Intraprocedural Imaging of Cardiovascular Interventions

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Preface

While many forms of non-invasive cardiac imaging have been used in association with cardiac procedures for many years, their use gained prominence in the mid-1980s when transthoracic echocardiography became an important component of the percutaneous mitral valvotomy procedure, especially to identify appropriate patients for this procedure. Soon thereafter, the use of transesophageal echocardiography to exclude left atrial thrombus was shown to be a valuable procedure enabling safe DC cardioversion without the need to wait for weeks of pre-cardioversion therapeutic anticoagulation. Most recently, many new and complex transcatheter cardiovascular procedures have been introduced and are radically changing the face of clinical cardiology. A critical part of the success of these new treatments is their optimal intraprocedural guidance by non-invasive imaging. This guidance helps reduce complications and improves outcomes. In fact, a new specialty of "Interventional Non-Invasive Imaging" has evolved and these imagers are a vital member of the treatment team. Three-dimensional echocardiographic imaging is an important skill for these imagers.

In this book, we present a practical approach for the use of imaging in a variety of cardiovascular procedures. We are fortunate to have contributions from the leaders in this field.

We present a wide array of procedures. In addition to the newest techniques such as transcatheter aortic valve replacement and transcatheter mitral valve edge-to-edge repair, we have included guidance of more established procedures such as pericardiocentesis. Also in light of the accelerating use of mechanical circulatory devices in the treatment of advanced heart failure, we discuss the evolving role of imaging in association with these devices. We have also included the technique of optical coherence tomography which is gaining applications in diagnosis of coronary artery disease and as an adjunct to coronary artery interventions.

While we expect that this book will be of value to imagers, we hope that other clinicians and interventionalists can use this as a resource to understand imaging needs during various cardiovascular procedures.

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Transesophageal Echocardiographic-Guided Cardioversion

1

David B. Min and Allan L. Klein

Abstract

The incidence and prevalence of atrial fibrillation (AF) continues to grow especially as the population ages. The uncoordinated atrial activation and subsequent ineffective atrial contraction is a strong stimulus for left atrial (LA) thrombus formation especially in the LA appendage. AF is associated with increased risk of stroke, heart failure, and all-cause mortality. The presence of LA thrombus in AF portends a poor prognosis. Direct current cardioversion (DCCV) is the most effective method of restoring sinus rhythm and, with it there is relief of symptoms, improved LV filling, reversed atrial remodeling, and possibly reduced cardio-embolic risk. However, there are significant risks of systemic embolization following DCCV if LA thrombus is present. Transesophageal echocardiography (TEE) is an ideal non-invasive imaging modality to detect thrombus in the LA and especially in the LA appendage. Its proper use can lead to earlier DCCV for AF and improve safety of DCCV in AF. A key part of this strategy is proper use of anticoagulation therapy. This chapter illustrates the important role of TEE in patient evaluation and risk stratification prior to cardioversion.

Keywords

Optical coherence tomography • Intra-coronary imaging • Acute coronary syndrome • Coronary artery disease • Intravascular ultrasound • Percutaneous coronary intervention • Plaque rupture • Calcific nodule • Plaque erosion • Instent restneosis • Neointimal proliferation • Neoatherosclerosis

Introduction

Since Willem Einthoven's first electrocardiograph description of "pulsus inaqequalis et irregularis" in 1906 [1], atrial fibrillation (AF) is now recognized as the most common arrhythmia worldwide [2]. With the prevalence expected to rise to 5.6–12.0 million in the United States by 2050 [3], AF is a growing epidemic, with the greatest impact felt on the aging population [4]. Furthermore, the national financial liability of AF has been estimated to be $26.0 billion annually, with greater than $5 billion attributed to direct AF treatment costs [5, 6]. The magnitude of this public health problem is likely substantially underestimated, as AF often manifests little to no symptoms and can go unrecognized.

AF represents a common final phenotypic pathway resulting from a multitude of pathophysiologic processes [7] which alter atrial tissue thus promoting uncoordinated atrial activation and subsequently ineffective atrial contraction [8]. The resultant blood stasis within the left atrium (LA) and left

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atrial appendage (LAA) in conjunction with AF-associated endothelial and extracellular atrial abnormalities and inflammatory-mediated coagulation changes fulfill Virchow's triad for thrombogenesis [9]. It is in this hypercoagulable milieu that intra-atrial thrombi form. The Framingham Heart Study found that AF is associated with a four to fivefold increased risk of thromboembolic events [10]. Multiple studies have linked AF to increased long-term risks of stroke, heart failure, and all-cause mortality [11, 12]. More specifically, the identification of thrombus in AF portends a poor prognosis with studies showing an embolic risk of up to 10.4 % per year and an annual mortality rate of 15.8 % [13].

A variety of diagnostic and therapeutic paradigms have been deployed to understand and alter, if not prevent, the downstream consequences of AF, including systemic thromboembolism and impaired diastolic left ventricular function. First described by Dr. Bernard Lown in 1962 [14], direct current cardioversion (DCCV) has been utilized to restore normal sinus rhythm to relieve symptoms, improve LV filling, reverse atrial remodeling, and possibly reduce cardioembolic risk [15]. DCCV is the most effective method of restoring sinus rhythm with a reported short-term success rate of 75–93 % [16, 17]. However, DCCV is not completely benign; aside from the risks associated with sedation, there is significant a risk of systemic embolization after DCCV [18–21]. Since the 1930s [22], the LAA has known to be a location of thrombus formation. Since then, consideration effort has been made to detect the presence of thrombi within the LA and LAA and alter the risk of systemic embolization.

While transthoracic echocardiography (TTE) has excellent overall image quality, transesophageal echocardiography (TEE) offers superior spatial resolution to TTE [23] and is the reference standard for assessing the LAA for thrombus formation with high sensitivity (92–100 %), specificity (98–100 %), and negative predictive value (98–100 %) [8, 24–27]. Through case studies, this chapter aims to illustrate the important role of TEE in patient evaluation and risk stratification prior to cardioversion.

Left Atrial Appendage Structure and Function

Located lateral to the LA and extending over the atrioventricular groove towards the left circumflex artery in close proximity to the left ventricular free wall [28], the LAA is a blind, trabeculated cul-de-sac. The trabeculations (or pectinate muscles) reflect its embryological origins. Whereas the smooth-walled LA, with whom the LAA is contiguous, is formed from the primordial pulmonary veins, the LAA is a remnant of the embryonic LA [29, 30]. There is a fold of serous pericardium that forms a ridge between the LAA orifice and the left superior pulmonary vein; this structure is also known as the warfarin ridge, Q-tip sign, posterolateral ridge, and ligament (or fold) of Marshall [31]. This prominence can be mistaken for a tumor or thrombus due its undulating motion and/or side lobe artifacts arising from echoes reflecting off the ridge [32, 33].

The LAA has a highly variable morphology; an autopsy study noted that 90 % of the LAA examined had multiple lobes, with the presence of two (80 %) distinct lobes being the most common and some specimens having as many as five lobes [34]. Based on its lobular/angular morphology, the LAA shape has been classified as: "chicken wing" (48 %), "cactus" (30 %), "windsock" (19 %), and "cauliflower" (3 %). In a multimodality imaging study of AF-patients, LAA shape correlated with risk of stroke; patients with atrial fibrillation who had "chicken wing" morphology were 79 % less likely to have a stroke/TIA history compared to the other shapes [35]. Consistent with such lobular heterogeneity, the LAA has great variation in length (ranging from 16 to 51 mm) and volume (0.7–19.2 mL) [36]. Likewise, the orifice connecting the LA and LAA, which typically lies superiorly and laterally, is an eccentric, elliptical structure with a minimal diameter ranging from 4 to 27 mm and a maximum diameter from 10 to 40 mm [36, 37]. The complexity and variability of LAA shape, size, and number of lobes underscores the importance of fully interrogating the LAA from multiple different angles/planes to detect possible thrombus prior to cardioversion.

Whereas the key function of the LA in modulating left ventricular filling as a reservoir, conduit, and pump is well recognized [38], the role of the LAA is less clear. It has been postulated given its increased distensibility, the LAA may serve as a decompression chamber during ventricular systole and in states of increased LA pressure and volume overload [39]. The LAA may further regulate volume homeostasis by secreting atrial natriuretic peptide and b-type natriuretic peptide in response to activation of stretch receptors within the LAA that ultimately results in diuresis [30, 40–43].

The trabeculated pectinate musculature of the LAA actively contracts in young, healthy individuals with normal intracardiac conduction with a distinctive pattern [34, 39]. LAA flow velocities can be assessed using pulsed wave Doppler by placing the sample volume 1–2 cm from the orifice within the LAA chamber, ideally in the midesophageal 0° or 90° planes [31]. In patients with a normal sinus rhythm, the characteristic pattern is quadriphasic (Fig. 1.1), as described below [44–46]:

- Early diastolic LAA emptying: In early diastole, a low-velocity positive wave occurs following the mitral-inflow E wave that represents the drop in LA pressure following opening of the MV and external compression of the LAA due to LA distension. The average early diastolic emptying velocity is 20–40 cm/s and is related to mitral E and PV diastolic velocities.

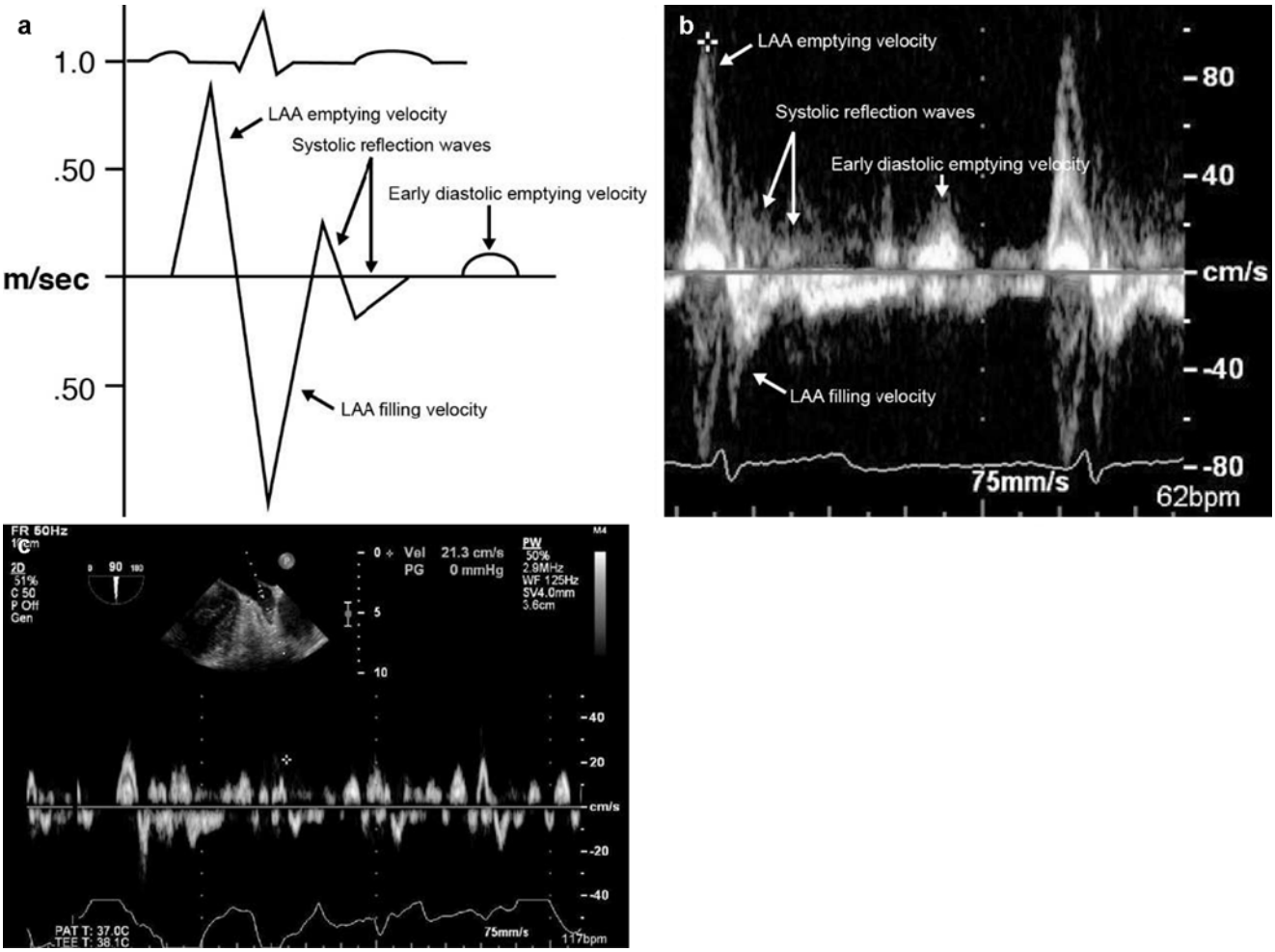


Fig. 1.1 LAA flow pattern in sinus rhythm (**a**, **b**) and AF (**c**)

- Late diastolic LAA emptying: Starts in late diastole immediately after the p-wave and represents LAA contraction and emptying. The late diastolic emptying velocity, which is typically 50–60 cm/s, is the largest wave in sinus rhythm and is a marker of LAA contractile function. This velocity correlates with LAA ejection fraction, LA size, and LA pressure.
- LAA filling: The retrograde velocity wave following late diastolic emptying in early systolic reflects LAA elastic recoil and relaxation. The average LAA filling velocity is 40–50 cm/s; the magnitude of the LAA filling velocity is inversely related to velocity of LAA contraction.
- Systolic reflection waves: After LAA filling in early systole, multiple low-velocity alternating inflow-outflow waves are seen; the functional significance of these waves has yet to be definitively determined.

In AF, the LAA develops endocardial thickening with fibrosis and loses pectinate muscle; this negative remodeling results in stretching, dilation, and decreased LAA contractility predisposing it to stagnant blood flood and thrombosis [47]. Some of most devastating consequences of AF including thromboembolic sequelae; over 90 % of thrombi in non-valvular AF have been localized to the LAA [48]. Thrombi have been found in patients who have had AF for <3 days [49] as well as patients who had been receiving oral anticoagulant therapy for 4 weeks [50]. Point scoring systems such as $CHADS_2$ and CHA_2DS_2-VASc have been developed and validated assessing the risk of ischemic stroke in non-valvular AF patients [51, 52]. A subgroup analysis of a transesophageal study found that patients who had a $CHADS_2$ score ≥3 had significantly higher mortality than those with lower scores; however, the $CHADS_2$ scoring system was not reliable in predicting risk for left atrial appendage thrombus formation, especially in patients with low $CHADS_2$ scores [53]. Given the significant stakes involved, careful scrutiny of the LAA using TEE and/or other non-invasive modalities is often necessary to exclude thrombi prior to cardioversion.

Transesophageal Echocardiography Evaluation of LA/LAA

Indications for TEE in AF

Given the risk of clinical significant thromboembolism in AF, risk reduction strategies have been employed to reduce the chance of an embolic event before and after DCCV through the use of anticoagulation and/or imaging modalities [54, 55]. Up until the early 1990s, the conventional strategy to reduce the risk of thromboembolic events after DCCV for AF or atrial flutter ≥48 h was to anticoagulate with warfarin for at least 3 weeks before DCCV [56, 57]; this strategy is still a Class I recommendation from the AHA/ACC/HRS [8]. Anticoagulation post-cardioversion is necessary for AF of >48 h duration (and AF <48 h with risk factors for thromboembolism) due to the variability of return of coordinated atrial function ("atrial stunning"); retrospective analysis of pooled data from 32 studies found that 98 % of embolic events occurred within 10 days of DCCV [54].

With the advent of TEE and through the landmark study entitled Assessment of Cardioversion Using Transesophageal Echocardiography (ACUTE), TEE was shown to be effective in guiding and expediting DCCV with short-term anticoagulation [58]. This large, multicenter, randomized, prospective trial of 1,222 patients undergoing DCCV for AF of >48 h duration compared a TEE-guided short-term anticoagulation strategy prior to DCCV to a conventional (3 week) anticoagulation strategy and found no significant difference in embolic events. The TEE-guided group had less hemorrhagic events and shorter time to DCCV, thus reducing the time for possible adverse atrial remodeling due to delays in the return of atrial function. Similarly, the follow-up ACUTE II trial investigated the safety of TEE-guided DCCV using low-molecular weight heparin (enoxaparin) versus unfractionated heparin [59]. The study found that there were no differences in safety (embolic events, bleeding, or deaths) between the two groups but that the enoxaparin TEE-guided approach had a shorter median length of hospitalization. The Anticoagulation in Cardioversion using Enoxaparin (ACE) trial also similarly showed non-inferiority of enoxaparin to unfractionated heparin plus a vitamin K antagonist in reducing significant events post TEE-guided DCCV [60].

Since the ACUTE I trial, TEE-guided cardioversion has become increasingly popular; in the setting of increasing costs of general diagnostic imaging, appropriateness criteria were developed. In 2011, a multi-society writing committee, including the ACC, AHA and American Society of Echocardiography (ASE), published updated appropriate use guidelines regarding the use of TEE-guided cardioversion: TEE is appropriate to "facilitate clinical decision making with regards to anticoagulation, cardioversion, and/or radiofrequency ablation"; TEE is inappropriate when "the decision has been made to anticoagulate and not to perform cardioversion" [61]. An analysis by Grewal et al. in 2012 found that the vast majority of TEEs performed before DCCV were appropriate (only 2.7 % were found to be inappropriate); the most common indications were symptomatic AF and CHF/hemodynamic compromise [62]. The 2014 AHA/ACC/Heart Rhythm Society (HRS) Guideline for the Management of Patients with Atrial Fibrillation provides further recommendations on the evaluation and treatment of AF, including guidance on direct-current cardioversion. Per this set of guidelines, TEE prior to cardioversion is appropriate (Class IIa recommendation) for patients with AF or atrial flutter of 48 h or longer who have not been anticoagulated for the proceeding 3 weeks, provided that anticoagulation is achieved before TEE and can be maintained after potential cardioversion for at least 4 weeks [8].

Performing TEE in AF

Despite being considered a semi-invasive procedure that is generally performed with conscious sedation, the safety profile of TEE has been well documented with a <0.02 % major complication rate when performed by an experienced clinician [63–65]. The American College of Cardiology (ACC) and American Heart Association (AHA) have published guidelines establishing TEE competence [66]. To help guide the sonographer, the American Society of Echocardiography and the Society of Cardiovascular Anesthesiologists have published joint guidelines on performing a comprehensive TEE examination that provides recommendations on TEE indications, patient selection, monitoring during sedation, and general imaging views [31]. One caveat to consider when monitoring sedated patients with atrial fibrillation is that blood pressure (BP) measurements may fluctuate due to high beat-to-beat variability in ventricular filling time, stroke volume, and contractility [67].

Once the TEE probe is safely intubated into the mid-esophagus in a neutral position, the LA is the near-field structure, which will make it difficult to image the entire structure from a single plane [31]. Thus, multi-planar assessment and sweeping from the inter-atrial septum to lateral wall (rotating clockwise to counterclockwise) looking for potential thrombi or masses is needed. The LA is a muscular structure with small pits and troughs; abnormal increases in wall thickness may be sequelae of mural thrombus or endocarditis [31, 68].

The LAA is frequently best visualized in the mid-esophageal window starting at 0°. As a lateral structure, it may require slight counterclockwise rotation and flexion or withdrawal of the probe to a more cranial position to center

the LAA within the plane [69]. Given the complexity of its shape and the importance of excluding a potential thrombus, it is imperative that the LAA be systematically imaged from multiple imaging planes (i.e. mid-esophageal 0°, 45°, 90°, 120°, 160°); often the additional lobe(s) of the LAA will only be appreciated at angles >100° [69] (Fig. 1.2). Simultaneous, multiple-plane assessment modalities (such as biplane or multiplane) can be very helpful in assessing the LAA for abnormalities from orthogonal views.

In evaluating possible thromboembolic sources in AF, the TEE should not be limited solely to the LA and LAA. Complex aortic plaque (4 mm and the presence of mobile components) has been linked to increased stroke risk; furthermore, aortic atheroma or plaque is prevalent in patients with AF [70–72] (Fig. 1.3a). While assessment of the LV via TEE is limited due to foreshortening of the apical segments, abnormal findings on TEE may prompt assessment via alternative modalities. Thorough evaluation of the MV and AV is also indicated, for rheumatic valvular heart disease is a well-recognized stroke risk factor, as is the presence of a mechanical prosthetic valve, even in the absence of atrial fibrillation [73]. For patients with atrial fibrillation who may undergo pulmonary vein isolation in the future, one can also assess the pulmonary vein anatomy at time of TEE prior to DCCV to guide potential electrophysiology procedures; the Role of Transeosphageal Echocardiography Compared to Computed Tomography in Evaluation of Pulmonary Vein Ablation for Atrial Fibrillation (ROTEA) study showed that TEE provided both anatomical and function information that complemented CT [74]. Other major sources of cardioembolic strokes are infective and non-infective endocarditis, atrial myxomas, atrial and ventricular septal defects, and mitral annular calcification; thus a detailed TEE prior to DCCV can further identify potentially relevant sources of embolism beyond the LAA that can significant impact clinical management [75] (Fig. 1.3b–e).

The LAA is often surgically excised or excluded at time of concomitant open-heart surgery procedures in an attempt to prevent future thromboembolic events. However, TEE studies have reported patent flow between the LA and the residual LAA following surgical LAA intervention, up to 60 % [76–78]. Patients who had high persistent flow into the LAA after exclusion were also found to have a high prevalence of LAA thrombus [77]. Furthermore, differences in LAA patency rates have been found depending on technique and that the differences can been seen in the early (<30 day) post-operative period [77]. Given reports of early thrombus formation <48 h of onset of atrial fibrillation in patients following open-heart surgery [79], it is important not to assume that a history of "LAA ligation" is protective. Careful scrutiny of the structure, function, and flow characteristics of the remnant LAA is needed to exclude the presence of thromboembolic risks.

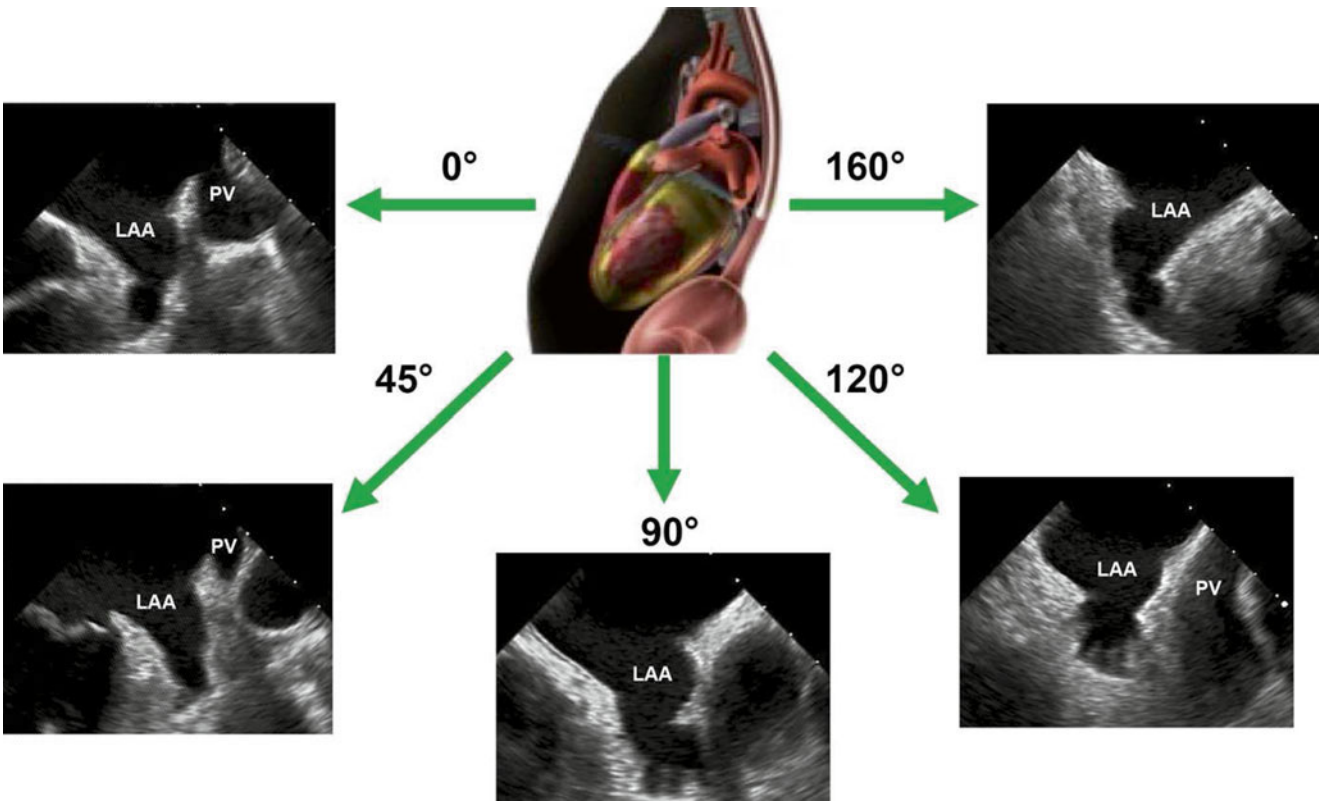
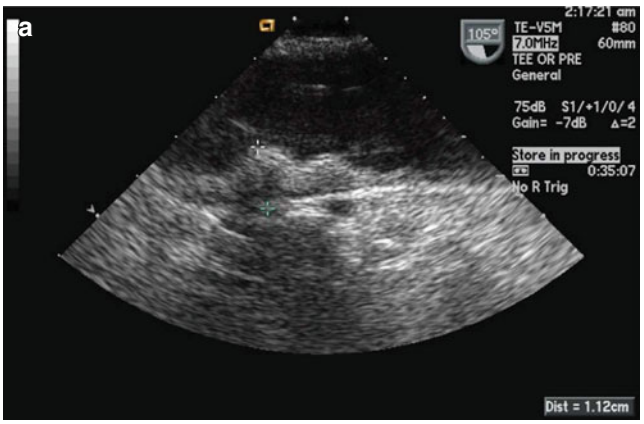


Fig. 1.2 Multiplanar imaging of LAA at different angles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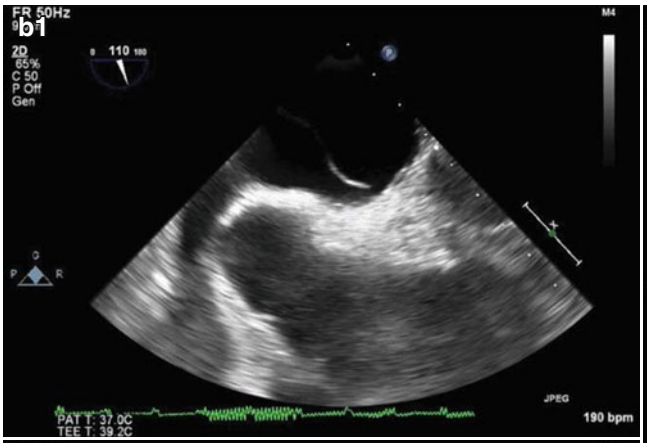
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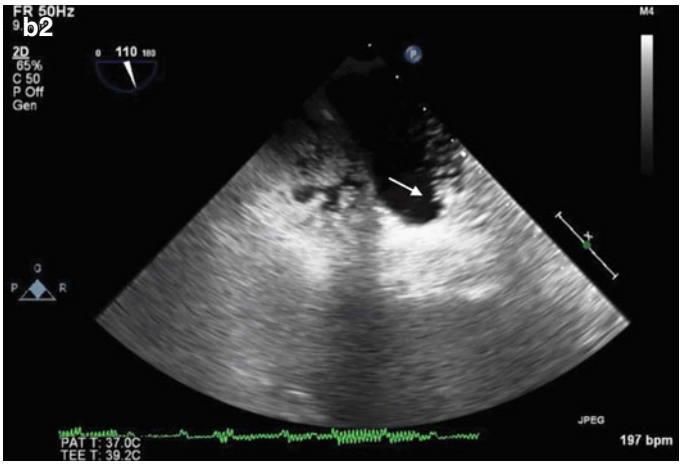
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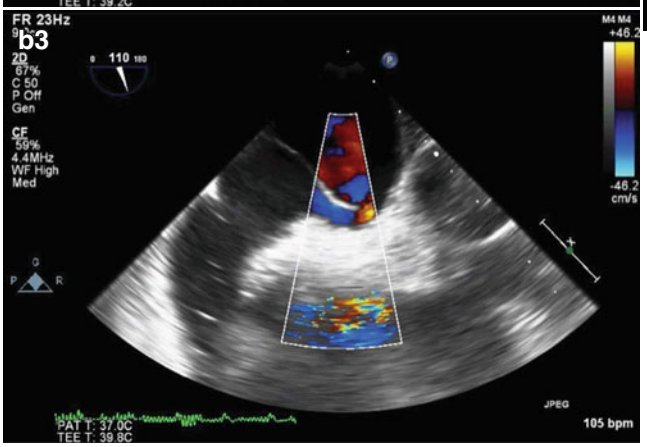
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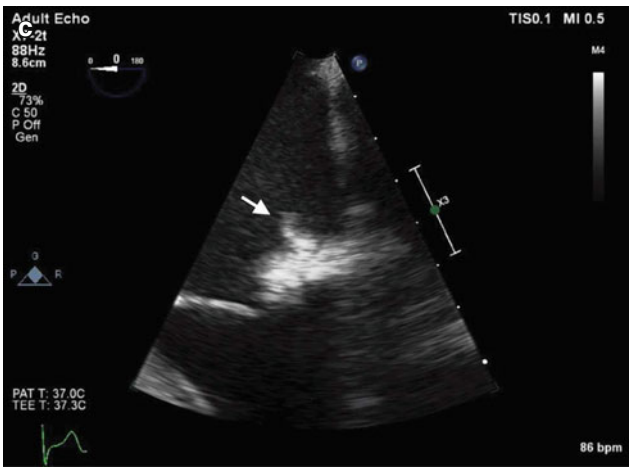
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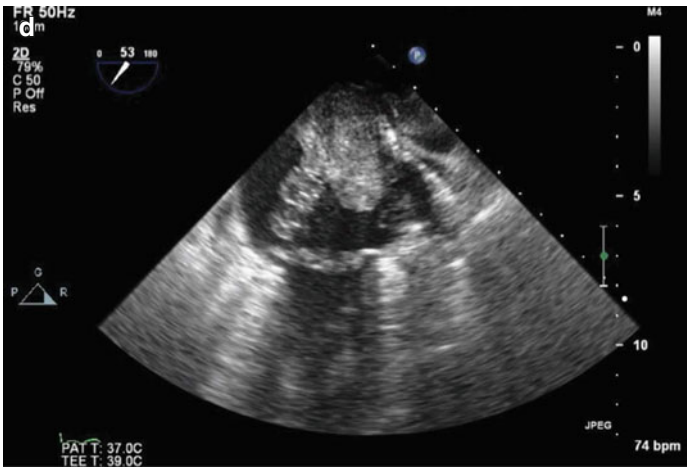
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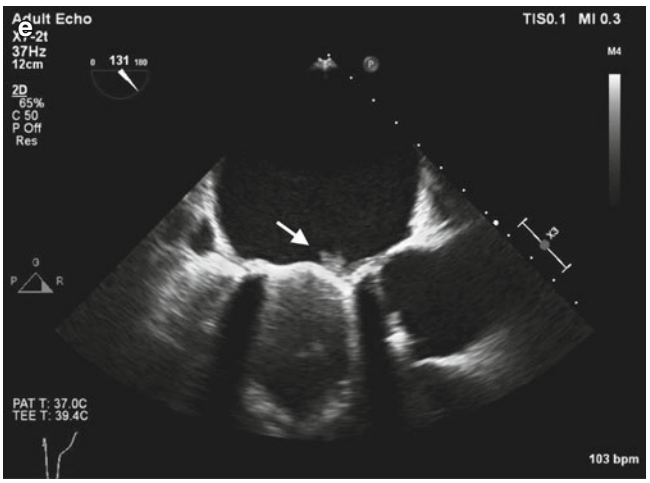

Fig. 1.3 (continued)

TEE Abnormalities in AF

Blood stasis secondary to ineffective LAA contraction in AF can be visualized on two-dimensional (2D) TEE as a spectrum ranging from spontaneous echocontrast (SEC) to sludge to thrombus formation [80, 81] (Fig. 1.4). SEC, also known as "smoke", has a slow, swirling motion of variable echodensity that represents non-laminar, low blood flow velocities [82, 83]. Reflecting increased erythrocyte aggregation, and presence of fibrinogen, SEC has been seen in up to 60 % of patients with AF [84–86]. A precursor of thrombus formation, SEC has been strongly associated with subsequent thrombotic events [58, 87]. However, the use of aspirin or warfarin does not influence the presence of SEC as the underlying hemodynamic abnormalities are persistent despite anticoagulation therapy [88]. Despite attempts to quantitative the degree of

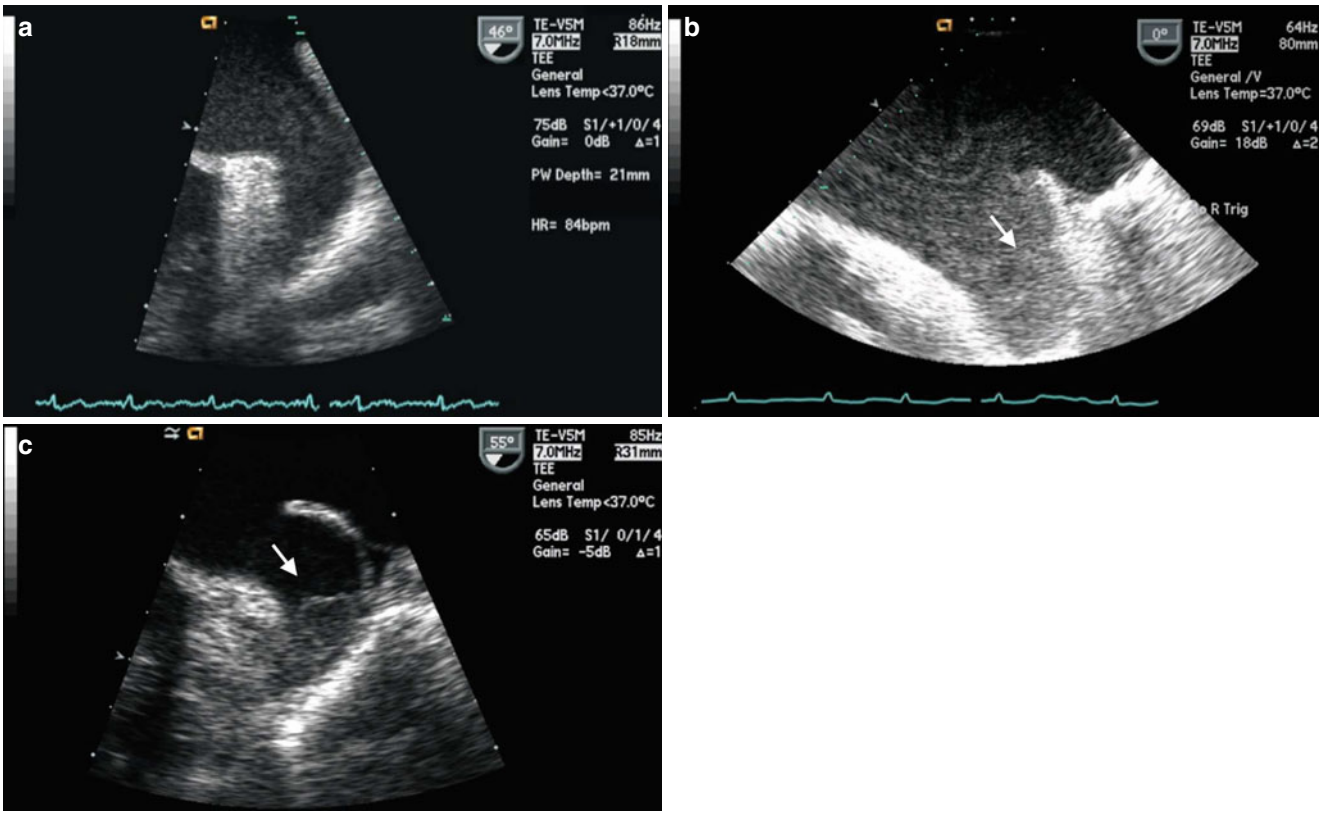

Fig. 1.4 Spectrum of LAA Thrombogenic Milieu (**a**) shows severe spontaneous echocontrast (*SEC*) in the LAA. (**b**) Shows severe SEC and layered sludge within the LAA. (**c**) Shows thrombus that has formed in the LAA

Fig. 1.3 Potential sources of emboli. (**a**) Shows severe atheroma (1.2 cm) in the mid-aortic arch. (**b**1) Shows a patent foramen ovale (PFO). (**b**2) Shows agitated saline flowing through the PFO. (**b**3) Shows color flow through the PFO. (**c**) Shows mobile mitral annular calcification. (**d**) Shows a large mass in the LA that on pathology was revealed to be sarcoma. (**e**) Shows a vegetation on a prosthetic mitral valve consistent with prosthetic endocarditis