Chapter 1
Echocardiographic Assessment of Acute Chest Pain in the CCU

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Abstract The field of critical care cardiology has evolved considerably over the past few decades. Cardiac units in the 1970s and the 1980s were practically coronary care units, where the units most frequently were populated by patients with acute and often uncomplicated myocardial infarction or unstable angina. Detection and rapid treatment of arrhythmias were the primary goals of therapy. Contemporary cardiac care units (CCU) have transformed into Cardiac intensive care units (CICU) where the focus has since shifted towards the management of patients with multisystem diseases, advanced hemodynamics compromise, complex ventricular arrhythmias, and established or incipient multi-organ failure. The two most common clinical scenarios leading to acute chest pain syndrome in CCU patients are acute coronary syndrome and acute aortic syndrome.

Keywords Chest pain · Aortic dissection · Acute coronary syndrome · Myocardial infarction

Introduction

The field of critical care cardiology has evolved considerably over the past few decades. Cardiac units in the 1970s and the 1980s were practically coronary care units, where the units most frequently were populated by patients with acute and often uncomplicated myocardial infarction or unstable angina. Detection and rapid treatment of arrhythmias were the primary goals of therapy. Contemporary cardiac care units (CCU) have transformed into cardiac intensive care units (CICU) where the focus has since shifted towards the management of patients with multisystem diseases, advanced hemodynamics compromise, complex ventricular arrhythmias, and established or incipient multi-organ failure. In addition, at many institutions,

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E. Herzog, E. Argulian (eds.), *Echocardiography in the CCU*,
https://doi.org/10.1007/978-3-319-90278-4_1

CCUs manage an increasing number of patients undergoing advanced therapies such as therapeutic hypothermia, transcatheter valve procedures, or ventricular assist device therapies.

Advances in echocardiography paralleled the progress made in critical care cardiology. The initial use of echocardiography was to detect pericardial effusion and cardiac tumors. However, the current applications of various forms of echocardiography include an extended list of pathological and therapeutic indications. Currently, echocardiography is fundamental to the management of patients with acute cardiovascular diseases. It has been shown that timely performed echocardiography can lead to a change in therapy in up to 80% of patients with an acute cardiovascular condition [1].

Different ultrasound modalities, from basic point-of-care ultrasound to advanced echocardiographic imaging, can be used for evaluation of patients with an acute cardiovascular condition (Table 1.1). Transthoracic echocardiography (TTE) is usually the frontline imaging modality. Transesophageal echocardiography (TEE) commonly follows a nondiagnostic TTE. However, in certain clinical scenarios, such as acute aortic syndrome, acute valvular regurgitation, acute prosthetic value dysfunction, atrial fibrillation or atrial flutter, and stroke, TEE could be performed first. Contrast echocardiography allows improved visualization of the endocardium and it has become an integral part of echocardiography in the modern critical care units. Pocket-sized imaging devices are occasionally used as a fast initial screening in an emergency setting, as well as an extension of physical examination in the intensive care units [1].

The two most common clinical scenarios leading to acute chest pain syndrome are acute coronary syndrome and acute aortic syndrome.

Table 1.1 Ultrasound modalities in evaluation of patients with suspected cardiac causes of acute cardiovascular conditions

Point-of-care cardiac ultrasound
Comprehensive transthoracic echocardiogram
Limited/focused transthoracic echocardiogram (intracardiac device positioning, etc.)
Transesophageal echocardiogram
Contrast echocardiography
Stress echocardiogram including dobutamine echocardiography
Speckle-tracking echocardiography for assessment of myocardial mechanics
3D echocardiography
Lung ultrasound
Vascular ultrasound for intravenous access

Echocardiography in Acute Coronary Syndrome

The last few decades have witnessed remarkable progress in the understanding of the pathophysiology of acute coronary syndrome (ACS). ACS incorporates a spectrum of clinical entities, ranging from unstable angina and non-ST-elevation ACS to ST-elevation myocardial infarction. Developments in the field of echocardiography have paralleled the progress made in ACS.

Assessment of Regional Systolic Function in Acute Coronary Syndrome

Occlusion of an epicardial coronary artery at the time of acute coronary syndrome leads to a loss of contractile function in the myocardial segments subtended by that vessel. The magnitude and duration of wall motion abnormalities depend on the severity, extent, and duration of the coronary occlusion.

In unstable angina (UA) or in non-ST-elevation ACS, left and right ventricular wall motion may be normal unless TTE is performed during an episode of chest pain.

ST-elevation myocardial infarction (STEMI) often results from an acute occlusion of a major coronary vessel. If the total cessation of coronary flow lasts for more than 3–6 h, myocardial necrosis will occur and the myocardium in the affected segments will be replaced with a fibrous tissue over the subsequent weeks [2].

The magnitude of regional contractile loss in acute coronary syndrome is usually assessed semiquantitatively. It is usually interpreted clinically as follows [2]:

1. Interpretation of wall motion abnormalities as seen in Table 1.2
2. Extent and location of affected segments
3. Suspected coronary artery distribution (left anterior descending artery vs. right coronary artery vs. left circumflex artery)

Table 1.2 Left ventricular wall motion scoring

	Score
Normal or hyperkinetic	1
Hypokinetic (reduced thickening)	2
Akinetic (absent or negligible thickening)	3
Dyskinetic (systolic thinning or stretching, aneurysmal)	4
Wall motion score index = Sum of individual segment scores/number of evaluated segments	

Assessment of Global Systolic Function in Acute Coronary Syndrome

Global left ventricular systolic function in acute coronary syndrome is assessed by both wall motion scoring and left ventricular ejection fraction.

Wall Motion Scoring

Wall motion scoring analysis assigns a numeric value to the degree of contractile dysfunction in each segment. The scoring criteria endorsed by American Society of echocardiography (ASE) are seen in Table 1.2.

Once all segments are assigned individual scores, a total score is calculated as a sum of individual scores. A wall motion score index (WMSI) is then calculated as a ratio between the total score over the number of evaluated segments. The WMSI is a dimensionless index. A 17-segment model is commonly used to allow for standardized communication within echocardiography and with other imaging modalities [3]. For a fully visualized normal ventricle the total score is 17 (all segments have normal contractility). However, ASE does not recommend to score the true apical segment of the 17 segments. Since all 17 segments are evaluated, the wall score index of a normal heart is 17/17 = 1. For abnormal ventricles, the higher the WMSI, the more significant the wall motion abnormality.

Figures 1.1 and 1.2 depict echocardiographic views of the left ventricle, demonstrating the left ventricular wall segments and their typical corresponding coronary distributions [4]. The utilization of image-enhancing agents (echocardiographic contrast microbubbles) has improved the detection of wall motion abnormalities, and therefore the confidence in delineation of the affected segments.

Assessment of Left Ventricular Ejection Fraction

Left ventricular ejection fraction (LVEF) has been consistently shown to be one of the most powerful predictors of mortality and morbidity in patients with heart disease [5]. LVEF is the single most powerful predictor of mortality and the risk for developing life-threatening ventricular arrhythmias after myocardial infarction [6]. Furthermore, once the acute coronary syndrome resolves, the residual LVEF is important for treatment options as LVEF cutoff values are built into recommendations for both medical and implantable device therapies. By definition, LVEF is the percentage of the end-diastolic volume that is ejected with each systole as the stroke volume. Thus, to calculate the LVEF one needs to estimate the end-systolic and end-diastolic volumes of the left ventricle.

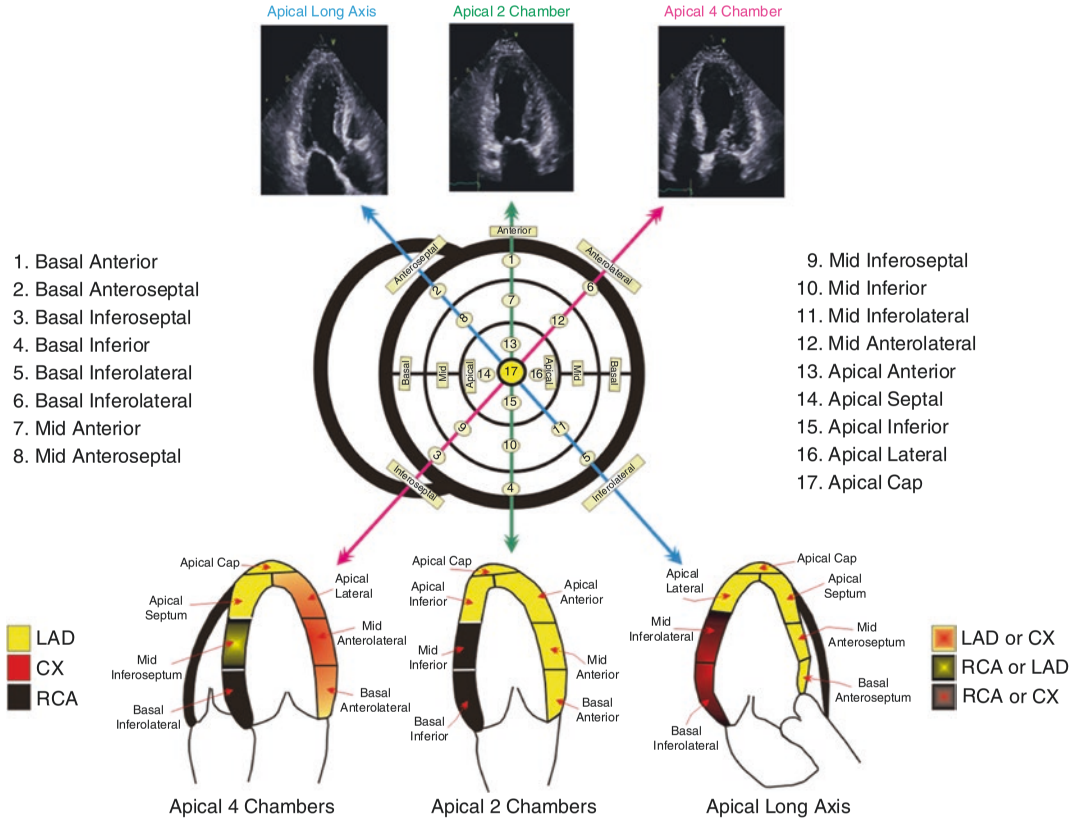

Fig. 1.1 Apical four-chamber (A4C), apical two-chamber (A2C), and apical long-axis (ALX) views in relation to the bull's-eye display of the LV segments (center). Top panels show echocardiographic images and bottom panels depict the LV wall segments visualized in each view

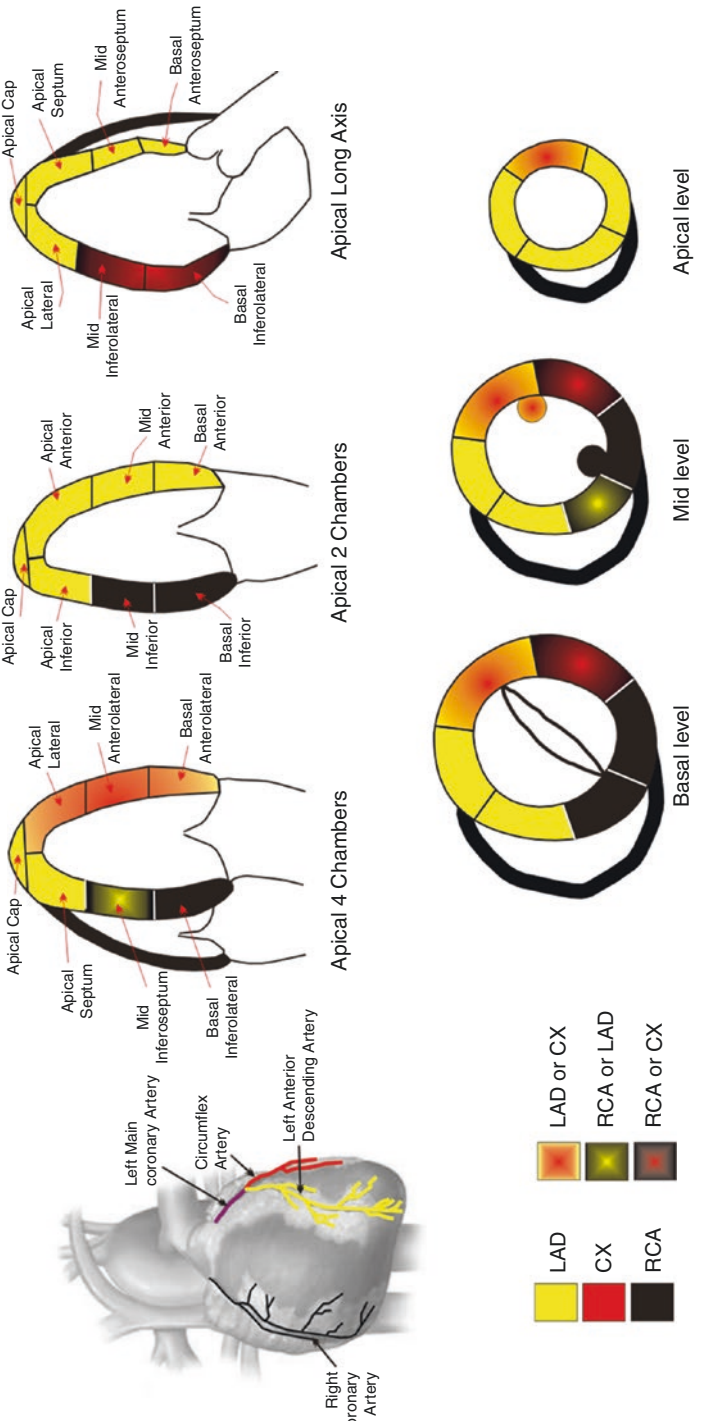

Fig. 1.2 Distributions of the right coronary artery (RCA), left anterior descending artery (LAD), and circumflex coronary artery (CX)

The Ischemic Cascade

The ischemic cascade refers to a sequence of events that occur in the myocardium after the onset of ischemia [7]. Myocardial perfusion is determined by coronary blood flow and myocardial oxygen consumption. Any imbalance in this supply and demand relationship results in myocardial ischemia. The mechanical, electrographic, and clinical events that follow the development of ischemia were formally described in 1985 by Hauser et al. [8], and were later termed the "ischemic cascade" [9]. Classically the observable changes occur sequentially (Fig. 1.3) starting with perfusion abnormalities leading to abnormalities in wall function, then ischemic electrocardiogram (ECG) changes, and finally angina. Echocardiography has the ability to detect these pathophysiologic changes in the myocardium at the early stages and therefore is more sensitive than history, physical examination, and ECG for identification of myocardial ischemia.

Echocardiography for the diagnosis of suspected acute ischemia is most helpful in subjects with a high clinical index of suspicion but with nondiagnostic ECG because it allows real-time assessment of myocardial function.

Of note, troponin elevation with chest pain may not be related to an acute coronary syndrome, but rather be secondary to other cardiovascular pathologies such as valvular disease (e.g., severe aortic stenosis or regurgitation) which can induce ischemia by causing LV wall stress; acute heart failure due to systolic and/or diastolic dysfunction; or acute pulmonary embolism with right ventricular strain. Echocardiography is a valuable tool to detect and quantify these abnormalities, regardless of whether the patient is having an acute coronary syndrome.

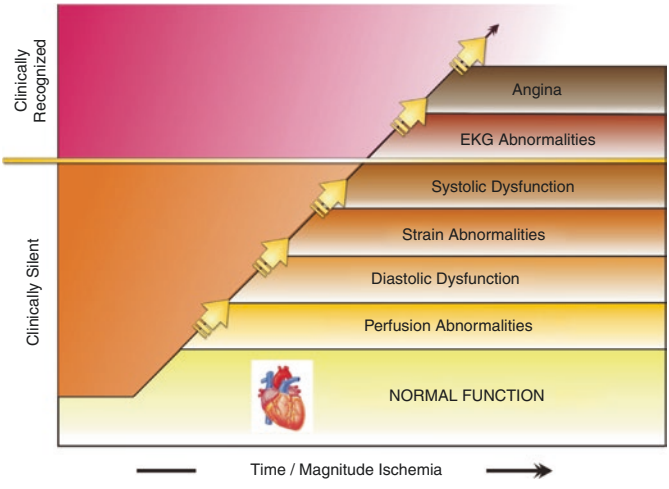

Fig. 1.3 The ischemic cascade. Myocardial dysfunction occurs in a predictable sequence that is detectable prior to clinical symptoms

Pathway for the Use of Echocardiography in Patients with Cardiac Chest Pain (Fig. 1.4)

In the United States it is estimated that about eight million people present each year to the emergency departments with complaints of acute chest pain. If the chest pain is clearly not of a cardiac origin, the patients should be treated accordingly and transthoracic echocardiography (TTE) is seldom needed. If the cause of chest pain appears to be of a cardiovascular etiology then the initial evaluation should include history, physical examination, 12-lead ECG, chest X-ray (CXR), and laboratory tests including cardiac biomarkers (CPK and troponin).

As outlined earlier, ACS incorporates a spectrum of clinical entities ranging from unstable angina and non-STE ACS to STEMI.

The AHA/ACC guidelines in 2014 combined the term unstable angina and the non-STEMI to one group defined as non-STE ACS [10]. The European Society of Cardiology (ESC) guidelines in 2015 still recommend keeping both entities separate [11]. Out of all patients presenting with chest pain only the minority actually have ACS. Among ACS patients, most patients have non-STE ACS.

TTE is an integral part of the management of patients with ACS as seen in Fig. 1.4.

In patients with STEMI, it is now well accepted that patients should preferably be referred to a site that can perform primary percutaneous coronary intervention (PCI). TTE should not be done immediately as it may delay the transfer to the cardiac catheterization laboratory for PCI. TTE should be performed *only after* the intervention is completed.

Patients with non-STE ACS should be treated per current guidelines [10, 11] and *TTE should be performed as soon as possible* to exclude any structural heart disease.

Currently, the use of contrast echo is a standard of care for patients in whom two of more consecutive left ventricle (LV) segments are not well visualized, or when there is a need to rule out an apical thrombus.

For patients whose chest pain is of a cardiovascular etiology but not due to ACS, TTE should be performed *immediately* if the symptoms persist and the patient has active ongoing chest pain.

If the symptoms of chest pain had subsided we recommend to continue the serial evaluation for ACS [10, 11] and perform *TTE as needed*.

If TTE shows an evidence of new wall motion abnormalities, a diagnosis of coronary artery disease is likely. Similarly, contrast echocardiography is necessary when the endocardial definition is poor.

For patients in whom a diagnosis of ACS is ruled out, and they do not have evidence of new wall motion abnormalities, other cardiac etiologies should be considered. These include acute aortic syndrome, acute pericardial disease, pulmonary embolism, aortic stenosis, or hypertrophic cardiomyopathy.

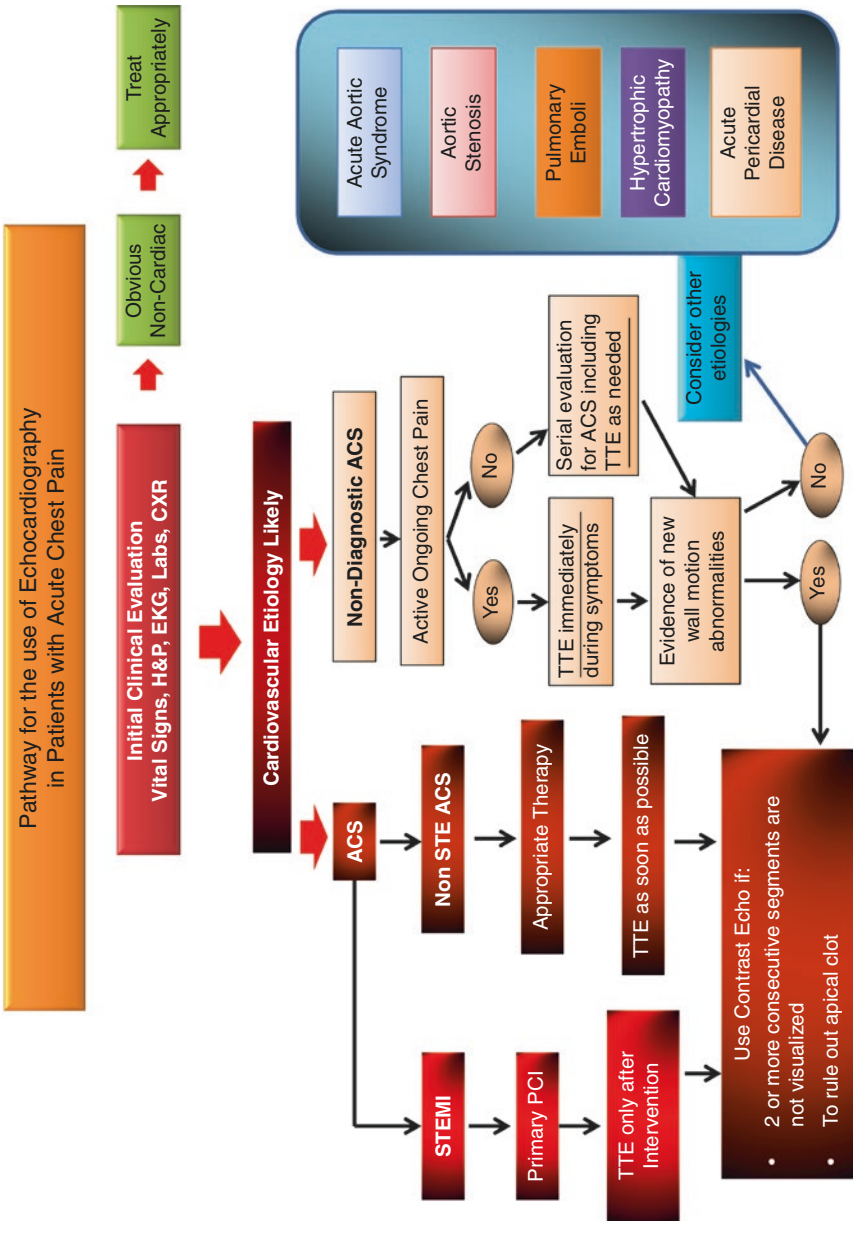

Fig. 1.4 Pathway for the use of echocardiography in patients with acute chest pain

Echocardiography in the Diagnosis and Management of Mechanical Complications of Myocardial Infarction

Mechanical complications of acute myocardial infarction (AMI) result in some of the deadliest cardiovascular outcomes. It is difficult to assess the true incidence of these complications as both clinical and autopsy series differ considerably. It is important to realize that these catastrophic events can occur within minutes to hours after the inciting event, or even days to weeks later. Mechanical complications of acute myocardial infarctions can be divided into two major categories: acute-phase complications and chronic-phase complications. Table 1.3 outlines acute-phase complications and the chronic-phase complications of AMI.

Echocardiography is the key imaging modality in the assessment of these complications.

Left Ventricular Free Wall Rupture

Left ventricular free wall rupture (LVFWR) is an almost universally fatal complication of AMI. Despite advances in the reduction of both mortality and morbidity from AMI, death related to LVFWR remains high.

Myocardial rupture more often involves the left ventricle rather than the right ventricle, and rarely involves the atria [12]. The infarct commonly affects the anterior and lateral walls of the left ventricle near the junction of the infarcted and normal myocardium.

Figure 1.5 demonstrates a TTE of a patient with inferolateral free wall rapture.

Figure 1.6 demonstrates a case of LV free wall rupture causing pericardial tamponade with thrombus formation in the pericardial space.

Ventricular Septal Rupture

The rupture of the interventricular septum is another deadly complication of AMI. Rapid diagnosis, aggressive medical therapy, and prompt surgical intervention are essential to increase the chances of survival.

In the pre-reperfusion era, rupture of the interventricular septum was estimated to occur in 1–2% of patients with acute myocardial infarction and to account for

Table 1.3 Acute-phase vs. chronic-phase complications in acute myocardial infarction

Acute phase	Chronic phase
Left ventricular free wall rupture	True ventricular aneurysm
Ventricular septal rupture	Ventricular pseudoaneurysm
Right ventricular infarction	Left ventricular thrombus
Acute mitral regurgitation	

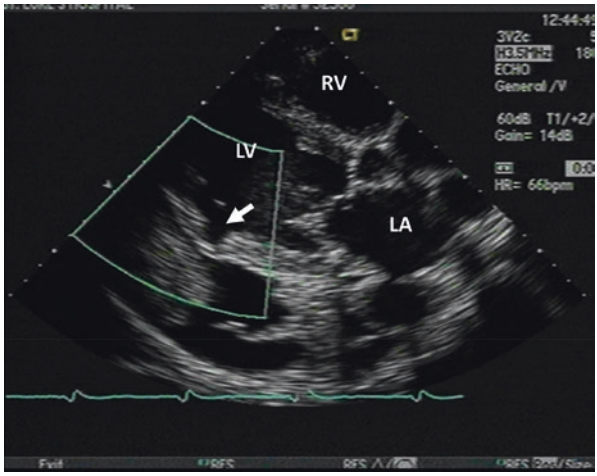

Fig. 1.5 Parasternal long-axis view of a transthoracic echocardiogram demonstrating a inferolateral free wall rupture (*arrow*); *RV* right ventricle, *LV* left ventricle, *LA* left atrium

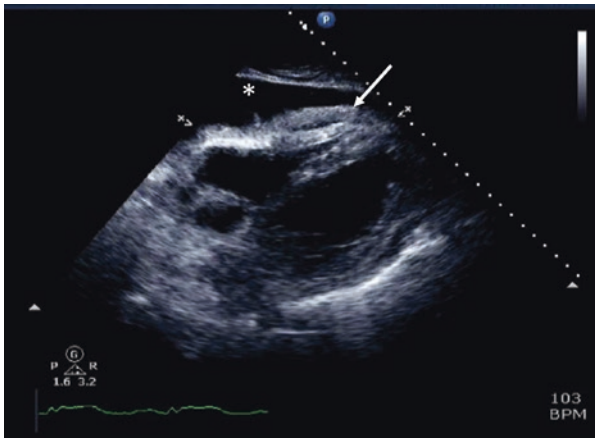

Fig. 1.6 Subcostal view showing pericardial effusion (*asterisk*) with a thrombus in the pericardial space (*arrow*). This was caused by a ventricular free wall rupture after an acute myocardial infarction and the patient developed cardiac tamponade physiology

approximately 5% of deaths in this setting. This complication typically occurs within the first week after infarction, with a mean time from symptom onset of 3–5 days. The classic risk factors for septal rupture in the pre-reperfusion era included hypertension, advanced age, female sex, and absence of a history of myocardial infarction or angina [13]. Prognosis for ventricular septal rupture in the pre-reperfusion era was very poor. With the advent of thrombolysis and percutaneous intervention, incidence and outcomes have changed.

In the current era, early reperfusion therapy may prevent the extensive myocardial necrosis that is associated with ventricular rupture. Patients generally have a mean time of 1 day from infarction to development of a ventricular septal rupture (VSR).

The ventricular septum is a very vascular structure. The rarity of septal rupture and the variable infarct location relate to the fact that the interventricular septum has a dual

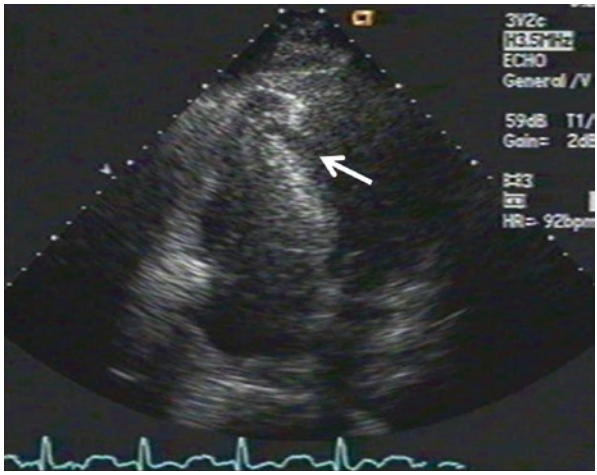

Fig. 1.7 Apical four-chamber view of a transthoracic echocardiogram demonstrating a ventricular septal rupture in the distal portion of the interventricular septum (*arrow*)

blood supply. The anterior two-thirds are supplied by the left anterior descending coronary artery and its branches. The posterior one-third is supplied by branches of the posterior descending artery, which arises from the right coronary or the left circumflex artery, depending on the dominance of the circulation [14]. Similar to the free wall rupture, interventricular septal rupture occurs most frequently with a first myocardial infarction when there is less likely to be collateral blood flow. In this setting, with an abrupt cessation of blood flow in the infarct-related artery, no collateral flow exists to support the infarcted zone, thereby making the septum prone to rupture.

Figure 1.7 demonstrates an apical four-chamber view of a transthoracic echocardiogram demonstrating a ventricular septal rupture in the distal portion of the interventricular septum (arrow).

Figure 1.8 demonstrates a case of apical ventricular septal rupture in a patient with an acute LAD territory MI.

Right Ventricular Infarction

Right ventricular infarction (RVI) complicates up to half of inferior wall left ventricular infarctions [15].

Coronary blood flow to the right ventricle is unique in that it occurs in both systole and diastole. In the majority of patients the right coronary artery (RCA) supplies the right ventricle through the acute marginal branches, as well as the inferior wall and posterior interventricular septum through the posterior descending artery in right dominant systems. Typically, RVI occurs when there is occlusion of the RCA proximal to the acute marginal branches. It can also occur with an occlusion of the left circumflex in patients who have a left dominant system. Although quite rare, occlu-

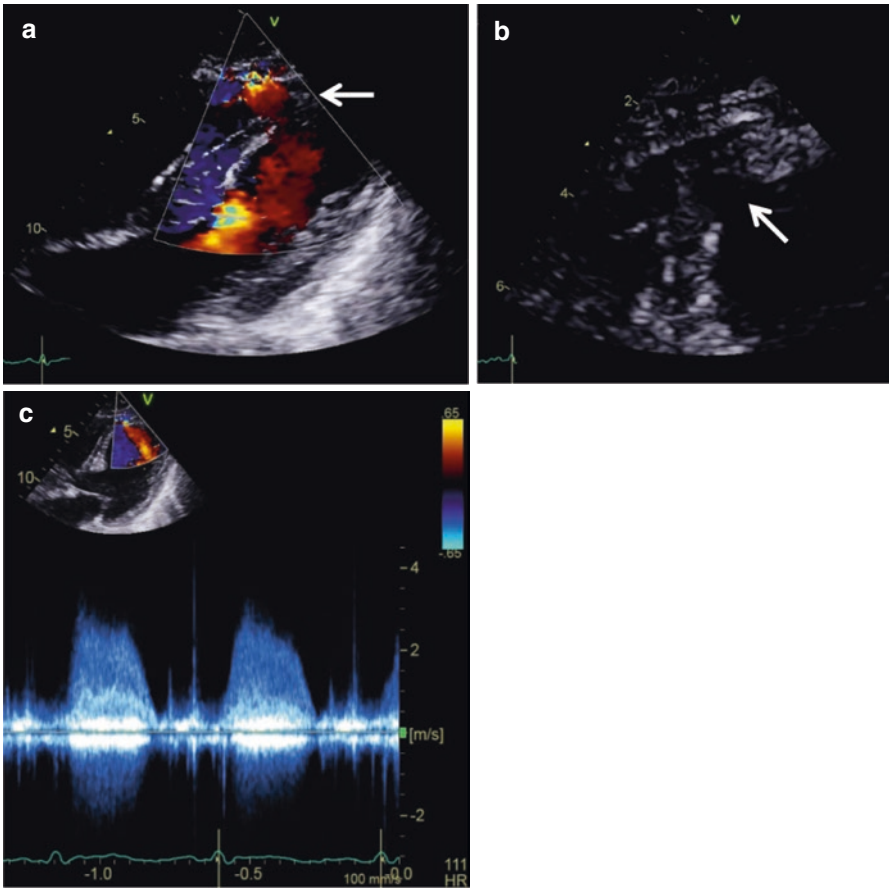

Fig. 1.8 Subcostal view showing apical ventricular septal rupture with flow from the left ventricle to right ventricle: (**a**) color Doppler flow, (**b**) VSR defect seen with 2D imaging, (**c**) continuous-wave Doppler demonstrating gradient across VSR

sion of the left anterior descending artery may result in infarction of the anterior right ventricle [15].

Acute underperfusion of the right ventricular free wall and adjacent interventricular septum leads to a stunned and noncompliant right ventricle. Loss of the right ventricular contractility results in a serious deficit in the left ventricular preload with a resultant drop in cardiac output, thereby causing systemic hypotension.

Acute right ventricular dilatation causes a leftward shift of the interventricular septum, increasing left ventricular end-diastolic pressure with a resultant decrease in left ventricular compliance and cardiac output (Fig. 1.9).

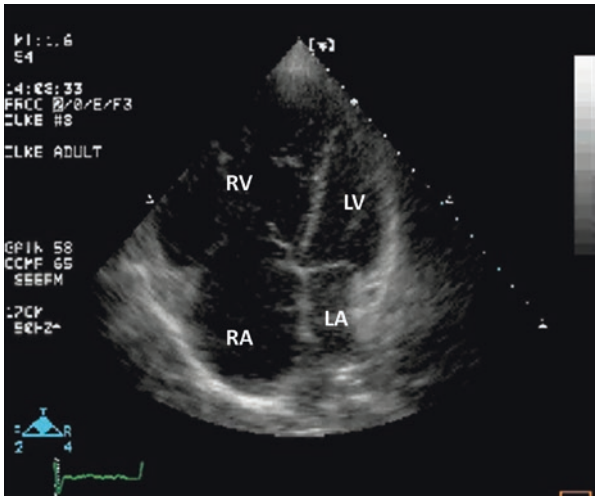

Fig. 1.9 Apical four-chamber view of a transthoracic echocardiogram demonstrating a dilated and severely hypokinetic right ventricle compressing the left ventricle. *RV* right ventricle, *RA* right atrium, *LV* left ventricle, *LA* left atrium

Acute Mitral Regurgitation

Acute mitral regurgitation (MR) is another major fatal mechanical complication of acute myocardial infarction. The three main causes of MR in the setting of an AMI include ischemic papillary muscle dysfunction, papillary muscle or chordal rupture, and left ventricular dilatation [16].

Figure 1.10 demonstrates an apical four-chamber view of a transthoracic echocardiogram showing mitral regurgitation as a result of an inferior wall myocardial infarction.

Figure 1.11 shows a ruptured papillary muscle resulting in severe mitral regurgitation, requiring urgent surgical intervention.

True Ventricular Aneurysm

In contrast to the acute complications of myocardial infarction, the chronic complications of AMI are not immediately life threatening. They have a different presentation and require different treatment. A true left ventricular aneurysm (LVA) is a common chronic complication of AMI that is important to diagnose.

Approximately 70–85% of LVAs are located in the anterior or apical walls, and in most cases are due to complete occlusion of the LAD coronary artery in the absence of collateralization. However, 10–15% of cases involve the inferior and basal walls due to right coronary artery occlusion. A rare finding is a lateral LVA, which is the result of an occluded left circumflex coronary artery.

LVA has been described as a well-delineated and distinct break ("hinge point") in the LV geometry and contour present in both systole and diastole. The pathognomonic feature is a wide mouth that enables communication with the aneurysmal cavity (Figs. 1.12 and 1.13).

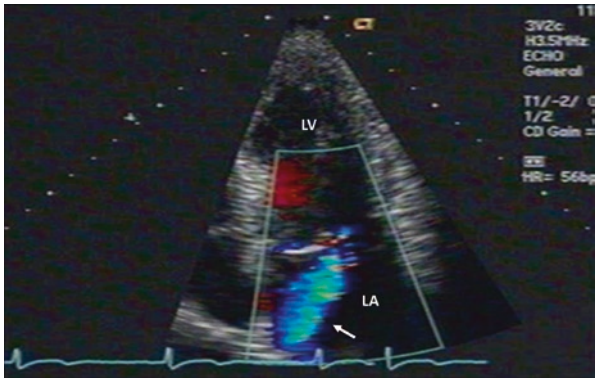

Fig. 1.10 Apical four-chamber view of a transthoracic echocardiogram showing mitral regurgitation as a result of an inferior wall myocardial infarction (arrow)

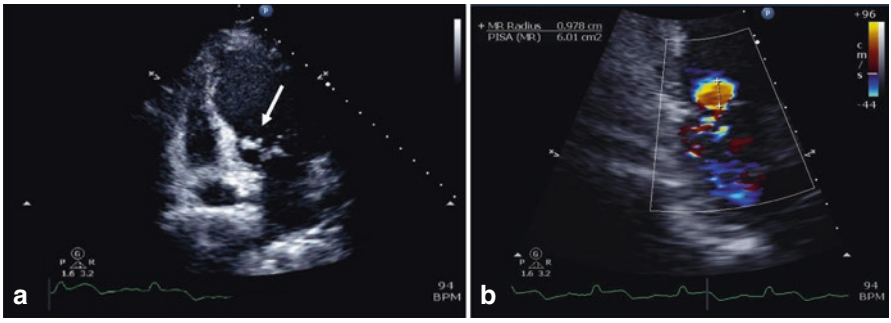

Fig. 1.11 Papillary muscle rupture. (**a**) Apical four-chamber view showing ruptured head of the papillary muscle (arrow). (**b**) Apical three-chamber view with color Doppler demonstrating severe mitral regurgitation with large PISA radius. *PISA* Proximal isovelocity surface area

Although the size of an aneurysm varies widely, most are within 1–8 cm in diameter. The wall of the aneurysm typically consists of a hybrid of necrotic myocardium and white fibrous scar tissue. This wall is extremely thin and delicate and may calcify over an extended period of time. Of note, it is imperative to distinguish between a LVA and a pseudoaneurysm, which is characterized by a narrow neck and has a distinct "shelf-like" opening.

Left Ventricular Pseudoaneurysm

A left ventricular pseudoaneurysm (LVPA) or false aneurysm is a less common form of a ventricular aneurysm and presents in <1% of patients post-myocardial infarction.

An LVPA forms when cardiac rupture is contained by adherent pericardium or scar tissue. Unlike a true aneurysm, a LVPA is devoid of endocardium or myocardium and since these aneurysms are prone to rupture a quick and accurate diagnosis is of extreme importance. Unlike a true LVA whereby the walls consist of dense fibrous

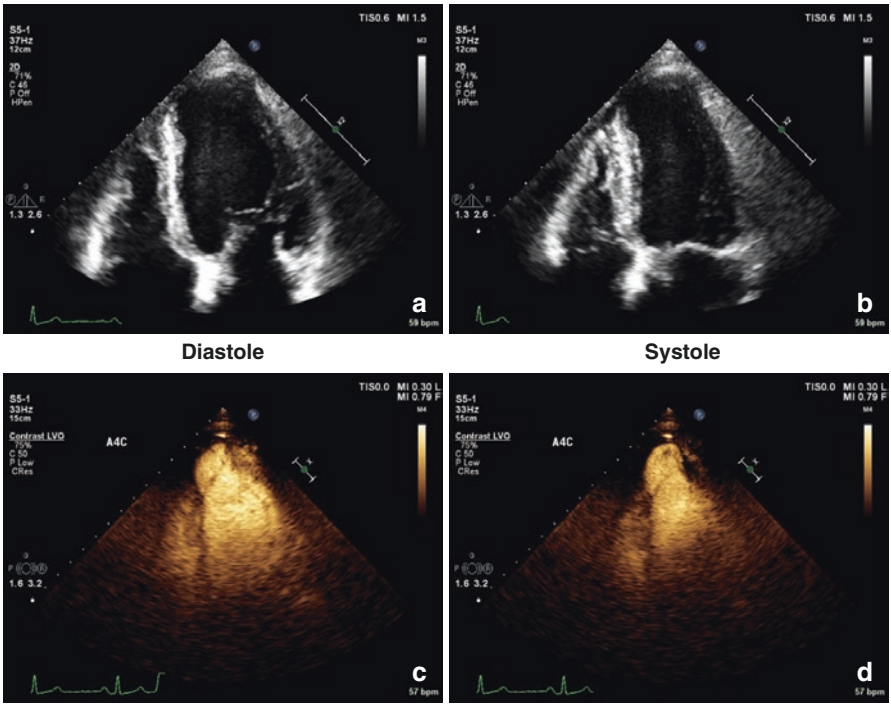

Fig. 1.12 (**a**–**d**) Apical four-chamber view in diastole (**a**) and systole (**b**) and with echo contrast, respectively (**c**, **d**), showing a large apical AMI with a large apical aneurysm with no clot

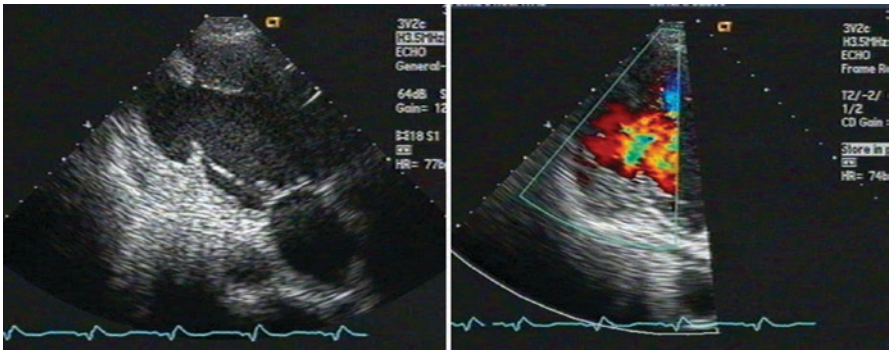

Fig. 1.13 Apical three-chamber view (left) obtained from transthoracic echocardiogram revealing a large left ventricular aneurysm, enhanced with color flow Doppler (right) establishing an area of communication between the normal left ventricle and the aneurysmal portion

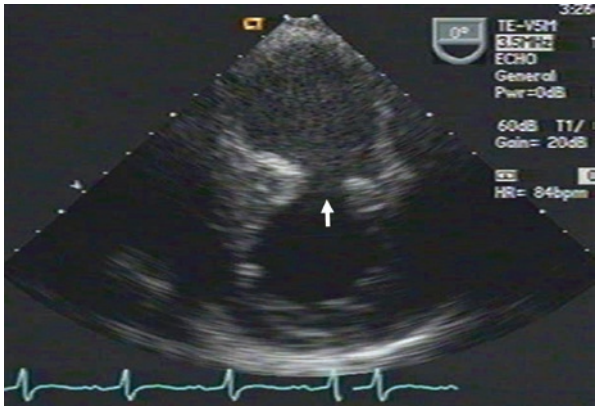

Fig. 1.14 Short-axis view of the left ventricle (lower cavity) during transesophageal echocardiography. Large pseudoaneurysm is seen (higher cavity). Notice the narrow "bottleneck" opening (arrow)

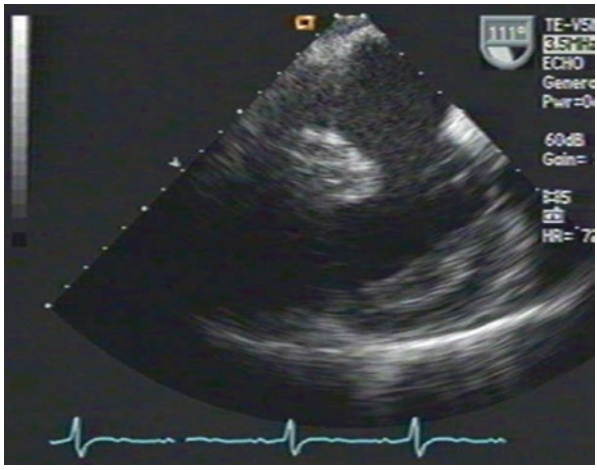

Fig. 1.15 Short-axis view from transesophageal echocardiogram depicting a large pseudoaneurysm (higher cavity). Notice the narrow "shelf-like" opening into the aneurysmal cavity

tissue with excellent tensile strength, the wall of a LVPA is comprised of thrombus and varying portions of the epicardium and parietal pericardium. It is the result of an AMI (typically an inferior or posterolateral wall AMI) with myocardial rupture and hemorrhage into the pericardial space, becoming progressively compressive. Cardiac tamponade occurs, thereby preventing further hemorrhage into the pericardium. Over time, thrombus organizes with overall poor structural integrity, and thus is prone to inevitable rupture, which can be a fatal event [17].

Echocardiography can usually distinguish a pseudoaneurysm from a true aneurysm by the appearance of a connection between the aneurysm and ventricular cavity. LVPA have a narrow neck, typically less than 40% of the maximal aneurysm diameter, which causes an abrupt interruption in the ventricular wall contour (Figs. 1.14 and 1.15). In contrast, true aneurysms are nearly as wide at the neck as they are at the apex.

Left Ventricular Thrombus

A mural LV thrombus is a common sequela of an AMI and most commonly develops in the presence of a large infarction. Thrombi are prone to originate in regions of stasis; they are most commonly noted to occur in the apex but may also occur in lateral and inferior aneurysms. Characteristically, a thrombus has a nonhomogeneous echo density with a margin distinct from the underlying wall, which is akinetic to dyskinetic (Figs. 1.16 and 1.17). A thrombus is more likely to occur following an AMI in the LAD artery distribution (up to 33%) than in regions supplied by the right coronary or circumflex coronary arteries (<1%).

Echocardiography Acute Aortic Syndrome

Acute aortic syndrome (AAS) represents a spectrum of life-threatening conditions with similar clinical presentation and the need for urgent management. It includes classic acute aortic dissection (CAAD), intramural hematoma (IMH), and penetrating aortic ulcer (PAU) [18, 19].

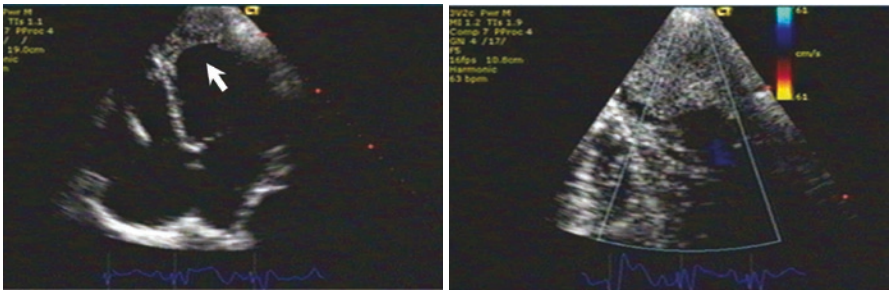

Fig. 1.16 Apical four-chamber views of a transthoracic echocardiogram from a 74-year-old patient 3 months following AMI (top). Notice the large protruding apical thrombus (*arrow*). Color flow imaging (bottom) may be used to demonstrate abnormal flow patterns

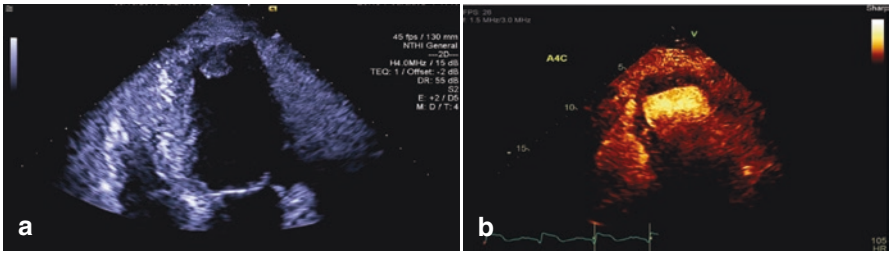

Fig. 1.17 Apical four-chamber view of a transthoracic echocardiogram demonstrating a large left ventricular apical thrombus (**a**). Injection of an echo-contrast agent delineates the linear appearance of this large apical thrombus (**b**)

AAS is characterized by disruption of the media layer of the aorta and typically presents with acute chest pain.

Although the incidence of AAS is lower than that of ACS, AAS carries a higher mortality, and is therefore a critical component of the differential diagnosis of chest pain in the CCU. The International Registry of Acute Aortic Dissection (IRAD) was created in 1996 as a way to combine data acquired from multiple institutions in Europe, North America, and Asia [19]. The 2010 intersocietal guidelines for the diagnosis and management of patients with thoracic aortic disease propose a standard approach to the diagnosis and treatment of AAS [20].

While clinical history and physical exam are important, imaging is essential in the diagnosis of AAS. Transesophageal echocardiography (TEE), computed tomography (CT), and magnetic resonance imaging (MRI) are the preferred imaging modalities and angiography is rarely needed.

AAS is classified based on the location and extent of involvement of the aorta. Two commonly used classifications include the DeBakey and the Stanford systems. The DeBakey system divides aortic dissection into three types based on their anatomical location. Type I originates in the ascending aorta and propagates beyond the aortic arch, type II is limited to the ascending aorta only, and type III is limited to the descending aorta [21]. The Stanford system divides aortic dissections into two types. Type A includes any dissection that involves the ascending aorta, while type B dissections are limited to the descending thoracic aorta [22]. The Stanford classification appears to have wider acceptance and is now used for all three AAS types: CAAD, IMH, and PAU.

Intramural Hematoma

IMH is defined by crescentic or circumferential thickening of the media layer of the aortic wall. IMH is likely due to ruptured vasa vasorum resulting in intramural bleeding but without a detectable intimal tear.

According to the IRAD experience, IMH typically presents with the symptoms of severe chest and back pain, similar to CAAD. However, IMH is less likely to result in severe aortic regurgitation and pulse deficits. IMH is usually unstable and may evolve into CAAD or regresses spontaneously; thus serial imaging is crucial. Stanford type B lesions in the descending aorta are more common than type A lesions in the ascending aorta (65% vs. 35% of all IMH, respectively) [19].

On TEE, IMH is diagnosed if there is regional thickening of the aortic wall >5 mm in a crescentic or circumferential pattern without an intimal flap or tear (Fig. 1.18a–c). Limitations of TEE in diagnosing IMH arise from the TEE's inability to visualize all portions of the aorta including the area around the origin of the brachiocephalic artery and all but the most proximal portions of the abdominal aorta. TEE is very useful in diagnosing complications of IMH, such as pericardial effusion or aortic regurgitation.

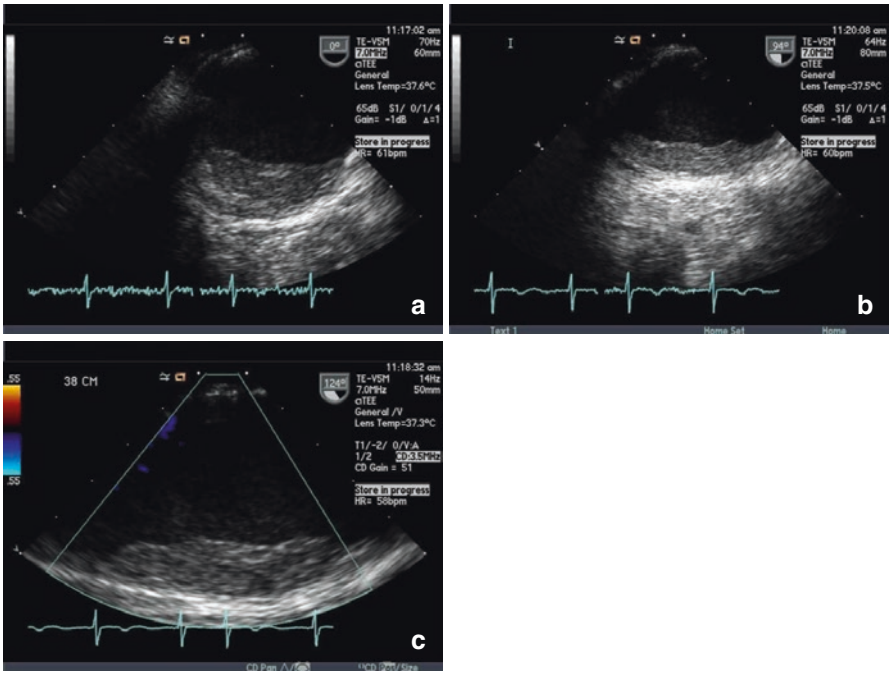

Fig. 1.18 IMH on TEE, showing marked thickening of the aortic wall in a crescentic pattern without an intimal flap or tear, at different TEE angles: (**a**) 0°, (**b**) 94°, (**c**) 124°

Classic Acute Aortic Dissection

CAAD is the most common form of AAS [20]. It occurs in approximately 66–75% of all AAS. The overall incidence of CAAD is low, and is estimated at 0.5–4.0 cases per 100,000 per year, and is thought to affect men more than women in a 2:1 ratio.

CAAD is characterized by an intimal tear, which leads to abnormal blood flow from the aortic lumen into the media. Consequently, there is longitudinal separation of the media layers by blood flow which tears an intimomedial flap from the remainder of the aortic wall. This flap separates the abnormal false lumen from the true aortic lumen. Intimal tears typically occur at the locations within the aorta with the highest shear stress. These are the right side of the ascending aorta immediately distal to the ostium of the right coronary artery (type A dissections) (Fig. 1.19a–c), and immediately distal to the ostium of the subclavian artery adjacent to the insertion of the ligamentum arteriosus (type B dissections).

Complications such as aortic regurgitation and pericardial tamponade can occur, and over time chronic changes such as false lumen thrombosis and aneurysm are common.

The typical symptom of acute aortic dissection is “aortic pain” similar to other forms of AAS. Acute, severe, tearing chest pain is the hallmark symptoms of

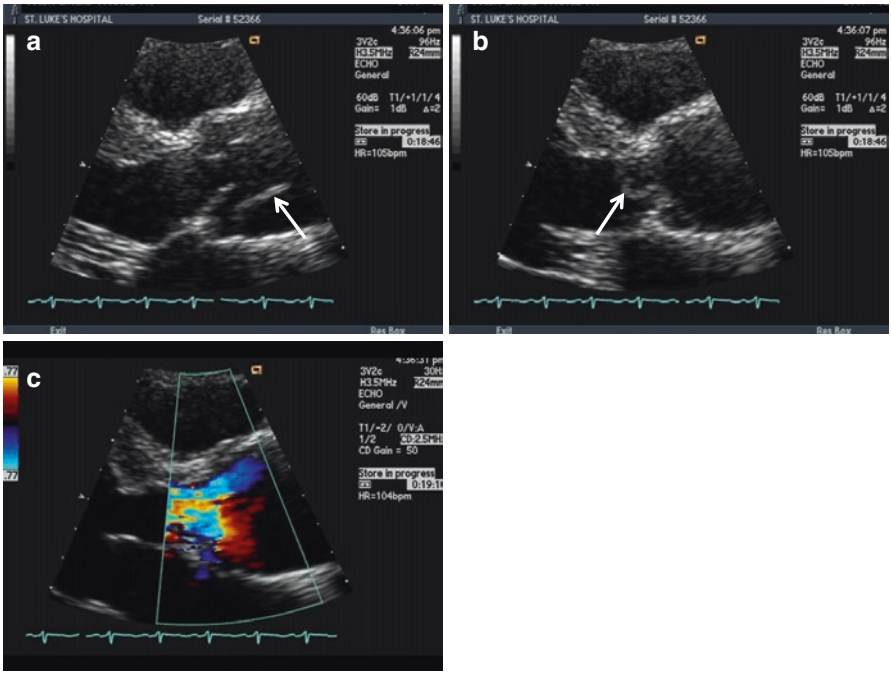

Fig. 1.19 Type A ascending aortic dissection with a distinct dissection flap seen in the ascending aorta (**a** with *arrow*) prolapsing into the aortic valve (**b** with *arrow*) with evidence of severe aortic regurgitation (**c**)

CAAD. Pain limited to the chest is typical of type A CAAD and pain in the back is more often the symptom of type B CAAD.

TEE is useful in the diagnosis of CAAD especially when a CT with contrast cannot be performed, such as in hemodynamically unstable patients or in patients who cannot tolerate iodinated intravenous contrast such as those with renal insufficiency or severe allergy. The reported sensitivity of TEE is 98% and specificity is 63–93%. Findings on TEE are a dissection flap separating the true and false lumen as well as the site of intimal tear represented by flow from the true lumen into the false lumen on color Doppler (Fig. 1.20a, b). Spectral Doppler may help corroborate the diagnosis by demonstrating "to and fro" flow into and out of the false lumen.

The true lumen is identified by its expansion with systole and contraction in diastole. The true lumen is often smaller than the false lumen. In early stages, the false lumen may be echo free or may contain spontaneous echo contrast (also known as "smoke") due to stasis of blood flow. In later more chronic stages, the false lumen may be partly or completely obliterated by thrombus formation (Fig. 1.21a–d).

Complications of CAAD may be seen on echocardiography such as aortic regurgitation, pericardial effusion/tamponade, and wall motion abnormalities indicative of ischemia if there is coronary ostial involvement.

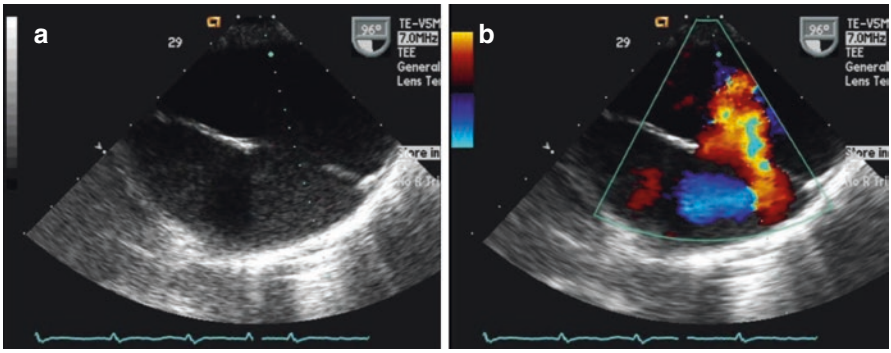

Fig. 1.20 A dissection flap is seen in the descending thoracic aorta separating the true and false lumens; the site of intimal tear is represented by the flow from the true lumen into the false lumen on color Doppler

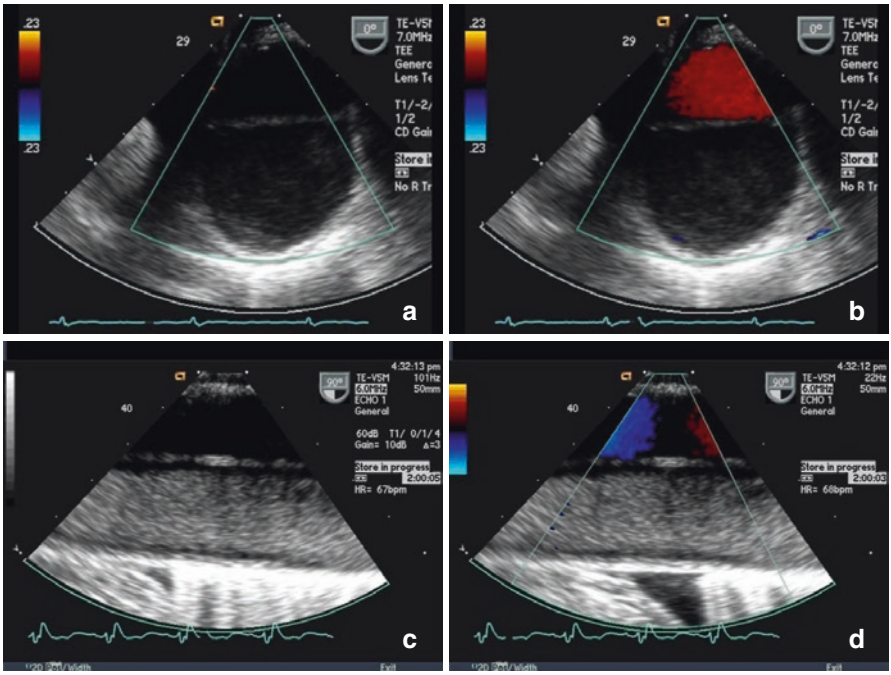

Fig. 1.21 Type B descending aortic dissection with a distinct dissection flap seen in the descending aorta (panel **a**). The true lumen is often smaller than the false lumen. In early stages (panel **b**), the false lumen may be echo free or may contain spontaneous echo contrast (also known as "smoke") due to stasis of blood flow. In later more chronic stages, the false lumen may be partly or completely obliterated by thrombus formation (panels **c**, **d**)

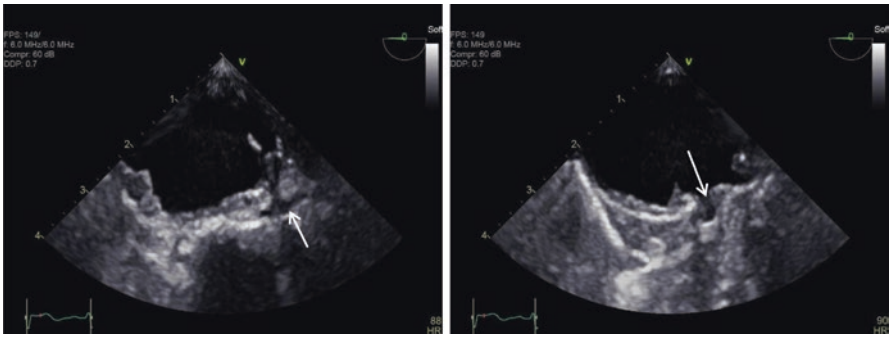

Fig. 1.22 (**a**, **b**): Penetrating aortic ulcer (PAU) develops as an atherosclerotic plaque erodes and penetrates through the elastic lamina into the media layer of the aorta, causing ulceration

It is important not to confuse the intimomedial flap of CAAD with either artifacts or surrounding vascular structures. Linear reverberation artifacts in the ascending aorta should not be mistaken for type A aortic dissection. Typically, reverberation artifacts are located twice as deep as the anterior aortic wall. In addition, a dilated azygos vein adjacent to the descending thoracic aorta may give an illusion of a type B dissection. Color or spectral Doppler imaging in both instances may help distinguish true aortic dissection from its masqueraders.

Penetrating Aortic Ulcer

Penetrating aortic ulcer (PAU) represents the process by which an atherosclerotic plaque erodes and penetrates through the elastic lamina into the media layer of the aorta, causing ulceration (Fig. 1.22a, b). PAU may further erode through the adventitia leading to either focal (pseudoaneurysm) or complete aortic rupture. Thrombus occasionally forms within PAU. In addition, PAU may lead to either IMH or aortic dissection, which is why PAU is characterized as an AAS.

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