulted in a reduction of t−IVT from 16.8 s/min to 10.0 s/min, and a corresponding
also in s/min) is calculated as 60−(t−FT + t−ET). A heart rate reduction of 10 bpm
imaging at 100 bpm. The filling time (FT) is measured from the start to the end of
Doppler imaging at 90 bpm.

Dyspnoea resulting from left-sided cardiac disease is likely to be associated with elevated left atrial pressure (LAP) and pulmonary oedema. Historically, pulmonary capillary wedge pressure has been measured using a pulmonary artery catheter as a substitute for LAP measurement. The use of the pulmonary artery catheter has greatly declined over the past decade, owing to a number of studies that showed potential harm or no improved outcomes in the perioperative and critical care settings. Although absolute pressure values cannot be measured using echocardiography, a drive has occurred to find an echocardiography-derived parameter that can be used to estimate the LAP noninvasively. Indices that have been proposed include interrogation of the transmitral left ventricular (LV) filling pattern (E/A ratio, E wave deceleration time, and the isovolumic relaxation time), pulmonary venous Doppler diastolic deceleration time (Fig. 2), M-mode colour Doppler propagation velocities, the time interval between the onset of early diastolic mitral inflow (E) and annular early diastolic velocity (e’) by tissue Doppler imaging, and the E/e’ ratio. None of these measures has been well-validated in the context of emergency medicine; they all present technical challenges that must be carefully considered for accurate interpretation, and provide only estimates of a potential range of corresponding LAP values. Even when used in combination (as proposed in critical care), they can at best only indicate that the LAP is probably very high or normal.

LV ejection fraction has been the main parameter used for the diagnosis, treatment, and stratification of patients with heart failure. However, this parameter has several limitations that are particularly relevant in the acute setting, such as load-dependency and inotropy-dependency. Even in the absence of high-quality 2D images, Doppler abnormalities in transmitral filling might provide an early indicator of important pathology.

Unlike LUS, echocardiography might be challenging to perform well and interpret accurately, as a number of considerations add to the complexity of its application in the acute setting. First, in all parameters described for LAP estimation, the confounding factors imposed by critical illness (changes in heart rate, cardiac output, LV compliance, and volume and ventilatory status) have not been fully evaluated. Second, not only might patients with a relatively normal LAP have radiographic and sonographic evidence of pulmonary oedema, but conversely, patients with chronically elevated LAP might have no evidence of pulmonary oedema. Similarly to LUS, however, the echocardiographic findings should be integrated with those from clinical examination, laboratory investigations, and lung imaging data (radiographic and/or sonographic), and be assessed within the clinical context. The main value of echocardiography in this setting is to diagnose or exclude an underlying cardiac cause for dyspnoea and guide subsequent interventions.

Right-sided disease: pulmonary embolism

The diagnosis of pulmonary embolism can be challenging, because symptoms and signs are nonspecific. The transthoracic echocardiogram is normal in approximately
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 technique19. 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 dilatation78 (FIG. 3a). Secondary tricuspid regurgitation might be present, allowing estimation of pulmonary arterial systolic pressure using the simplified Bernoulli equation41 (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)85. 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 resistance41,82.

**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 pericardiocentesis83. 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 tamponade44.

**Monitoring of therapy**

Echocardiography is not recommended for the monitoring of therapy in patients with AHF in the absence of cardiogenic shock8,9,11, 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 discharge88.

**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)89,10,11. Precise definitions of cardiogenic shock can vary; however, the syndrome generally results from inadequate cardiac output for peripheral organ requirements85,86. 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 saturation86. 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 fatal85,86 (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 isotropic and ventilatory support, as well as metabolic and arterial blood gas status, because these variables might have profound effects on echocardiographic findings.
**CONSENSUS STATEMENT**

**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\(^{67,88}\). Static echocardiographic parameters are widely used to predict volume responsiveness in critically ill patients (FIG. 4\(^b\)), 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\(^{85}\). 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 |
| 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 not tolerated haemodynamically)  
• Support for BAV (experimental) | • Nonrecoverable disease and not suitable for transplantation or VAD  
• Severe neurologic injury or intracerebral bleeding  
• Ventricular septal defect, or interatrial defect, severe aortic stenosis, and severe aortic regurgitation  
• Mechanical aortic valve  
• Sepsis  
• Bleeding diastasis  
• Severe peripheral vascular disease (left-sided device) | • Validation of underlying cause  
• Biventricular function assessment  
• Adequate device position  
• Positioning of inlet and outlet of device |
| Tandem Heart (Cardiac Assist, USA) | • High-risk PCI and acute MI  
• AMI complicated by cardiogenic shock | • Bleeding diastasis  
• 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 |
| 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.
CONSENSUS STATEMENT

findings published in the past 3 years. 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.

**Inotropes and vasoactive agents.** Although inotropes and vasoressors 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. Dobutamine is generally the first-line inotrope of choice in the clinic. The detrimental effects of positive inotropic agents have been extensively described in the literature, 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).

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. 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. Echocardiographic demonstration of a dominant or isolated A wave on transmittal Doppler in combination with post-ejection shortening can also be diagnostic, and indicates that aortic root pressure should be increased and/or LVEDP reduced. 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.

Echocardiography can be used to estimate pulmonary artery systolic pressure and pulmonary vascular resistance, as well as measure RV dimensions and performance. 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. Echocardiography can help to diagnose LV outflow tract obstruction (with or without associated mitral regurgitation). Treatment in this context involves reduction or cessation of positive inotropic agents, in combination with volume and pressor support.

**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. 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.

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**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 intraventricular 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 torrrential 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.
### 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**[121,122]                                              |                                      |                                     |                                                                          |                                                                          |
| Alternative diagnoses for patient’s signs and symptoms?                             | Pericardial effusion                 | Subxiphoid, paraстernal 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[121,124] |
|                                                                                   | RV dilatation/systolic function      | Subxiphoid, paraстernal long-axis and short-axis views, apical four-chamber view  |                                                                           | RV dysfunction (various criteria): sensitivity 74%, specificity 54% for diagnosis of acute PE[13] |
| Evidence of impaired systolic function?                                           | Global LV systolic function          | Subxiphoid, paraстernal 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[125,126] |
| 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[125] |
| Gross structural abnormality as AHF aetiology?                                     | * Gross valvular abnormality*        | Subxiphoid, paraстernal long-axis and short-axis views, apical four-chamber view  | AHF aetiology might be identified in rare cases                          | NA                                                                      |
|                                                                                   | * Intracardiac mass                  |                                                                                   |                                                                          |                                                                          |
| **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[12]          |
| 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[15,18] |
| 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[14,15] |

*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[111]. 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 unloaded.
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.25,32,12h

Other indications
Transoesophageal echocardiography 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.6,12,13–18. 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 physiology, 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.
Oh, J. K. Echocardiography as a noninvasive

52. Temporelli, P. L., Scapellato, F., Eleuteri, E., Imparato, A. & Gheorghiade, M. Doppler echocardiography in advanced systolic heart failure: a noninvasive alternative to Swan–Ganzer catheter.

53. Ritzema, J. L. et al. Serial Doppler echocardiography and tissue Doppler imaging in the detection of elevated diastolic arterial pressure and in ambulant subjects with chronic heart failure.

54. Lester, S. J. et al. Unlocking the mysteries of diastolic function: an update on LAD Ultrasound Stone 10 years later. J. Am. Coll. Cardiol. 51, 679–689 (2008).

55. Vignon, P. et al. Echocardiographic assessment of pulmonary artery occlusion pressure in ventilated patients: a transoesophageal study. Crit. Care 12, R18 (2008).

56. Vignon, P. Hemodynamic assessment of critically ill patients using echocardiography Doppler. Curr. Opin. Crit. Care 11, 227–234 (2005).

57. Cikes, M. & Solomon, S. D. Beyond ejection fraction: the importance of the long axis dynamics of the human left ventricle. Br. Heart J. 65, 215–220 (1990).

58. Henni, M. Y. & Gibson, D. G. Long axis function in heart disease. Heart 81, 229–231 (1999).

59. Tavazzi, G. et al. A 50-year-old woman with ongoing dyspnea. Chest 150, e9–e11 (2016).

60. Pfeifer, A., Tammi, C., Unger, P. F., Leh, R. & Sztajzel, J. Diagnostic accuracy of Doppler echocardiography in unselected patients with suspected pulmonary embolism. Int. J. Cardiol. 65, 101–109 (1998).

61. Casazza, F., Bongarzoni, A., Capozi, A. & Agostoni, O. Regional right ventricular function in acute pulmonary embolism and right ventricular failure. Eur. J. Echocardiogr. 6, 11–14 (2005).

62. Amalsalem, M. et al. Addressing the controversy of estimating pulmonary arterial pressure by echocardiography. J. Am. Soc. Echocardiogr. 29, 95–102 (2016).

63. Chapman, 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 future research implications. Circulation 120, 992–1007 (2009).

64. Lindqvist, P., Cutucattea, A. & Henein, M. Echocardiography in the assessment of right heart function. J. Am. Soc. Echocardiogr. 29, 225–234 (2008).

65. Bossone, E. et al. Echocardiography in pulmonary arterial hypertension: from diagnosis to prognosis. J. Am. Soc. Echocardiogr. 26, 731–739 (2013).

66. Imazio, M. & Adler, Y. Management of pericardial effusion. Eur. Heart J. 36, 1186–1197 (2015).

67. 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).

68. Thiele, H., Olshen, E. M., Desch, S., Ette, I. & de Waala, S. Management of cardiogenic shock. Eur. Heart J. 36, 1223–1230 (2015).

69. Reynolds, H. R. & Hochman, J. S. Cardiogenic shock: current concepts and improving outcomes. Circulation 117, 688–697 (2008).

70. Monnet, X. & Teboul, J. Assessment of volume responsiveness during mechanical ventilation: recent advances. Crit. Care 17, 213 (2013).

71. 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).

72. Charron, C., Callens, A. & Veillard–Baron, A. Echocardiographic measurement of fluid responsiveness. Curr. Opin. Crit. Care 12, 249–254 (2006).

73. Mebazaa, A., Karpadi, P., Renaud, E. & Algòtsson, L. Acute right ventricular failure — from pathophysiology to new treatments. Intensive Care Med. 30, 185–196 (2004).

74. Inohara, T., Kutsaka, S., Fukuda, K. & Menon, V. The challenges in the management of acute pulmonary infarction. Eur. Heart J. Acute Cardiovasc. Care 2, 226–234 (2013).

75. Bendjif, K. & Desvigne, J. A. Fluid responsiveness in mechanically ventilated patients: a review of indices used in intensive care. Intensive Care Med. 29, 352–356 (2003).

76. 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).

77. Reus, C., Vincent, J. L. & Payen, D. Management of right ventricular volumes during fluid challenge. Chest 98, 1450–1454 (1990).

78. Francis, G. S., Bartos, J. A., Perrier, A., Unger, P. F. & Tamm, C. Inotropes. Br. J. Pharmacol. 165, 2015–2033 (2012).

79. Singer, M. Catecholamine treatment for shock — equally good or bad? Lancet 370, 656–657 (2007).

80. Duncan, A. M., Francis, D. P., Gibson, D. G., & Henni, 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).

81. Duncan, A. M., O’Sullivan, C. A., Gibson, D. G., & Henni, 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 (2000).

82. Duncan, A. M., Francis, D. P., Henni, 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).

83. Thysgen, K. et al. Third universal definition of myocardial infarction. Eur. Heart J. 33, 2551–2567 (2012).

84. Gibson, D. C. & Francis, D. P. Clinical assessment of left ventricular diastolic function. Heart 89, 231–238 (2003).

85. Henni, M. Y. & Gibson, D. G. Suppression of left ventricular early diastolic filling by long axis asynchrony. Br. Heart J. 87, E175 (2002).

86. 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. 90, 217–225 (1992).

87. Rudski, L. G. et al. Guidelines for the echocardiographic assessment of the right heart in adults: a report from the American Society of Echocardiography. J. Am. Soc. Echocardiogr. 25, 685–710 (2012).

88. Hooper, M. G. & Granton, J. Intensive care management of patients with severe pulmonary hypertension and right heart failure. Am. J. Respir. Crit. Care Med. 184, 1114–124 (2011).

89. Chockalingam, A., Teivani, L., Aggarwal, K. & Dellisperger, K. C. Dynamic left ventricular outflow tract obstruction in acute myocardial infarction with shock: cause, effect and coincidence. Circulation 116, e10–e11 (2007).

90. Soar, J. et al. European Resuscitation Council Guidelines for Resuscitation 2015: Section 8. Adult advanced life support. Resuscitation 95, 100–147 (2015).

91. Monnet, X. et al. European Resuscitation Council Guidelines for Resuscitation 2015: Section 1. Executive summary. Resuscitation 95, 1–80 (2015).

92. Beaulieu, Y. Bedside echocardiography in the assessment of the critically ill. Curr. Med. Res. Opin. 25, S235–S249 (2007).

93. Beaulieu, Y. Bedside echocardiography in the assessment of the critically ill. Curr. Med. Res. Opin. 25, S235–S249 (2007).

94. Shah, K. B. et al. Mechanical circulatory support devices in the ICU. Chest 146, 848–857 (2014).

95. Stewart, G. C. & Givertz, M. M. Mechanical circulatory support for advanced heart failure: patients and technology in evolution. Circulation 125, 1504–1515 (2012).
