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The use of echocardiography in acute cardiovascular care: Recommendations of the European Association of Cardiovascular Imaging and the Acute Cardiovascular Care Association

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Echocardiography is one of the most powerful diagnostic and monitoring tools available to the modern emergency/ critical care practitioner. Currently, there is a lack of specific European Association of Cardiovascular Imaging/Acute Cardiovascular Care Association recommendations for the use of echocardiography in acute cardiovascular care. In this document, we describe the practical applications of echocardiography in patients with acute cardiac conditions, in particular with acute chest pain, acute heart failure, suspected cardiac tamponade, complications of myocardial infarction, acute valvular heart disease including endocarditis, acute disease of the ascending aorta and post-intervention complications. Specific issues regarding echocardiography in other acute cardiovascular care scenarios are also described.

Keywords Acute cardiovascular care • Critically ill patients • Echocardiography • Recommendations

Introduction

Echocardiography is one of the most powerful diagnostic and monitoring tools available to the modern emergency/ critical care practitioner, and the provision of echocardiography is fundamental to the management of patients with acute cardiovascular disease. Echocardiography can provide important information throughout the whole patient pathway, having been shown to change therapy in 60–80% of patients in the pre-hospital setting, improve diagnostic accuracy and efficiency in the emergency room, reveal the aetiology of unexplained hypotension in 48% of medical intensive care patients and provide information additional to that obtained from the pulmonary artery catheter. Echocardiography is now included in the universal definition of acute myocardial infarction (AMI), and in international guidelines regarding the management of cardiac arrest. In the critical care setting echocardiography can be used to measure/monitor cardiac output and to determine abnormalities of cardiac physiology and coronary perfusion, as well as providing more standard anatomical information related to diagnosis. Although the potential scope of echocardiography is evident, specific recommendations for its use in acute cardiac care are currently lacking from the European Association of Cardiovascular Imaging (EACVI) and the Acute Cardiovascular Care Association (ACCA). In this document, we describe the practical applications of echocardiography in patients with acute

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cardiovascular conditions, in particular with acute chest pain, acute heart failure, suspected cardiac tamponade, complications of MI, acute valvular heart disease including endocarditis, acute disease of the ascending aorta and post-intervention complications. Specific issues regarding echocardiography in different acute cardiac care scenarios are also described.

Types of echocardiographic imaging

The numerous challenges of imaging in the acute setting are well-documented, and consist in a number of factors including patient habitus, supine/prone positioning, positive pressure ventilation, lung injury and related features (pneumothorax/pneumo-mediastinum), trauma (head and neck, thoracic) and the presence of lines/dressings and/or drains. Further, the echocardiographic data must be interpreted in the context of the acutely/critically ill patient, thus incorporating a number of factors that are not normally considered by the echocardiographic practitioner (*Table 1*). Additionally, there may be time-critical factors that further challenge the echocardiographer (i.e. cardiac arrest).

The choice of imaging modality in the acutely/critically ill patient population will not only depend upon the sensitivity and specificity of the modality for a given potential diagnosis, but will also include the risks of transportation and to potentially remote parts of the hospital [i.e. for computed tomography (CT) or cardiac magnetic resonance (CMR)]. For this reason, echocardiography, performed as a point-of-care imaging technique, is particularly important in acute cardiovascular care.

Transthoracic echocardiography

Transthoracic echocardiography (TTE) is generally the initial imaging modality in the assessment of acute cardiac conditions. It is widely available, most cardiologists are trained in TTE, and it is indicated in the majority of clinical scenarios associated with cardiac emergencies.[1] An optimal TTE study in the acute cardiac care setting may not be achievable, and echocardiography in this setting frequently requires non-standardized echocardiographic views. If the study is restricted to standard imaging planes only,[2] important findings, crucial for imaging and understanding altered pathology – and/or physiology – can be easily overlooked. Despite these challenges, echocardiographic examination in the acute cardiac care setting should be as comprehensive as possible, and undertaken with a fully equipped echocardiographic machine.[2,3] This approach must be clearly distinguished from point-of-care-focussed cardiac ultrasound and/or examination with pocket-sized imaging devices.

Table 1 Considerations that may influence echocardiographic findings and interpretation in critically ill patients

Positive pressure ventilation
• ntubation/ventilation
• Different ventilatory modalities
• Weaning
Filling status
Inotropic status
Metabolic status
Effects of sedation on myocardial function
O_2 and CO_2 levels
Mechanical circulatory support
Extracorporeal respiratory support
Differential effects on right and left heart
Ventricular–ventricular interaction in the context of respiratory support
Definition of normal range in the critical care setting
Exclusion of most patients from studies/randomized controlled trials

Transoesophageal echocardiography

Transoesophageal echocardiography (TOE) will usually follow a non-diagnostic transthoracic study. However, in the acute cardiac care setting TOE may be chosen as a first-line imaging technique when it is anticipated that TTE images will be non-diagnostic, and in certain clinical scenarios when optimal imaging of specific structures of the heart and/or great vessels is mandatory. Reliance on sub-optimal TTE images may lead to missing/misinterpretation of findings and subsequent misdiagnosis, with potentially detrimental consequences for the patient. Here, TOE is mandated, particularly in the case of acute aortic syndromes, acute valvular regurgitation, acute prosthetic valve dysfunction, chest trauma/aortic transection and atrial fibrillation/flutter for exclusion of thrombus.[1,4] Studies should not be undertaken in patients who are hypoxic and/or unable to protect their own airways without prior intubation and ventilation. In the acutely/critically ill population, care must be taken to exclude/correct significant coagulopathy prior to TOE probe insertion, if necessary intubating the oesophagus using direct laryngoscopy, and considering the use of paediatric probes to minimize potential trauma, particularly in those receiving mechanical cardiac and/or respiratory support. During the study, the airway and haemodynamics should be managed by a practitioner trained in intensive/acute cardiac care/anaesthesia, and independent from the TOE practitioner.

Contrast echocardiography

Contrast echocardiography with second generation contrast agents allows improved visualization of the endocardium,[5] which is useful in the assessment of left ventricular (LV) systolic function in patients with poor endocardial border delineation of LV pseudoaneurysms and intracardiac masses.[6,7] In the setting of acute cardiac failure, contrast injection can improve the Doppler signal envelope and be helpful for detecting severe aortic valve stenosis in patients with bad quality imaging. It may also be used to facilitate the diagnosis of aortic dissection.[8] Despite some earlier safety concerns regarding the use of contrast agents, recently published data revealed no increase in mortality in patients who underwent contrast-enhanced echocardiography examinations, including critically ill patients.[9–12] Agitated saline may be useful to differentiate between intracardiac and intrapulmonary shunting and may be used to confirm correct placement of the cannula/drain during echocardiographic-guided pericardiocentesis. Details on the clinical use of contrast echocardiography can be found in EACVI recommendations.[13]

Lung ultrasound examination

Lung ultrasound examination can be performed with any commercially available two-dimensional echocardiographic machine, including pocket-sized devices, and may be helpful in the differential diagnosis of acute dyspnoea, especially in diagnosing/excluding pneumothorax, pleural effusions and in the demonstration of interstitial oedema.[14,15]

Focused cardiac ultrasound

Focused cardiac ultrasound (FoCUS) protocols[16,17] have been proposed for the rapid detection of significant cardiac pathology and assessment of volume status and biventricular function, in particular in time-critical scenarios including cardiac arrest and trauma. A number of studies have shown that FoCUS may facilitate decision-making information in the acute setting.[16–18] Echocardiography is now recommended (where appropriately trained practitioners are available) in the management of cardiac arrest.[18] However, FoCUS should always be used and interpreted thoughtfully, since this fundamentally limited approach may lead to missing/misinterpretation of important findings unless the practitioner is aware of its (and their) limitations.[16,19]

Pocket-sized imaging devices

Pocket-sized imaging devices have been recommended as a tool for a fast initial screening in an emergency setting, as well as an extension of physical examination in the coronary and intensive care unit.[20] Technical characteristics and image quality of these new miniaturized echocardiographic systems are usually sufficient for the qualitative (but not quantitative) evaluation of ventricular and valvular function, pericardial and pleural effusion or extravascular lung water;[20] however, they have marked limitations of which the practitioner must be aware, and they must not be used to substitute for a comprehensive echocardiography study.

Level of competence

Performing echocardiography (TTE and TOE) and interpreting images in patients with acute/critical cardiac conditions requires a level of competence and training of the operator *at least* equivalent to the level necessary to perform elective studies.[21] The experienced echocardiographer will generally use the two techniques (TTE and TOE) interchangeably in order to obtain the information required. The operator must take into account the pathophysiological status of the patient, frequently with rapidly changing haemodynamic support, and synthesize all information to provide the relevant guidance to the attending physician. For performing TOE studies and advanced echocardiography techniques, operators should fulfil advanced echocardiography training requirements[21] and undergo specialized additional training in undertaking studies in the acute setting. Since echocardiographic examinations in patients with an acute cardiovascular condition are frequently requested as urgent/emergency, it is suggested that all such studies should be supervised by an expert cardiologist with an advanced level of competence in echocardiography[19,21] and experienced in performing and interpreting echocardiography in the acute/ critical care setting.

Principles, practice and specific considerations related to the use of echocardiography in emergency settings are outlined elsewhere.[19] Briefly, two levels of competence are recommended: the independent operator level and the expert operator level.[19] It is strongly recommended that all cardiologists who are involved in emergency/acute cardiac care on a daily or regular basis complete an additional training programme consisting in interpreting/reporting at least 150 echocardiographic examinations in critical or life-saving scenarios, in order to further improve technical skills and build experience.[19] An adequate case-mix is essential, and at least 50 of the additional cases should be personally performed, documented and all interpreted under close supervision. For non-cardiologists the requirements are essentially the same; additional theoretical learning on certain emergency cardiovascular diseases/ conditions is, however, highly recommended. Of note, it is strongly recommended that sonographers and fellows should not routinely perform echocardiography in the acute/critical care setting unsupervised.

Competence can be formally assessed through a certification process. Currently, individual certification for various echocardiographic modalities is offered by the EACVI.[22] Both individual competence and the competence of the team, facilities and appropriate logistics acknowledged by successful EACVI laboratory accreditation[23,24] are likely to guarantee high-standard service in all echocardiographic modalities and clinical settings, including echocardiography in acute cardiovascular care. In encompassing acute/critical care echocardiography, the certification process therefore supports the concept of echocardiography 'without walls', mirroring the patient-centric approach which is pivotal to acute/critical care medicine.

It is recognized that FoCUS may be helpful in selected cases, but it should be emphasized that EACVI in general strongly advocates systematic training in echocardiography and emergency echocardiography.[16,19] Specific training and certification is recommended for all users of FoCUS and pocket-sized imaging devices, with the exception of cardiologists who are certified for TTE according to national legislation.[20] This FoCUS certification should be limited to the clinical questions that can potentially be answered in such settings. The echocardiographic examination with the current generation of pocket-size imaging devices does not allow performance of, nor replacement of, a complete echocardiogram[20] and their limitations must therefore be recognized.

Clinical scenarios

A number of clinical scenarios present diagnostic challenges to the acute cardiac care cardiologist, with patient presentation potentially ranging from the pre-hospital setting through the emergency department, the cardiac catheterization laboratory and the cardiac intensive care unit (*Table 2*).

Cardiac arrest

The most extreme presentation of the critically ill cardiac patient is cardiac arrest. Throughout the echocardiography literature there is evidence that the technique can be used to diagnose/exclude some of the causes of cardiac arrest, not diagnosable using any other point-of-care technique (hypovolaemia, tamponade, pulmonary embolism, severe LV/RV dysfunction, MI and tension pneumothorax).[18,25]

Table 2 Echocardiographic signs indicative or suggestive of the cause of clinical admission in acute cardiovascular conditions

Systolic heart failure	Heart failure with preserved left ventricular ejection	Pulmonary embolism	Tamponade[e]
(1) LVEF<45–50%[a]	(1) LVEF ≥ 50%[a]	(1) Thrombus into right chambers	(1) Usually large pericardial effusion
(2) LVEDD >55 mm and/or >32 mm/m^2	(2) LVEDV<97 mL/m^2	(2) Abnormal septal motion	(2) Swinging heart
(3) LVESD >45 mm[a] and/or 25 mm/m^2	(3) LVESV<43 mL/m^{2a}	(3) Dilatation RA, RV (end-diastolic RV/LV diameter>0.6 or area>1.0)	(3) RA collapse (rarely LA)
(4) LVEDV>97 mL/m^2	(4) E–e′ ≥13[b]	(4) Global RV hypokinesia	(4) Diastolic collapse of the anterior RV-free wall (rarely LV)
(5) LVESV>43 mL/m^2	(5) Ar – A ≥ 30 ms	(5) McConnell sign hyperkinesia[d]	(5) IVC dilatation (no collapse with inspiration)
(6) Abnormal wall motion	(6) LA volume ≥ 34 mL/m^2	(6) Mild to severe TR	(6) TV flow increases and MV flow decreases during inspiration (reverse in expiration)
(7) Functional MR and/or TR	(7) Peak tricuspid velocity>3 m/s	(7) Pulmonary hypertension around 40–50 mmHg (>60 mmHg in the case of pre-existing pulmonary hypertension)	(7) Systolic and diastolic flows are reduced in systemic veins in expiration and reverse flow with atrial contraction is increased
(8) Peak tricuspid velocity >3 m/s	(8) Ultrasound lung comets[c] + signs and symptoms of heart failure		
(9) Aortic time velocity integral <15 cm[a]			
(10) Diastolic dysfunction (E/A ≥ 2 + DT<150 ms indicates increased LV filling pressures)[b]			
(11) Ultrasound lung comets[c]			

[a]May be profoundly affected by use of vasoactive agents.
[b]May be affected by the filling status of the presence and the use of vasoactive agents.
[c]Not specific for heart failure, merely indicates interstitial oedema.
[d]Specificity increasingly questioned.
[e]All echo features must be interpreted in the clinical context, and in light of the level of cardiorespiratory support. In patients who have undergone recent cardiac surgery these features may be absent. Features that vary with respiration are reversed with positive pressure ventilation.
LVEF: left ventricular ejection fraction; E: early mitral inflow velocity; e′: early diastolic mitral annular velocity; A: duration of the pulmonary flow reversal; Ar: duration of the A-wave; LA: left atrium; LV: left ventricle; RA: right atrium; RV: right ventricle; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; LVEDV: left ventricular end-diastolic volume; LVESV: left ventricular end-systolic volume; DT: deceleration time; IVC: inferior vena cava: TV: tricuspid valve: TR: tricuspid regurgitation.

Where appropriate training is undertaken, peri-resuscitation echocardiography does not impact upon high quality cardiopulmonary resuscitation (CPR), and may potentially improve diagnosis and alter management throughout the whole pathway of the acute cardiac care patient. Specific training in Advanced cardiac life support (A(C)LS) compliance is required even with experienced practitioners, in order to ensure images are obtained and recorded only during the pulse/rhythm check. International evidence-based guidelines support the use of echocardiography in an A(C)LS-compliant manner, by appropriately trained practitioners in order to diagnose/exclude potentially reversible causes of cardiac arrest, and guide immediate post-resuscitation management.[18]

Acute chest pain

Patients with acute chest pain represent a significant proportion (20–30%) of emergency department visits, have a high mortality and require rapid assessment, as treatment may be time-critical. Of the potential differential diagnoses, acute coronary syndromes (ACSs) are the most likely important underlying cause. ACSs are frequently characterized by the presence of chest pain, electrocardiogram (ECG) changes and a characteristic change in the cardiac enzyme/protein profile. However, it has been shown that these parameters alone may detect only approximately 30% of acute ischaemic events as a large majority of patients have atypical chest pain, a normal or inconclusive ECG and an early normal serum troponin level. Correct and early identification of ACS by traditional methods is therefore challenging in a significant number of patients. Here, echocardiography is a valuable bedside technique in the triage of patients with acute chest pain. Echocardiography can be very useful to identify acute myocardial ischaemia and other major causes of chest pain such as acute aortic dissection, pericardial effusion and pulmonary embolism and for evaluation of chest pain in patients with unresponsive/persistent haemodynamic instability despite intervention. Further, myocardial ischaemia is frequently under-recognized in the intensive care unit, where patients may be intubated and ventilated and receiving sedation/analgesia as a part of their routine management. Here, any haemodynamic instability in an at-risk patient should prompt recording of a 12-lead ECG, with echocardiography used to facilitate the diagnosis. Of note, the performance of echocardiography should never delay the initiation of treatment.

ACSs

Rest echocardiography

In acute ischaemic chest pain, the primary role of rest echocardiography is to assess the presence and extent of regional wall motion abnormalities, encountered in different types of myocardial injury (ischaemia, stunning, hibernation or necrosis). Echocardiography alone cannot distinguish between ischaemia and infarction; however, the absence of wall motion abnormalities, especially in patients with ongoing or prolonged chest pain (> 45 min), excludes major myocardial ischaemia. Of note, normal resting echocardiography cannot definitively rule out a transient episode of ischaemia, especially in patients with chest pain of short duration. In patients with

suspected ACS, deformation imaging of the LV (strain and strain rate) is a potentially useful technique to reveal subtle wall motion abnormalities (including post-systolic shortening) when standard visual assessment of wall motion fails to detect any abnormalities.[26,27] It is important to remember that segmental wall motion abnormalities are not synonymous with ischaemia, and can also occur in other conditions, such as myocarditis, right ventricular (RV) pressure/volume overload states, LV pre-excitation, Takotsubo cardiomyopathy, left bundle branch block or in the presence of a paced rhythm. During the hospital stay, echocardiography is used to assess LV function.

Contrast echocardiography

Myocardial contrast echocardiography is the only technique that allows immediate and simultaneous point-of-care assessment of LV wall motion and myocardial perfusion. Several studies have reported a high sensitivity of myocardial contrast echocardiography, as compared with standard echocardiography and gated single-photon emission CT, to detect an ACS in patients presenting to the emergency room with chest pain and a non-diagnostic ECG.[28] This technique also provides accurate additional prognostic information. Indeed, patients with normal myocardial perfusion and function at rest have an excellent outcome, while the presence of perfusion defects at rest identifies a subset of patients at high risk for ACS.[29] However, the choice of appropriate technical settings and correct interpretation of images is highly specialized and requires experience and technical expertise which is usually outside the practice of emergency department and intensive care physicians, as well as many cardiologists.

Stress echocardiography

Pre-discharge exercise testing is currently recommended in patients without recurrent chest pain, normal or non-diagnostic ECG findings and serial negative troponin measurements. Stress echocardiography is indicated in patients in whom exercise ECG testing is submaximal, not feasible or non-diagnostic. It is also preferred over exercise ECG when facilities are available. Both exercise and pharmacological stress echocardiography have been shown to be feasible and safe when performed in the acute setting. They provide short-term prognostic information comparable to SPECT in the triage of patients with chest pain, allowing safe early discharge,[30] with a negative predictive value of approximately 97%. Pharmacological stress echocardiography (dobutamine infusion with addition of atropine if necessary or high-dose dipyridamole and atropine) can be used in patients unsuitable for exercise testing. Dobutamine stress echocardiography is more cost-effective[31] than exercise ECG testing. Stress myocardial contrast echocardiography may also be used to determine prognosis in patients with significant cardiac risk factors presenting with chest pain, but a negative 12-h troponin and non-diagnostic ECG. In these patients, a negative stress myocardial contrast echocardiography predicts an excellent outcome.[32]

Myocarditis

Acute myocarditis is a potentially serious condition with a widely variable presentation and clinical course. To date, 2D echocardiography has played a limited role in the diagnosis of acute myocarditis because of a lack of specific diagnostic features and/or the apparently normal examinations encountered in its less severe forms.[33] Echocardiographic findings in patients with acute myocarditis are non-specific and may consist in: LV systolic and diastolic dysfunction, resting regional wall motion abnormalities, exercise-induced wall motion abnormalities (usually due to microvascular dysfunction) and unspecific changes in image texture.[34] The echocardiogram may also demonstrate intracardiac thrombi, secondary mitral and/or tricuspid regurgitation and co-existent pericardial involvement. Although the presence of myocardial interstitial oedema leads to a thickening of the ventricular wall in acute myocarditis, especially in more fulminant forms,[35] echocardiography is not able to accurately differentiate myocardial oedema from wall hypertrophy.

Speckle tracking imaging is a promising non-invasive method that might help to identify areas of intramyocardial inflammation in patients with acute myocarditis and no visible wall motion abnormalities/LV systolic dysfunction measured by standard parameters. A reduction in global systolic longitudinal strain and strain rate, as assessed by speckle tracking analysis, correlates with intramyocardial inflammation in endomyocardial biopsies of patients with acute myocarditis.[34] However, speckle tracking analysis is not able to differentiate inflammation-induced systolic longitudinal strain reduction from other causes that lead to alteration of LV longitudinal contraction, such as subendocardial ischaemia, infiltrative disease, toxin-related myocardial damage and others. Real-time, low-mechanical-index myocardial contrast echocardiography is now recommended for studying myocardial perfusion in various settings,[13] including myocarditis. Areas of necrosis and inflammation have been demonstrated to result in myocardial perfusion defects,[36] and the presence of perfusion defects that do not match a known coronary distribution territory should raise the clinical suspicion of myocarditis in the appropriate clinical setting.

Recommendations for echocardiography in patients with acute chest pain

Recommended:

(1) Evaluation of acute chest pain in patients with suspected myocardial ischaemia, non-diagnostic ECG and cardiac necrosis biomarkers, and when resting echocardiogram can be performed during the pain;
(2) Evaluation of acute chest pain in patients with underlying cardiac disease (valvular, pericardial or primary myocardial disease);
(3) Evaluation of patients with chest pain and haemodynamic instability unresponsive to simple therapeutic measures;
(4) Evaluation of chest pain in patients with suspected acute aortic syndromes, myocarditis, pericarditis or pulmonary embolism.

Not recommended:

(1) Evaluation of chest pain in patients for which a non-cardiac aetiology is apparent;
(2) Evaluation of ongoing chest pain in patients with a confirmed diagnosis of myocardial ischaemia/infarction.

Note: TOE may be indicated when TTE studies are non-diagnostic.

Stress-induced cardiomyopathy (Takotsubo syndrome)

Takotsubo cardiomyopathy was originally described 20 years ago in Japan, as a transient, stress-induced dysfunction of the LV apex.[37] This cardiomyopathy accounts for approximately 2% of all patients admitted with a potential diagnosis of ACS. Patients are typically female (>90%) and perimenopausal, but the condition can affect all patient groups.[38]

Takotsubo cardiomyopathy mimics an ACS, with patients presenting with chest pain and ECG changes, but with no angiographic evidence of ACS.[39] It is characterized by reversible LV dysfunction with regional wall motion abnormalities that do not fully correspond to typical coronary artery perfusion territories. The typical case of Takotsubo cardiomyopathy presents with LV apical akinesia (*Figure 1*), making echocardiography an ideal clinical diagnostic tool in many of these patients. However, coronary angiography is mandatory in suspected cases of Takotsubo cardiomyopathy to rule out obstructive coronary artery disease. Takotsubo has a more heterogeneous clinical presentation than initially considered, with akinesia demonstrated in the LV mid-cavity, LV base and RV, with or without sparing of the other LV segments. Biventricular involvement is described in about one-quarter of patients,[40] and involvement of the mid-ventricular segments has been recently reported in 40% of all cases.[41] LV function must completely recover to confirm the diagnosis of Takotsubo cardiomyopathy, with the recovery time ranging from several days to many weeks.[42]

Aortic dissection and other acute aortic syndromes

Dissection of the aorta is a life-threatening emergency condition for which early diagnosis and prompt management significantly impact upon outcomes.[43,44] Visualization of an intimal flap within the aorta separating the true and false lumens is considered diagnostic. The false lumen can be identified by systolic compression, spontaneous echo contrast, reversed systolic flow, delayed or absent flow, and thrombus formation (*Figure 2*). Specific criteria for identifying the true lumen include systolic expansion and diastolic collapse of the lumen, the absence or low intensity of spontaneous echo contrast, systolic jets directed away from the lumen, and systolic anterograde flow. Identification of the originating entry tear and involvement of the ascending aorta are essential to distinguish between type A and type B aortic dissections, as their management strategies are strikingly different.

A normal TTE examination cannot exclude aortic dissection; however, TTE can potentially demonstrate the intimal flap in the aortic root and arch and identify complications (acute aortic regurgitation, pericardial effusion or regional wall motion abnormalities suggestive of involvement of a coronary artery). Reverberation artefacts are a major pitfall with echocardiography, and the imager must be experienced in order to avoid misdiagnosis. TOE is a more sensitive diagnostic procedure;[45,46] however, focused/rapid transthoracic scanning is strongly advised before each TOE to screen for cardiac tamponade and LV wall motion abnormalities. Cardiac tamponade may be present in type A aortic dissection and in this case, when TTE demonstrates both the dissection and the pericardial collection,

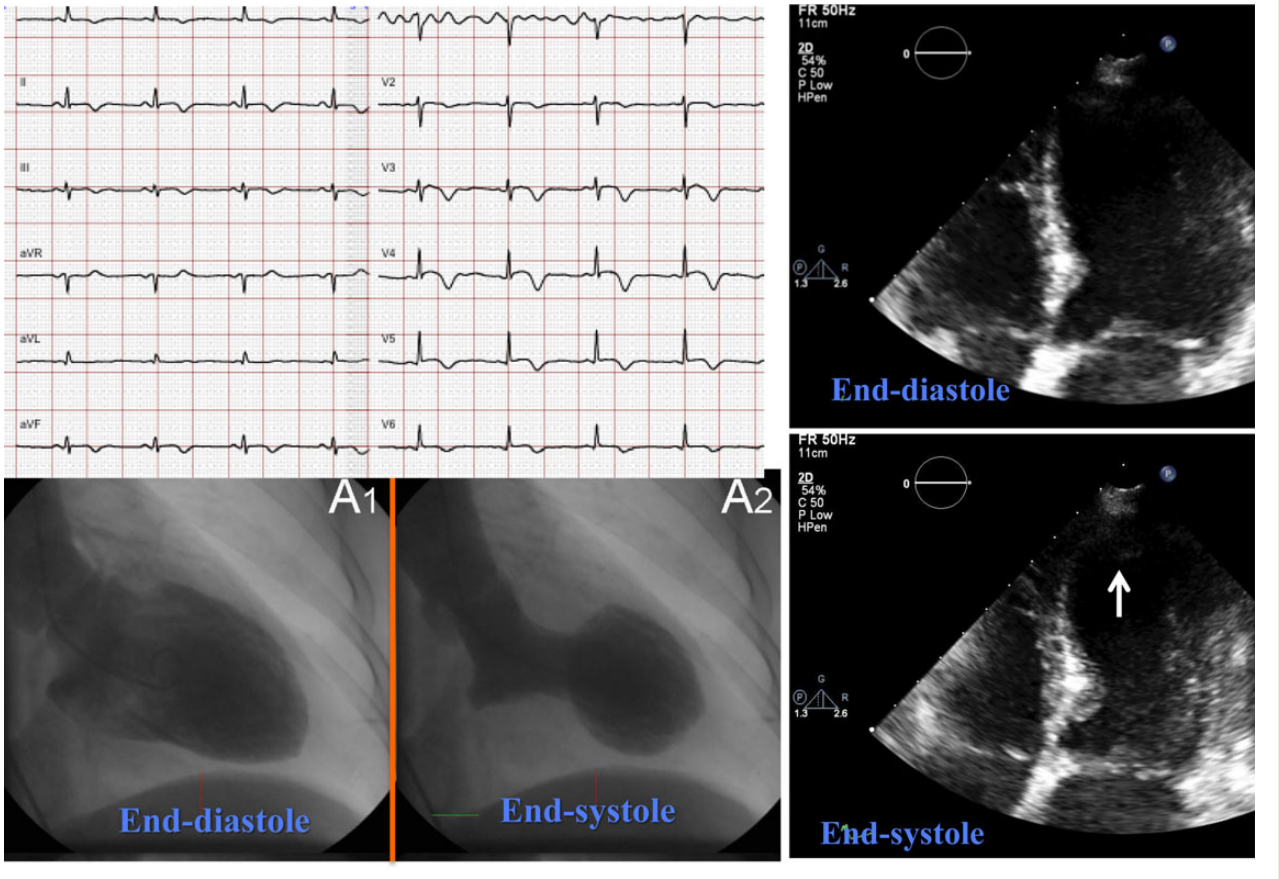

Figure 1: Electrocardiogram tracing, echocardiography and ventriculography in a patient presenting with acute chest pain, extensive apical wall motion abnormality (arrow) and mild elevation of troponin. Takotsubo cardiomyopathy was diagnosed after exclusion of significant coronary artery disease by angiography.

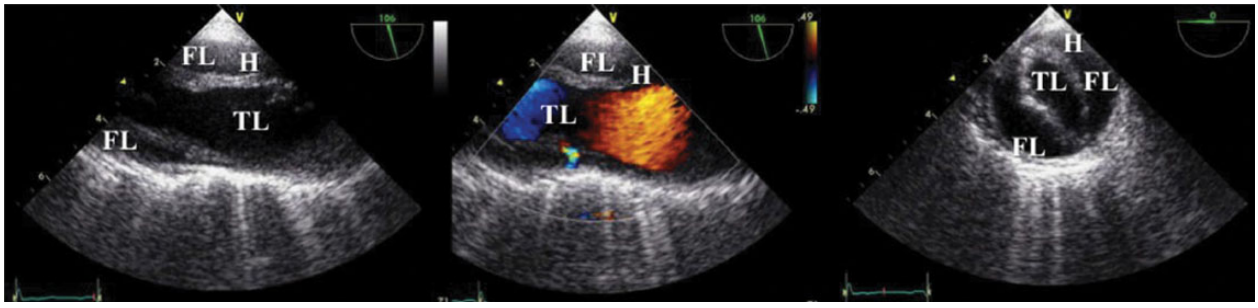

Figure 2: Transoesophageal echocardiographic examination obtained in a patient with type B acute aortic dissection. TL: true lumen; FL: false lumen; H: intense spontaneous contrast + thrombus.

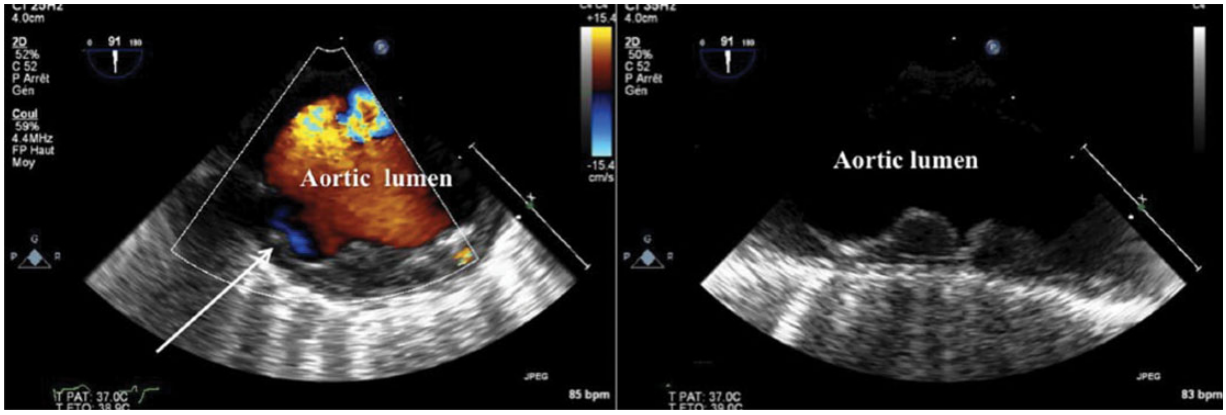

Figure 3: Severe atherosclerotic disease of the descending thoracic aorta in an elderly patient. Note the increased thickness of the aortic walls, protrusion of the atherosclerotic plaques into the aortic lumen and the anfractuosity of its contours. The white arrow indicates the presence of a penetrating ulcer of the aortic wall.

TOE is unnecessary and potentially dangerous, as it may provoke haemodynamic decompensation. Here, TOE can be performed in the operating room to document the extension of the aortic dissection.

As compared with CMR or CT, TOE cannot visualize the most distal parts of the ascending aorta near the proximal arch, nor the abdominal aorta. However, aortic dissection strictly limited to this segment of the ascending aorta is extremely unusual, as generally the intimal tear extends into the aortic arch and can be readily demonstrated with TOE. In patients presenting with acute dissection of the abdominal aorta, clinical symptoms (i.e. abdominal pain) will favour CT or CMR over TOE.

Other causes of acute aortic syndrome include intramural haematoma and penetrating atherosclerotic ulcers[47] (*Figure 3*). TTE remains of limited value and if echocardiography is the only modality of diagnosis available, TOE is the recommended approach, provided it is appropriate for the patient's clinical status. Aortic intramural haematoma is considered as a precursor of classic dissection (Class 2 Aortic Dissection), originating from ruptured vasa vasorum in media layers. It can progress to acute aortic dissection or regress in some patients. Echocardiographically, intramural haematoma is characterized by >5 mm crescentic or circumferential heterogeneous thickening of the aortic wall. Sometimes, an echo-free region may be observed, suggesting haemorrhage or liquefaction of the haematoma. If the diagnosis is questionable, other imaging modalities, such as CMR, may be necessary. A penetrating atherosclerotic ulcer (Class 3–4 Aortic Dissection) most frequently occurs in the descending aorta. In this situation, CT and CMR are the diagnostic modalities of choice. Blunt chest trauma is discussed in *Traumatic injuries of the heart and aorta*, below.

Pericarditis

Acute pericarditis is the most common disorder involving the pericardium. It may be the first manifestation of an underlying cardiac/extracardiac disease or an isolated disease involving the pericardium alone. In patients presenting with acute chest pain, pericarditis must be differentiated from an ACS. A small pericardial effusion is a frequent complication of AMI (especially in patients where reperfusion of the culprit coronary artery was not performed) and may also present during the subacute phase (Dressler's syndrome). The diagnosis primarily relies upon clinical history (chest pain changing with inspiration and position), examination (pericardial friction rub; audible, however, in only one-third of patients), ECG (diffuse concave upwards ST segment elevation and PR segment depression)

and TTE features. However, a normal study does not exclude the diagnosis, with pericardial effusion detectable in only 60% of cases.[48] Pericardial thickening (>3 mm) may be present, and although echocardiography is not accurate, TOE is superior to TTE (*Figure 4*). When elevated cardiac troponin is detected (up to 50% of patients presenting with acute pericarditis,[49] the term perimyocarditis is applicable. Here serum troponin elevation reflects myocardial involvement in the inflammatory process, and there may be associated regional wall motion abnormalities.

Chronic pericarditis related to longer-term inflammation with fibrosis and calcification can lead to pericardial constriction and may be a cause of severe dyspnoea. However, in this case dyspnoea is gradually progressive and is rarely the presenting complaint in the emergency department. All modalities of echocardiography are very helpful for the diagnosis of constrictive pericarditis and for differential diagnosis with restrictive cardiomyopathy.

Recommendations for echocardiography in patients with suspected pericardial disease

Recommended:

(1) Patients with suspected pericardial disease, including effusion, constriction or effusiveconstrictive process;
(2) Pericardial friction rubs developing in acute myocardial infarction accompanied by symptoms such as persistent pain, hypotension, and nausea;
(3) Patients with suspected bleeding in the pericardial space (i.e. trauma, perforation);
(4) Guidance and follow-up of pericardiocentesis.

Acute dyspnoea

Heart failure

Acute dyspnoea is a frequent condition in emergency rooms. Heart failure (HF) is the most common cardiac cause of acute dyspnoea,[50] which can be related to either new-onset HF or to worsening of pre-existing HF.[51] Patients may present with a spectrum of conditions ranging from acute pulmonary oedema, cardiogenic shock, isolated RV dysfunction or HF complicating an ACS. The work-up for acute dyspnoea begins with a complete and thorough history and physical examination. However, the definitive diagnosis of HF may be hampered by the lack of specificity or sensitivity of the signs and symptoms.[52] Furthermore, as HF is not a diagnosis *per se*, but rather a syndrome, additional investigations are required to determine the underlying cause. Echocardiography is an essential tool for the evaluation of functional and structural changes causing and/or associated with HF. TTE should be performed shortly following suspicion of HF in a patient presenting with dyspnoea.[18] Rapid diagnosis of the underlying cause, and distinction between HF due to systolic *vs.* isolated diastolic dysfunction, should be obtained since identification of these features determines immediate treatment in the acute setting.

Echocardiographic features of systolic HF (*Figure 5*) are listed in *Table 1*. It is important to remember that in critically ill patients treated with positive inotropic agents and/or mechanical circulatory support the 'normal' values quoted from non-critical care studies may not be valid. Here, every parameter must be interpreted in the clinical context, including the level of cardiorespiratory support. In patients with dyspnoea and bilateral pulmonary infiltrates on plain chest radiography, echocardiography can be used to distinguish between elevated and low left atrial pressure using a combination of techniques (*Figure 6*). In patients with an abnormal relaxation pattern (E/A <1), and peak E velocity <50 cm/s, LV filling pressures are usually normal. With restrictive filling (E/A ≥2, mitral E deceleration time <150 ms), mean LA pressure is often increased. The use of additional Doppler parameters is recommended in patients with E/A ratios ≥1 to <2 to distinguish those with increased LV filling pressures,[53] and in ventilated patients a combination of Doppler parameters (mitral inflow, Doppler myocardial imaging, pulmonary vein Doppler and colour Doppler M-mode flow propagation velocity) is recommended.[54]

The diagnosis of HF with normal ejection fraction (HFnEF), largely corresponding to diastolic HF, is more challenging. HFnEF accounts for more than 50% of all HF patients.[55] It refers to patients with

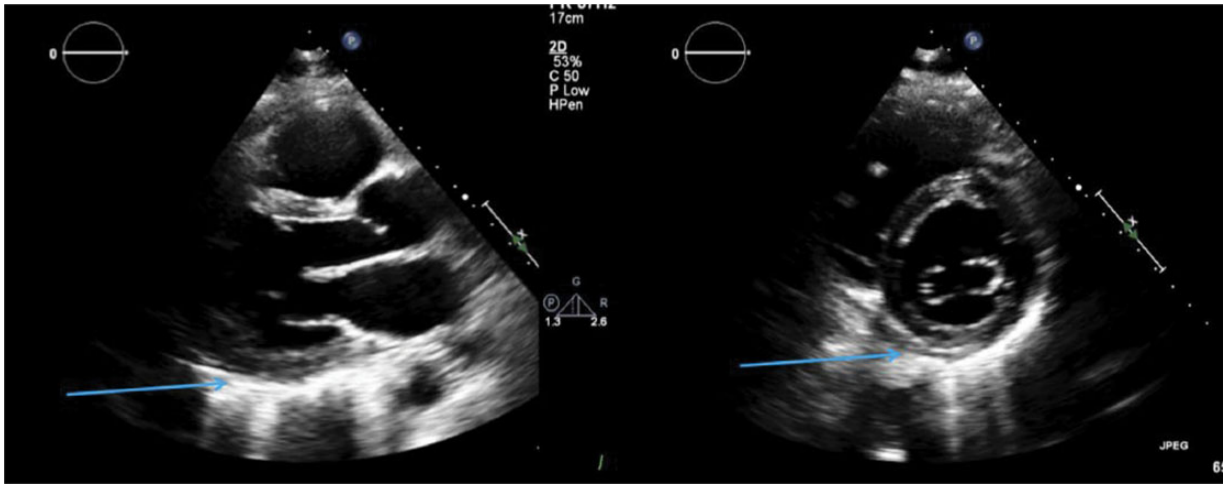

Figure 4: Echocardiographic examination of a patient admitted for acute pericarditis. Note the increased thickness of the pericardial layer close to the inferolateral and anterolateral wall of the left ventricle and absence of pericardial effusion in this patient with acute pericarditis (arrows).

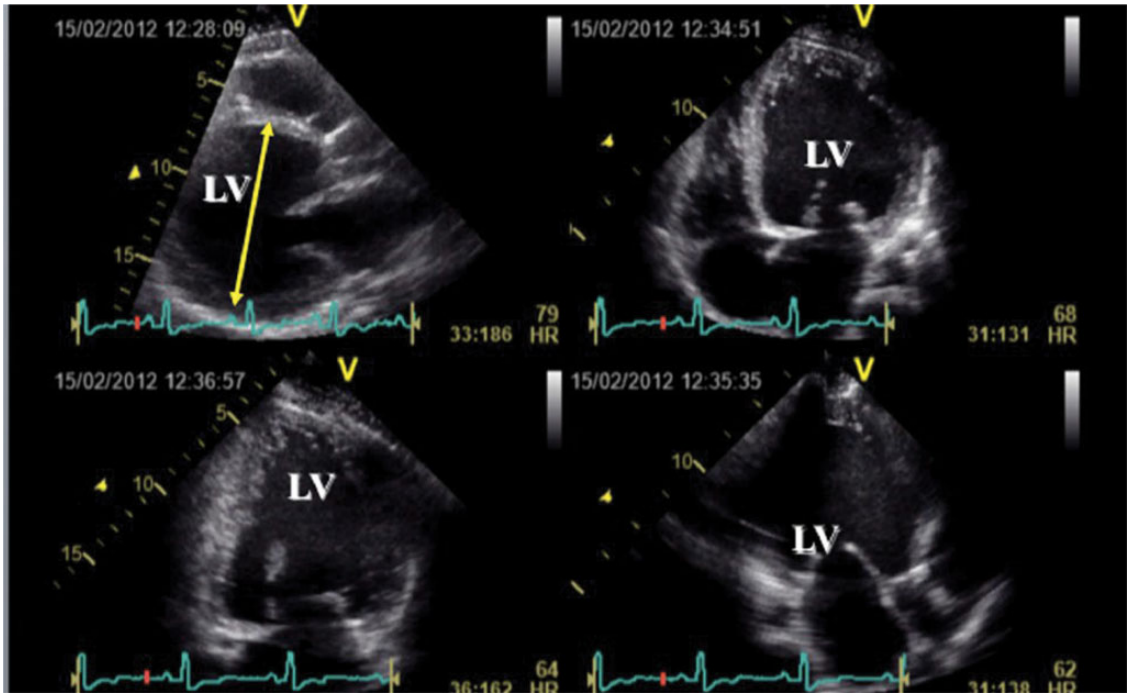

Figure 5: Echocardiographic examination showing dilated cardiomyopathy and depressed left ventricular (LV) systolic function in a patient admitted for acute dyspnoea. (see Supplementary data online, *Video 1*).

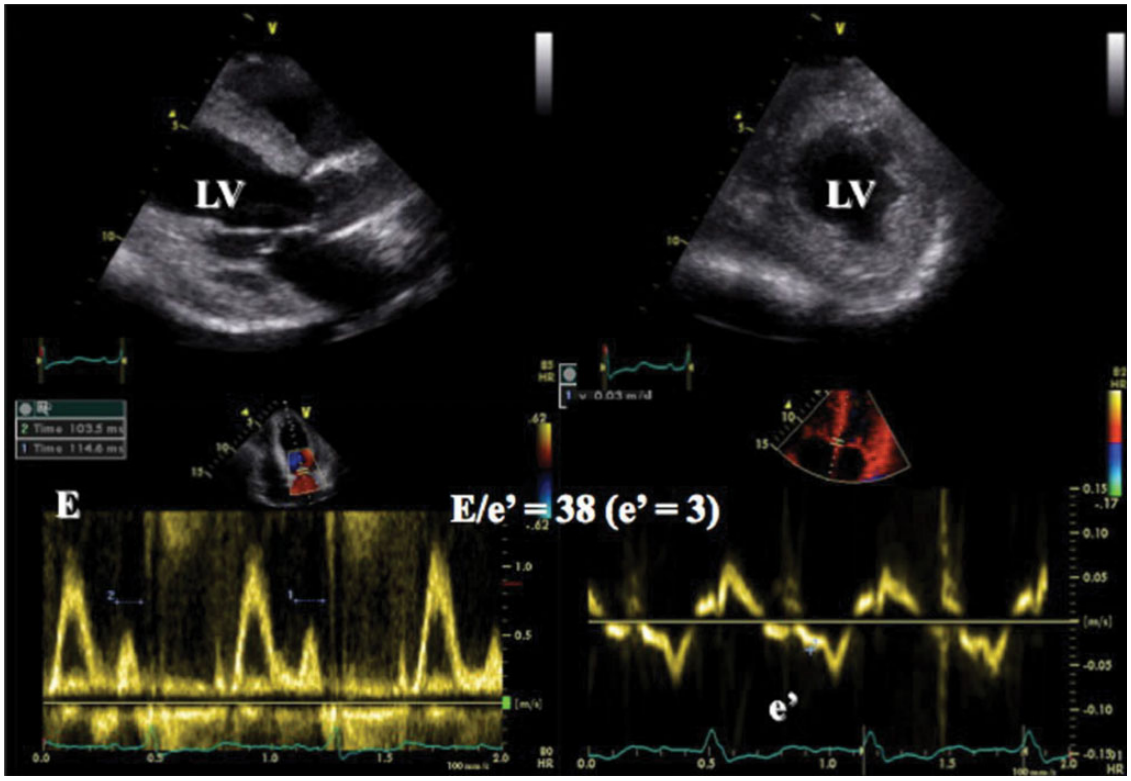

Figure 6: Echocardiographic examination showing preserved left ventricular (LV) systolic function in a patient admitted for acute dyspnoea. The diagnosis of hypertrophic cardiomyopathy was made. The spectral tissue Doppler-derived E–e′ ratio revealed an increased LV filling pressure confirming the diagnosis of diastolic heart failure. (see Supplementary data online, *Videos 2* and *3*). E: early mitral inflow velocity; e′: early diastolic mitral annular velocity.

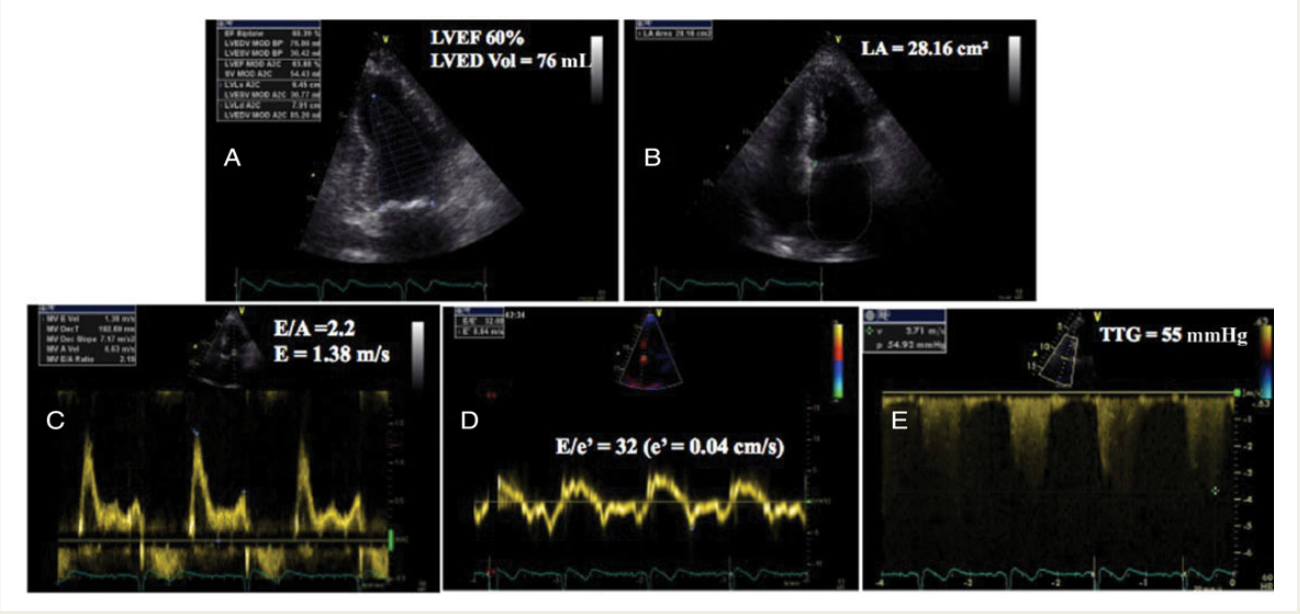

Figure 7: Diagnosis of heart failure with preserved left ventricular (LV) ejection fraction (EF) in a patient presenting with dyspnoea. Note the preserved LVEF [>50%, (A)], the left atrium (LA) dilatation (B), the restrictive pattern of the transmitral flow (C), the high E–e' ratio (D) and the increase in pulmonary systolic pressure (E). ED Vol: end-diastolic volume; E: early mitral inflow velocity; e': early diastolic mitral annular velocity; A: duration of the pulmonary flow reversal; TTG: transtricuspid pressure gradient.

HF and preserved LV ejection fraction (*Figure 7*) and requires the presence of signs and/or symptoms of HF and a number of echocardiographic parameters, listed in *Table 3*.[51] Conventional echocardiographic parameters derived from the mitral inflow pattern are classically poorly correlated with haemodynamics in patients with preserved LV function.[56,57] In the acute setting and before treatment, diastolic dysfunction in a patient presenting with dyspnoea implies almost exclusively an increase in LV filling pressures. Advanced echocardiographic evidence of increased LV filling pressure includes an increased ratio of peak E-velocity to early mitral annular velocity (e') using pulsed-wave tissue Doppler imaging (E–e' ≥13).[53] An outline of interpretation of LV diastolic function is represented in *Figure 8*. The presence of ≥ 2 abnormal measurements increases the diagnostic confidence. Of note, the ratio of peak E-velocity to colour M-mode flow propagation velocity is less accurate in this setting.

Advantages and limitations of the various echo Doppler parameters in assessing diastolic function have been detailed elsewhere.[53] Atrial fibrillation and sinus tachycardia are two frequently associated conditions in patients presenting with acute HF that make analysis of diastolic function more challenging. In general, when LV ejection fraction is depressed, mitral E deceleration time <150 ms has reasonable accuracy for the prediction of increased LV filling pressures.[58] In both reduced and preserved LV ejection fraction, a ratio of E–e' (lateral mitral annulus) >10–11 can still be used to predict high LV filling pressures when LA volume index is increased (≥ 34 mL/m^2) or Ar – A duration is ≥ 30 ms (A: duration of the pulmonary flow reversal; Ar: duration of the A-wave) or the delta E/A ratio with Valsalva manoeuvre is >0.5.

Table 3 Echocardiographic contraindications to extracorporeal support

Absolute contraindications to VA ECMO/LVAD	Absolute contraindications to VV ECMO
• Aortic dissection (unrepaired) • Severe aortic regurgitation • Coarctation of the aorta (unrepaired)	• Severe ventricular dysfunction • Cardiac arrest • Severe pulmonary hypertension
Relative contraindications to VA ECMO/LVAD	**Relative contraindications to VV ECMO**
• Severe aortic atheroma • Abdominal/thoracic aortic aneurysm with intraluminal thrombus	• Large PFO/ASD • Significant TV pathology (TS/TR)

PFO/ASD: patent foramen ovale/atrial septal defect; TS/TR: Tricuspid stenosis/tricuspid regurgitation; VA ECMO/LVAD: veno-arterial extracorporal membrane oxygenation/left ventricular assist device; VV ECMO: venous-venous extracorporeal membrane oxygenation

In both acute systolic and diastolic HF, interstitial oedema may be diagnosed at the bedside by the demonstration of an abnormally high number of bilateral sonographic B-lines (also called ultrasound lung comets). B-lines originate from water-thickened interlobular septa, may occur very rapidly in response to an increase in pulmonary venous pressure and can be detected with a cardiac ultrasound probe positioned over the chest[14,19] (*Figure 9*). A number of recognized protocols exist for lung ultrasound in the identification of interstitial

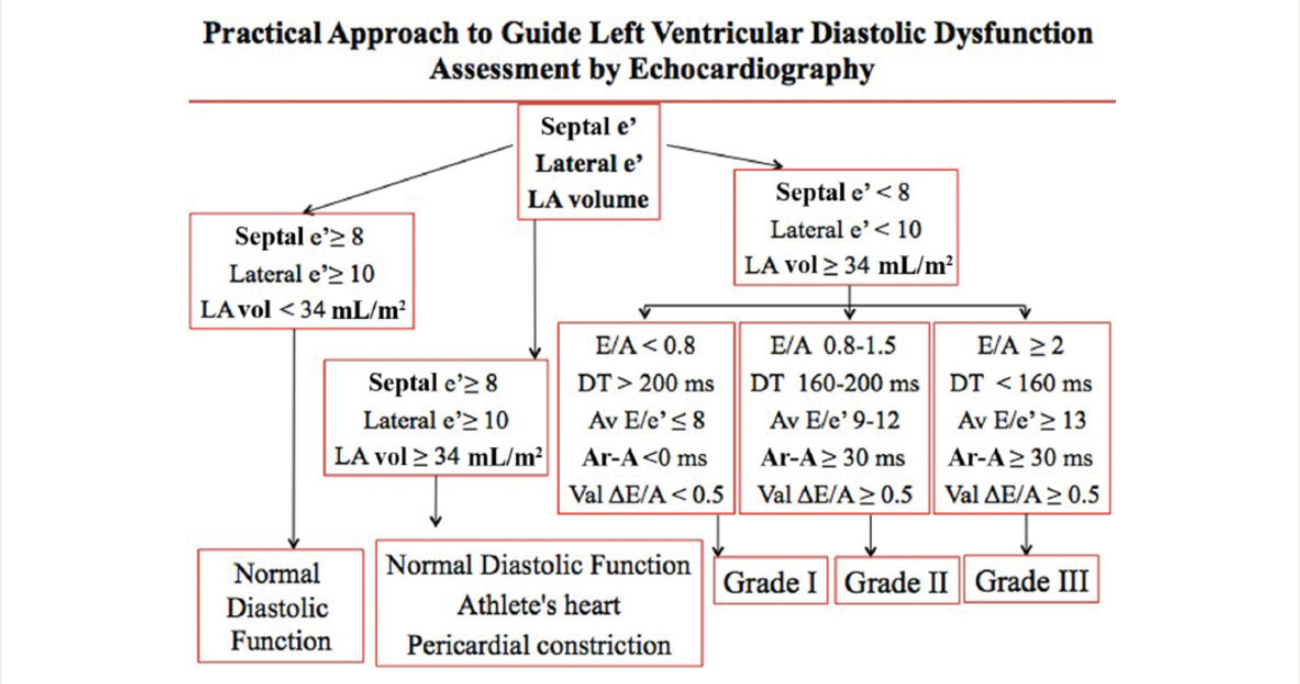

Figure 8: Practical approach to grade diastolic dysfunction by echocardiography. Adapted from the EACVI/ASE recommendations for the evaluation of left ventricular diastolic function by echocardiography.53 LA: left atrial; vol: volume; DT: E wave velocity deceleration time; Av: average, Val: Valsalva manoeuvre; E: early mitral inflow velocity; e′: early diastolic mitral annular velocity; A: duration of the pulmonary flow reversal; Ar: pulmonary venous atrial flow reversal.

oedema, and physicians working in the acute cardiac care environment should consider undertaking additional training in this field. Of note, lung ultrasound merely describes the presence of interstitial oedema, not its underlying cause.

Cardiomyopathies

The main use of echocardiography in acute cardiac care of patients affected by cardiomyopathies relates to the diagnosis and management of acute HF. All cardiomyopathies can lead to acute episodes of HF, either in the presence of a reduced ejection fraction or when the ejection fraction is still normal since the main determinant of cardiac symptoms and prognosis is represented by the increase in LV filling pressure.[59] Here, echo Doppler examination represents the key cardiac imaging modality because of its unique capacity to identify the presence of elevated filling pressures, and the mechanism(s) of acute deterioration.

Ejection fraction is not particularly helpful as a measure of ventricular function in the critically ill patient population since normal values under the conditions of ventilatory support are not known. Beyond ejection fraction, the application of two-dimensional speckle tracking echocardiography (STE) offers potentially useful information in acute HF patients with underlying cardiomyopathies,[60] in particular when ejection fraction appears preserved. While in patients with depressed systolic function all the strain components and LV twisting are severely reduced, in the presence of preserved LV systolic function (such as in early stages of restrictive cardiomyopathy (RCM) and hypertrophic cardiomyopathy (HCM)), radial strain is relatively reduced and longitudinal strain is markedly depressed, similar to that of patients with reduced ejection fraction, but circumferential strain is maintained and LV twisting appears to be normal or supra-normal, acting as a balancing mechanism to maintain ejection fraction in the normal range.[61] Global longitudinal strain (GLS) of subendocardial fibres should be therefore assessed in all patients with HF, especially when LV ejection fraction is preserved. Values $< -16\%$ indicate mild depression of GLS and values $< -10\%$ are consistent with a severe reduction of GLS. Of note, these values still require to be validated in the acute settings and are not applicable to patients with systolic HF being treated with inotropic agents or mechanical circulatory support. The evaluation of GLS is highly feasible and reproducible in this clinical setting while circumferential and radial strain, as well as LV twisting, are less reproducible.[60] In certain cardiomyopathies a reduction of regional longitudinal strain is encountered. Thus, infiltrative cardiomyopathies, such as cardiac amyloidosis or Loeffler's cardiomyopathy (eosinophilic infiltration), reveal a prominent reduction of regional longitudinal strain of the basal LV segments (*Figure 10*) while the reduction of GLS appears to be more generalized in HCM (*Figure 11*).

In patients with HCM, a comprehensive approach to assessing LV filling pressure is recommended, with consideration of all echocardiographic data (i.e. pulmonary arterial pressures, mitral inflow pattern, E–e′, etc.) according to the individual clinical context. In the acutely unwell HCM patient, LV outflow tract obstruction should be always excluded. Here continuous wave (CW) Doppler assessment of the LV outflow tract is used to determine the peak velocity at the site of obstruction, with an excellent correlation of pressure differences as measured by the CW Doppler method and

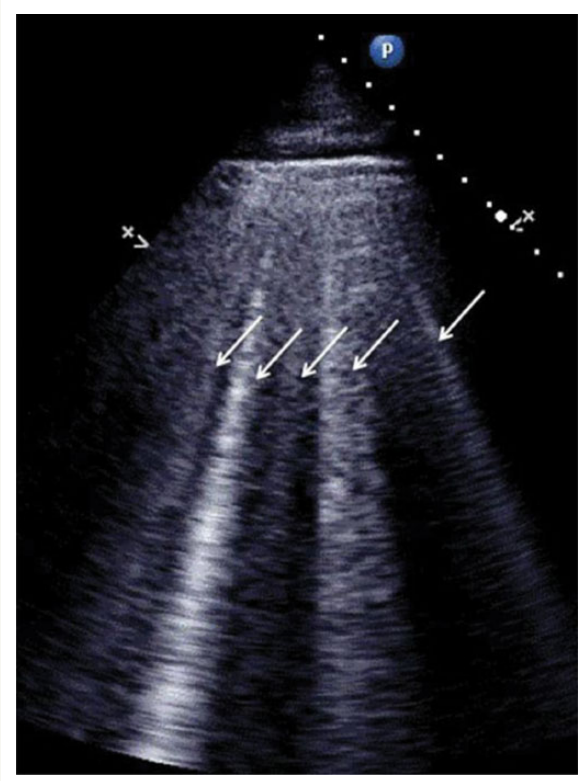

Figure 9: Transthoracic lung ultrasound reveals multiple sonographic B-lines (ultrasound lung comets, white arrows) in a patient with acute pulmonary oedema.

by cardiac catheterization. Colour flow mapping can be used to characterize the level of obstruction, either in the LV outflow tract (LVOT) or in the LV midcavity. In patients with significant LV hypertrophy and potential for LVOT obstruction, this may be exacerbated in the presence of positive inotropic agents or hypovolaemia. This may be particularly important in the context of concomitant right HF, where resultant underfilling of the LV exacerbates the risk of dynamic LVOT obstruction. Here echocardiography is essential for diagnosis and monitoring the response to interventions.

The echocardiographic detection of intracardiac thrombi in patients affected by idiopathic cardiomyopathy (IDCM) and LV non-compaction is common when they present acutely with an ischaemic stroke. Spontaneous echo contrast ('smoke') is considered a pre-thrombotic condition, associated with an increased risk for thromboembolic events.[62] As LV thrombi develop predominantly apically or in akinetic regions, TTE has superior diagnostic accuracy (sensitivity = 90%, specificity = 85%)[62] compared with TOE. The accuracy of TTE is further increased by using colour Doppler and/or intravenous contrast agents.[13] Several echocardiographic features must be evaluated in patients with LV thrombi (*Figure 12*) including shape (thrombus may be mural or protruding within the cavity), motion (thrombus may be fixed or present an independent motion to a variable extent) and also the presence of any adjacent LV aneurysm—a localized area of akinesia or dyskinesia that deforms the LV chamber during both systole and diastole.[63] A higher risk of embolization is found in patients with larger thrombus size and/or thrombi which are mobile and protrude into the LV cavity, particularly in older patients.

In cardiomyopathy patients with atrial flutter and/or atrial fibrillation, atrial thrombi involve most frequently the LA cavity and LA appendage (LAA). TOE is the 'gold standard' for diagnosis of LA and LAA thrombi, with high sensitivity and specificity. By using TOE, LAA thrombi appear as echogenic masses, distinct from the underlying endocardium, observed in more than one imaging plane. They should be distinguished from pectinate muscles by using multiple planes of imaging. Here biplane imaging may be of use, particularly when evaluating an anatomically complex LAA. Patients with mechanical circulatory support should be evaluated for intracardiac thrombi, particularly related to cannulae, and also valves (including prosthetic valve) when the heart is not ejecting. This is highly specialized echocardiography and should only be undertaken by experts in the field.

Key points regarding the emergency echocardiographic evaluation in patients with suspected cardiomyopathies

- Calculate 2D LV ejection fraction and additional signs of LV systolic dysfunction (sphericity index, pulsed tissue Doppler derived s' velocity of mitral annulus, indexed stroke volume).
- Take into account LV geometry and possible regional differences of myocardial wall thickness.
- Estimate LV filling pressure (E–e′ ratio, AR – A duration difference, LA volume index, pulmonary arterial systolic pressure).
- Take into account reduction of GLS, even in the presence of normal ejection fraction.
- Actively diagnose/exclude LVOT obstruction in patients with HCM/LV hypertrophy.
- Take into account the level and degree of cardiorespiratory support.

Pulmonary embolism

The diagnosis of acute pulmonary embolism is challenging in the emergency room since both symptoms (dyspnoea and/or chest pain) and clinical signs are not specific.[64] If available, TTE can help to establish a prompt diagnosis and to identify patients with high-risk features. Overall, the sensitivity of TTE for the diagnosis of pulmonary embolism is about 50–60% while the specificity is around 80–90%. In some situations, that is, critically ill patients, TOE may improve the sensitivity.[65] Of note, TTE is normal in about 50% of unselected patients with acute pulmonary embolism, but it can provide direct and/or indirect evidence for the diagnosis. The visualization of a large, mobile, serpentine thrombus trapped in the right heart chambers or pulmonary artery is rare, but makes the diagnosis evident.[66] In general, although other diagnostic tests (CT, D-dimer, V/Q scanning) are used to confirm the diagnosis, echocardiography is valuable as a complementary imaging technique. Where the patient is catastrophically haemodynamically unstable, TTE may be the only immediately available and appropriate imaging investigation.[67]

The main indirect findings for pulmonary embolism are the consequences of acutely increased pulmonary artery/ right heart pressures. Although non-specific, they include: dilatation of right heart chambers (i.e. abnormal ratio of RV diameter or area to LV diameter or area and of the inferior vena cava), RV hypokinesia, abnormal motion of the interventricular septum. In a patient with a relevant history and clinical findings, a ratio between end-diastolic RV to LV

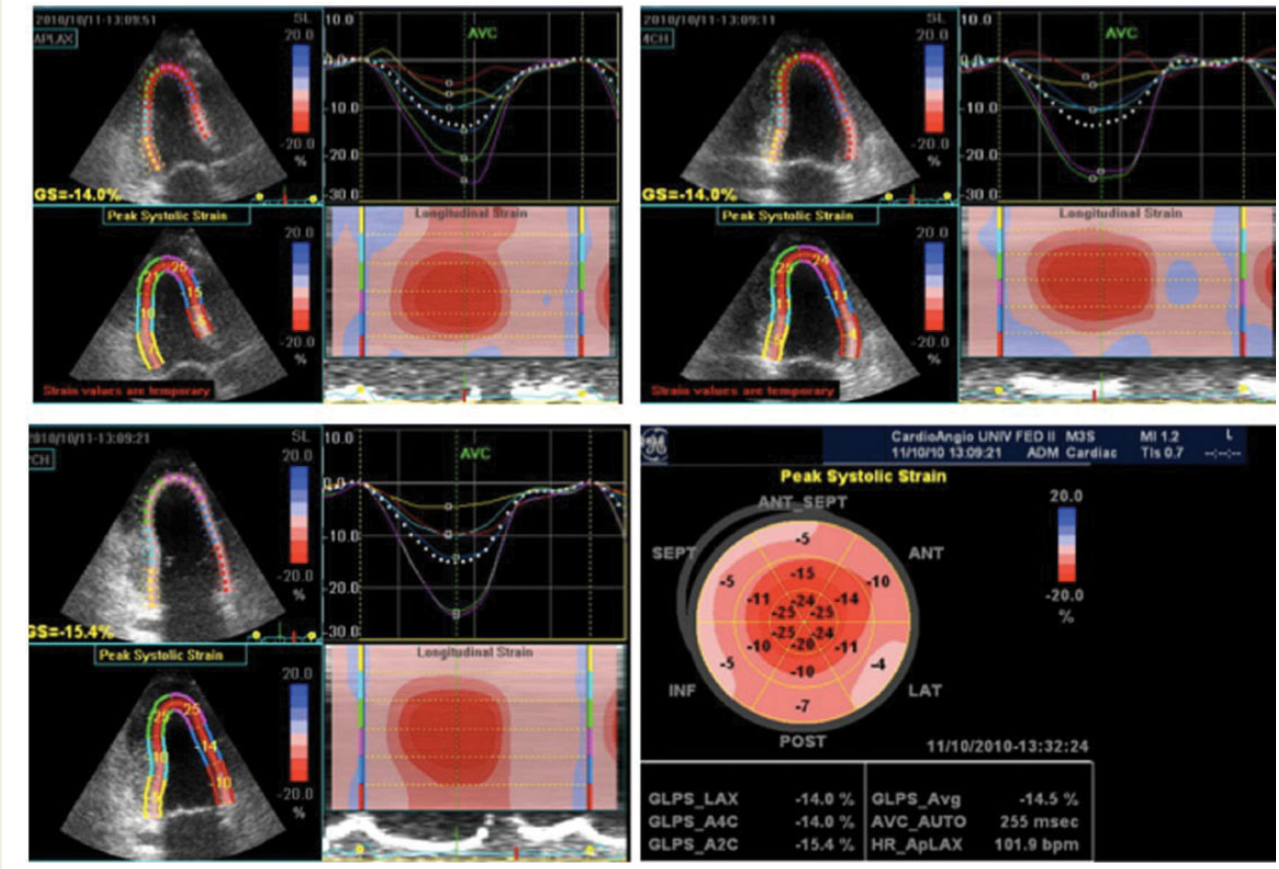

Figure 10: Sample of regional longitudinal strain (APLAX: apical long-axis, 4CH: four-chamber view, 2CH: two-chamber view) a bulls-eye representation in a patient with cardiac amyloidosis. In the presence of a normal ejection fraction (56%), the reduction of regional longitudinal strain involves predominantly the basal segments of the left ventricle. In the left section of each view colour representation of quantitation of peak regional strain values (normally negative) referring to six myocardial regions is depicted. In the right upper section of each view regional colour systolic curves of systolic strain are marked while the dotted white line corresponds to average strain (GLPS). In the right lower section qualitative colour M-mode strain representation refers to the six consecutive myocardial segments: at the bottom left, ventricle (LV) basal right segments (red colour); at the central part, LV apex; at the top, LV basal segments (yellow colour); red and pink colour refers to systolic deformation.

diameter >0.6, and a ratio of end-diastolic RV to LV area >1.0 are consistent with massive pulmonary embolism[68] (*Figure 13*). In pulmonary embolism, RV hypokinesia is not necessarily global but can be limited to the mid-RV free wall while contraction of the RV apex may be normal or hyperdynamic (McConnell sign).[69] Although previously thought to be specific for the diagnosis of pulmonary embolism, this is now questioned, since it can be seen in other conditions. Where pulmonary embolism is diagnosed, echocardiography can be used to differentiate those patients not at high risk into intermediate risk (evidence of RV dysfunction) *vs.* low risk (no RV dysfunction). In patients with suspected high-risk pulmonary embolism presenting with shock or hypotension, the absence of echocardiographic signs of RV pressure overload or/and dysfunction virtually excludes massive pulmonary embolism as a cause of haemodynamic instability.

Secondary tricuspid regurgitation is frequent in patients with intermediate-to-high-risk pulmonary embolism. It allows the estimation of RV systolic pressure and thus of pulmonary arterial systolic pressure (PAsP) in the absence of pulmonary valve stenosis. PAsP can be estimated from the peak velocity of the tricuspid regurgitant jet (V) according to the simplified Bernoulli equation, but may underestimate when tricuspid regurgitation is very severe. Right atrial pressure is estimated by clinical examination of the jugular veins, by the diameter of the inferior vena cava and its respiratory changes, or potentially by direct measurement from central venous catheterization in the critically ill. As the RV is only able to generate a PAsP of up to 60 mmHg acutely, in the acute setting the tricuspid regurgitant jet velocities are expected to be no higher than 2.5–3.5 m/s, corresponding to a PAsP of about 40–50 mmHg in acute pulmonary embolism. Conversely, a PAsP >60 mmHg may suggest a more chronic process, relating to repeated episodes of pulmonary embolism (*Figure 13*) or a chronic pulmonary parenchymal disease and/or super-added pulmonary embolism. Other modalities such as Doppler myocardial imaging and strain may add significant information relating to RV function evaluation; however, these techniques remain experimental, in particular in the acute setting.

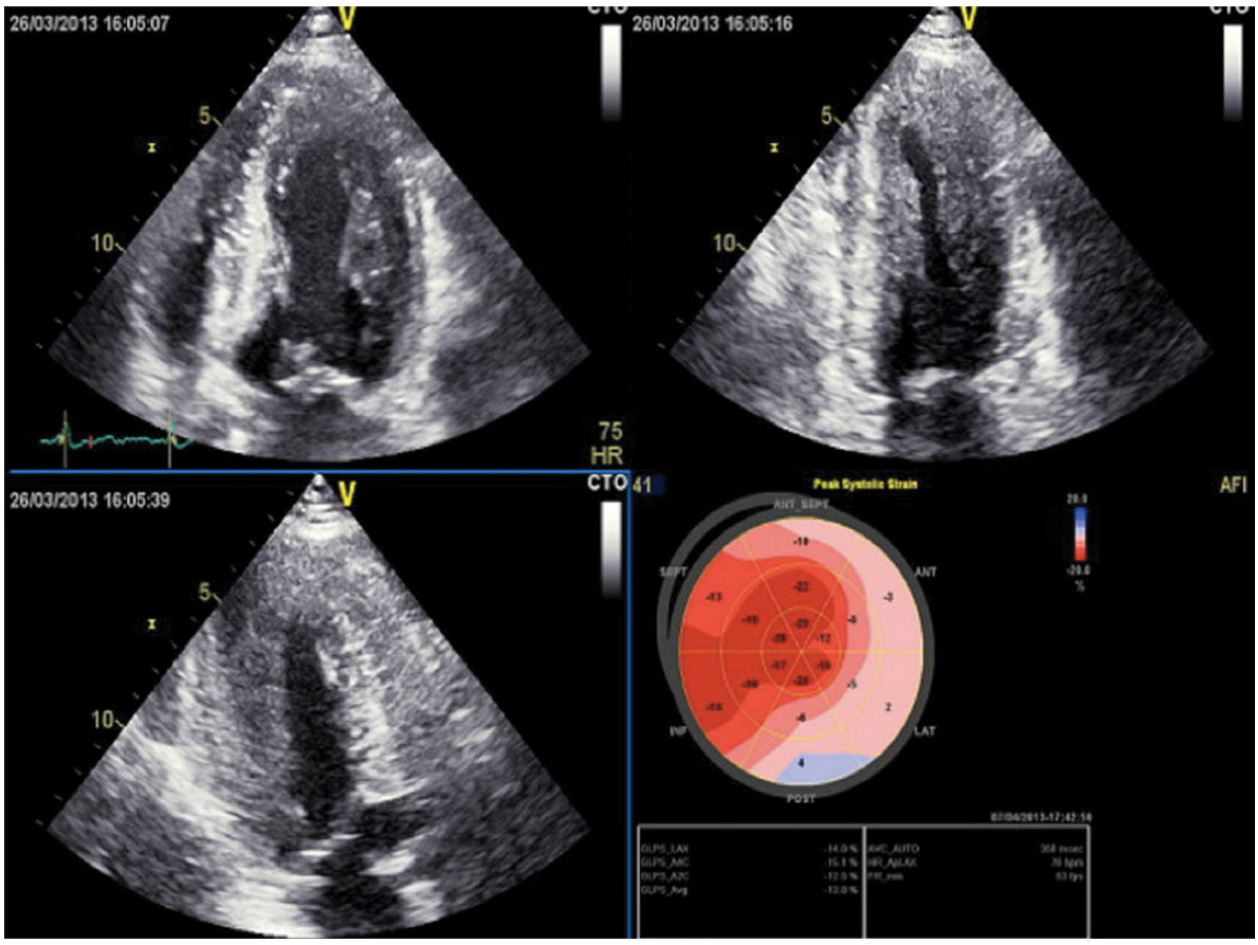

Figure 11: Regional longitudinal strain (bulls-eye representation) in a patient with hypertrophic cardiomyopathy. In the presence of a normal left ventricular ejection fraction (57%), the reduction of regional longitudinal strain involves left ventricle walls more homogeneously than in a patient with cardiac amyloidosis.

Recommendations for echocardiography in patients with suspected/confirmed pulmonary embolism

Recommended:

(1) Suspected high risk of pulmonary embolism where shock or hypotension are present and CT is not immediately available (#);
(2) For distinguishing cardiac *vs.* non-cardiac aetiology of dyspnoea in patients in whom all clinical and laboratory clues are ambiguous;
(3) For guiding the therapeutic option in patients with pulmonary embolism at intermediate risk.

Reasonable:

(1) Search for pulmonary emboli and suspected clots in the right atrium or ventricle or main pulmonary artery branches;
(2) For risk-stratification in non-high risk pulmonary embolism.

Not recommended:

(1) For elective diagnostic strategy in haemodynamically stable, normotensive patients with suspected pulmonary embolism.

#TOE may be indicated when TTE studies are nondiagnostic, catastrophic decompensation may occur with sedation.

Pneumothorax

Pneumothorax (PTX) is a potentially lifethreatening condition in patients admitted to the emergency department for acute dyspnoea with or without chest pain, in patients following central line insertion, and/or in patients with lung injury undergoing positive pressure ventilation. Over the last decade, the use of ultrasound as a technique to evaluate PTX has rapidly evolved.[14,70] In a normal lung, the two pleural layers are closely opposed, and ultrasound shows the movement of the parietal over the visceral pleura synchronized with respiration (lung/pleural sliding). When air separates the two layers the parietal pleura is still visualized, but lung sliding is not seen. Absence of lung sliding is required for the sonographic diagnosis of PTX, but its absence does not necessarily confirm PTX, since several other conditions (massive atelectasis, main bronchus intubation, pleural adhesions) may also result in absence of lung sliding. Additional sonographic signs of PTX, which increase sensitivity of ultrasound and are required for diagnosis, include the following: (i) absence of B-lines, (ii) absence of lung pulse, and (iii) presence of lung point. Opposition of the parietal and visceral pleura is necessary to visualize B-lines, therefore the presence of even one isolated B-line

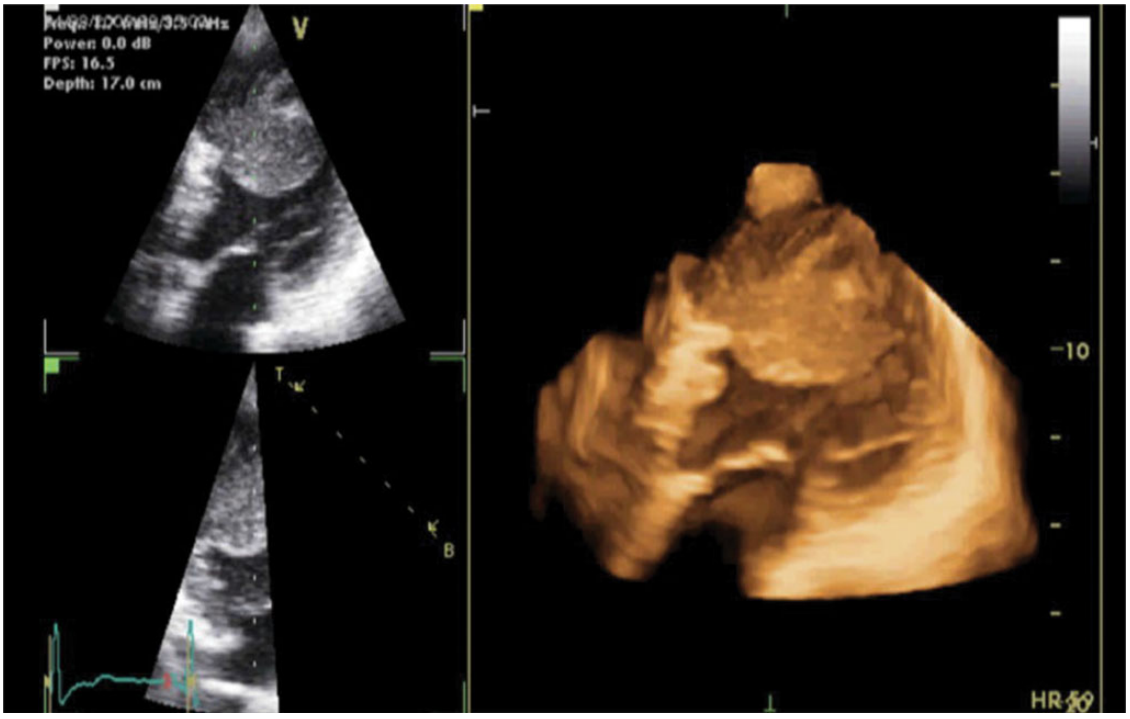

Figure 12: 3D transthoracic echocardiography showing a thrombus located in the left ventricular apex in a patient with recent ST segment elevation myocardial infarction of the anterior wall. (see Supplementary data online, *Video 4*).

excludes PTX in the area scanned.[71] The *lung pulse* refers to the rhythmic movement of visceral upon parietal pleura and underlying lung tissue, synchronous with the cardiac rhythm. PTX is characterized by the absence of both lung sliding and the lung pulse as the intrapleural air does not allow transmission of any movements to the parietal pleura. Thus, visualization of lung pulse excludes PTX in the area scanned.[14] The lung point is the point on the chest wall where the normal pleural interface contacts the edge of the PTX. Using B-mode ultrasound, the lung point will appear as the boundary between normal lung sliding and PTX pattern (absence of lung sliding and of B-lines). The lung point represents the physical limit of PTX as mapped on the chest wall and is the most specific sonographic sign of PTX.[72] In the emergency setting, the combination of lung sliding and lung pulse coupled with the presence of B-lines allows prompt and safe exclusion of a PTX (in the area scanned) without the need for searching the lung point. If tension pneumothorax is suspected in the haemodynamically unstable patient, it should be treated clinically, rather than relying on ultrasound features, in particular where the sonographer is not an expert in the field.

Heart–lung interactions and ventilation

Most of the patients who are ventilated have problems in weaning from mechanical ventilation, depending upon the type of intensive care unit concerned. Although alveolar oedema is the most frequently cited cardiac cause of failure to wean, additional cardiac-related factors also contribute. Determining the cardiac contribution can be challenging and echocardiography can be pivotal to weaning success. However, this demands understanding the effects of normal and mechanical ventilation upon the pressure/volume relations of the heart, and the physiological changes when transitioning from mechanical to spontaneous ventilation.

Cardiac output is predominantly determined by the pressure difference between mean systemic pressure and right atrial pressure, and so during normal inspiration, as this pressure difference increases, venous return correspondingly increases. The increased volume in the right heart results in correspondingly reduced flows in the left heart due to ventricular–ventricular interaction. During positive pressure ventilation, these changes are reversed, and application of constant positive pressure [i.e. positive end-expiratory pressure (PEEP)] results additionally in a fall in venous return throughout the cardiac cycle. Additionally, the increase in transpulmonary pressure increases RV afterload, resulting in reduced right heart ejection. The corresponding increase in intrathoracic pressure decreases LV afterload and increases LV preload, resulting in an increase in left heart ejection. These cyclical changes are readily demonstrated using Doppler echocardiography, and form the basis of diagnosing echo features of tamponade, excessive ventilatory pressures and predicting volume responsiveness.

Evaluation of failure to wean from mechanical ventilation therefore involves first demonstrating/excluding the presence of significantly elevated LA pressure (either at rest or on weaning), and then demonstrating/excluding the underlying cardiac causes of weaning failure that are potentially reversible. Estimation of LA pressure in this patient population remains, however, complex, and few techniques

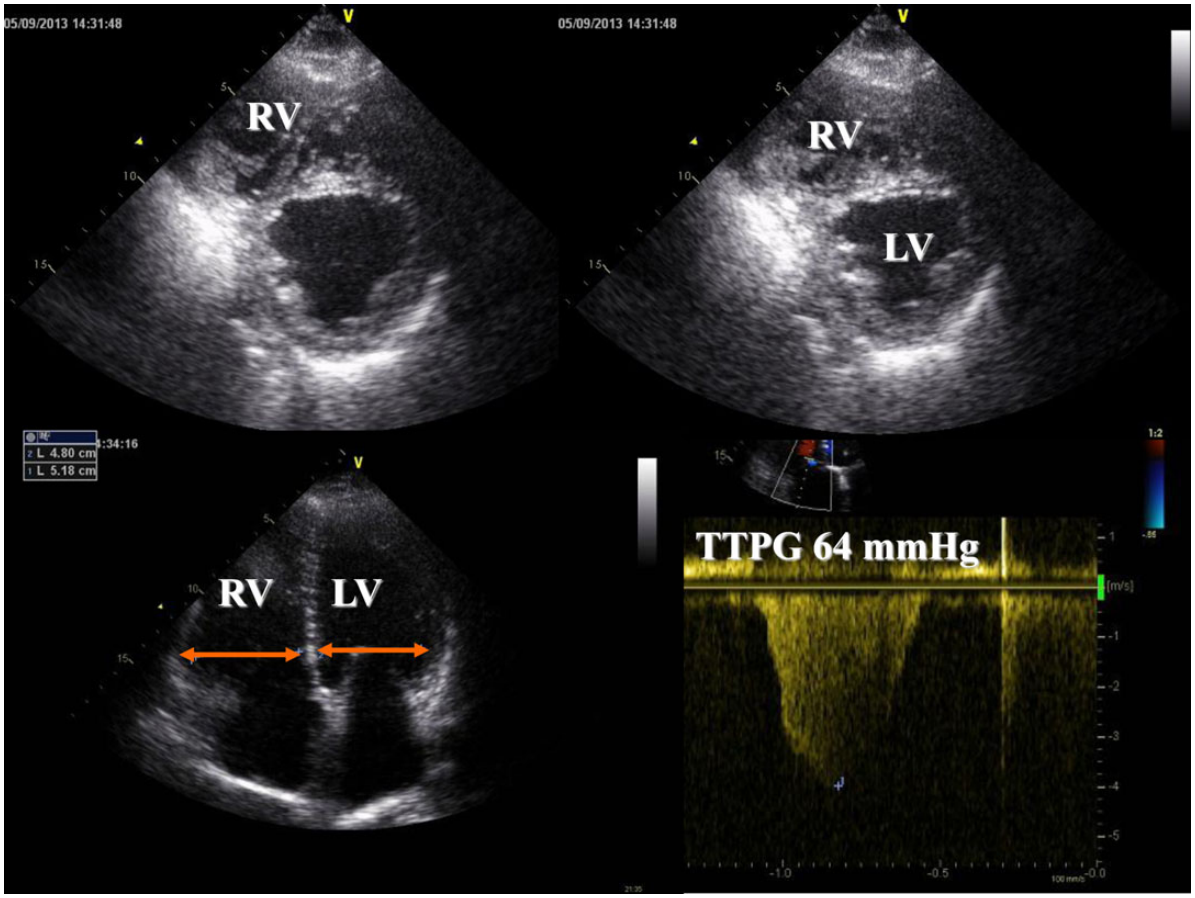

Figure 13: Echocardiographic examination of a patient admitted for recurrent episodes of pulmonary embolism. The right ventricle (RV) was enlarged as compared with the left ventricle (LV) and the ratio between end-diastolic RV to LV diameter >0.6. The transtricuspid pressure gradient (TTPG) was markedly increased, indicating chronic severe pulmonary hypertension. Tricuspid annular plane systolic excursion was found to be lower (8 mm *vs.* 11 mm) than the one measured on the echocardiogram one month previous to this admission. On the basis of the decrease in RV systolic function, recurrent pulmonary embolism was suspected. (see Supplementary data online, *Videos 5* and *6*).

have been fully evaluated in this context. Factors to be considered have been described in *Cardiomyopathies*, above. In all cases, the confounding factors of critical illness (heart rate, cardiac output, LV compliance, volume status and ventilation) have not yet been fully evaluated in this context.[73]

Physiological stress echocardiography (targeted echocardiography during a weaning trial) may be limited by tachycardia, tachypnoea and patient agitation. Here, other modalities of stress may be required. Pharmacological stress echocardiography has been used in the critically ill, and can be applied even in patients on positive inotropic support (*ACCs*, above). Where dynamic mitral regurgitation is suspected (Cardiogenic shock complicating AMI, below), volume and pressure loading may be used to reveal the nature, severity and dynamic nature of regurgitation. Other causes of weaning failure should be actively sought including progressive LV and/or RV dysfunction, excessive tachycardia/bradycardia for the patients' pathology (*Cardiac arrhythmias* section, below), or development of outflow tract obstruction (*LVOT obstruction*, below). Additional features that should be sought include exclusion of dynamic intra-cardiac shunting with resultant disproportionate hypoxia, and exclusion of the presence of intrapulmonary shunting.

Recommendations for echocardiography in patients with acute dyspnoea

Recommended:

(1) For distinguishing cardiac *vs.* non-cardiac aetiology of dyspnoea in patients in whom clinical and laboratory clues are ambiguous/ non conclusive;
(2) Assessment of LV size and function in patients with suspected clinical diagnosis of HF;
(3) To assist in determining the cause of failure to wean from mechanical ventilation.

Not recommended:

(1) Evaluation of dyspnoea in patients for which a non-cardiac aetiology is apparent.

Note: TOE is indicated when TTE studies are not diagnostic.

Haemodynamic instability and shock

When dealing with patients with hypotension and shock, prompt determination of the underlying cause can be lifesaving as it allows timely initiation of appropriate treatment. Here, echocardiography permits rapid assessment of cardiac structure and function, global and regional ventricular wall motion, cardiac chambers size, valvular disease and the presence or absence of a pericardial collection. Currently, TTE rather than TOE should be considered as the initial imaging in unstable patients. TOE is indicated when TTE is of non-diagnostic value and when the patient is intubated and ventilated. Shock can be broadly categorized into hypovolaemic, cardiogenic, distributive (e.g. anaphylactic, septic, neurogenic) and obstructive.

Hypovolaemic shock

Hypovolaemic shock is caused by a critical decrease in intravascular volume (i.e. haemorrhage, inadequate fluid intake, external fluid loss). Diminished venous return (preload) results in decreased ventricular filling and reduced stroke volume. Unless compensated for by an increase in heart rate, cardiac output falls. In hypovolaemia, echocardiography can rapidly document a small hyperdynamic unloaded ventricle, with a reduced LV end-diastolic area. There are a number of caveats, however, in using this feature to diagnose hypovolaemia and potential volume responsiveness, and the technique should only be applied in the context of a normal LV. In profound hypovolaemia (likely to respond to volume loading by an increase in cardiac output) the inferior vena cava diameter may be small (<10 mm) with inspiratory collapse in spontaneously breathing patients. In mechanically ventilated patients with hypovolaemia, the inferior vena cava might also be of small diameter but at end expiration and with variable respiratory change (depending on adaptation to ventilator).[74] As with interpretation of LV end-diastolic area to predict volume responsiveness, there are a number of exclusions to application of this technique in the critically/ acutely ill patient population, particularly when cardiac/cardiopulmonary pathology co-exists. They are not applicable when the patient is interacting with the ventilator.

Distributive shock, sepsis and the sepsis syndromes

Distributive shock is caused by an insufficient intravascular volume of blood secondary to vasodilation. Septic shock is the commonest cause. It is frequently associated with relative hypovolaemia (loss of intravascular volume due to capillary leak), relatively high cardiac output (except potentially in the presence of cardiac disease) and hyperkinetic biventricular systolic function. The LV is rarely dilated, unless an underlying chronic cardiac dysfunction in present. In some patients, diffuse transient hypokinesia might be observed, but the cardiac output may still be high or normal.[75] In other conditions, the absence of hyperdynamic LV and RV systolic function often underlines relative myocardial functional impairment. Some degree of pulmonary hypertension and/or elevated pulmonary vascular resistance is common. RV systolic dysfunction may develop in up to one-third of patients. Intrinsic depression of RV myocardial function is detected as RV hypokinesia, and semi-quantitatively appreciated as variable degree of RV dilatation. Septic shock can be associated with endocarditis or line/cannula infection, as well as related to other systemic causes of infection. In sepsis accompanying pneumonia, when respiratory failure supervenes assessment of ventricular function should anticipate the potential requirement for extracorporeal respiratory support, with the requirement that the RV is able to tolerate a significant additional volume load (up to approximately 4 L/min). Here, advanced assessment of RV function is required. Emerging studies have suggested the potential use of speckle tracking to detect ventricular dysfunction in septic shock not appreciated by conventional echocardiography. In these studies, despite no demonstrable difference in fractional shortening and ejection fraction between controls and children with sepsis, significant abnormalities in circumferential and longitudinal strain, strain rate, radial displacement and rotational velocity and displacement have been demonstrated.[76]

In patients with clinical features of endocarditis and/or at high risk of endocarditis expert echocardiography should be undertaken to exclude valvular endocarditis as a cardiac source of sepsis (*New cardiac murmur* section, below). Detection of infection in the presence of indwelling lines and cannulae can be challenging. It is common for patients to develop thrombus associated with indwelling central venous cannulae, and fibrin strands are frequently seen on implanted pacemaker devices. Determination of line/wire/cannula infection can be challenging and may not be possible using echocardiography only. Here, the presence of new/persisting sepsis with progressive enlargement of mobile masses despite anticoagulation may suggest the diagnosis. Where endocarditis is suspected, but TOE is negative in patients with permanently implanted devices, intracardiac echocardiography has been proposed; however, its use in critically ill endocarditis patients has not been evaluated.[77]

Obstructive shock

Mechanical factors that interfere with filling (tamponade, mediastinal masses, inferior or superior vena cava compression or thrombosis, tension PTX, severe asthma, intracardiac tumours or clot) or emptying (acute massive pulmonary embolism, aortic stenosis, LVOT

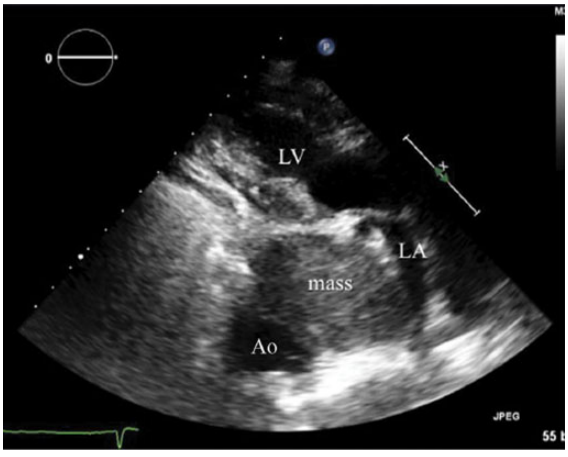

Figure 14: Mediastinal mass in a patient presenting with obstructive shock. Note the compression of the left atrium (LA) by a round mass localized behind the LA but in front of the descending thoracic aorta. Ao: aorta; LV: left ventricle.

obstruction) of the heart or great vessels may cause obstructive shock (*Figure 14*). As a result of the low cardiac output, the patient will be tachycardic with signs of tissue hypoperfusion and will have an increase in systemic vascular resistance to compensate.

LVOT obstruction

There is a subset of patients (many of them with a history of hypertension and LV hypertrophy, or with previous aortic valve replacement) who with volume depletion develop dynamic LVOT obstruction, with systolic anterior motion of the mitral valve and secondary mitral regurgitation, resulting in a progressive fall in cardiac output. Here, right heart catheterization (increased pulmonary capillary wedge pressure resulting from mitral regurgitation and the hyperdynamic non-compliant LV) may be misleading and may result in inappropriate decision-making (i.e. administration of inotrope agents instead of a beta-blocker and intravenous fluids). There should be a high index of suspicion in such patients, especially where they fail to increase cardiac output in response to escalating inotropic support. Echocardiography is diagnostic.

Cardiac tamponade

Cardiac tamponade results from important and/or rapid accumulation of fluid in the pericardial space that increases intrapericardial pressure, above intracavitary pressure, resulting in an important fall in transmural pressure. Therefore, chamber filling is reduced and cardiac output is compromised. Compensatory tachycardia and vasoconstriction initially maintain a normal cardiac output; however, at a critical level of intrapericardial pressure, cardiac output and arterial pressure fall. The inspiratory increase in venous return to the heart increases RV volumes, and because of the resultant septal shift (ventricular interdependence) LV filling is reduced, such that LV stroke volume subsequently falls, and pulsus paradoxus may result ($>$10 mmHg fall in systolic blood pressure with inspiration). These clinical features correspond to some of the echocardiographic findings.[78]

There are several 2D-echo findings that suggest a haemodynamically significant pericardial collection. Although a large pericardial collection is more likely than a small collection to be associated with increased intrapericardial pressure, this depends mainly upon the rate of accumulation. Hence, a patient with malignant pericardial effusion may have minimal/no haemodynamic compromise, while a patient who has sustained RV perforation during pacing may develop cardiogenic shock with only a small/minimal collection. The most sensitive sign of tamponade is cyclic compression, inversion or collapse of the right atrium, where chamber collapse in late diastole persisting into early ventricular systole occurs in the case of severely increased intrapericardial pressure. This sign is of only moderate specificity, however. Although diastolic RV collapse (inward diastolic motion of the RV free wall) occurs later, it is a more specific sign and is best appreciated from the parasternal or subcostal long-axis views. Dilatation of the inferior vena cava without change during deep inspiration has a reasonable sensitivity but only a moderate specificity for the diagnosis of tamponade (in the absence of concomitant right-sided disease), which is further reduced in the positive pressure ventilation setting. Pulsed wave Doppler provides additional information in the diagnosis of tamponade. Here changes in transvalvular velocities during respiration (best recorded at low speed) include inspiratory increase in RV inflow velocity ($>$ 35–40%) as well as a parallel (but slightly delayed for transpulmonary transit) reduction during inspiration of LV inflow and aortic ejection velocities. Of note, such phasic changes in flow with respiration are reversed in positive pressure ventilation.

If the patient's condition requires urgent pericardiocentesis, the procedure may be echocardiographically guided, as this has been shown to reduce complications.[78] Echocardiography can be used to determine the deepest part of the collection and the distance between the patient's skin and the collection. If necessary, the position of the pericardial needle can be visualized and in the case of doubt, agitated saline contrast can be injected to confirm the intrapericardial position of the needle/cannula. Echocardiography can additionally be used to verify whether the collection has been completely drained. TOE is rarely indicated in this setting.

Post-cardiac surgery is a special consideration, where collections may be small and localized, and early postoperative echocardiographic features of tamponade may be absent (even in the presence of profound haemodynamic compromise). Reliance on TTE potentially leads to misdiagnoses, and where suspected, the patient should undergo either immediate surgical exploration or TOE prior to intervention where the surgeon requires.

Cardiogenic shock

Cardiogenic shock is a state of inadequate cardiac output to meet the demands of the tissues. The commonest cause remains severe LV systolic dysfunction secondary to AMI. Shock due to LV dysfunction remains the leading cause of mortality in AMI (50–70%).[79] Other causes include RV dysfunction, mechanical complications of AMI, cardiomyopathy, severe valvular heart disease, myocarditis, myocardial contusion and acute aortic dissection. Echocardiography is an excellent initial tool for confirming the diagnosis of the cause of cardiogenic shock, providing additional information regarding the haemodynamic status of the patient (including filling pressures and stroke volume) and ruling out other causes of shock; therefore, immediate TTE should be performed when cardiogenic shock is suspected. Where TTE is suboptimal, TOE may be indicated.

Cardiogenic shock complicating AMI

In the setting of AMI, there are a number of causes of cardiogenic shock, including reduction in LV function, RV infarction, acute severe mitral regurgitation and mechanical complications: LV free wall rupture, ventricular septal rupture, and papillary muscle rupture.

LV dysfunction

Cardiogenic shock may result from the acute loss of a major percentage of cardiomyocyte function (at least 40% of the LV) or the additive loss of myocardial function in a previously damaged LV. Either extensive myocardial necrosis or stunned nonfunctional but viable myocardium may contribute to post-AMI shock. This is manifested on echocardiogram by a depressed LV global (ejection fraction) and regional function (regional wall motion abnormality), a decrease in stroke volume and cardiac output, elevated LV filling and pulmonary pressures with or without secondary mitral regurgitation. Both LV ejection fraction and the severity of mitral regurgitation are major predictors of mortality; however, admission ejection fraction does not correlate with outcome.[80]

RV infarction

RV infarction is most often associated with inferior AMI but rarely may be an isolated condition. Echocardiography may show RV dyssynergy, dilatation, paradoxical septal motion, and McConnell sign. A decrease of tricuspid annulus systolic excursion (TAPSE) is usually present. The diagnosis may also be suspected when a tricuspid regurgitation with low pulmonary pressure, associated with pulmonary regurgitation with a steep pressure half-time, is demonstrated. Assessment of LV function in the context of acute, severe RV dysfunction can be challenging as the LV may be relatively unloaded. Care must be taken when extracorporeal support is being considered, as when a right-ventricular assist device (RVAD) is used, the increased volume delivered to the LV may reveal significant LV dysfunction.

Free ventricular wall rupture

Free ventricular wall rupture is the most serious complication following AMI but its presentation may range from an acute sudden catastrophic collapse with electrical-mechanical dissociation to an insidious pericardial effusion with or without accompanying hypotension. Echocardiography may sometimes demonstrate overt cardiac tamponade or only a pericardial collection in subacute free wall rupture (30% of rupture). Thus, the presence of pericardial collection in the setting of AMI should raise the suspicion of subacute ventricular wall rupture, and careful scanning for wall rupture should be performed. A pericardial collection in this setting may consist of blood/thrombus and be localized inferiorly, presenting a potential diagnostic challenge.

Acute mitral regurgitation

Acute mitral regurgitation is a potentially life-threatening complication of AMI.[80] It is most commonly secondary (functional) to LV dysfunction without structural valvular abnormalities, resulting from increased leaflet tethering and decreased mitral closing forces. When present, secondary mitral regurgitation may exhibit a range of severity, and is frequently dynamic, being reduced in the presence of positive pressure ventilation. Even a mild degree of mitral regurgitation in the acute setting of MI is associated with a worse prognosis. More rarely mitral regurgitation can result from acute papillary muscle dysfunction or rupture. Rupture of the posterior papillary muscle (supplied by a single coronary artery) is by far the more common, and can be complete or partial. Echocardiography demonstrates a partial or complete flail of the mitral valve. The diagnosis is made when a triangular mobile structure representing the head of the papillary muscle attached to the tip of the flail leaflet prolapses into the LA during systole. Colour Doppler usually shows an eccentric jet directed away from the defective leaflet (anteroseptally directed jet of mitral regurgitation in the case of posterior flail leaflet). Where catastrophically severe, the colour jet may underestimate the severity of mitral regurgitation, as pressures between the two chambers rapidly equilibrate.

Acute mitral regurgitation can additionally result from acute systolic anterior motion of the mitral valve secondary to dynamic LVOT obstruction. Here the echo reveals an increased flow velocity across the LVOT and a typical latepeaking dagger-shaped CW Doppler profile (from the apical view). The severity of mitral regurgitation can range from mild to severe. The jet typically arises centrally but may take an eccentric course in the LA (directed towards the LA lateral wall). Flow duration and Doppler spectral configuration help to differentiate mitral regurgitation from LVOT obstruction. In mitral regurgitation, the rising slope at mid-systole is usually perpendicular to the baseline, whereas it is curvilinear until it reaches the highest velocity in the LV outflow signal. Moreover, the mitral regurgitation signal extends beyond the ejection period while its onset occurs earlier than the flow of dynamic LV obstruction. The evaluation of the severity of acute mitral regurgitation is described elsewhere.[81] The other mechanical complications of AMI have been discussed elsewhere.[80,81]

Recommendations for echocardiography in patients with shock

Recommended:

(1) For differential diagnosis of the cause of hypotension or shock, by detecting cardiac or non-cardiac aetiologies
(2) For differentiating the different cardiac causes of cardiogenic shock and guiding appropriate therapy, including surgical intervention when indicated.

Note: TOE is indicated when TTE studies are not diagnostic.

New cardiac murmur

Cardiac murmurs may be due to valvular heart disease, increased flow across a normal valve or shunts related to congenital or acquired defects. A murmur in a patient with acute cardiorespiratory symptoms or clinical signs suggesting structural heart disease is a definite indication for echocardiography. It provides valuable information regarding valvular morphology and function, aetiology of valve disease, quantification of valvular lesions, chamber size and ventricular function, wall thickness and pulmonary artery pressures.[82] Echocardiography also readily detects associated structural abnormalities such as fibrosis, calcification, thrombus or vegetation and abnormalities of valvular motion such as immobility, flail or prolapsing leaflets, or prosthetic valve dehiscence. The evaluation of valvular disease is extensively described in appropriate EACVI recommendations.[81]

Valvular disease leading to intensive care admission is likely to be severe, and predominantly affecting the leftsided valves. Both aortic and mitral regurgitation are the most common relevant acute valvular regurgitation; however, with increasing surgery for valvular disease, the incidence of prosthetic valve dysfunction is increasing.[81]

Critical aortic stenosis may lead to intensive care admission where acute decompensation occurs, or may be an incidental finding in a patient admitted for other reasons. Acute decompensation due to mitral stenosis is generally related to the onset of atrial fibrillation, and/or an increase in circulating volume (for example in pregnancy). Right-sided valvular pathology rarely leads to decompensation of the patient to the extent that admission is indicated *per se*, but the underlying pathology (i.e. endocarditis) may be the trigger for admission to the intensive care unit.

Valvular regurgitation

Acute mitral regurgitation can complicate an AMI or develop during an episode of transient myocardial ischaemia involving the posterior

and lateral LV wall. Acute mitral regurgitation can also result from spontaneous chordal rupture (degenerative or rheumatic mitral valve), infective endocarditis, acute HF (systolic or diastolic), and trauma, or mitral prosthetic dysfunction. Acute aortic regurgitation usually results from acute aortic dissection, trauma, infective endocarditis and rupture of a congenital valve fenestration or degeneration of previous bioprosthetic replacement. Where new mitral or aortic regurgitation occurs in the setting of acute clinical symptoms, there should be a high index of suspicion for infective endocarditis. TTE is mandatory in this setting,[83] and should be performed as soon as the diagnosis of infective endocarditis is suspected. TOE should be undertaken where there is diagnostic doubt and/or in planning the surgical strategy. TOE is indicated in particular when interrogation of a valve prosthesis is required. In addition to its major diagnostic value, echocardiography is also useful for assessing the cardiac consequences of the valve destruction, the prognosis and the risk of embolism. Finally, in the setting of acute cardiac care, echocardiography will help the clinician in the decision and timing for surgery.

Echocardiographically, the major difference between acute and chronic severe valvular regurgitation is the extent of cardiac chamber enlargement and the magnitude of LV or LA pressure increase. With acute mitral regurgitation, and in the absence of intrinsic LV systolic or diastolic dysfunction, the LV and the LA dimensions are normal or mildly increased. The evaluation of regurgitation severity is complex, should integrate several parameters and be interpreted in the context of the cardiorespiratory support of the patient, as positive pressure ventilation and pharmacological agents used for sedation/analgesia can significantly reduce the severity of mitral regurgitation. TOE might be necessary to accurately determine valve anatomy in some cases. Mitral regurgitation is also almost always severe in cases of papillary muscle rupture. Although colour flow imaging is helpful in detecting the presence and direction of regurgitant jet, colour Doppler may underestimate the severity in very severe regurgitation, and eccentric jets are more difficult to qualitatively examine. Therefore, the colour flow area of the regurgitant jet is not recommended to quantify the severity of acute aortic or mitral regurgitation. When feasible, measurement of the vena contracta and the PISA method are the recommended approaches to quantify both aortic and mitral regurgitation. For aortic regurgitation, a vena contracta width >6 mm or an effective regurgitant orifice area ≥ 30 mm^2 indicates severe regurgitation. For mitral regurgitation, a vena contracta width >7 mm or an effective regurgitant orifice area ≥ 40 mm^2 indicates severe regurgitation. However, it should be emphasized than a modest mitral regurgitant volume that develops acutely into a small non-compliant LA may cause severe pulmonary congestion and systemic hypotension, and that manipulation of the circulation and positive pressure ventilation may significantly alter the haemodynamics of the patient and echocardiographic findings.

Other more valve specific Doppler approaches can help in consolidating the impression of the degree of regurgitation. In mitral regurgitation, an early truncation of mitral regurgitant velocities and pulmonary venous systolic flow reversal are particularly informative. In aortic regurgitation, a short aortic insufficiency pressure half-time (<200 ms) and a diastolic flow reversal in the descending aorta (end-diastolic velocity >20 cm/s) are helpful. In acute severe aortic regurgitation, premature diastolic opening of the aortic valve and premature closure of the mitral valve with diastolic mitral regurgitation are particularly associated with catastrophic regurgitation. In the case of very severe regurgitation, echocardiographic parameters will additionally suggest a high LA pressure (short isovolumetric relaxation time (IVRT), LV mitral inflow showing a restrictive pattern with an increased E velocity >1.5 m/s, lung ultrasound suggesting interstitial oedema). Advantages and limitations of the various echo Doppler parameters used in valvular regurgitation are defined in the ESC/EACI guidelines, but evaluation must always consider the ventilatory, pharmacological and mechanical support of the patient before grading severity.[81,84]

Careful evaluation of valvular regurgitation is required in patients in whom mechanical circulatory support is indicated. Here, even mild aortic regurgitation may be important as it becomes continuous when the heart does not eject, and may lead to progressive LV dilatation.[81]

Valvular stenosis

Once the diagnosis of severe valvular disease demanding intervention is made[85] echocardiography provides information that shows whether the patient's anatomy is suitable for transcatheter intervention.[86,87] In the acutely unwell cardiac patient, the indications for transcatheter intervention are limited, but potentially lifesaving, and include percutaneous mitral commissurotomy (PMC) and balloon aortic valvuloplasty (BAV), potentially with a view to definitive procedural intervention at a later date.

Significant mitral stenosis

Significant mitral stenosis may mimic acute respiratory distress syndrome (ARDS), presenting with poor gas exchange and bilateral pulmonary infiltrates; however, the history and clinical examination will usually suggest the underlying diagnosis. A precipitant for acute deterioration may be the onset of atrial fibrillation. Where the patient is *in extremis*, PMC is performed with good effect. Indeed, PMC is the treatment of choice as a bridge to surgery in high-risk, critically ill patients. Echocardiography is the main method to assess the aetiology, severity and consequences of mitral stenosis, in addition to determining anatomical suitability and excluding contraindications to PMC. The details of valvular assessment and intra-procedural monitoring using echocardiography are published elsewhere.[85]

Critical aortic stenosis

Critical aortic stenosis may be the primary reason for intensive care/emergency admission, or may be an incidental finding in a patient presenting with other symptoms (i.e. fractured hip requiring surgery). The diagnosis may be challenging on the intensive care unit, particularly in a low cardiac output state; however, TTE findings are characteristics. Critical aortic stenosis can be managed using BAV as a bridge to surgery or transcatheter aortic valve implantation (TAVI) in haemodynamically unstable patients who are at high risk for surgery, or in patients with symptomatic severe aortic stenosis who require urgent major non-cardiac surgery.[87] As in mitral stenosis, guidelines and recommendations for assessment of the suitability of the aortic valve for BAV have been published elsewhere.[81–85]

Recommendations for echocardiography in patients with cardiac murmur

Recommended:

(1) Acute cardiac/intensive care patients with cardiac murmurs and symptoms or signs of heart failure, myocardial ischaemia/infarction, syncope, thromboembolism, infective endocarditis, or clinical evidence of structural heart disease;
(2) Where mitral regurgitation is suspected in a patient on positive pressure ventilation, stress echocardiography (volume and/or pressure loading) may be indicated;
(3) In patients with critical aortic stenosis and cardiogenic shock echocardiography should be used to assess suitability for BAV;
(4) In patients with severe mitral stenosis and cardiogenic shock and/or pulmonary oedema echocardiography should be used to assess for PMC;
(5) In patients requiring extracorporeal mechanical circulatory support minor degrees of valvular regurgitation (particularly aortic regurgitation) may be important and should be evaluated by an expert.

Prosthetic valve dysfunction

Haemodynamic instability in the presence of prosthetic valve replacement should raise suspicion of massive prosthetic obstruction or regurgitation, in addition to the possibility of prosthetic endocarditis in patients with sepsis. It is extremely unusual for right-sided valve disease to result in acute deterioration/decompensation. Left-sided prosthetic dysfunction should be suspected where there is normalization of septal motion, in particular in the presence of a dynamic LV and features of pulmonary oedema and/or cardiogenic shock. For the general evaluation of prosthetic haemodynamics the reader is referred to the corresponding recommendations.[88] Prosthetic valves are more difficult to evaluate than native valves, and therefore in the setting of a possible prosthesisrelated emergency should be evaluated by an expert in both TTE and TOE.[44]

Prosthetic valve regurgitation

Prosthetic regurgitation is frequently paraprosthetic, although sudden structural deterioration of bioprosthetic valves may cause severe transvalvular regurgitation. In rare cases, fracture of mechanical prostheses may occur, leading to occluder (disc) embolization with massive regurgitation. Large paraprosthetic leaks are often associated with abnormally increased mobility/instability of the entire prosthesis (rocking) and are termed dehiscence (*Figure 15*). Abnormal mobility of the prosthesis sewing ring nearly always implies severe paraprosthetic regurgitation. The severity of regurgitation may be difficult to judge especially in aortic prostheses using standard measures. The extent of the prosthetic circumference on short-axis views showing leakage may be used to estimate severity, with >20% (corresponding to >72°) of the circumference indicating severe regurgitation (*Figure 16*).[89,90] In acute regurgitation of an aortic prosthesis, premature mitral valve closure may be detected and indicates catastrophic regurgitation. New paraprosthetic leakage presenting in the acute/ emergency setting is predominantly due to endocarditis. In contrast to endocarditis for native valves, vegetations are frequently absent, with the only echocardiographic abnormalities being paraprosthetic regurgitation and/or valve instability.

Prosthetic valve obstruction

Since all prostheses represent some degree of obstruction to flow even if functioning normally, detection of true prosthetic obstruction can be challenging. Obstruction is generally due to either thrombosis (most frequently in mitral or tricuspid mechanical valves) or pannus. Direct observation of the valve mechanism is usually feasible for mitral and tricuspid prostheses, but may be challenging for valves in the aortic position, even with TOE, and in particular where mitral valve replacement

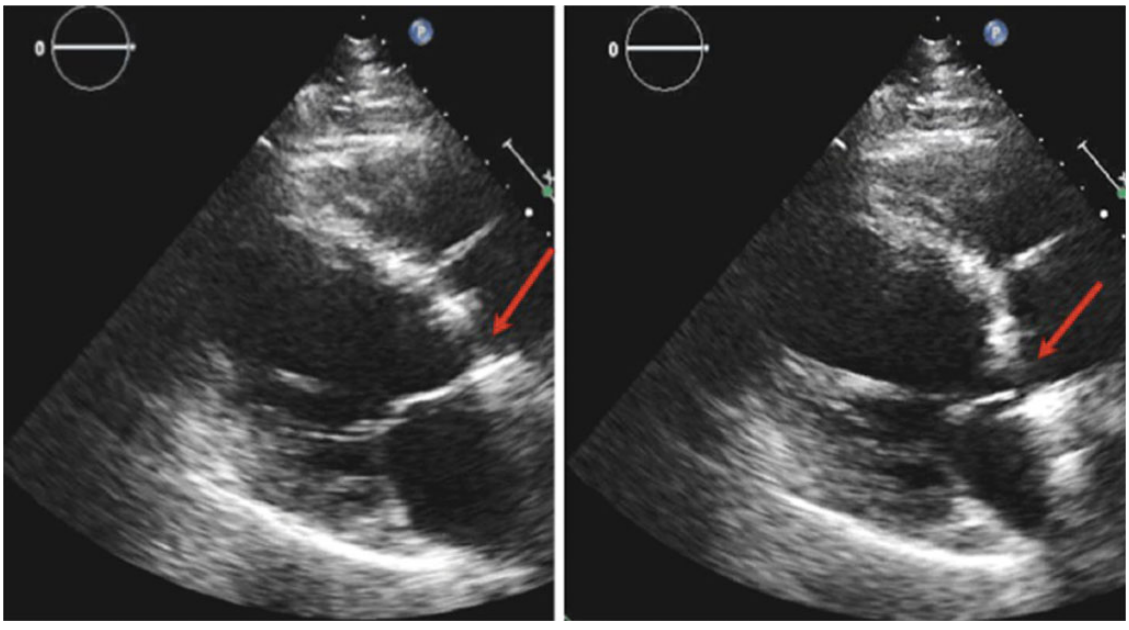

Figure 15: Mechanical aortic prosthesis with rocking motion, indicating dehiscence (see arrows). Transthoracic parasternal long axis view, left: in systole, right: in diastole. (see Supplementary data online, *Video 7*).

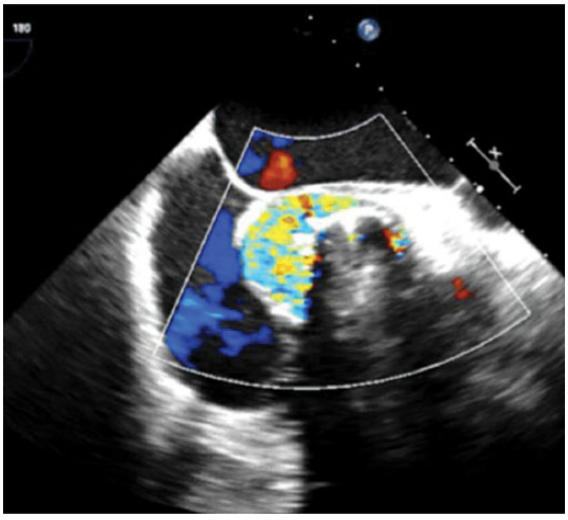

Figure 16: Same case as in *Figure 15*. The transoesophageal short-axis view of the aortic prosthesis shows on colour Doppler severe paraprosthetic regurgitation (approximately from 7 to 1 o'clock and thus 50% of the circumference). (see Supplementary data online, *Video 8*).

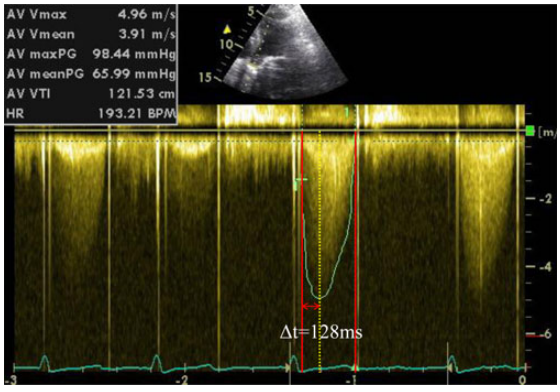

Figure 17: Transprosthetic continuous wave Doppler recording of a bi-leaflet mechanical aortic prosthesis with partial thrombotic obstruction (surgically confirmed). High flow velocities (peak 496 cm/s) and acceleration time (between red arrows) of 128 ms.

coexists. In all cases, expert evaluation is indicated. Transprosthetic gradients have wide normal ranges, and true obstruction may present with gradients well within those limits. Further, concomitant prosthetic regurgitation or small-for-patient prostheses (patient–prosthesis mismatch) will lead to abnormally high velocities without true mechanical obstruction. This is more frequent with aortic mechanical prostheses than with other prostheses. Ideally, for each valve, comparison with previous (ideally at time of implant) velocities should be undertaken and pressure recovery should be calculated where indicated.[91] In particular where a patient is in a high cardiac output state, or septic, the increased cardiac output may lead to correspondingly increased velocities that do not relate to an increase in valve obstruction. Here, increased velocities across all valves will be noted. Prolonged acceleration times (interval from onset of ejection to peak ejection velocity $>$100 ms; *Figure 17*) correspond well with prosthesis obstruction in mechanical aortic valve replacement. TOE and CT are particularly helpful in identifying thrombus or pannus in aortic prostheses and on the atrial side of mitral prostheses, and where doubt remains fluoroscopy of the mechanical prosthesis should be undertaken.

Recommendations for echocardiography in patients with suspected prosthetic valve dysfunction

Recommended:

(1) Urgent TTE is mandatory in all patients with suspected acute prosthetic valve dysfunction to define the extent and mechanism of regurgitation and/or obstruction.
(2) TOE is required, except in cases where TTE is entirely conclusive.
(3) Echocardiography should be used in parallel with other diagnostic techniques where uncertainty exists regarding the mechanism of dysfunction.
(4) If prosthetic obstruction is suspected, fluoroscopy and/or CT should be considered.

Cardiac arrhythmias

In the critically ill patient population, heart rates that might be acceptable in the outpatient setting may be inadequate. Thus, where stroke volume is limited (for example restrictive RV) a relative tachycardia may be required to maintain adequate cardiac output. In this patient population heart rates of 100–120 beats/min may be required. Other pathologies represent different challenges: a long diastolic time may be required to maintain a low LA pressure in mitral stenosis, a short diastolic time only required in LV/ RV restriction, and in any patient with temporally long mitral and/or tricuspid regurgitation, tachycardia may severely limit ventricular filling. When assessing any critically ill patient the optimal heart rate for that patient and their individual pathology at that time should be considered in order to maintain the optimal cardiac output at the lowest filling pressure. This may make echocardiographic evaluation challenging, but it is unavoidable in some patients.

Atrial arrhythmias

Atrial arrhythmias, common in the acute setting, present challenging conditions for assessing cardiac function and haemodynamics especially when irregular (as in atrial fibrillation). The loss of atrial contraction and the beat-tobeat changes in pre-load and LV contractility affect diastolic filling and ejection. Excessively rapid and irregular heart rhythms really challenge accurate assessment of cardiac function and filling pressures.[92] In atrial fibrillation, respecting an average of about 10 consecutive heart beats ($\geq$13 has been suggested[93]) with a view to allowing the use of echocardiographic parameters usually used in sinus rhythm to predict elevated filling pressures, except for the difference between duration of pulmonary vein A-velocity and mitral inflow A-velocity. E–e′ ratio ($>$13) and E/Vp ($>$1.4) indicate increased LV filling pressures. The use in critically ill patients should still be applied with caution. The pulmonary venous flow D-velocity deceleration time can also be measured ($<$220ms). Instead of measuring 10 consecutive beats, measuring three consecutive beats at a heart rate around 70 beats/min might

be adequate. A value of the E-velocity deceleration time ≤ 150ms has been demonstrated to predict elevated filling pressure in patients having a decrease in LV ejection fraction.[58,94] The averaging remains crucial for systolic as for diastolic function parameters assessment; however, other approaches have been proposed.[95] The 'index beat' method uses the measurement performed on the cardiac cycle following a pair of equal preceding cardiac cycles.

If atrial fibrillation duration is >48 h or there is doubt about its duration, TOE can be used to rule out intracardiac thrombus prior to cardioversion. This has been discussed elsewhere.[44]

Ventricular arrhythmias

Patients with ventricular arrhythmias require clarification of the aetiology of their arrhythmias for decision-making and ongoing care. Echocardiography is one of the first investigations to be performed as soon as the arrhythmia is successfully terminated.

Ischaemia

Ischaemia is the most frequent cause of ventricular arrhythmias, which can induce ventricular fibrillation (requiring immediate defibrillation), ventricular tachycardia or atrial fibrillation. Echocardiography in the acute setting may reveal globally or regionally reduced ventricular function related to the occluded/obstructed coronary vessel and immediate revascularization should be achieved [see *Acute Coronary Syndromes (ACSs)*, above]. In more chronic post-MI patients, scar tissue and reduced cardiac function are important foci for arrhythmogenecity. In the non-intensive care setting, cardiac function by LV ejection fraction is the most used parameter for risk stratification of ventricular arrhythmias, future prognosis and decision-making for implantable cardioverter defibrillator (ICD) in patients with previous MI. The best parameters in the critical care setting, in the context of inotropic and possibly ventilator support, are not known.

Non-ischaemic causes

Non-ischaemic causes should be considered in particular in individuals <35 years of age with ventricular arrhythmias. Inherited genetic cardiac disease is the most frequent cause of life-threatening arrhythmias in this population, including HCM, arrhythmogenic RV cardiomyopathy and familial dilated cardiomyopathy (DCM). In arrhythmogenic right ventricular cardiomyopathy (ARVC), echocardiography may reveal a dilated RV with reduced function, dilated RVOT and in progressive cases a thinning of the RV wall along with RV aneurysms and/or increased trabeculation of the RV (*Figure 18*). In HCM, echocardiography typically shows symmetric or asymmetric hypertrophy, normal or slightly reduced ventricular volumes, diastolic dysfunction and increased atrial volumes.[96] A hypertrophied interventricular septum or any segments with a wall thickness >15mm in the absence of other explanatory diagnoses, such as hypertension or amyloidosis, makes the diagnosis of HCM probable.[97] Importantly, if no echocardiographic or other imaging abnormalities are found in a young patient with ventricular arrhythmias, ion channelopathies, the use of toxics or doping have to be considered. Echocardiography will be normal by standard means, but recent studies have shown that a prolonged and dispersed myocardial contraction can be visualized using strain echocardiography.[98,99]

Detailed recommendations regarding standard echocardiography in these cases are outlined in the appropriate EACVI guidelines.[100]

Traumatic injuries of the heart and aorta

Blunt or penetrating chest trauma may cause severe injury to the heart and great vessels. Both TTE and TOE play an important role in the assessment of patients with chest trauma, and TOE may be indicated in patients with polytrauma and/or on mechanical ventilation or when a traumatic acute aortic syndrome is suspected. Of all cases of blunt trauma resulting in substantial injury to the thorax, motor vehicle collisions account for the majority, followed by falls from height, pedestrian–automobile collisions and crush injuries. It is important to distinguish aortic from cardiac injuries. With modern trauma centres and management, in severe trauma/polytrauma, rapid CT scanning allows rapid and wider visualization, and when immediately available is the imaging modality of choice. In those patients with unequivocal evidence of aortic injury, no further diagnostic imaging evaluation is necessary. Unlike other imaging techniques, TOE can be used intraoperatively, supporting and informing surgical and anaesthetic decisions. Finally, TOE can help evaluate the myocardium for wall motion abnormalities and assess the haemodynamic consequences of pericardial collection.[8,101] However, 5–25% of trauma patients have cervical spine fractures and in these patients TOE is contraindicated.[8] The correct diagnosis additionally requires differentiation of aortic rupture from other conditions that mimic its echocardiographic findings, such as aortic debris or atheromatous

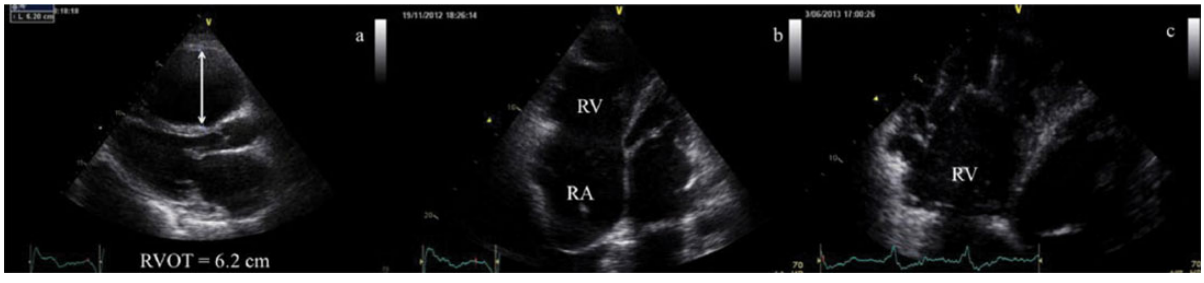

Figure 18: Dilated right ventricle (RV) with reduced function, dilated right ventricular outflow tract (RVOT) and thinning of the RV wall along with highly trabeculated RV apex and free wall are all signs of arrhythmogenic RV cardiomyopathy. RA: right atrium.

plaques. Also, traumatic pseudoaneurysms must be differentiated from true aneurysms.[8]

Acute traumatic aortic injuries

It is estimated that 75–80% of thoracic aortic injuries are a result of motor vehicle collisions, either head-on or serious lateral impact. Acute traumatic aortic injury from blunt trauma is a substantial cause of morbidity and mortality, being immediately lethal in 80–90% of cases. If detected early, it is estimated that 60–80% of patients who reach the hospital alive (usually showing an aortic pseudoaneurysm) will survive following definitive therapy. Therefore, prompt recognition and treatment of these injuries are critical for survival.[101] Though open surgical repair continues to be the mainstay of therapy, percutaneous endovascular stent-grafting repair is becoming frequent in many trauma centres. Trauma may cause aortic rupture, dissection or intramural haematoma. Partial disruption of the aortic wall may lead to pseudoaneurysm. Although TOE is described in trauma patients with suspected aortic injury, many patients will be undergoing CT routinely, and aortic imaging would be a part of the thoracic assessment protocol.

Understanding the mechanism of aortic injury is crucial to interpreting imaging findings. The sites of relative immobility of the aorta (the aortic isthmus and ligamentum arteriosum, the aortic root and the diaphragmatic aorta) are the most affected by blunt trauma. The exact pathophysiology of thoracic aortic injuries is complex and probably results from the interplay of multiple mechanisms. An osseous pinch is caused by posterior translation of the sternum, which traps the aorta at the narrowest point of the thorax. Torsion is depicted in the ascending aorta resulting in twisting, above the fixed aortic valve. Compression of the lumen forces blood to run inferiorly, toward the aortic valve, resulting in a water hammer effect. At the ligamentum arteriosum, the aorta is affected by both bending and shearing stresses, augmented by the anatomic changes from the osseous pinch against the thoracic spine and pressure changes from the water hammer effect. The combination of forces is concentrated at the fixed point of the descending thoracic aorta, at the level of the ligamentum arteriosum.[101]

Aortic injury most commonly results from transverse tears, and can be segmental or circumferential, and partial or transmural. Partial lacerations usually involve only the inner two wall layers, resulting in a contained rupture. The adventitia may be injured in up to 40% of cases and adventitial injury is almost universally fatal because of rapid exsanguination. Temporary tamponade may be achieved by surrounding mediastinal soft tissues.[8,101]

Recommendation for echocardiography in patients with suspected acute traumatic aortic injuries

Recommended:

(1) TOE is one of the first-line imaging methods recommended for diagnostic confirmation in patients with suspected acute traumatic aortic injury. (In practice, in most trauma centres, rapid trauma CT protocols are used as the first line of imaging.)
(2) TOE is also recommended intraoperatively to guide surgical and anaesthesic decisions.

Not recommended:

(1) TOE is relatively contraindicated in patients with cervical spine fractures.

Acute traumatic cardiac injuries

In blunt cardiac trauma, several forces may be involved, including compression of the heart between the spine and sternum, abrupt pressure fluctuations in the chest and abdomen, shearing from rapid deceleration, and blast injury. In addition, fragments from rib fractures can directly traumatize the heart. Unless the treating clinicians have experience in trauma, it is easy to underestimate the potential tissue damage in blunt trauma, in particular in blast injuries.

In patients with minor injuries and no tachycardia, hypotension, respiratory difficulty, chest pain or other concerning symptoms, no intervention besides clinical assessment may be needed. In patients with significant trauma, clinical assessment is difficult and often not sensitive or specific enough for detection of cardiac injury.[102,103] Clinicians must assume that hypotension and tachycardia in the trauma patient results first from the haemorrhage, but the presence of hypotension and tachycardia in the setting of isolated chest trauma suggests that pericardial tamponade and/or tension PTX/haemothorax should be excluded. Here, the clinician should perform a standard bedside ultrasound examination [i.e. Focused Assessment with Sonography for Trauma (FAST)], which includes screening for clinically significant haemopericardium and pleural collection.[16]

Once pericardial tamponade is excluded, a standard echocardiogram is useful in patients with signs of HF or abnormal heart sounds to diagnose the cause of dysfunction, estimate the need for volume resuscitation or inotropic support and identify other injuries (including RV dysfunction) requiring intervention or monitoring for arrhythmia. TOE is superior to TTE[104] for investigating the cause of persistent haemodynamic instability or other problems potentially related to cardiac injury. TOE provides a clearer view of wall motion abnormalities and valvular (i.e. valvular tear) and septal (i.e. septal rupture) injuries. Blunt cardiac injury includes a spectrum of pathology, ranging from clinically silent transient arrhythmias to deadly cardiac wall rupture.

Cardiac contusion/dysfunction

The most common expression of cardiac contusion is cardiac dysfunction (diminished contractility in the absence of arrhythmia or haemorrhage). The right heart is most commonly affected, due to its anterior position and ventricular injuries are as common as atrial injuries. Global dysfunction or abnormal regional wall motion may be seen. TAPSE should be always reported in the first echocardiographic protocol and used for follow-up of RV function, taking into account haemodynamics, ventilator, and inotropic and pressure support, as well as the arterial blood gases at the time of the study. Careful scanning of the RV free wall should be performed, looking for thinned myocardial segments at risk of rupture. In the presence of pericardial collection there is a significant risk of RV free wall rupture and careful screening for a pseudoaneurysm should additionally be performed using all available acoustic windows. Paradoxical septal movement might be the result of a right bundle branch block (RBBB) and if the ECG confirms its presence, cardiac contusion should be suspected.

Myocardial rupture

Myocardial rupture is the most serious type of blunt cardiac injury, where the majority of patients do not reach the emergency

department alive. Of those who do, rapid diagnosis by echocardiography followed by appropriate intervention can be life-saving. Less severe injuries to the ventricular wall may lead to delayed necrosis and manifest as late rupture, several days postadmission, and patients should be screened to determine those at risk.

Septal and valvular injury

Septal and valvular injury are both rare. Valvular injury occurs at the aortic/mitral/tricuspid valves. The lesion may consist of a tear of the leaflet, the papillary muscle and/or chordae tendineae with acute valvular regurgitation, clinically suspected and confirmed by echocardiography.

AMI

Although rare, AMI may result from coronary artery dissection, laceration and/or thrombosis. The left anterior descending artery is the most frequently affected artery. An ECG and an echocardiogram should be obtained early in the case of suspected MI.

Arrhythmia

Arrhythmia raises the suspicion of cardiac trauma if haemorrhage is excluded, in particular if unexplained, persistent tachycardia, new bundle branch block and minor arrhythmia (i.e. occasional premature ventricular contractions) occur, and it is an indication for rapid echocardiographic evaluation.

Recommendation for echocardiography in patients with suspected acute traumatic cardiac injuries

Recommended:

(1) Focused cardiac ultrasound examination should be immediately performed in patients with isolated chest trauma, hypotension and tachycardia to exclude pericardial tamponade or tension PTX.
(2) Emergency echocardiographic examination is necessary in patients with isolated chest trauma, no cardiac tamponade or PTX but having persistent tachycardia or hypotension, signs of HF, abnormal auscultatory findings, abnormal ECG tracings or recurrent arrhythmias. In such patients, echocardiography can help diagnose the cause of abnormal findings, estimate the need for volume resuscitation or inotropic support and identify other injuries requiring intervention.
(3) TOE is superior to TTE in the assessment of traumatic cardiac injury.

Not recommended:

(1) In patients with minor chest injuries and no tachycardia, hypotension, respiratory difficulty, chest pain or other concerning symptoms, no intervention besides clinical assessment may be needed.

Post-procedural complications

The indications and processes for intra-operative and intraprocedural echocardiography are widely recognized and documented. Following cardiac surgery or any catheter laboratory intervention, if the patient presents with haemodynamic instability and/or inadequate cardiac output, echocardiography should be performed as a first step examination to identify the underlying diagnosis. A complete TTE examination should be performed, but frequently TOE will be preferred due to the poor quality of transthoracic acoustic windows, in particular immediately after cardiothoracic surgery.

Post-surgical complications

Pericardial collection and cardiac tamponade are common complications after cardiac surgery. Diagnosis is clinical; however, when echocardiography is required, if TTE is negative, TOE should be performed if there is diagnostic uncertainty prior to return to the theatre for surgical evacuation. Frequently, echocardiographic features of tamponade will be absent, and pericardial collections are small and localized (and may be missed using TTE).

Myocardial function can be significantly impaired after a long bypass run and/or where cardioplegia has been challenging. Where there has been coronary intervention, there should be a high index of suspicion for coronary disruption, and a low threshold for coronary angiography. As the first 2–4 cm of the coronary arteries can be identified using TOE, any studies performed in this context should specifically image the coronary arteries, and where mitral valve surgery has been undertaken, specifically address the course of the circumflex coronary artery in the region of the posterior mitral valve (MV) annulus.

The RV is particularly susceptible to post-bypass dysfunction, the mechanisms of which are unclear. As RV dysfunction may seriously compromise the prognosis of the patient, RV size and function must be carefully checked in any cardiac post-operative patient. TAPSE is one of the parameters of RV function that is strongly recommended to be reported in the preand perioperative echocardiographic assessments—always taking into consideration the haemodynamics and degree of respiratory and inotropic support. Because TAPSE diminishes immediately after cardiac surgery it is recommended to measure TAPSE intra-operatively (preand post-cardioplegia, after intervention, post-protamine administration and in certain circumstances, i.e. RV to PA conduit, post-chest-closure). When coronary artery manipulation has been part of the procedure it should not be assumed that the cause of RV dysfunction is solely cardioplegia related.

Echocardiography immediately after heart transplant is essential, particularly in haemodynamically unstable patients. It will allow ruling out early rejection, early RV dysfunction, tamponade or other causes of instability.

The details of echocardiographic diagnosis of post-cardiac surgical complications are fundamental to perioperative echocardiographers, warrant separate consideration and are described elsewhere. Similar considerations are given for heart transplant patients.

Post-catheter/electrophysiology laboratory complications

Acute complications relevant to the echocardiographer generally include tamponade, ventricular failure and cardiogenic shock, occlusion of coronary stents and displacement of implanted devices. The commonest of these are covered in the recommendations outlined in this document.[87] When intracardiac devices have been implanted, haemodynamic instability should prompt initially TTE imaging. However, this will usually be followed by an expert TOE study that should be reviewed with the implanting cardiologist. In addition to demonstration of device displacement, echocardiography should be used to determine the time, urgency and strategy for further intervention.

Extracorporeal support

Extracorporeal support is increasingly used to support critically ill patients with severe cardiac and/or respiratory failure. It may be used as a bridge to recovery, transplantation, decision for further intervention, or in a very few patients with cardiac failure, as a destination therapy. Although echocardiography for extracorporeal support is highly specialized, certain key principles apply.[105,106] First, extracorporeal support is not a treatment *per se*, but rather a supportive therapy whilst awaiting resolution of the underlying pathological process. Thus echocardiography has a vital role in excluding any potentially treatable underlying cause for cardiorespiratory failure. Second, echocardiography is essential to determine the requirement for the RV and/or LV support and level of support required, and in assessing the ability of the right and left ventricles to support the extracorporeal circuit. This demands that the practitioner understands the different types of circuit, and the load that each will place on the heart. Third, echocardiography is mandatory to exclude cardiovascular contraindications to initiation of support (*Table 2*). Echocardiography subsequently has a vital role in its successful implementation, including confirming/guiding correct cannula placement, ensuring the goals of support are met, detecting complications and assessing tolerance to assistance. Finally, in patients requiring extracorporeal cardiac support, various echocardiographic parameters have been proposed to be used in conjunction with clinical and haemodynamic assessment in order to attempt to predict those patients who can be successfully weaned. Any practitioner intending to undertake echocardiography in patients on or requiring extracorporeal support should undergo specialized training in the techniques and their application.

Conclusions

Echocardiography has become the primary imaging tool for bedside diagnosis and monitoring of patients in acute cardiovascular conditions. It is non-invasive, provides rapid and accurate assessment of cardiac morphology and haemodynamics under stressful situations and is very useful in assisting therapeutic procedures. The fact that echocardiography can be repeated when required makes it highly useful in emergency/critical care circumstances.

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