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The ESC Textbook of Intensive and Acute Cardiovascular Care (2 ed.)

Edited by Marco Tubaro, Pascal Vranckx, Susanna Price, and Christiaan Vrints

Latest update

This online textbook has been comprehensively reviewed for the February 2018 update, with revisions made to 28 chapters. Find out more about the updates made.



ACCA
Acute Cardiovascular
Care Association

 European Society of Cardiology

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Echocardiography and thoracic ultrasound

Chapter: Echocardiography and thoracic ultrasound

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February 22, 2018: This chapter has been re-evaluated and remains up-to-date. No changes have been necessary.

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Update:

Updates to references

Updated on 27 July 2017. The previous version of this content can be found **here**.

Summary



For the emergency management of cardiovascular disorders, echocardiography and thoracic ultrasound are indispensable imaging techniques at the bedside. In the intensive care environment, crucial questions, such as left and right ventricular function, valvular heart disease, volume status, aortic disease, cardiac infection, pleural effusion, pulmonary oedema, pneumothorax, and many others, can be sufficiently and reliably answered by using these techniques; in fact, it is almost impossible to manage patients with acute severe haemodynamic impairment reasonably well without a prompt and repeated access to echocardiography. This is confirmed by the prominent place that echocardiography has in the guideline-based diagnosis and treatment of all major cardiovascular emergencies, from acute heart failure to the acute coronary syndrome to pulmonary embolism, etc. Moreover, it is the ideal tool to follow the patient, since repeat examinations pose no risk to the patient and demand relatively little logistics and resources. To benefit from the wealth of information that echocardiography and thoracic ultrasound can provide, modern equipment (including a transoesophageal probe) and systematic training of echocardiographers must be ensured. The availability of prompt and experienced echocardiography and thoracic ultrasound services at all times is fundamental for sound contemporary cardiovascular intensive care.

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Introduction



The intensive care environment poses specific and rigorous demands on the availability, speed, versatility, and accuracy of cardiac imaging. Echocardiography is by far the technique which best matches these demands in the vast majority of cases and thus is clearly—as in cardiology in general—the ‘first-line’ imaging technique in intensive care. Prompt availability of echocardiography is indispensable to any intensive care unit (ICU) and, of course, central to cardiac care units. In this chapter, the basic knowledge of echocardiographic techniques and examination procedures is assumed; strengths, weaknesses, pitfalls, and particularities of echocardiography in specific situations are discussed. New techniques are briefly presented, but the main emphasis rests on the most appropriate use of standard armamentarium, corresponding to the typical needs of intensive care management. The text has been arranged according to clinical scenarios.

Technical and equipment considerations



While echocardiography has always been a relatively small, portable, and deployable technique at the bedside, in contrast to other imaging modalities, technical progress in recent years has led to further

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reductions in size, even of state-of-the-art machines, making them well usable in an intensive care setting. Technical considerations in the selection of the best ultrasound machine for your coronary care unit (CCU) are provided in  Fig. 20.11. Besides, the industry has produced miniaturized echocardiography machines which have begun to differentiate into two main lines. The first line of products comprises laptop-type devices which essentially have all the capabilities of larger machines, including full digital storage and transoesophageal imaging. These devices typically still need a power supply or additional battery package and, although eminently mobile, a table or similar support platform. The other line of products is further miniaturized to allow effortless portability ('ultrasound stethoscope'), with a minimal weight of <1 kg; they can be carried in a coat pocket or around the physician's neck (see  Figure 20.1). These devices have quite limited options, if any, e.g. for Doppler interrogation and storage, and cannot be used for transoesophageal imaging [1, 2]. They still provide reasonable 2D quality sufficient to identify, for example, impaired LV function or a pericardial effusion.



Figure 20.11
Modern mobile, miniaturized echocardiographic devices from different manufacturers. Weights are indicated.

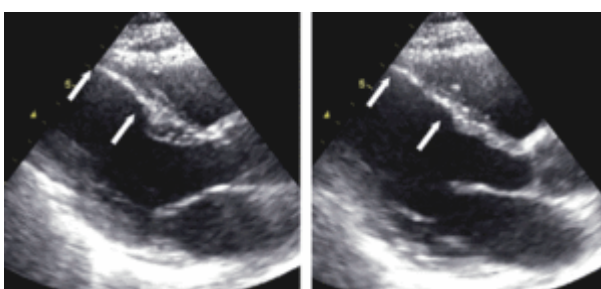


Figure 20.1
Wall motion abnormality in the anteroseptum (LAD territory). Left, systole; right, diastole. The wall segment between the white arrows does not thicken or move, in contrast to the basal septum. This is a sign of ischaemia or scar.

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The selection of an ultrasound machine for an ICU should consider the following fundamental issues:

- ◆ 2D image quality remains the cornerstone of echocardiographic diagnosis
- ◆ Reasonably good colour Doppler and, often neglected, continuous-wave Doppler are very important (e.g. to assess aortic stenosis (AS) or pulmonary systolic pressure via the tricuspid regurgitation profile)
- ◆ Trans oesophageal echocardiography (TOE) is an essential option
- ◆ Newer techniques are of limited importance; basic tissue Doppler is useful to assess left ventricle (LV) diastolic pressures, but more sophisticated techniques rarely are necessary to deal with emergencies
- ◆ Digital storage, including long-term storage, is mandatory, ideally via a digital network. Technically, even wireless options today are already available, although still rare. It is very important to be able to promptly recall previous examinations to monitor changes, e.g. in wall motion, and, given the life-and-death character of daily decisions in the intensive care environment, full documentation is vital to protect patients and physicians

In the intensive care setting, the maintenance of echocardiography equipment is a constant headache, often because no clear responsibilities exist. This relates to equipment maintenance, cleaning, proper disinfection of transoesophageal probes, housekeeping of reports and other documentation, management of digital storage, including protection against loss of data, regular software updates, providing sufficient space, and other issues.

Training



Minimal training requirements are provided in the online data supplement. The ICU is often the first place where a cardiology or internal medicine fellow in training operates an echocardiography machine, usually under informal supervision of a more experienced colleague. Because of the frequent direct impact of the echocardiographic diagnosis on management, this is a particularly impressive and enlightening situation to learn echocardiography. However, this must be accompanied or followed by systematic training, in particular because the time pressures of the intensive care setting often lead to incomplete and hurried examinations. Also, crucial competencies, such as eyeballing LV pump function or assessing valvular regurgitation, are not acquired sufficiently without systematic training and comparison to other techniques. In a study of the diagnostic accuracy of 'point-of-care' echocardiography with portable devices, the diagnosis of impaired LV function agreed in only 75% of cases between fellows with limited training and fully trained echocardiographers [1]. Therefore, the training recommendations of the European Association of Cardiovascular Imaging

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[3] (see  Table 20.1) or national societies should be followed, and it should be ensured that definitive reports are reviewed beforehand by a trained echocardiographer. For a fully trained cardiologist subspecializing in intensive cardiac care, the curriculum of the Acute Cardiac Care Association (ACCA) requests the ability to fully and independently perform trans thoracic echocardiography (TTE) and TOE (level III competence, including performance of at least 350 transthoracic and 50 transoesophageal studies).

Table 20.1 Training requirements in echocardiography of the European Association of Echocardiography to achieve basic and advanced level of competence

Echocardiography technique	Minimum number of examinations performed to become competent	Level of competence	Minimum number of examinations performed per year to maintain competence
TTE	350 (basic)	III	Reasonable exposure
	750 (advanced)	III	100
TOE	75 (advanced)	III	50
Stress echocardiography	100 (advanced)	III	100

Level III, the ability to independently perform the procedure (unsupervised).


From Popescu BA, Andrade MJ, Badano LP, Fox KF et al . European Association of Echocardiography recommendations for training, competence, and quality improvement in echocardiography. *Eur J Echocardiogr* 2009;10:893-905).

Data storage, preferably digital, and written reporting of echocardiographic findings are of critical importance in the intensive care setting, not only for patient management, but also for medicolegal reasons. Understandably, there is a tendency to neglect these tasks in the context of life-threatening scenarios, which nevertheless should be counteracted. (See also  Chapter 2).

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Acute coronary syndromes

Echocardiography is extremely valuable in acute coronary syndrome (ACS), and therefore an echocardiographic examination should be performed at the earliest possible point in time [4, 5]. The echocardiographic hallmark of ACS is the wall motion abnormality as a marker of ongoing or recent myocardial ischaemia (see  Figure 20.2). Wall motion abnormalities result from impaired systolic wall thickening and reduced systolic endocardial inward motion. They range in degrees, from hypokinesia (reduced thickening and inward motion) through akinesia to dyskinesia (systolic outward movement and thinning) and aneurysm (systolic and diastolic outward bulging and thinning), and in extent by the number of wall segments affected, which most conveniently are described by the standard 16- or 17-segment schemes [6]. The wall motion score is a semi-quantitative way to express this; each segment receives a score from 1 (normokinetic) to 4 (dyskinetic), and the sum of all scores divided by the number of segments, called the 'wall motion score index', is a dimensionless semi-quantitative parameter of wall motion impairment, 1 being for a normal ventricle and increasing in value with increasing wall motion abnormalities. The pattern of affected segments may indicate which coronary artery is affected. The degree and extent of wall motion abnormalities depend on the severity of ischaemia, which, in turn, depends mainly on the location of the occlusive thrombus and the duration of ischaemia. However, it is frequently not possible to decide whether a wall motion abnormality is new or old, although myocardial thinning or increased echogenicity implying fibrosis are signs of an older scar. Also, whether a new wall motion abnormality is reversible by an acute intervention (myocardial hibernation) is not immediately possible to decide, although some newer techniques, like left heart contrast echocardiography or deformation imaging, may be helpful. Although echocardiography is quite good at detecting acute myocardial ischaemia, wall motion abnormalities may be missed, depending on the image quality, and therefore echocardiography is not 100% sensitive for ACS. However, a good-quality echocardiography without any wall motion abnormalities makes acute myocardial ischaemia highly unlikely. On the other hand, the extent and severity of a detected, and presumably new, wall motion abnormality is important for global LV function and also predicts the prognosis and likelihood of post-infarction remodelling.

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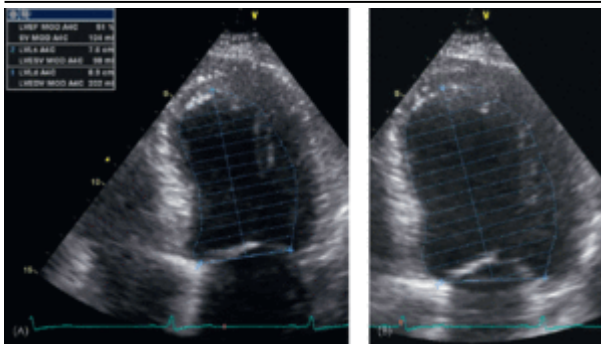


Figure 20.2

Calculation of EF by monoplane modified Simpson's rule (summation of discs) in a patient with mildly impaired LV pump function. Apical four-chamber view. (A) End-systolic frame, yielding a volume of 104 ml. (B) End-diastolic frame, yielding a volume of 202 ml. The EF is $(202 - 104)/202 = 51\%$. Note that the papillary muscle is included in the volume.

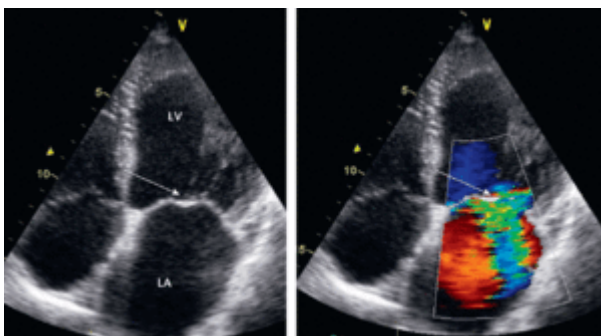



Figure 20.3





Ischaemic cardiomyopathy. All heart chambers are dilated, particularly the LV and LA. Note typical configuration of the closed mitral valve on the left, with closure line of leaflets (arrow) pulled into the LV by eccentric tug of the papillary muscles. Right, colour Doppler of functional, or 'ischaemic', mitral regurgitation (arrow).

A second crucial piece of information is global LV pump function, usually expressed as ejection fraction (EF), with well-known prognostic and management implications (see  Figure 20.2). EF can be eyeballed by experienced (!) observers and can be measured by 2D and three-dimensional (3D) methods with sufficient image quality. A useful surrogate parameter for EF in patients difficult to image is the excursion of the mitral annulus (mitral annular plane systolic excursion, MAPSE) or the peak systolic longitudinal (apico-basal) tissue velocity at the base of the LV, e.g. the basal septal segment in the four-chamber view. Besides this information, echocardiography is able to provide evidence for increased filling pressures, e.g. restrictive transmitral filling pattern,


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which carries considerable prognostic weight, independently of the EF [7]; for more detail, the reader is referred to the literature [8, 9].

Finally, complications of an acute myocardial infarction (AMI) can be quickly detected in the acute phase of an ACS:

- ◆ **Thrombus formation:** this is quite frequent in patients with large wall motion abnormalities, especially anterior aneurysms (see  Figure 20.4). Thrombi are often accompanied by spontaneous echocardiographic contrast or 'smoke', a marker of thrombogenic flow conditions caused by red blood cell (RBC) aggregation. To detect apical thrombi, they should be systematically sought, especially in two-chamber views, to minimize apical foreshortening, which is usually present, to some degree, in four-chamber views. Left heart contrast application can help in delineating thrombi. Fresh LV thrombi are potential sources of systemic embolism and require anticoagulation
- ◆ **Aneurysm formation:** aneurysms are large wall motion abnormalities due to myocardial scar, with an outward bulging shape that does not change during the cardiac cycle. They most frequently occur at the LV apex and are prone to thrombus formation ( Figure 20.12)
- ◆ **MR: acute ischaemic mitral regurgitation (MR)** may develop due to several mechanisms. Most frequently, LV global dilatation and remodelling lead to functional regurgitation (see  Figure 20.5). In rarer cases, the subvalvular apparatus of the mitral valve may be directly damaged by ischaemia, most dramatically in papillary (head) muscle rupture, with sudden onset of torrential regurgitation (see  Endocarditis, acute valvular regurgitation, and prosthetic valve dysfunction section). Chordal rupture may also occur, leading to less dramatic presentations. In any case, new ischaemic MR is a recognized negative prognostic sign and typically leads to pulmonary congestion or oedema. MR is detected easily by colour Doppler, and the underlying mechanism should always be sought. In papillary muscle rupture, hypermobile mitral leaflets flapping back and forth from the LV to the left atrium (LA), with an attached solid structure representing the ruptured distal papillary structure, are seen, together with signs of severe MR
- ◆ **Right ventricle (RV) infarction:** this complication of inferior infarcts manifests as an enlarged, hypokinetic RV, with new onset of tricuspid regurgitation. Recognition is important, because fluid restriction in this scenario is deleterious

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◆ Ischaemic ventricle septum defect (VSD) (see  Figure 20.6): rupture of the ventricular septum occurs in the muscular part of the interventricular septum, and the murmur is often taken for MR. Colour Doppler shows the jet in the RV, and the rupture site can often be seen directly on 2D images, but sometimes the course of the rupture line is tortuous and not directly visualizable. Subcostal images are very useful to detect VSDs, since the echocardiographic beam is almost coaxial to the shunt jet. VSDs are easily overlooked if the colour Doppler sector is not positioned in the appropriate region, which is the apical and mid RV

◆ Ventricular free wall rupture (FWR) and pseudoaneurysm formation: complete rupture of the LV myocardium leads either to a rapidly lethal tamponade, detectable as pericardial fluid and (usually) asystole, or, if the rupture is contained by the parietal pericardium, to pseudoaneurysm formation. Typical signs of a pseudoaneurysm are an abrupt decrease of myocardial thickness, an abrupt outward course (as if around a sharp corner) of the endocardial contour, and often a 'neck' which is narrower than the maximal diameter of the body of the pseudoaneurysm ( Figure 20.13). There may be paradoxical systolic inflow into the pseudoaneurysm and diastolic outflow. Distinction between a true aneurysm and a pseudoaneurysm is important, because the latter is, in principle, an urgent indication for surgery; the situation is not always clear, and additional imaging modalities may be necessary

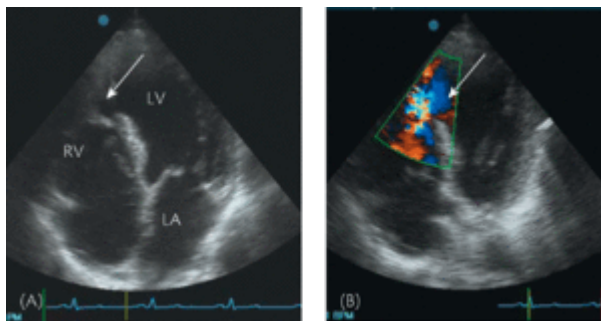


Figure 20.4

Post-infarction ischaemic VSD. (A) Apical four-chamber view. The discontinuity in the muscular ventricular septum is visible (arrow). (B) There is a colour Doppler jet towards the RV, indicating left-to-right shunting. Note that this jet is easily missed if the septum is not investigated by colour Doppler.

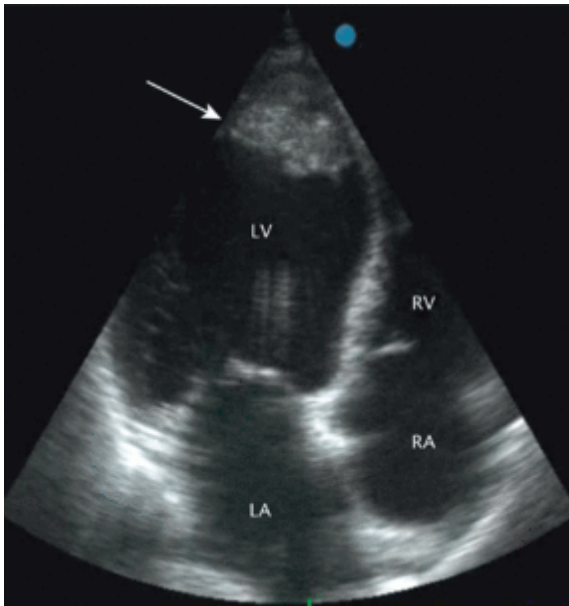


Figure 20.12
Anterior MI, apical aneurysm, and large apical thrombus (arrow).
Inverted apical four-chamber view.

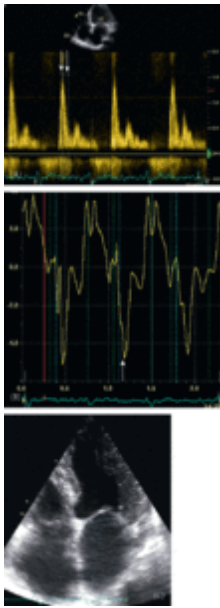


Figure 20.5

(A) Transmitral restrictive filling pattern with high and short E wave and small A wave; the peak E wave is more than double as high as the peak A wave. The E wave deceleration (time interval between arrows) is 103 ms. (B) In the same patient, basal myocardial velocities by tissue Doppler. Reduced early diastolic peak velocity e' (5 cm/s, arrow) and also low peak systolic velocities (4 cm/s). E/e' in this patient was 29, indicating massively elevated LV filling pressures. (C) LA volume calculation from the four-chamber view by summation of discs (different patient from panels A and B). The LA is mildly enlarged at 36 mL/m².

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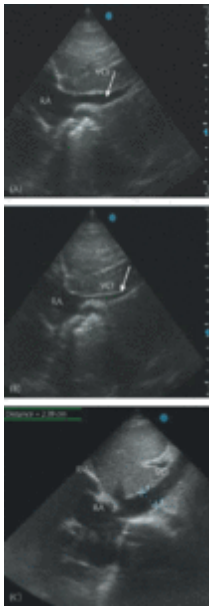


Figure 20.6
Respiratory variability of the diameter of the inferior vena cava, modified subcostal view. (A) Expiration. (B) Inspiration. (C) Dilated inferior vena cava (IVC; 24 mm) due to right heart failure.

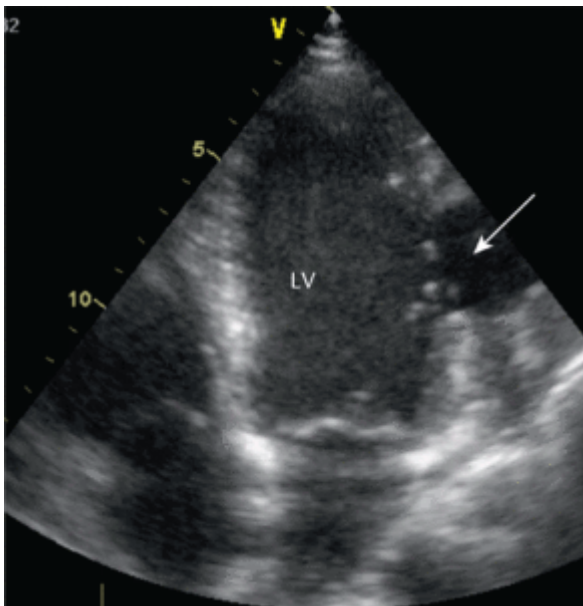


Figure 20.13
Post-infarction pseudoaneurysm arising from the lateral wall (arrow). Four-chamber view. The discontinuity of the LV free wall is well evident.

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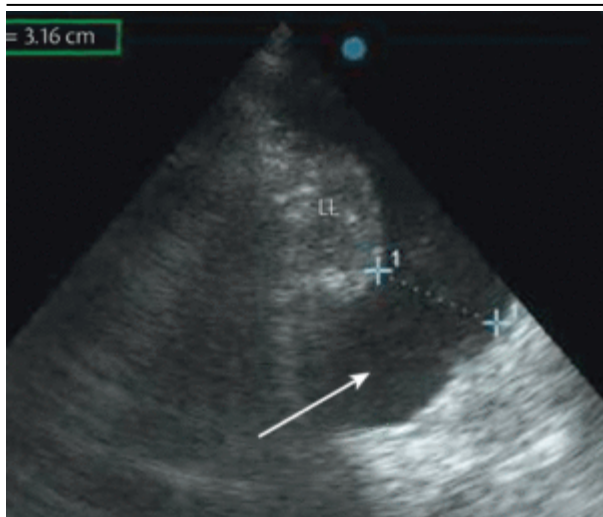


Figure 20.7
Large left pleural effusion (arrow) and collapsed left lung (LL).
Transducer placed in the left posterior axillary line.

In the subacute phase and before release from hospital, it is important to provide a baseline study of wall motion and ventricular function for later follow-up. Regional and global function may still undergo changes in this phase, both in the direction of improvement if there is still myocardial stunning or of deterioration due to LV post-infarction remodelling. Other concerns are the presence of inducible ischaemia from coronary territories other than the culprit one and the question of myocardial viability in areas of wall motion abnormality; these questions can also be addressed by (stress) echocardiography. (See also → Chapters 43 and 46.)

Shock and hypotension

Severe hypotension, shock, and cardiac arrest all call for immediate echocardiographic support. When performing such emergency echocardiography, it is useful to have a list of the most important echocardiographic features in mind to search for (see → Table 20.2). The imaging conditions are almost always suboptimal, with many individuals surrounding the patient, frequently in cramped conditions of an invasive laboratory, with simultaneous procedures such as establishing intravenous (IV) access or frank resuscitation going on in parallel. Obviously, portable echocardiographic machines are beneficial in these circumstances. Contrary to the typical protocol of echocardiography that begins with parasternal imaging, often only apical or subcostal windows are usable and should be quickly sought. In the ventilated patient, TOE, if quickly available, is very helpful. In the patient in cardiac arrest undergoing resuscitation, or shortly thereafter, it is typical to see diffusely hypokinetic, dilated ventricles. This per se does not establish a cause for the cardiac arrest. However, the presence of a pericardial effusion or a disproportionately large RV points to tamponade (see

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→ Chapter 27) or PE (see → Chapter 64) as the most likely causes of arrest, and these two conditions can be detected or excluded by echocardiography within seconds, even in very unfavourable circumstances, and may lead to lifesaving treatment.

Table 20.2 Typical echocardiographic features of shock and hypotension of different aetiologies



Aetiology	Key echocardiographic signs	Remarks
LV pump failure	Enlarged hypokinetic, spheroid LV, functional mitral regurgitation	
Pulmonary embolism (PE)	Enlarged RV apex reaching cardiac apex, hypokinetic RV, tricuspid regurgitation, elevated pulmonary pressure (degree varies with pre-existing pulmonary hypertension (PH), RV function, and extent of embolism)	
Pericardial tamponade	Pericardial fluid compressing the RV and/or right atrium; exaggerated respiratory variation in LV and RV inflow	Look for signs of aortic dissection, MI, trauma, or other thoracic disease, e.g. tumours
Acute left-sided valvular regurgitation	Structural damage of aortic or mitral valve, e.g. papillary muscle rupture; Doppler signs of severe aortic or mitral regurgitation; hyperkinetic LV, often of normal size	Look for signs of inferior MI in papillary muscle rupture and for signs of aortic dissection in aortic regurgitation
Acute right heart failure	Enlarged hypokinetic RV, may occur with PE, chronic PH, or as RV infarction complicating inferior myocardial infarction (MI)	

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Aortic dissection or rupture	Aortic enlargement, aortic valvular regurgitation, dissection flap in the aorta, pericardial tamponade	The typical site of aortic rupture, e.g. after deceleration trauma, is the proximal descending aorta, which is visualizable by TOE
Sepsis	Endocarditic vegetation on valve or pacemaker electrode, abscess, or destruction of valves	In sepsis due to non-cardiac causes, the discrepancy between systemic hypotension in the presence of an often hyperkinetic heart is typical
Prosthetic valve obstruction	'Frozen' occluder position in mitral prostheses, often with clear thrombus; in the aortic position, difficult to see, even by TOE. Massive transvalvular gradient elevation by Doppler	Always use TOE; compare to earlier transprosthetic gradients, if possible
Prosthetic valve regurgitation or dehiscence	Abnormal mobility ('rocking') of prosthesis, (colour) Doppler signs of severe regurgitation, premature mitral valve closure in aortic regurgitation	
AS	Severe AS, typically with a severely depressed LV	Continuity equation must be used to evaluate stenosis severity; gradients may be deceptively low

Most aetiologies of an acute drop in blood pressure (BP) are discussed in more detail in the pertinent sections of this chapter. Volume depletion, which is a frequent cause of hypotension, does not have specific echocardiographic signs; typically, the RV is relatively small (underfilled),

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and the inferior caval vein collapses with inspiration. The value of echocardiography in this scenario lies mainly in the exclusion of cardiac pathology. See  Assessment of cardiac output and volume status section for more detail. (See also  Chapter 49.)

Heart failure

See also  Chapter 51.

Assessment of systolic left ventricular function ( Table 20.6)

Echocardiography provides crucial information on the mechanisms, severity, and therapeutic options in congestive heart failure [10]. Global LV function is typically categorized into systolic or pump function, which assesses the ability of LV to create an adequate stroke volume, and diastolic function, which relates to the ability of the LV to fill adequately at low diastolic pressures. The evaluation of global LV systolic function consists of:

- ◆ EF, calculated from end-diastolic and end-systolic LV volumes (see  Figure 20.2). It may be visually estimated from several cross-sections or preferentially measured by tracing the LV in end-diastole and end-systole in the four-chamber view (monoplane EF) or additionally in the two-chamber view (biplane EF), enabling the calculation of LV volumes and EF by the modified Simpson's rule method. If 3D echocardiography is available, volumes can be calculated from the full 'volume dataset' without any geometric assumptions
- ◆ End-systolic (LV end-systolic diameter, LVESD) and end-diastolic (LV end-diastolic diameter, LVEDD) LV short-axis diameters (by M-mode or by 2D echocardiography measured from a parasternal long-axis view) and the shortening fraction $(LVEDD - LVESD)/LVEDD$ are the oldest quantitative parameters of global LV function. However, they only take into account wall motion at the base of the LV
- ◆ The systolic excursion (normally >12 mm) of the AV plane of the LV, i.e. the apical displacement of the mitral annulus during systole, can serve as a measure of global systolic function
- ◆ On tissue Doppler recordings from the mitral annular region of the septal and lateral wall in the apical four-chamber view, peak systolic longitudinal velocities are normally >5 cm/s (see  Figure 20.5). Strain values averaged over all LV segments ('global strain') may also be used to evaluate LV function

See  Table 20.3 for normal values.



Table 20.3 Echocardiographic signs of cardiovascular trauma

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- ◆ Pericardial effusion
- ◆ Wall motion abnormalities
- ◆ Rupture of valve leaflets or papillary muscles, with signs of acute regurgitation (especially in aortic or mitral valve damage)
- ◆ LV or RV FWR
- ◆ Intramural haematoma of the aorta
- ◆ Aortic dissection
- ◆ Aortic rupture/periaortic haematoma

Assessment of diastolic left ventricular function

Practically all patients with impaired global systolic function also have elevated LV filling pressures, and hence impaired diastolic function. Echocardiography provides an estimate of elevated filling pressures and is able to detect markers of impaired prognosis such as the restrictive transmitral filling pressure [8, 9]. On the other hand, there is a large group of patients suffering from symptoms of heart failure, although the EF is preserved, especially in the presence of hypertension and LV hypertrophy. This has been termed 'heart failure with normal EF' or 'diastolic heart failure'. The following signs and parameters should be systematically used to evaluate diastolic LV filling pressures:

- ◆ Impaired systolic function (EF) invariably leads to elevated filling pressures
- ◆ The presence of LV hypertrophy, independent of cause, indicating impaired relaxation and often also reduced chamber compliance, which, in advanced hypertrophy, requires higher than normal filling pressures
- ◆ The size of the LA, measured as LA volume from the apical four-chamber view or in both apical four-chamber and long-axis views (see  Figure 20.5). Normal size ($\leq 34 \text{ mL/m}^2$) excludes chronic elevation of LV filling pressures. However, the LA may also enlarge in other conditions, e.g. AF.
- ◆ The ratio E/e' (where E is the peak transmitral early diastolic flow velocity, divided by e' , the peak early diastolic mitral annular tissue velocities averaged from the septal and lateral mitral annular region) (see  Figure 20.5). A ratio < 8 largely excludes elevated filling pressures, whereas a ratio > 15 largely proves substantially elevated filling pressures. Between these values, other parameters have to be used to evaluate filling pressures [6]. These include a longer duration of the retrograde pulmonary atrial wave than that of the transmitral A wave, an increase in the peak velocity of the retrograde pulmonary

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atrial wave >35 cm/s, a reduction in pulmonary systolic forward flow, a delay in the onset of e' in relation to E, and others (📷+ Figure 20.14)

- ◆ Pulmonary artery systolic pressure, measured by the maximal tricuspid regurgitation velocity (see 📷 section on PE), is elevated in diastolic heart failure and is a measure of the severity of heart failure
- ◆ A restrictive transmitral flow pattern (peak E $>2 \times$ peak A wave velocity and E wave deceleration time <150 ms) indicates severely impaired prognosis (see 📷 Figure 20.5); however, this is usually accompanied by systolic dysfunction. Isovolumic relaxation time, a highly preload-dependent time interval, measured from the cessation of aortic flow to the onset of transmitral inflow, is severely shortened (<60 ms). A pseudorestrictive pattern may be observed in young, perfectly healthy individuals due to very vigorous relaxation
- ◆ A transmitral flow pattern with E $<$ A peak velocities is very frequent, with isovolumic relaxation prolonged (>100 ms). This is normal in patients aged >60 years. The pattern excludes substantially elevated filling pressures, since these would increase the peak E wave

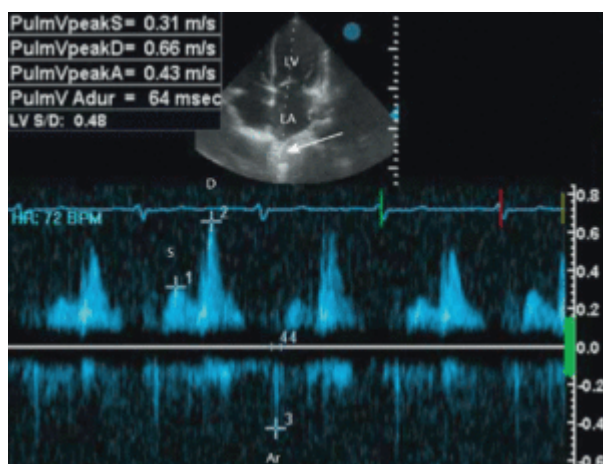


Figure 20.14


Pulmonary venous flow recorded from an apical window from the right upper pulmonary vein (arrow) by pulsed-wave Doppler. Reduced systolic flow and augmented atrial reverse wave velocity in the presence of ischaemic cardiomyopathy. S, systolic wave; D, diastolic wave; Ar, atrial reverse wave. Note, in upper panel, the calculated S/D ratio and peak reverse velocity VpeakA.

Other aspects of heart failure

Further important information in patients with heart failure gleaned from echocardiography includes:

- ◆ The presence and degree of MR



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- ◆ Signs of coronary artery disease (CAD) (e.g. regional wall motion abnormalities), cardiomyopathy (especially the dilated form), myocarditis, or constrictive pericarditis causing heart failure
- ◆ Signs of RV dysfunction: isolated RV dysfunction most frequently is due to chronic PH or acute pulmonary pressure elevation following PE (see  Chapter 66). Hallmarks are an enlarged and globally hypokinetic RV, with tricuspid regurgitation and elevated peak tricuspid velocity, which enables an estimate of peak systolic RV, and thus pulmonary, pressure. Importantly, in severe right heart failure after PE, the RV may not be able to generate high pressures, leading to deceptively low peak transtricuspid regurgitant velocities and RV pressure estimates. The ventricular septum is shifted to the left side, giving the cross-section of the LV a 'D' shape, instead of a circular appearance ('D sign'). RV size and function are usually eyeballed, due to the unreliability of M-mode or 2D measurements. A useful practical measure of RV function is the systolic excursion of the tricuspid annulus, with values <16 mm indicating impairment of RV function. RV end-diastolic free wall thickness >5 mm indicates chronic PH. RV infarction, a complication of inferior LV infarction, is discussed in the section on the ACS. Rarely, advanced cardiomyopathy, e.g. RV arrhythmogenic cardiomyopathy, may be the cause of right heart failure

Echocardiography also plays a critical role in identifying candidates for therapies and procedures which may improve heart failure or its prognosis [10]. The most important issues are:

- ◆ Measurement of EF to identify candidates for implantable defibrillator therapy (EF <35%)
- ◆ Diagnosis of hibernating myocardium with the potential to improve function after revascularization. Hibernating, i.e. dysfunctional, but viable, myocardial regions can be identified by dobutamine stress echocardiography. The identification of hibernating myocardium predicts improvement of EF and overall prognosis after revascularization

Assessment of cardiac output and volume status

Knowledge of cardiac output and of intravascular volume status is crucial in intensive care. These parameters can be estimated, to a degree, from echocardiography. Cardiac output is most easily calculated by measuring LV outflow tract (LVOT) diameter (D) and the time velocity integral of LVOT flow by pulsed-wave Doppler (VTI), taking care to align the Doppler beam as well as possible with the long axis of the outflow tract ( Figure 20.15) (see  Chapter 66). Provided there is no aortic regurgitation (AR), the systemic stroke volume (SV) is (assuming a circular outflow tract cross-section) approximated as:

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$$SV = \pi \times D^2 \times VTI / 4$$

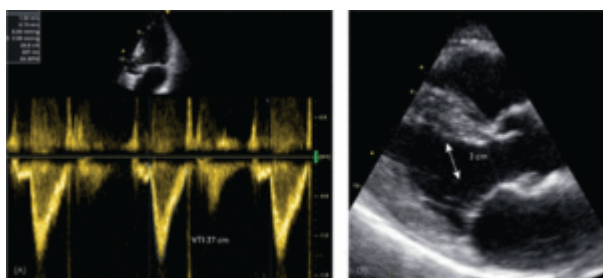




Figure 20.15

Stroke volume calculation from LVOT diameter (2 cm) and velocity time integral of the systolic flow profile in the LVOT, measured by pulsed-wave Doppler (27 cm), yielding a stroke volume of 85 mL. See text for further details.

A similar calculation can be made at the pulmonary valve level to calculate the pulmonary stroke volume. The product of the stroke volume and heart rate is the cardiac output. The calculation is far from precise but will give a reasonable estimate of the cardiac output.

The estimation of the intravascular volume status, specifically of central venous pressure (CVP) and of pulmonary capillary pressure, relies on indirect signs and is more complex.

◆ CVP may be estimated by assessing the respiratory variation of the diameter of the inferior vena cava from a subcostal echocardiographic window (see → Figure 20.6) (see also → Chapter 66). Exact measurements are rather difficult, since the inferior vena cava itself moves in and out of the cross-section during respiration, but a total obliteration of the vena cava during inspiration ('sniff') is usually associated with mean right atrial pressures of 5 mmHg or less. A less than 50% inspiratory decrease in caval diameter is associated with mean right atrial pressures above 10 mmHg, and no diameter change at all with severely elevated mean right atrial pressures ≥ 20 mmHg. Furthermore, the systolic portion of hepatic venous flow, which usually shows a systolic and diastolic forward wave towards the right atrium, decreases with increasing right atrial pressures. This is accentuated if there is substantial tricuspid regurgitation, which will lead to backward systolic flow in the hepatic veins. The hepatic veins can be easily sampled by pulsed-wave Doppler in held respiration from the subcostal window. PEEP elevates right atrial pressures and makes it difficult to use them to assess volume status

- ◆ Pulmonary artery pressure (PAP): systolic pulmonary pressure is assumed to be equal to peak RV pressure, which can be estimated using the tricuspid regurgitation Doppler signal (see  section on PE). In the absence of a usable tricuspid regurgitation signal, the acceleration time (time from the onset of pulmonary ejection to peak velocity) of the transpulmonary pulsed-wave Doppler flow profile may be used as a very rough estimate of pulmonary pressure; acceleration times >100 ms makes substantial PH unlikely
- ◆ Assessment of elevated LV filling pressures, and thus elevated pulmonary capillary pressures: this is detailed in the section on diastolic heart failure. Elevated pulmonary capillary pressures due to LV dysfunction usually are associated with E/e' ratios >15 [8]. Furthermore, high pulmonary capillary pressures and high LV filling pressures will generate a tall, short transmitral E wave; a transmitral E/A wave ratio <1 makes high filling pressures unlikely and may be normal for age or caused by hypovolaemia or slowed LV relaxation
- ◆ Pleural effusions are easily diagnosed and semi-quantitated by applying the echocardiographic transducer to the intercostal spaces over the lungs, preferentially with the patient sitting (see  Figure 20.12)

A chapter on TTE is available in the online data supplement.

Endocarditis, acute valvular regurgitation, and prosthetic valve dysfunction

See  Chapter 59.

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Infective endocarditis

Infective endocarditis may necessitate intensive care for several reasons: sepsis, acute valvular regurgitation, and central or peripheral systemic embolism. The echocardiographic hallmark of infective endocarditis are new mobile mass lesions (vegetations) (➡ Figures 20.16 and 20.19) attached to valvular structures and valvular destruction, leading to regurgitation; in fact, echocardiographic evidence of infective endocarditis is a major diagnostic criterion. Mitral and aortic valves are affected with similar frequency. Tricuspid endocarditis occurs mainly in drug addicts and patients with long-standing disease necessitating the insertion of indwelling central catheters and ports. Vegetations may also be attached to cardiac foreign bodies, such as pacemaker electrodes, and, in rare cases, directly to the endocardium, e.g. in the proximity of a VSD. Abscess formation in the perivalvular tissue may be observed, especially in aortic valve endocarditis (📷+ Figure 20.17) and in prosthetic valves. Further sequelae of endocarditis are fistulae, perforations, or the formation of a mitral pseudoaneurysm. Valve endocarditis may induce regurgitation of all degrees by perforation or rupture of valvular structures. Transoesophageal examination, because of its higher spatial resolution and better image quality, is superior to TTE and should always be used if there is a substantial suspicion of infective endocarditis (e.g., positive blood cultures) and no definite diagnosis can be established by TTE. In the presence of prosthetic valves, the use of TOE is mandatory [11], since infective endocarditis is frequent, difficult to detect, and more prone to complications, especially ring abscesses, in the presence of prosthetic valves. (See also 📷+ Chapter 59 and ➡ Figure 20.18.)

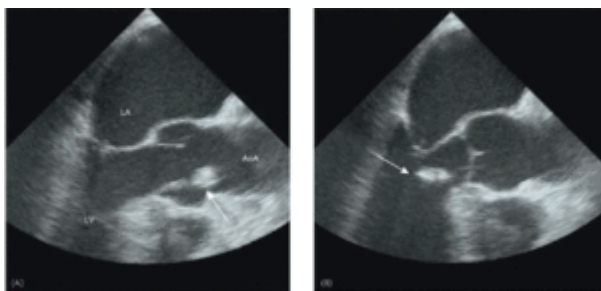


Figure 20.16

Infective endocarditis of the aortic valve, with vegetation attached to the right coronary cusp. Transoesophageal long-axis view at 120°. Note vegetation (arrow) changing position during the cardiac cycle. The vegetation is echo-dense, suggesting chronic or healed endocarditis. (A) Diastole. (B) Systole.

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Figure 20.19
Unusual presentation of mitral valve endocarditis. Modified apical four-chamber view. A ball-like mass, unusual for a vegetation, is attached to the atrial aspect of the anterior mitral leaflet (arrow). The surgical specimen confirmed a vegetation caused by *Staphylococcus aureus*.



Figure 20.17
Infective endocarditis. Abscess of the posterior aortic ring (arrow) in a patient with a mechanical aortic prosthesis. Transoesophageal long-axis view.

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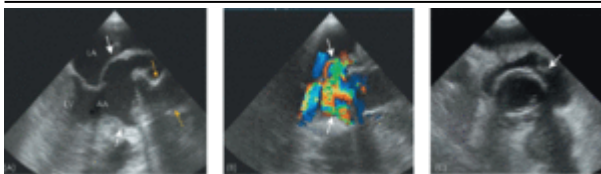


Figure 20.18

Prosthetic infective endocarditis after Bentall operation. TOE long-axis view at 120°. (A) Systolic image showing ring dehiscence and migration of the entire valved conduit (yellow arrows) above the plane of the aortic annulus (AA). The native aorta (white arrows) developed a pseudoaneurysm. (B) Colour Doppler in diastole showing a circular paraprosthetic leak (arrows). (C) TOE short-axis view of conduit replacing of the ascending aorta showing a periprosthetic abscess (arrow) in the proximal remnant aorta.

Acute mitral or aortic regurgitation

Acute severe MR occurs in several scenarios. Due to systolic regurgitation into the LA, there is acute volume overload, and consequently pressure overload of the LA, leading to increases in pulmonary capillary pressures and pulmonary oedema. Forward stroke volume is low, causing hypotension and ultimately CS. Key echocardiographic findings are [12, 13]:

- ◆ A clear structural abnormality of the mitral valve, with excessive mobility of a part of the valvular apparatus, such as a flail leaflet, or papillary muscle or chordal rupture (📷+ Figures 20.20 and 20.21)
- ◆ A large, consistently imaged proximal acceleration zone ($>1 \text{ cm}^2$ at aliasing velocities $\geq 50 \text{ cm/s}$) on the ventricular side of the mitral valve; the regurgitant jet in the LA is often hard to interpret due to tachycardia, low systemic pressure, and other factors
- ◆ Systolic pulmonary venous flow reversal
- ◆ In extreme cases, the typically symmetrical, bell-shaped continuous-wave Doppler profile of MR becomes triangular due to dramatic late systolic pressure increase in the LA
- ◆ The LV is often hyperkinetic due to massive volume overload. The LA may be of normal size due to the acuteness of regurgitation

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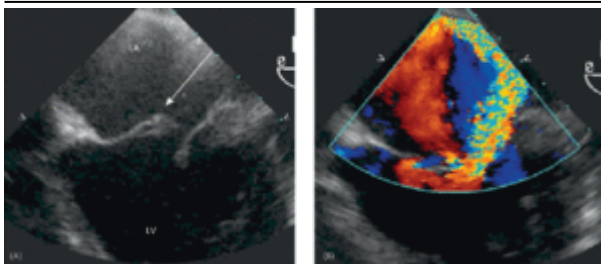


Figure 20.20
(A) Flail (arrow) of the anterior mitral leaflet, with severe eccentric mitral regurgitation. (B) TOE, four-chamber view.

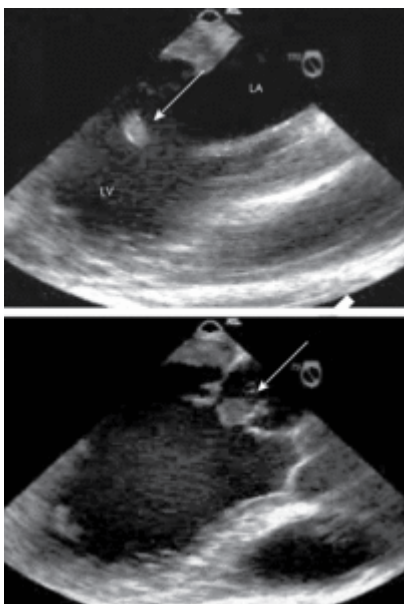


Figure 20.21
Papillary muscle rupture. Transoesophageal, transgastric two-chamber view. The ruptured head of the papillary muscle (arrow) is seen in the LV in diastole (top) and in the LA in systole (bottom).

Acute severe AR causes sudden LV volume overload and increased diastolic pressures, leading ultimately to pulmonary oedema, and, with reduced forward stroke volume, also leads to CS. Key echocardiographic findings are:

- ◆ A clear structural abnormality such as endocarditic destruction, rupture of a leaflet, or an aortic dissection membrane prolapsing into the outflow tract and precluding diastolic closure of the valve
- ◆ Doppler and colour Doppler signs of severe AR, including marked holo-diastolic backward flow in the descending aorta and, if present, a

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large proximal acceleration zone ($>1 \text{ cm}^2$ at aliasing velocities $\geq 50 \text{ cm/s}$) on the aortic side of the valve

- ◆ Premature closure of the mitral valve on mitral M-mode recording or the transmitral Doppler profile

Prosthetic mitral or aortic valves may develop sudden severe regurgitation due to ring dehiscence (e.g. in the course of infective endocarditis, endocarditic destruction or degenerative rupture of bioprosthetic leaflets) or thrombotic fixation or embolization of a mechanical occluder (📷+ Figure 20.22) (e.g. after strut fracture of a tilting disc prosthesis). (See also ➡ Chapter 59 and 📷+ Table 20.7.)

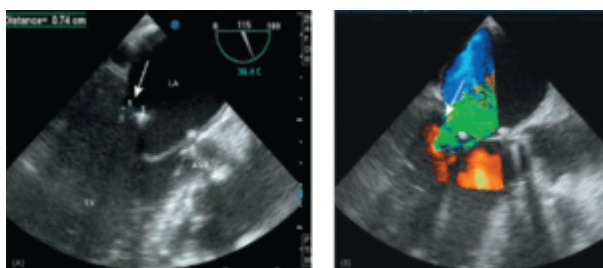


Figure 20.22

Severe mitral regurgitation due to ring dehiscence of mitral annuloplasty. The patient presented with AHF 6 months after aortic valve replacement and mitral annuloplasty for moderate/severe mitral regurgitation. (A) TOE long-axis view at 115° showing the paravalvular dehiscence of 7 mm width (arrow) at the posterior sewing ring. (B) Colour Doppler depicting severe mitral regurgitation, mostly outside the ring (arrow), combined with a smaller transmitral leak.

Table 20.7 Aetiology and echocardiographic features of acute valvular and prosthetic regurgitation

Aetiology in native regurgitation

- ◆ Infective endocarditis with valvular destruction
- ◆ Papillary muscle rupture following MI (mitral regurgitation)
- ◆ Degenerative chordal rupture (e.g. in mitral valve prolapse)
- ◆ Trauma

Aetiology in prosthetic regurgitation

- ◆ Bioprosthetic degeneration

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- ◆ Bioprosthetic endocarditis with leaflet destruction
- ◆ Prosthetic dehiscence (large paraprosthetic leak)
- ◆ Prosthetic thrombosis with fixed occluder
- ◆ Fracture of prosthetic valve with occluder embolization
- ◆ Trauma

Prosthetic valve thrombosis

Prosthetic valve thrombosis occurs almost exclusively in mechanical valves, leading to predominant stenosis, with or without regurgitation, depending on the dynamics of the prosthetic leaflet(s). Symptoms and clinical findings depend on how rapidly the prosthetic obstruction develops and on the severity of the obstruction [14, 15]. The incidence of prosthetic valve thrombosis is twice as high in the mitral as in the aortic position, and even higher in the tricuspid position, independently of the prosthesis type. Echocardiographic signs are increased transprosthetic Doppler gradients and, especially in mitral prostheses, absent motion of one or both occluders. TOE is mandatory and sometimes can distinguish obstruction by thrombus or by pannus. Fluoroscopy is very helpful and indispensable in aortic prosthetic thrombosis (📷 Figure 20.23). (See also 🔄 Chapter 59.)

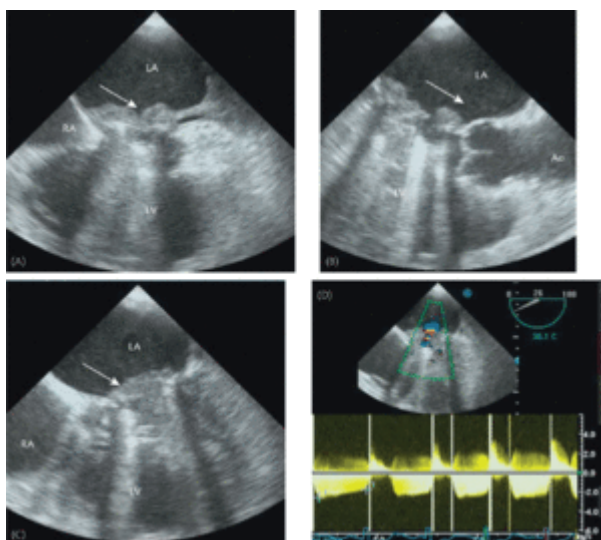





Figure 20.23

(A) to (C) Extensive obstructive thrombosis of a mechanical mitral prosthesis (arrow) in different transoesophageal views. (D) Transoesophageal continuous-wave Doppler profile across the mitral prosthesis, demonstrating obstruction (note very slow deceleration slope and a mean gradient of approximately 15 mmHg).

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Pericardial disease and trauma

The two main pericardial pathologies important for cardiac intensive care are pericardial effusion, including tamponade, and constrictive pericarditis. Pericardial effusions occur in many circumstances, including infectious, immunological, and malignant diseases, as well as in perforation or rupture of coronary vessels or cardiac chambers and traumatic damage.

Cardiac tamponade is a life-threatening complication of pericardial effusion and can occur even with small, but acutely developing, effusions, e.g. after coronary artery perforation or rupture due to PCI (see also  Chapter 27). Due to the limited intrapericardial space and low compliance of the pericardium, compression of cardiac chambers ensues, typically the right atrium (in late diastole and early systole) and RV (in early diastole) due to their relatively low inside pressures. This 'collapse', along with the effusion, can be seen directly on 2D echocardiography, especially in the four-chamber view and the subcostal view (see  Figures **20.8** and **20.9**). The duration of collapse during the cardiac cycle parallels the severity of haemodynamic compromise. While temporary inward displacement of the right atrial wall during the cardiac cycle is an early and sensitive sign of the haemodynamic significance of the pericardial effusion, its specificity for tamponade is low; on the other hand, RV collapse has lower sensitivity but higher specificity for haemodynamic compromise. In rare cases of localized pericardial effusions (e.g. post-operatively), the LA or LV may be primarily compressed; these localized effusions may be difficult to detect and may necessitate TOE, especially in the post-operative patient. The compression of the heart chambers leads to an exaggeration of the respiratory variation of inflow and outflow patterns of the ventricles; mitral inflow and aortic stroke volume decrease with inspiration, whereas tricuspid inflow increases. A >25 % decrease in peak transmitral E wave velocity with inspiration is considered a sign of haemodynamic compromise. A 'paradoxical' septal shift to the left in early diastole, created by the increase in transtricuspid flow, is also observed. In expiration, these changes are reversed. Furthermore, the inferior vena cava is usually distended and does not collapse with inspiration ( Table 20.8).

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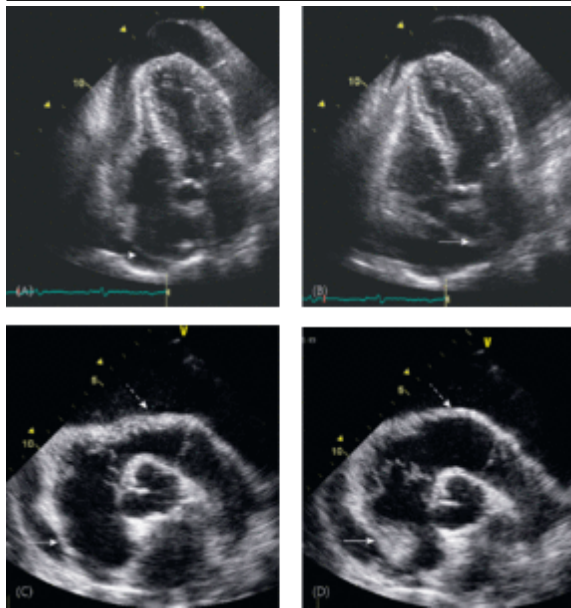


Figure 20.8

Circular pericardial effusion with onset of haemodynamic compromise (tamponade). (A) Apical four-chamber view in diastole, with normal convex contour of the right atrial free wall, and (B) systole, with compression of the right atrium (arrows). (C) Parasternal short-axis view in diastole, with compression of the RV (dotted arrows) and no compression of the right atrium (continuous arrow), and (D) systole, with compression of the right atrium and no compression of the pressurized RV.

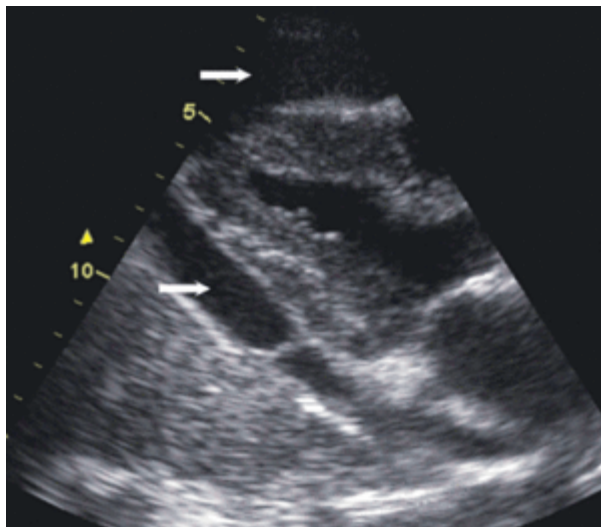


Figure 20.9


Large, circular pericardial effusion (arrows). Parasternal long-axis view.

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Table 20.8 Echocardiographic signs of cardiac tamponade

- ◆ Exaggerated respiratory variation of LV and RV filling: decreased inspiratory early transmitral filling and increased early transtricuspid filling; reciprocal changes in expiration
- ◆ Right atrial collapse
- ◆ RV collapse
- ◆ LA collapse
- ◆ LV collapse
- ◆ Inferior vena cava plethora

To prepare for pericardial puncture, the subcostal view is useful to determine the location, angle, and depth of the puncture. If the subcostal approach is not feasible, echocardiography helps to select alternative sites for puncture, i.e. sites where the distance between the skin and pericardial fluid is minimal, e.g. the apex. After puncture, the location of the tip of the needle or of the catheter introduced in the pericardial space may be confirmed by injecting an agitated infusion solution, which will create a bright contrast echo in the pericardial space.

In constrictive pericarditis, a thickened (>5 mm) or calcified pericardium may be apparent but often is not. The ventricles are of normal size, whereas the atria are enlarged. Global LV and RV systolic functions are normal, but paradoxical septal motion is present. In clear-cut cases, there is an inspiratory decrease in transmitral flow and increase in transtricuspid flow, with opposite changes in expiration. Sometimes, however, this sign is blunted by massive diuretic therapy. The transmitral flow in the typical case is characterized by tall, short E waves, with short deceleration time ('restrictive transmitral pattern'). (See also  Chapters 27 and 58.)


Traumatic cardiac and aortic injury

A detailed discussion on traumatic cardiac and aortic injury is provided in  Chapter 62. A focus on echocardiography is provided in the online data supplement.

Virtually all cardiac structures may be damaged by blunt trauma. The ventricles may develop intramyocardial haematoma, myocardial necrosis, or frank rupture, leading to usually a lethal tamponade, if a free wall is affected, or else to a VSD. The RV free wall is most often affected due to its anterior localization in the chest. The atria may also rupture. Coronary vessels may be lacerated, leading to haematopericardium and tamponade. Valvular structures may also be damaged, including leaflets and the support apparatus, most prominently papillary muscles or chordae. Tears


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of the pericardium, with or without other cardiac injury, may lead to herniation of other organs into the pericardial sac (e.g. intestinal herniation through an also ruptured diaphragm) or displacement of cardiac structures outside the pericardial sac.

The thoracic aorta is affected, especially by deceleration trauma (e.g. traffic accidents or falls) (see also  Chapter 61). Traffic accidents typically cause aortic damage at the aortic isthmus, the junction of the aortic arch, and the descending aorta. Falls may lead to aortic damage at the level of the innominate artery. The damage to the aorta ranges from intramural haematoma to complete transection of the vessel.

Echocardiography, and especially TOE, is very helpful after blunt chest trauma. In a study, more than half of 117 patients with blunt chest trauma showed clearly pathological findings on TOE, ranging from RV wall motion abnormalities to pericardial effusions; the ECG was often normal in these patients [16, Table 20.3]. Penetrating trauma of the heart or large vessels is typically rapidly deadly, precluding echocardiographic examination.

Pulmonary embolism

A dilated and hypokinetic RV is the typical echocardiographic finding in severe PE. Additionally, there is a variable increase in RV systolic pressure, as estimated by measuring peak tricuspid regurgitant velocity (see  Figure 20.10). This is done by measuring peak tricuspid regurgitant velocity using continuous-wave Doppler, calculating the peak pressure difference between the RV and right atrium from the Bernoulli equation ($\Delta p = 4 \times v^2$) and adding an estimate of the mean right atrial pressure, e.g. 10 mmHg (for more detail on right atrial pressure estimation, see section on heart failure). Note, however, that, in the acutely failing RV associated with fulminant PE, this pressure may be deceptively normal or only minimally elevated. Very high peak pulmonary pressures (e.g. >80 mmHg) cannot be generated by a previously normally loaded RV and do not occur in response to an acute embolism, unless there is previous PH. A shift of the ventricular septum to the left, flattening the cross-section of the LV into a 'D' shape, instead of the normal circular shape, in short-axis views, and paradoxical septal motion are important signs of acute RV pressure overload. Tricuspid regurgitation is invariably present, and the inferior vena cava may be distended and lack inspiratory collapse. Thrombi may sometimes be seen directly in the pulmonary artery imaged in parasternal or subcostal views. If TOE is performed, the right pulmonary artery can also be evaluated quite well, and thrombotic material may be seen there. The acute pressure increase in the right atrium leads to a shift in position of the atrial septum to the left side and may create continuous right-to-left shunt through a patent foramen ovale. In the presence of severe PE, paradoxical embolism of thrombotic material through a patent foramen ovale is a recognized and devastating complication. On the other hand, small PEs are not detectable on echocardiography. The role of echocardiography therefore is not in the definitive exclusion of a (small) PE, but in the assessment of whether a haemodynamically significant embolism has taken place and whether RV compromise warrants thrombolytic therapy.

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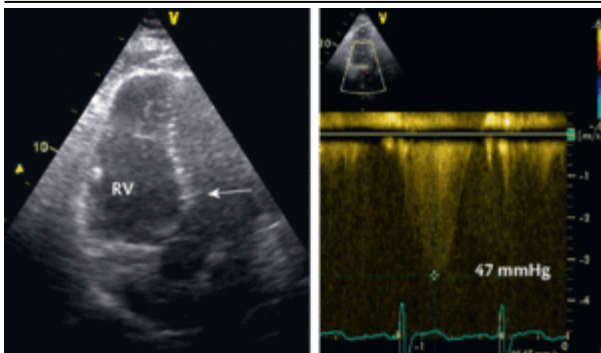



Figure 20.10

PE. (A) Dilated, hypokinetic RV which reaches the apex and appears larger than the LV. Note the shift of the basal septum to the left (arrow). (B) Continuous-wave Doppler of tricuspid regurgitation showing elevated peak RV pressure (peak tricuspid regurgitant velocity 47 mmHg).

The echocardiographic differentiation of chronic and acute pulmonary pressure elevation is difficult. RV hypertrophy, with an end-diastolic free wall thickness >5 mm, supports chronic PH but does not exclude an additional acute pressure increase. Several signs that have been described as relatively specific (but not sensitive) for acute PE [17] are:

- ◆ McConnell's sign: a hypokinetic RV free wall, together with a hyperkinetic or normokinetic RV apex
- ◆ The transpulmonary pulsed-wave Doppler flow profile may show shortened acceleration time (<100 ms) and notching, which is believed to result from reflected pressure waves created by a central pulmonary thrombus
- ◆ The '60/60' sign: a pulmonary systolic pressure <60 mmHg by tricuspid regurgitation and a pulmonary acceleration time <60 ms

It should be clear, however, that these signs are far from being ideally predictive for an acute PE. (See also  Chapter 66.)

Aortic emergencies

The ascending aorta can be seen over its first few centimetres from the left and right parasternal echocardiographic windows, and the aortic arch is seen from the suprasternal window, which, however, is often obstructed in elderly or emphysematous individuals. A much better evaluation of the thoracic aorta is afforded by TOE where almost the entire course is visible, except for a 'blind spot' created by tracheal and left bronchial interposition at the distal ascending aorta and proximal arch. Dilatation and aneurysms, atheromatous disease, plaque-adherent thrombi, aortic dissection or intramural haematoma, and traumatic damage (see section

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on cardiac trauma) can be diagnosed by TOE. The following are important pathological features of the aorta [18, Table 20.4]:




- ◆ Enlarged aortic diameters: these are important, since the risk of rupture and also of dissection depends on aortic diameters. Diameters >55 mm, in general, are an indication for surgery, even in asymptomatic patients, and, in patients with Marfan's syndrome or bicuspid aortic valve, lower values have been recommended for replacement [12]
- ◆ Atheromatosis is mainly observed in the descending aorta and the arch. Sometimes, mobile thrombi may be noted which may embolize ( Figure 20.24)
- ◆ Aortic dissection is diagnosed by identifying the pathognomonic dissection membrane, a thin and undulating membrane ('intimal flap') separating the true and false lumen. Reverberations of the posterior wall of the ascending aorta ( Figure 20.25) or the posterior wall of the right pulmonary artery may create aortic intraluminal linear, horizontal lines and need to be differentiated from true flaps, which are usually very well delineated and crisp. Entry and re-entry sites may be identified by 2D echocardiography and colour flow Doppler. The false lumen typically is larger, has slower flow (often spontaneous contrast or even thrombosis is present), and is convex towards the higher-pressurized, but smaller, true lumen ( Figure 20.26). The site of the intimal rupture and the extent of the dissection are crucial for the identification of the type of dissection and its management. Type A dissection typically is accompanied by some amount of AR and pericardial haemorrhage, which abruptly may progress to a lethal tamponade. These two signs are easily recognized on TTE and should raise the 'red flag' of a possible aortic dissection in the context of chest pain or other clinical presentations such as sudden hypotension or shock. When a patient with a suspected aortic dissection is examined by TOE, blood pressure must be controlled tightly, since death due to progressive rupture during TOE has been reported
- ◆ Intramural haematoma of the aorta is a variant or precursor of dissection, which often co-exists with areas of classic dissection (Table 20.5). It is characterized by a thickened aortic wall (>7 mm) with echolucent areas, a smooth intimal surface, and sometimes a displacement of superficial calcifications towards the lumen. Differentiation from severe atherosclerotic wall thickening or the thrombosed false lumen of a classic dissection can be difficult

Table 20.4 Classification of aortic dissection

- ◆ Stanford A: dissection involves the ascending aorta and may also involve the aortic arch and descending aorta

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- ◆ Stanford B: dissection involves only the descending aorta
- A more recent classification of acute aortic syndromes [17] distinguishes as follows:
- ◆ Class I: classic dissection of all types
 - ◆ Class II: intramural haematoma
 - ◆ Class III: subtle circumscribed dissection representing a localized tear without a clear-cut haematoma
 - ◆ Class IV: plaque ulceration (mostly in the descending aorta, and often in the abdominal aorta)
 - ◆ Class V: traumatic or iatrogenic (mostly catheter-induced, retrograde) dissection

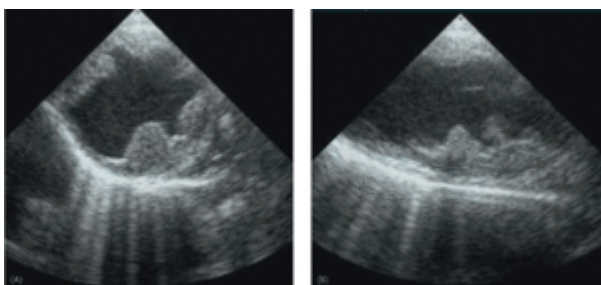


Figure 20.24
Thrombi in a non-aneurysmatic descending aorta. After a few weeks of anticoagulation, the thrombi dissolved, and only atherosclerosis of the aortic wall was seen. (A) Transoesophageal short-axis view. (B) Transoesophageal long-axis view.

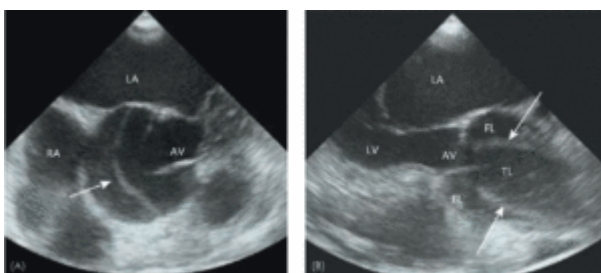


Figure 20.25
Dissection of the ascending aorta. (A) Transoesophageal short-axis view. Note the mobile flap (arrow). AV, aortic valve. (B) Transoesophageal long-axis view at 120°. Note the circular flap (arrows). FL, false lumen; TL, true lumen.

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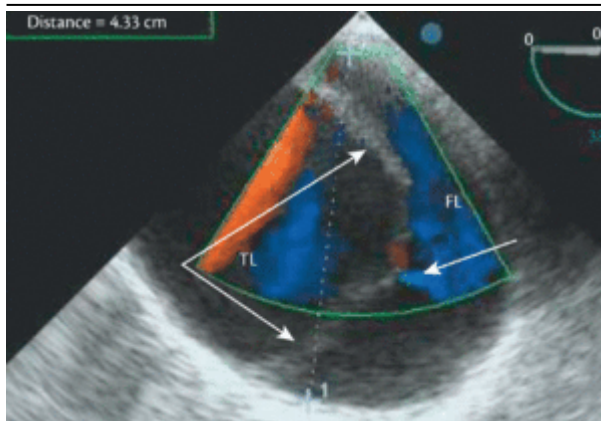




Figure 20.26

Dissection of the descending aorta; transoesophageal short-axis view. Note the dilated aorta (43 mm), intimal flap (white arrows), and a small communication (entry) between the true and false lumen (yellow arrow). FL, false lumen; TL, true lumen.

Table 20.5 Critical information to obtain by echocardiography in acute aortic dissection or intramural haematoma


1. Is there a dissection (i.e. the presence of a dissection membrane)? Does it involve the ascending aorta? Where is the entry tear?
2. Is there pericardial effusion?
3. Is there aortic regurgitation, and what is the mechanism (aortic dilatation, rupture of valvular apparatus, prolapse of dissection membrane through the aortic valve into the LV)? This is important to guide surgery as to whether aortic valve replacement is necessary
4. Does the dissection involve the coronary ostia, in particular the right ostium?
5. Is there intramural haematoma (may co-exist, or precede, classic aortic dissection), and does it involve the ascending aorta?

Dissection and intramural haematoma are life-threatening diseases which require emergency surgery if they affect the ascending aorta [19]. The questions that an echocardiographic examination should quickly and decisively address are summarized in  Additional online material. Importantly, non-classical forms of the acute aortic syndrome, such as an intramural haematoma or a penetrating ulcer, are often difficult to detect by echocardiography. CT or nuclear magnetic resonance should be liberally used in any cases of doubt. (See also  Chapter 61.)

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Systemic embolism

Echocardiography is the method of choice if a cardiovascular source of embolism is suspected [20]. TOE has a higher yield than TTE in identifying such sources and should be used if results may lead to a change in management. TOE is particularly valuable in assessing LA thrombi, endocarditic vegetations, tumours, and aortic atheromas. The presence of the following potential sources of embolism should be systematically sought:

- ◆ Atrial thrombi, in particular thrombi of the LA appendage, which frequently occur in AF (see  Chapter 56) and, in some instances (e.g. AS), also in sinus rhythm. However, in AF, which is by far the most frequent cardiac source of embolism, a negative TOE does not exclude that a LA or appendage thrombus was present before the embolism; therefore, independent of TOE findings, anticoagulation usually is indicated
- ◆ Infective endocarditis (see Endocarditis, acute valvular regurgitation, and prosthetic valve dysfunction section)
- ◆ LV thrombi from regions with severe wall motion abnormalities, e.g. apical aneurysm. TOE has no advantage in detecting LV thrombi, but left heart contrast echocardiography may be helpful
- ◆ Tumours, e.g. myxoma or fibroelastoma, best diagnosed or excluded by TOE
- ◆ Atrial septal defect or patent foramen ovale as the gate for paradoxical embolism
- ◆ Aortic atheromatosis with superimposed thrombi (TOE)

Personal perspective

Every intensive care patient with a cardiovascular disorder needs an echocardiography [21], and the sooner the better. Especially in the cardiac intensive care environment, there is simply no other imaging method even remotely as valuable as echocardiography. The challenge today, and in the future, is to ensure adequate training and to use the method, which is so conveniently available at the bedside, to the full extent of its diagnostic possibilities. This requires proper and dedicated training, which is not acquired 'on the fly'. There is no doubt that further echocardiographic refinements in haemodynamic assessment, diagnosis of myocardial ischaemia and viability, myocardial perfusion, coronary flow reserve, and others will arrive and further improve our diagnostic capabilities. The limiting factor,

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however, is often the 'human factor' that needs sufficient experience and expertise to harness the abundant imaging data for better patient care and outcomes.

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Additional online material



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Table 20.6 Normal and abnormal reference values of LV size

	Women				Men			
	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal	Reference range	Mildly abnormal	Moderately abnormal	Severely abnormal
LV dimension								
LV diastolic diameter	3.9-5.3	5.4-5.7	5.8-6.1	≥6.2	4.2-5.9	6.0-6.3	6.4-6.8	≥6.9
LV diastolic diameter/BSA (cm/m ²)	2.4-3.2	3.3-3.4	3.5-3.7	≥3.8	2.2-3.1	3.2-3.4	3.5-3.6	≥3.7
LV diastolic diameter/BSA (cm/m)	2.5-3.2	3.3-3.4	3.5-3.6	≥3.7	2.4-3.3	3.4-3.5	3.6-3.7	≥3.8
LV volume								
LV diastolic volume (ml)	56-104	105-117	118-130	≥131	67-155	156-178	179-201	≥201

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LV diastolic volume/BSA (ml/m ²)	35-75	76-86	87-96	≥97	35-75	76-86	87-96	≥97
LV systolic volume (ml)	19-49	50-59	60-69	≥70	22-58	59-70	71-82	≥83
LV systolic volume/BSA (ml/m ²)	12-30	31-36	37-42	≥43	12-30	31-36	37-42	≥43

From Lang R, Bierig M, Devereux R, Flachskampf FA et al Recommendations for Chamber Quantification. A report from the American Society of Echocardiography's Nomenclature and Standards Committee, the Task Force on Chamber Quantification, and the European Association of Echocardiography. Eur J Echocardiogr 2006;7:79-108).

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Introduction



Thoracic ultrasound (US) is a rapidly evolving method in assessing diseases of the lungs and pleura [1, 2]. It can be promptly applied at the bedside as a part of point of care testing (POCT) by a single clinician and minimal monitoring [2].

It is non-invasive, repeatable, relatively cheap, mobile, utilizes no radiation and has a short examination time that can augment physical examination with accurate and fast answers facilitating decision making in emergency conditions. [2, 3]. We will give an overview of the basic principles, technical aspects of thoracic US, cover clinical applications and conclude to current limitations.

Basic Principles - Equipment - Positioning



Basic Principles

In normal aerated lungs the ultrasound beam finds the lung air and no image is obtained, because US waves cannot be transmitted through air. The only visible structure is the pleura [1-4]. Thus the basic physical principle of lung US is that the less air in the lungs, the easier is the detection of lung abnormalities. When the air content decreases (e.g. pulmonary edema, pulmonary effusions) the acoustic mismatch needed to reflect the ultrasound beam is created, and some images appear. When the air content is further decreased, such as in lung consolidations, the acoustic window on the lung becomes completely open, and the lung may be directly visualized as a solid parenchyma, as the liver [5, 6].

Ultrasound Equipment

Appropriate probe depends on the location of the pathology and the patients' BMI [2]. Any modern 2-D black-and-white scanner unit is appropriate having a high frequency (7.5 - 10 MHz) linear transducer for surface structures (e.g. pleura), and a lower frequency (3.5 MHz) convex transducer for the evaluation of deeper lesions. Suitable modes are real-time B-mode to evaluate and scan organs in real-time, providing anatomic and functional information and time motion (M-mode) to image moving structures [1, 2, 3].

Patient Positioning and Scanning

The patient is scanned in the supine and sitting position, and if possible in lateral decubitus position [7, 8]. Patients are usually scanned using an intercostal approach from the 2nd to the 4th (left) or 5th (right) intercostal space of the anterior and the lateral chest using parasternal, mid-

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clavicular, anterior axillary, and mid-axillary lines as anatomical landmarks (Volpicelli's zones) [9, 10].

The transducer is moved longitudinally and transversely to visualize the lung surface through the intercostal spaces avoiding the acoustic restriction of the ribs. An abdominal window is used with a linear-convex probe for the sonographic imaging of caudal parts of the lungs, passing through the liver and diaphragm on the right and through the spleen and diaphragm on the left. A water-soluble US transmission gel is used to the skin as coupling medium and disinfection measures of the transducer and personnel hands are applied.

Lung signs, points, lines and patterns



Various US lung signs and lines can be recognized related to the normal lung and pleura. see Figure **20.27** ONLINE Localization of the pleura constitutes the basic anatomic landmark for the examination of the lung described as an intense hyper-echoic line that is created by the surface of visceral pleura against that of air-filled lung moving synchronously with respiration [4]. The thoracic wall is recognized by the characteristic posterior shadowing of the ribs. The intercostal muscles extend between the ribs, creating a useful intercostal monographic window. Due to the varying thickness of the subcutaneous tissues, the ribs constitute the most suitable anatomical landmark for recognition of the pleura, which can be found 0.5–1 cm in depth from the hyper echoic surface of the ribs. Lung signs, points, lines and patterns are shown in Table 20.9 (ONLINE) and include 'Lung sliding' (see Figure **20.28**, Video 1), 'A' lines (see Figure **20.29**), Seashore sign (see Figure **20.30**), 'B' lines (see Figure **20.31**), Lung point (see Figure **20.32**), Lung pulse, Air bronchogram (see Figure **20.33**) (all ONLINE) [6, 11–16].

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Figure 20.27

Normal anatomic pattern of lung US. Ribs with its acoustic shadow represent a well-defined landmark. Chest wall muscles appear as multiple layers of echogenicity. The outer two layers of intercostal muscles are usually visible in a rib space (between the rib shadows), through which can be seen the pleural space and the lung. Visceral pleura (Pv) is marked by the sharp bright, linear interface of the lung surface and together form the so-called 'pleural line' a roughly horizontal echogenic line 0.5 cm below the upper and lower ribs. Parietal pleura (Pp) is a less distinct, weakly echogenic line, the location of which is inferred by its relationship to the ribs and the visceral pleura. Pleural space (Ps) is a thin (< 1mm) hypoechoic line superficial to the Vp-air-filled lung interface, located within 1cm of depth from the rib interface.

Table 20.9 Normal and abnormal lung signs, points, lines and patterns

Signs, points, lines and patterns	Description
Normal	
<i>'Lung sliding' (gliding) sign</i>	Normal visceral and the parietal layers of the pleura sliding over each other illustrated as a hyperechoic line moving forward and backward during respiration, recognizable in B-mode in real-time imaging. Its presence excludes the presence of pneumothorax [11].
<i>Seashore or beach sign</i>	It is a dynamic monographic sign of normal lung seen in M-Mode having two parts: the superficial part is composed of multiple horizontal lines corresponding to motionless soft subcutaneous tissues ending to the pleural line. The other part corresponds to the motion

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	of normal lung giving a sandy 'granulous' pattern looking like the sand on the beach [12].
<i>'A' lines</i>	Constitute a basic artefact of the normally aerated lung. They are single or multiple horizontal hyperechoic repetition artefacts parallel to the pleural line. Each A line is separated by a distance equivalent to the thickness of the subcutaneous tissue between the ultrasound probe and the pleural interface. They result from the intense reflection between the surfaces of contact of soft tissue and air-filled lung. They are also found in pneumothorax when 'lung sliding' is abolished [11].
Pathologic	
<i>'B' lines or 'comet tail artefacts' lines or 'lung rocket'</i>	They are hyperechoic (white) lines vertical to the pleural line originating from the visceral pleura erasing A-lines on their passage. The number of vertical 'B' lines depends on the degree of lung aeration loss and their intensity increases with inspiratory movements. They are categorized depending on their average distance in lines B3 and B7, where the distance is ≤ 3 mm (ground glass pattern) and 7 mm (thickening of the interlobular septa, interstitial syndrome). Their presence excludes the existence of pneumothorax [6,13].
<i>Lung point</i>	The point that there is a clear change from one pattern (seashore sign) to the other (stratosphere or barcode sign) pathognomonic for pneumothorax [14].
<i>Lung pulse</i>	Small to and fro movement of the visceral on the parietal pleura on 2-D induced by the heartbeat implying an intact pleural interface. In M-Mode is seen as an intermittent vertical artefact synchronous with the ECG It is an early, dynamic and diagnostic sign of complete atelectasis [15].

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<i>Air bronchogram</i>	Hyperechoic punctiform or linear elements inside a tissue-like pattern of the pulmonary consolidation. The dynamic air bronchogram is characterized by presence of air inside the bronchi in dynamic movement within the tissue. Static air bronchogram is an indication of atelectasis and is characterized by the motionless entrapment of air inside the atelectatic region of lung [16].
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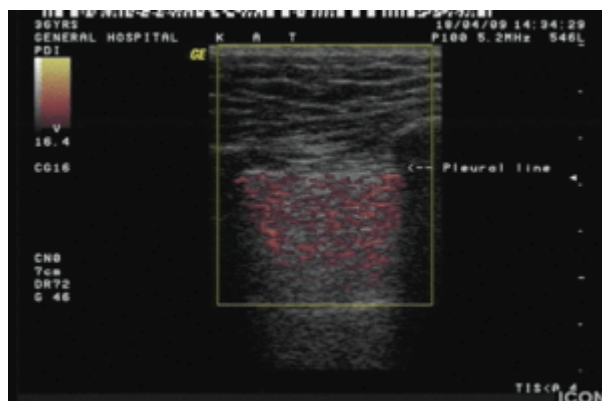


Figure 20.28
Lung sliding. Power Colour Doppler imaging. Normal visceral and the parietal layers of the pleura sliding over each other illustrated as a hyperechoic line moving forward and backward during respiration. The power colour Doppler signal along the pleural line reveals a breath-dependent movement of the lung.

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Figure 20.29

Horizontal artifacts - the A lines. Single or multiple horizontal hyperechoic repetition artefacts parallel to the pleural line. They result from the intense reflection between the surfaces of contact of soft tissue and air-filled lung.

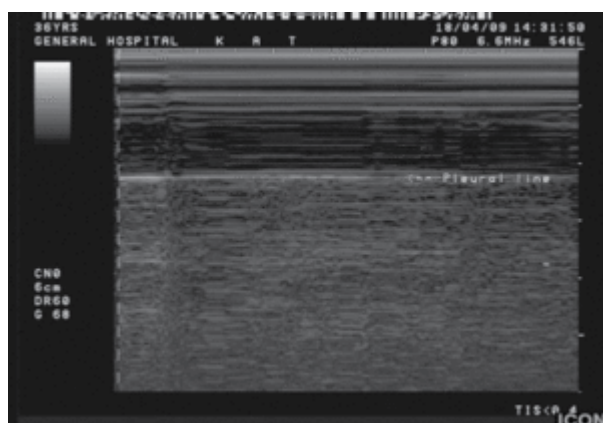


Figure 20.30

Time motion mode (M-mode). Seashore or beach sign. A dynamic monographic sign of normal lung seen in M-Mode having two parts: the superficial part is composed of multiple horizontal lines corresponding to motionless soft subcutaneous tissues ending to the pleural line. The other part corresponds to the motion of normal lung giving a sandy 'granulous' pattern looking like the sand on the beach.

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Figure 20.31

'B' lines or 'comet tail artefacts' (CTA) lines or 'lung rocket' hyperechoic (white). Lines vertical to the pleural line originating from the visceral pleura erasing A-lines on their passage: a) In normal lung, CTA are seen, b) In the case of pneumothorax CTA are not seen and reverberation artifacts generated by the pleural air form parallel horizontal echoic lines.

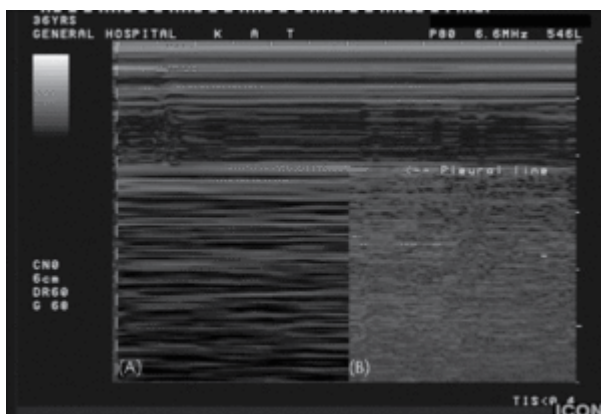


Figure 20.32

Lung point. Time motion mode (M-mode). a) In the case of pneumothorax (PTX) and absent lung sliding, only horizontal lines are visualised, the 'stratosphere sign', in contrast to b) the 'seashore sign' which is seen in normal lung. The point that there is a clear change from one pattern (seashore sign) to the other (stratosphere or barcode sign) pathognomonic for PTX.

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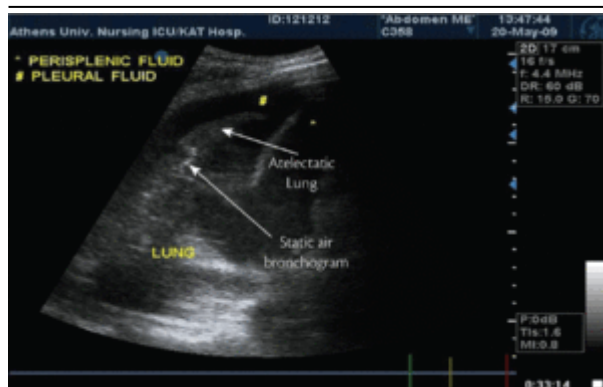


Figure 20.33

Air bronchogram Hyperechoic punctiform or linear elements inside a tissue-like pattern of the pulmonary consolidation. The dynamic air bronchogram is characterized by presence of air inside the bronchi in dynamic movement within the tissue. Static air bronchogram is an indication of atelectasis and is characterized by the motionless entrapment of air inside the atelectatic region of lung.

The most informative lung US sign for the cardiologist is the sum of B-lines denoting the extent of extravascular lung water (EVLW) [4]. In each scan, B-lines may range from zero to ten. A total score of severity is calculated ranging from 0 (≤ 5 B-lines, No sign), 1 (6 - 15 B-lines, Mild degree), 2 (16 - 30 B-lines, Moderate degree), 3 (> 30 B-lines, Severe degree) indicating the amount of EVLW [7].

Clinical applications, differential diagnosis and monitoring



Clinical applications of lung ultrasound in the ICU are shown in [Table 20.10 \(ONLINE\)](#).

Table 20.10 Clinical applications of lung ultrasound in the intensive care unit

1. Diagnosis of pulmonary consolidation
2. Diagnosis of atelectasis
3. Diagnosis of alveolar-interstitial syndrome
4. Differentiating between pulmonary oedema and ARDS
5. Differentiating between pulmonary oedema and COPD
6. Diagnosis of pulmonary embolism
7. Diagnosis of pneumothorax
8. Diagnosis and estimation of volume and nature of pleural effusion
9. Diagnostic and therapeutic ultrasound-guided thoracentesis

10. Differential diagnosis of acute dyspnea, acute respiratory failure
11. Evaluation of EVLW

Interstitial Syndrome

In this condition there is an increase in interstitial fluids characterized by the presence of B-lines [3, 4, 6]. B-lines cannot differentiate cardiogenic edema and acute respiratory distress syndrome (ARDS) but several findings are suggestive of ARDS: alterations of the pleura (subpleural consolidations); “spared areas”, defined as areas of normal sonographic lung appearance surrounded by areas of multiple B-lines; and large consolidations of various sizes [17, 18].

B-lines are present in patients with cardiogenic edema, but not in patients with exacerbation of chronic obstructive pulmonary disease (COPD) [19]. They are also reliable in predicting the cardiogenic origin of dyspnea, with accuracy comparable to natriuretic peptides [20]. In acute dyspnea, multiple B-lines associated to subpleural consolidations are highly suggestive of non-cardiogenic pulmonary edema.

B-lines are related to radiographic Kerley B-lines and lung water score on chest X-ray [9], to EVLW accumulation and pulmonary congestion [9, 20], and to the severity of diastolic dysfunction [21]. The number of B-lines increases with worsening New York Heart Association (NYHA) functional class [21] and allow monitoring of pulmonary congestion by observing its clearance [20, 22, 23].

B-line pattern facilitates the differential diagnosis of acute respiratory failure because respiratory, contrary to cardiogenic causes, show a non-interstitial pattern [9, 24–27].

Alveolar Syndrome

In this condition there is increase in alveolar fluids leading to lung consolidation even with complete air loss. When no aerated lung is interposed between consolidation and the probe, consolidation can be visualized on US as a hypoechoic region or a tissue-like echo pattern, which differs from the surrounding aerated pattern [28, 29].

Analysis of the shape, margin, distribution, vascularization, and some peculiar characteristics such as air and fluid bronchograms often allows for a differential diagnosis between different types of consolidation (i.e., pneumonia, infarctions in pulmonary embolism, contusions, and obstructive and compressive atelectasis) [17]. Ultrasound monitoring of

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these patients may show re-aeration of the consolidated area from alveolar pattern to the interstitial pattern and finally normal aeration [17, 30].

It should be mentioned that both alveolar and interstitial pattern may coexist according to different degrees of the affected lung aeration.

Pleural Syndrome

A conventional application of thoracic US is the diagnosis and quantification of pleural fluid see Figure 20.34 ONLINE [31-33] and subcutaneous emphysema see Figure 20.35, Video 2 ONLINE.

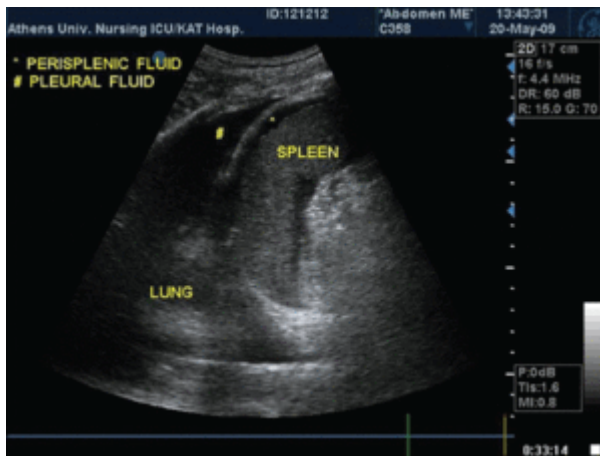


Figure 20.34
Pleural and perisplenic fluids.



Figure 20.35

Subcutaneous emphysema. E-lines artifacts. Reverberation echoes caused by subdermal gas collections (arrow) create a 'snow flurry' image. These reverberation artifacts, also referred as E-lines artifacts (arrowheads) are vertical laser-like lines that reach the edge of the screen usually not allowing the visualization of underlying structures but in contrast to the comet tail artifacts, they do not arise from the pleural line (pl) but from the chest wall. Similar artifacts can be generated by parietal shotgun pellets that along with parietal emphysema are two situations where PTX may be present.

Lung US is useful for the diagnosis of pneumothorax which is visible on US. The diagnostic hallmark of pneumothorax is the "lung point" with specificity of 100%, and sensitivity of about 65%. It corresponds to the point where visceral and parietal pleura regain contact with each other. M-mode performed at the lung point, shows a clear change from one pattern (seashore sign) to the other (stratosphere sign) indicating air in the pleural space [14]. (see Figure 20.32)

A simple algorithm by the International recommendations for POC lung US has been proposed for the exclusion of pneumothorax. Pneumothorax is present if "lung point" is present and there is no lung pulse sign [34]. However, absent lung sliding does not always mean pneumothorax. Pneumothorax is excluded if there is lung sliding, lung pulse or even a single B-line with negative predictive value of 100% [11, 35, 36].

Ultrasound guided interventions



Pleural Fluid Aspiration

US is superior to chest radiograph for the determination of the optimal site for thoracentesis having a success rate of up to 97% [37, 38]. US detect pleural fluid septations with greater sensitivity than CT scanning and also minimize the risk of visceral puncture [38, 39] and

pneumothorax following aspirations independently of the size of the effusion [39].

Closed Intercostal Tube Drainage

US is ideal and strongly recommended for identifying the optimal site for safe and effective intercostal fluid drainage [39, 40]. This is particularly relevant in patients with loculated parapneumonic effusions, where thickened parietal pleura, adhesions or loculations often complicate insertion [37]. Depending on operator experience, US may also guide further decisions regarding the need for intrapleural fibrinolytics, thoracoscopy or for surgical intervention [41].

Integrated cardio-pulmonary ultrasound evaluation and limitations



Lung US is much easier than echocardiography and minimal teaching and image recognition skill sessions are needed [42]. The learning curve for B-lines evaluation and grading is very short [43]. The addition of lung US to echocardiography provides insights on the eventual pulmonary involvement and requires only a few minutes.

Presence of multiple, diffuse, bilateral B-lines associated to left ventricular systolic and/or diastolic dysfunction or valvular heart disease is highly indicative of cardiogenic pulmonary congestion. Presence of multiple, diffuse, bilateral B-lines, associated to a normal heart, indicates a non-cardiac cause of pulmonary edema, such as ARDS, pneumonias, or pulmonary fibrosis.

It is important to distinguish the multiple, diffuse, bilateral B-lines pattern from focal multiple B-lines that can be present in normal lungs or may be seen around many pathologic conditions, as lobar pneumonia, pulmonary contusion, pulmonary infarction, pleural disease, and neoplasia.

Lung US limitations are essentially patient and operator dependent. Obese patients are frequently difficult to examine because of the thickness of their ribcage and soft tissues. The presence of subcutaneous emphysema or large thoracic dressings is a significant limitation. The main limitation of B-lines is the lack of specificity for cardiogenic pulmonary edema. We should have in mind that all imaging data should be evaluated within the clinical context and integrated with patient's history, clinical presentation and other laboratory data.

Conclusions



Lung US is a simple, fast, low-cost, reliable and repeatable examination for the evaluation of respiratory status contributing to the bedside diagnosis of lung and pleural disorders. Also, estimates EVLW through B-lines assessment representing a new, helpful tool for the cardiologist, for the management of HF patients. A combination of lung US with echocardiography may help to differentiate the main causes of acute dyspnea and life-threatening conditions in critically ill hypoxic patients.

Further reading

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