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EACVI DOCUMENT

The role of multi-modality imaging for the assessment of left atrium and left atrial appendage: a clinical consensus statement of the European Association of Cardiovascular Imaging (EACVI), European Heart Rhythm Association (EHRA) of the European Society of Cardiology (ESC)

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Structural, architectural, contractile, or electrophysiological alterations may occur in the left atrium (LA). The concept of LA cardiopathy is supported by accumulating scientific evidence demonstrating that LA remodelling has become a cornerstone diagnostic and prognostic marker. The structure and the function of the LA and left atrial appendage (LAA), which is an integral part of the LA, are key elements for a better understanding of multiple clinical conditions, most notably atrial fibrillation, cardioembolism, heart failure, and mitral valve diseases. Rational use of various imaging modalities is key to obtain the relevant clinical information. Accordingly, this clinical consensus document from the European Association of Cardiovascular Imaging, in collaboration with the European Heart Rhythm Association, provides comprehensive, up-to-date, and evidence-based guidance to cardiologists and cardiac imagers for the best practice of imaging LA and LAA for the diagnosis, management, and prognostication of the patients.

Keywords left atrium • left atrial appendage • imaging • atrial fibrillation • stroke • anticoagulation • echocardiography • cardiac magnetic resonance • cardiac computerized tomography • cardiomyopathy

Introduction

Structural, architectural, contractile, or electrophysiological alterations may occur in the left atrium (LA).[1] The concept of LA cardiopathy is supported by accumulating scientific evidence demonstrating that LA remodelling has become a cornerstone diagnostic and prognostic marker. The structure and the function of the LA and left atrial appendage (LAA), which is an integral part of the LA, are key elements for a better understanding of multiple clinical conditions, most notably atrial fibrillation (AF), cardioembolism, heart failure (HF), and mitral valve diseases. Rational use of various imaging modalities is key to obtain the relevant clinical information. Accordingly, this clinical consensus document aims to elucidate the state-of-the-art, disease-centred multi-modality imaging of LA and LAA to provide practical advice for the diagnosis, management, and prognostication of the patients. Intraprocedural guidance, technical aspects of the procedures, or the indications of interventions related to LA and LAA are out of the scope of this document. The clinical advice is based on evidence and/or consensus of the writing group and is classified into several categories, as shown in *Advice table 1*. Advice aims to encourage optimal use of imaging for the benefit of the patients.

Advice table 1 Categories of clinical advice

Strength of advice definition	Symbol
Clinical advice, based on robust evidence	
Clinical advice, based on uniform consensus of the writing group	
May be appropriate, based on published evidence	
May be appropriate, based on consensus within the writing group	
Area of uncertainty	

Morphology and function of LA and LAA

Normal morphology and function

LA consists of the main body and LAA. The main body of the LA consists of three components without clear anatomic demarcations: (i) the venous inflow component that receives blood from pulmonary veins (PVs); (ii) the vestibule, the outlet part surrounding the mitral orifice; and (iii) the inter-atrial septum (IAS).[2] Smooth endocardium lines the thin muscular walls of the LA body which can be described as superior (the roof), posterior, left lateral, septal (or medial), and anterior. Normal LA function has three phases: (i) PV forward flow (reservoir phase) during ventricular systole, (ii) PV forward flow during early diastole (conduit phase), and (iii) PV reverse flow by LA contraction during late diastole (absent in AF)[3] (*Figures 1* and *2*).

PVs enter the LA from the posterosuperior wall with frequent anatomic variations. Typically, two PVs (upper, lower) from each lung enter the LA with a funnel-shaped orifice which makes it difficult to see the clear demarcation of the ostium. An accessory right PV and common trunk of upper and lower PVs at entry are common variations (see CCT).

The LAA is a finger-like extension of the anterolateral LA wall located in the left atrioventricular groove, with a well-defined, usually oval orifice (the ostium), a neck region, and a lobulated body. Based on the shape of the central and secondary lobes, LAA morphology can be classified into four types with possible overlaps: windsock (single central lobe), chicken wing (bended central lobe), cauliflower (short central lobe and several lobes leading to a distal width larger than the proximal part), and cactus (central lobe leading to several secondary lobes superior and inferiorly).[3,4] The inner surface of the LAA is lined by the pectinate muscles with prominent indentations.

Remodelling and abnormal function of LA and LAA

The relationship between LV function and LA volume is complex and dynamic. Various factors such as volume and pressure overload in the context of mitral stenosis (MS), regurgitation, left ventricular (LV) systolic and/or diastolic dysfunction (DD), or AF contribute to the remodelling and dilatation of the LA. LA enlargement frequently occurs along the superoinferior axis more prominently than the anteroposterior axis. LA demonstrates phasic volume changes during cardiac cycle representing reservoir, conduit (passive emptying), and contractile (active emptying) functions. Abnormal LA function is typically characterized by decreased compliance and/or contractile dysfunction.[5] Thickening of the wall and fibrosis may contribute to LA dysfunction by increasing the stiffness of the LA with or without significant dilatation.[6]

LAA enlargement often accompanies LA dilatation. Diminished LAA contractile function is characterized by diminished emptying velocity and stasis. The LAA has contractile and endocrinological functions, while its distensibility contributes to LA pressure modulation.[3,7]

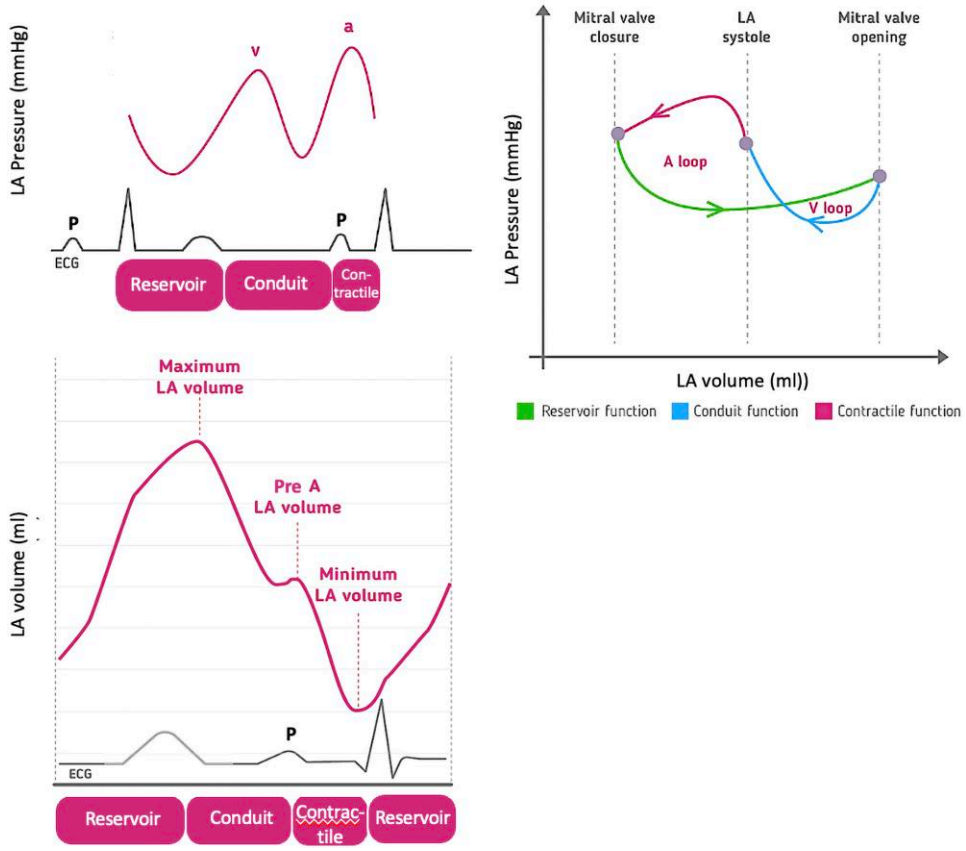


Figure 1 Phasic changes of LA: pressure, volume, and pressure/volume curves.

Multi-modality imaging for LA and LAA

The assessment of structural, architectural (tissue characteristics), functional, or electrophysiological changes associated with LA cardiopathy relies on the rational use of imaging modalities by appreciating their capabilities and limitations to derive clinically relevant information (*Table 1*).

Transthoracic echocardiography

Transthoracic echocardiography (TTE) is the primary imaging modality to evaluate LA size and function. Since LA enlargement is asymmetrical, anteroposterior LA diameter measured from the M-mode or 2D parasternal long-axis image can significantly underestimate LA size.[8,9] Yet, for some specific clinical conditions, the cut-offs for LA dilatation remain based upon anteroposterior linear measurements, such as in risk stratification in hypertrophic cardiomyopathy (HCM).[10,11] LA volume quantification by 2D echocardiography (2DE) and, preferentially, by 3D echocardiography (3DE) is the accurate way to evaluate LA size in clinical practice.[12] The measurement of LA volume by 2DE requires the acquisition of LA-focused apical views to maximize both the LA width and length in four- and two-chamber (should be similar in both views) to avoid foreshortening,[10] because the LA and the LV are not co-axial.[13] The bi-plane area–length method systematically yields larger LA volumes than the Simpson's bi-plane disc summation method as it assumes an ellipsoid shape.[14] The Simpson's method is preferred over the area–length method for clinical use because of fewer geometric assumptions than the area–length method.[5] LA volume calculated by 2DE correlates well with measurements obtained using 3DE, cardiac computed tomography (CCT), and cardiac magnetic resonance (CMR), despite some degree of systematic underestimation.[15] Automated tools based on 2D speckle-tracking echocardiography (STE) have high feasibility and yield LA volumes (average bias 1.5 mL, limits of agreement ± 8 mL) comparable to the standard manual tracing of LA endocardial contours.[15] By 2D echo, the upper limit of normal LA volume is defined as 34 mL/m^2 (range 16–34 mL/m^2) in both men and women.[10] Transthoracic 3DE has lower interobserver variability and higher accuracy than 2D, by avoiding foreshortening, geometric assumptions, and manual contour tracing errors.[16,17] LA volumes are significantly larger when measured by 3DE than by 2DE; therefore, cut-offs for detecting LA remodelling and dysfunction cannot be used interchangeably between the two techniques. Reference values for 3DE have been obtained from large cohorts of healthy volunteers (*Table 2*).[13,18,19] Recent studies testing the latest 3DE software tools dedicated for LA quantification showed that the measurement bias against CMR data is minimal and clinically negligible.[15,20] Fully automated 3D LA volume quantification using dedicated software packages also enables single-beat acquisitions with sufficient temporal resolution for reliable quantification of the LA even in patients with irregular rhythms.[21]

Maximal LA volume has been the most clinically used parameter; yet, there is an important body of evidence supporting the role of phasic LA volumes (particularly LA minimal volume) and function. LA function may demonstrate alterations prior to volume changes.[12] The same dedicated 2DE views used for maximal LA volume calculation can be employed to obtain LA phasic function parameters. From the LA volumes, the total emptying volume (EV) is calculated as (LAVmax – LAVmin), the passive EV as (LAVmax – LAVpreA), and the active EV as (LAVpreA – LAVmin). Total emptying fraction (LAEF); (total EV / LAVmax), passive LAEF;

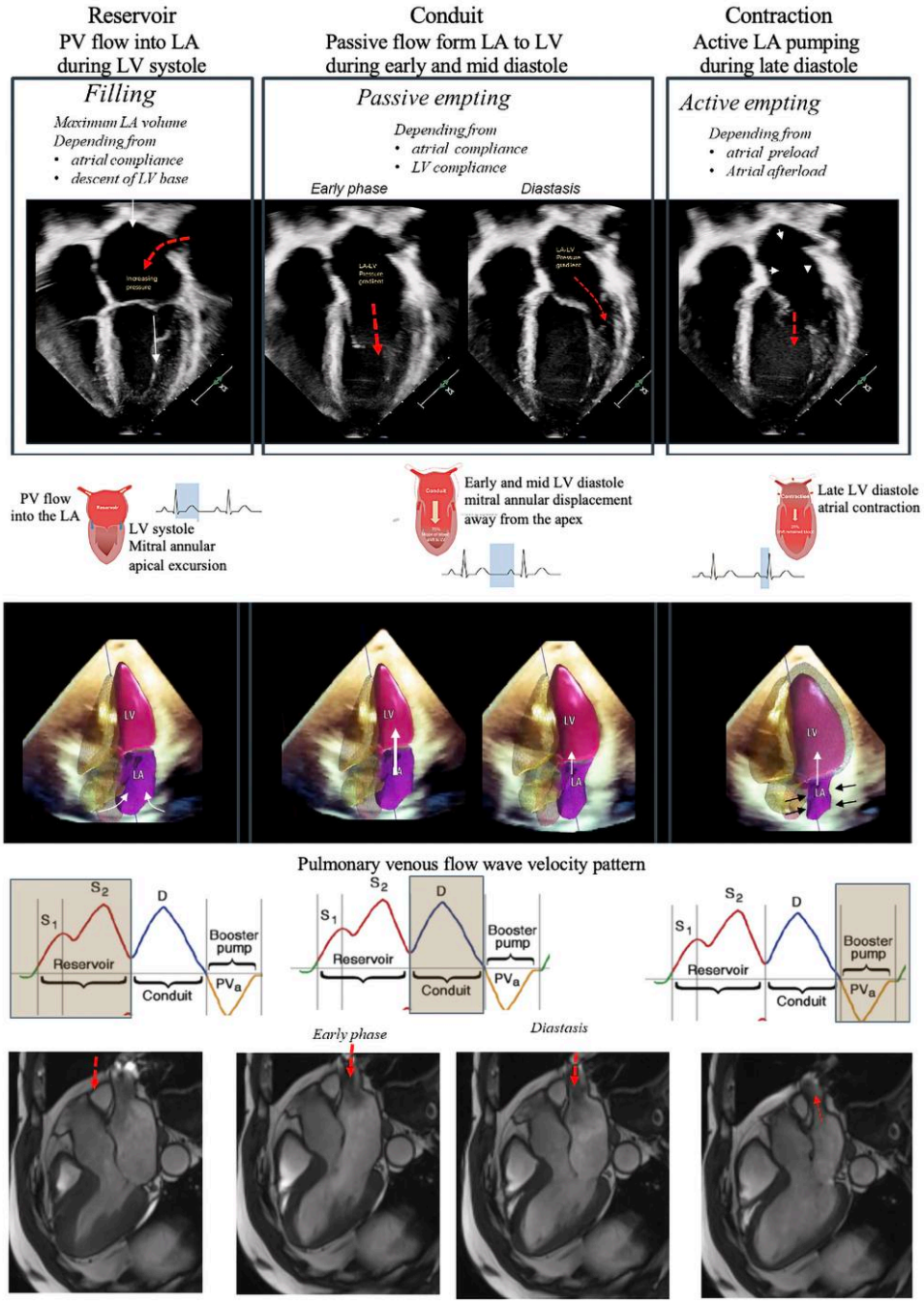


Figure 2 Translation of LA phasic function into imaging. Note the interaction between mitral annular descent by LV contraction, LA reservoir, atrial contraction, and PV flow.

(passive EV / LAVmax), and active LAEF; (active EV / LAVpreA) are calculated as indices of reservoir, conduit, and contractile function, respectively (*Figure 3*).[13] Because indexing LA size to body surface area accounts for gender difference, only the indexed value should be reported.[22]

Because of manual endocardial border tracing at three different time points of the cardiac cycle, assessment of LA phasic function by 2DE is time-consuming and prone to errors. LA function can be measured faster and automatedly, without geometric assumptions by using dedicated software packages for LA volume quantitation from 3DE datasets (*Figure 4*).[13] The limitations of 3DE at present include low spatial resolution and the need for dedicated equipment and training. There is paucity of data regarding its incremental prognostic value compared to 2DE.

Mitral inflow velocity during atrial contraction (A wave) and late diastolic mitral annular velocity (a′) are Doppler metrics of LA function. Blunted or absent A and a′ waves during sinus rhythm are typical findings of atrial stunning. Of note, these parameters are load- and angle-dependent and present during sinus rhythm only. Speckle-tracking strain has been a highly sought-after technique for LA function quantification. It is semi-automated, less angle-dependent, and less affected by artefacts than tissue Doppler-based measurements. LA strain

Table 1 Goal-directed multi-modality imaging for LA and LAA

	TTE	TOE	ICE	CCT	CMR	PET
LA volume	+++[a]	+	+	+++	+++	-
LA morphology	+++[a]	-	+	+++	+++	-
LA phasic function	+++[a]	-	-	++	+++	-
LA strain	+++	-	-	+	++	-
LAA morphology	-	++	++	+++	++	-
LAA function	-	+++	+	-	+	-
Pulmonary vein flow	++	+++	-	-	-	-
Pulmonary vein pattern	-	++	++	+++	+++	-
LAA thrombus	-	+++	+++	+++	++	-
LA fibrosis	-	-	-	-	++	++[b]
Peri-atrial epicardial fat	+[c]	-	-	+++	+++	-
Real-time image integration with EAM	-	-	-	+++	+++	-
Inflammation	-	-	-	-	-	++[d]

Abbreviations: CCT, cardiac computerized tomography; CMR, cardiac magnetic resonance; EAM, electroanatomic mapping; ICE, intracardiac echocardiography; PET, positron emission tomography; TTE, transthoracic echocardiography; TOE, transthoracic echocardiography.
[a]Ideally by 3DE.
[b]PET with fibroblast activating protein inhibitors.
[c]Only thickness.
[d]PET with fluorodeoxyglucose.

Table 2 Reference echocardiographic values of LA volumes[13]

	3D			2D
	Women	Men	Overall	Overall
LAVImax (mL/m^2)	31 (27–45)	31 (19–52)	32 (28–36)	24 (21–28)
LAVImin (mL/m^2)	10 (5–18)	11 (4–21)	10 (8–12)	8 (6–10)
LAVIpreA (mL/m^2)	18 (10–30)	18 (9–32)	18 (14–21)	14 (12–18)
Total LAEF (%)	68 (53–79)	66 (51–80)	67 (63–71)	67 (62–74)
Active LAEF (%)	40 (18–61)	41 (20–60)	41 (35–48)	46 (39–53)
Passive LAEF (%)	45 (22–60)	43 (23–61)	44 (38–49)	41 (32–48)

Values are given as median (Interquartile range). Abbreviations: LAVI, left atrial volume index; max, maximum; min, minimum.

assessment requires dedicated, focused image acquisitions to optimize lateral and temporal resolution. The use of dedicated software with an automated LA wall detection algorithm improves measurement reproducibility. The region of interest (ROI) should fit in the thin wall, accurately track the motion of the mitral annulus, and avoid the strong signals of the adjacent and stationary pericardial tissue, which—if included—may underestimate strain values.[23] LA function parameters [reservoir (LASr), conduit, and contraction strain] are computed as LA time–strain curves over the cardiac cycle (*Figure 5*). Analysis, reporting, and interpretation should follow the EACVI/ASE/Industry Taskforce consensus statement on LA strain analysis.[24] The reference timing of zero strain should be set at end-diastole (R–R gating). The alternative method is when zero strain is set at the onset of atrial contraction (P–P gating). Importantly, however, the R–R gating is less prone to errors

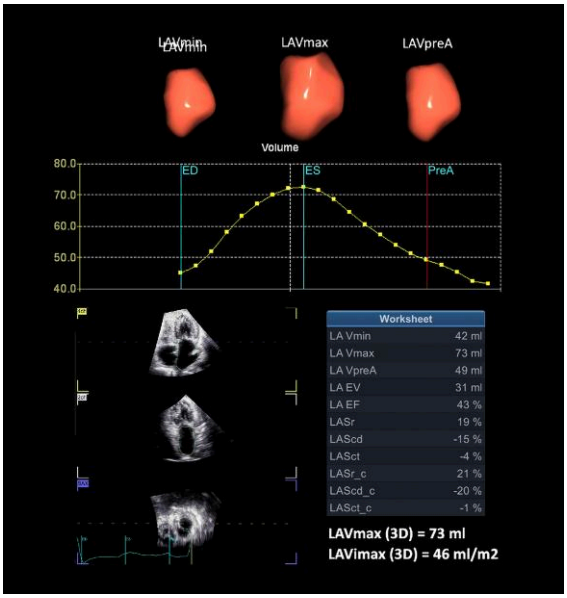


Figure 3 Phasic volumes by 3DE.

than the P–P gating and can be used during AF. Yet, high reproducibility (intraclass correlation 0.93 and 0.90, respectively) of both methods was shown in the MASCOT-HIT study.[25] Strain rate during ventricular systole, early diastole, and late diastole correspond to reservoir, conduit, and contractile function, irrespective of the R–R or P–P gating. Of note, LA load impacts volume and strain measurements regardless of the method used. Normal LA phasic strain values were reported in the healthy population from a meta-analysis including 40 studies in 2017 and from the NORRE study in 2018[19,26] (*Table 3*).

Additionally, reference ranges of LA phasic function by strain were published from 1329 healthy participants in the HUNT study (analyses performed by General Electric HealthCare EchoPAC system). Accordingly, bi-plane mean ±2SD reference values are as follows, in females and males, respectively: %LASr, 33.2 (17.2–49.2), 32.7 (16.0–49.3); %LA conduit strain, −17.0 (−30.8 to −3.3), −15.7 (−29.1 to −2.3); %LA contraction strain, −16.2 (−25.4 to −7.0), −17.0 (−26.9 to −7.0).[27]

Combining early diastolic mitral inflow (*E*) and annulus (*e′*) velocities with LASr enables obtaining an index for the estimation of LA stiffness by the formula: *E*/*e′*/LASr.[28]

Advice table 2 Use of TTE

Evaluation of the LA by TTE requires the acquisition of atrial-focused views.	
It is advised to assess LA size by volume (bi-plane 2D or 3D) quantification and to use the same tool for intra- and inter-individual comparisons.	
LA phasic volumes and strain by TTE are the mainstay of assessing LA function.	
Volumetric cut-offs obtained from different echo techniques cannot be used interchangeably.	
R–R gating is advised for the assessment of LA phasic function by 2D strain.	
LA size estimation should not be limited to M-mode or 2D linear measurements.	

Transoesophageal echocardiography

Transoesophageal echocardiography (TOE) is well suited to image the LA and the LAA, given their proximity to the oesophagus. However, LA sizing is performed by TTE rather than TOE, because the ultrasound scan sector cannot entirely encompass LA on TOE. Exclusion of thrombi, search of cardioembolic sources, characterization of the PVs before AF ablation, procedural planning, and guidance for LAA occlusion are the most common indications for imaging LA and LAA by TOE.[29] Although TOE is semi-invasive, it is safe in renal insufficiency. It provides adequate image quality in AF and offers information about associated cardiac pathologies and haemodynamics.

Technical considerations

Multiple TOE planes are used to explore the LA and LAA from the mid-oesophageal view. With 2D probes, LAA can be easily visualized between 45 and 90° with counterclockwise rotation and gentle anteflexion of the transducer. Once the LAA is visualized, it is centralized on the screen, and a thorough evaluation is performed by sweeping from 0 to 135°. However, 3D probes allow visualization of bi- or tri-plane simultaneously which facilitates the sweep process (*Figure 6*). A 3D zoom acquisition with a wide sector (since high temporal resolution is not specifically required for atrial structures) and excluding the atrial roof in the nearfield (avoiding obstruction of the view) is generally performed for a live assessment and further cropping. From the 3D zoom acquisition, a live 'en face' view of the LAA orifice can be obtained. Further perpendicular cropping and orientation of the 3D dataset enable the characterization of the LAA morphology. Photo-realistic rendering with light adjustments or CCT-like rendering with improved transparency (Glass) has also been recently introduced to improve the qualitative assessment by highlighting the tissue–blood interface (*Figure 7*).[30]

The TOE also provides anatomic and functional information about PVs. Notably, TOE underestimates PV dimensions, especially for the inferior veins.[31] The left upper PV is visualized in its long axis adjacent to LAA with further counterclockwise rotation between 45 and 110°. Visualization of the left lower PV requires slight vertical manipulation or further angulation to 120°. The right upper PV can be visualized after clockwise rotation of the probe with anteflexion from either 0, 45, or 120–135° (on the left and right side of the screen, respectively) adjacent to the IAS and superior vena cava. The right lower PV is the most difficult to align with the Doppler beam and is best seen from the extreme clockwise rotation of the probe at 0° without anteflexion immediately inferior to the right upper PV. PV Doppler interrogation shows systolic (S), diastolic (D), and atrial contraction (A) waves during sinus rhythm (*Figure 8A–D*). PV flow velocities are relevant for the assessment of LA pressure, mitral regurgitation (MR) severity, and PV stenosis after PV isolation. The left and right PV orifices are widely separated, and the pyramidal 3D data set cannot include them all in a single image. From the best 2D views obtained, the left and right PV orifices can be seen 2 by 2 by 3D zoom acquisition from an optimal orthogonal display, and rotation of the dataset as shown in figure (*Figure 8E* and *F*). The left PVs are adjacent to LAA, and the right PVs run adjacent to the IAS.

LAA function

Flow in the LAA is visualized using colour Doppler with a low Nyquist limit, and flow velocities are measured by pulsed-wave Doppler. Four phases have been described: emptying (contraction) velocity, filling velocity, a biphasic systolic reflection wave, and an early diastolic emptying wave[29] (*Figure 9*). Emptying velocity ranges from 63 ± 29 to 83 ± 25 cm/s and filling velocities range from 54 ± 17 to 61 ± 18 cm/s. Velocities can decrease because of high LA pressure. A sawtooth pattern with low velocities is observed during AF. Velocities <40 cm/s in sinus rhythm are associated with an increased risk of stroke.[32]

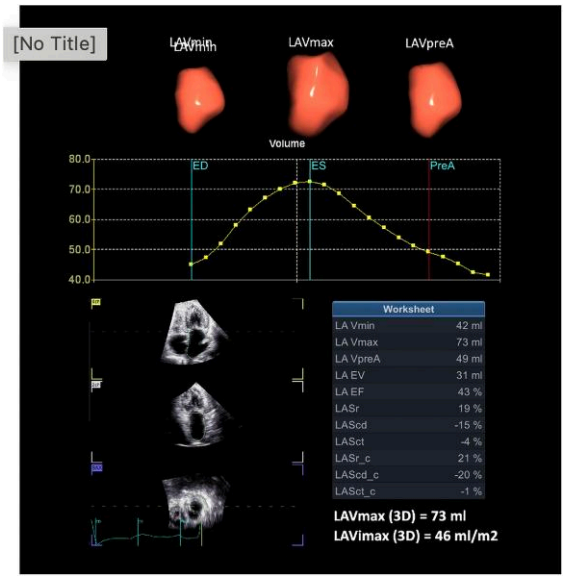


Figure 4 3D automated LA volume quantification. Note that the LA long axis is adjusted.

Advice table 3 For the use of TOE

Multiplanar imaging with or without 3D is indispensable for complete visualization of LAA morphology.	
TOE is the preferred technique for the assessment of LAA, function, and thrombus.	
TOE is the preferred technique for the assessment of PV flow.	
The use of TOE is inappropriate for the assessment of LA size or volume.	

CMR

CMR allows a comprehensive 3D evaluation of LA anatomy, structure, and function. Its potential to reveal the extent of atrial fibrosis by late gadolinium hyperenhancement (LGE) imaging is an advantage.[33]

Assessment of morphology

CMR steady-state free-precession (SSFP) cine images provide excellent blood-endocardium and epicardium-fat contrast, allowing good delineation of the atrial endocardial borders and LA size. This allows precise measurement of LA volumes, either using a bi-plane area–length method from two- and four-chamber cine images (*Figure 10A*) or using the Simpson's disc summation method on a stack of adjacent short-axis images from the atrioventricular ring to the roof of the LA. Since LV and LA are not co-axial, the LA volumes derived from the bi-plane area–length method may underestimate the true LA size because the long-axis cine images are normally aligned to the LV axis during

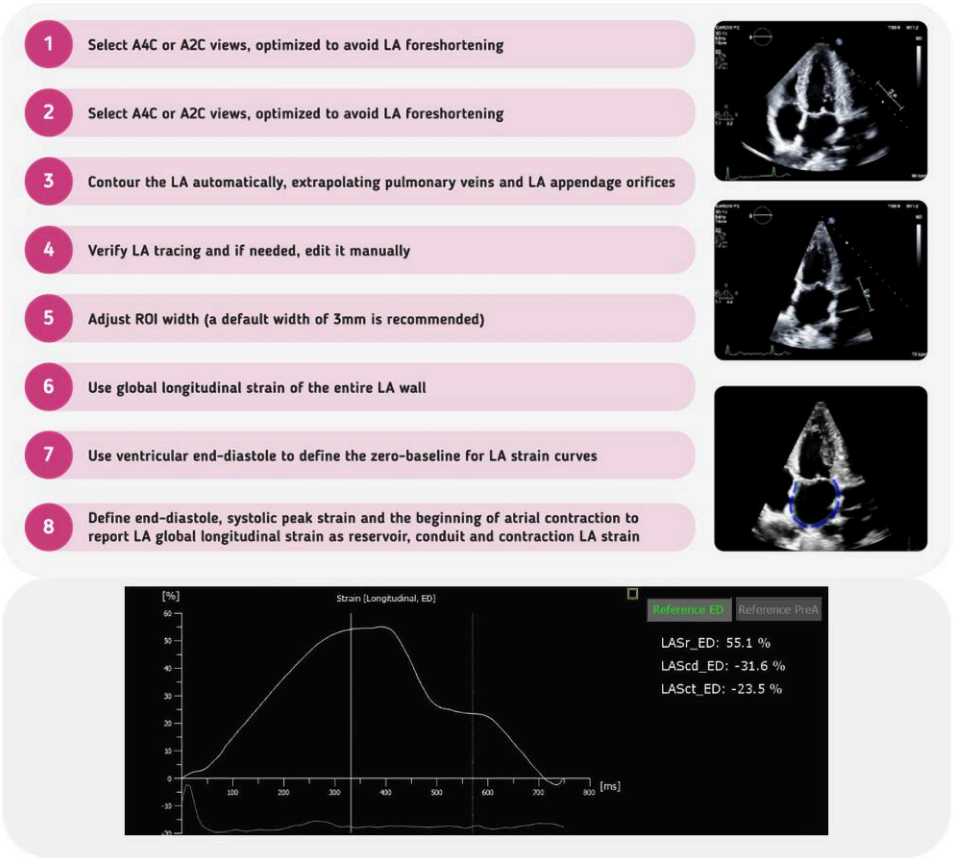


Figure 5 Image acquisition and computation of LA strain.

Table 3 Reference values of LA phasic strain (NORRE study)[19]

	All ages	Age 20–40 years	Age 40–60 years	Age >60 years
LAS reservoir	42.5 (36.1–48.0)	46.8 (42.3–52.4)	40.9 (35.4–46.1)	35.5 (30.9–41.9)
LAS contraction	−16.3 (−12.9 to −19.5)	−15.6 (−11.9 to −19.0)	−16.3 (−13.2 to −19.6)	−16.8 (−13.6 to −21.4)
LAS conduit	−25.7 (−20.4 to −31.8)	−30.6 (−26.8 to −36.5)	−24.1 (−19.7 to −29.3)	−18.6 (−14.7 to −22.6)

Values are given as median (interquartile range).

acquisition. The Simpson's method is more precise and overcomes the geometric assumption and limitations of the area–length method; however, it requires additional scanning time to acquire the LA short-axis stack. The reference values obtained from the two- and four-chamber cine images are given in *Table 4*.[34]

More precise evaluation of atrial morphology, especially of the LAA and PV, can be achieved by using 3D angiographic (MRA) images (*Figure 10C*) which can be acquired either using free breathing of navigator gated MRA after gadolinium injection or using non-contrast balanced SSFP MRA.[35] Like CCT, these images can be merged with 3D electro-anatomical maps using landmarks or surface registration for AF ablation[36] (*Figure 10E*). Evaluation of the anatomy of the PV pre-ablation and PV stenosis post-ablation is also feasible with CMR; however, less data is available as compared to CCT.[37]

CMR allows understanding of tissue characteristics and detection of atrial fibrosis by high-resolution 3D LGE imaging[38] (*Figure 10E*). LGE on the LA wall has been detected in several conditions such as AF,[39,40] mitral valve diseases,[41] and cardiac amyloidosis (CA).[42] Yet, assessment of atrial LGE is performed visually at best by experienced centres without a consensus for quantification and is not possible if the image quality is suboptimal. Variability among observers has been a significant disadvantage.[38] Further work is required to standardize atrial LGE imaging to enable its widespread clinical use.

Cine and 3D MRA allow also the assessment of LAA size and morphology and measurement of its ostial diameter and depth. For assessing LAA thrombus, CMR with inversion time myocardial delayed enhancement acquisition has the highest accuracy (99.4%) followed by contrast-enhanced (97.6%) and cine CMR 93.9%) tools.[43] Hence,

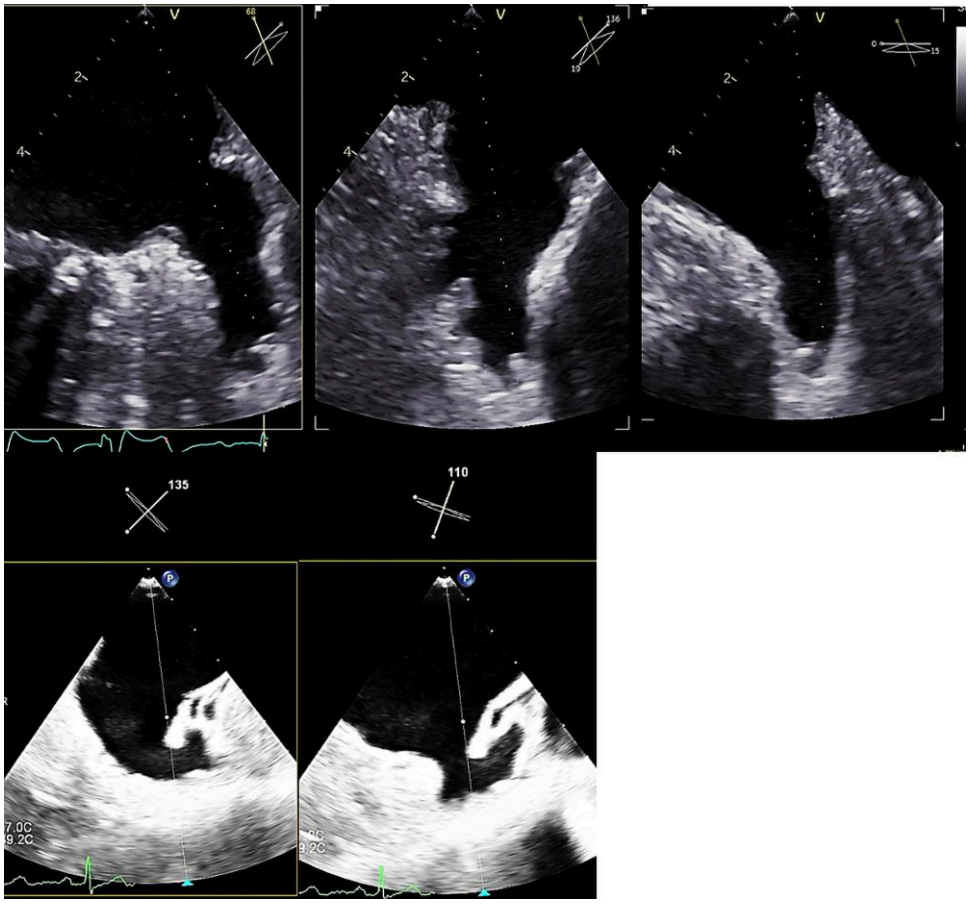


Figure 6 Multiplane images of the LAA showing different morphologies.

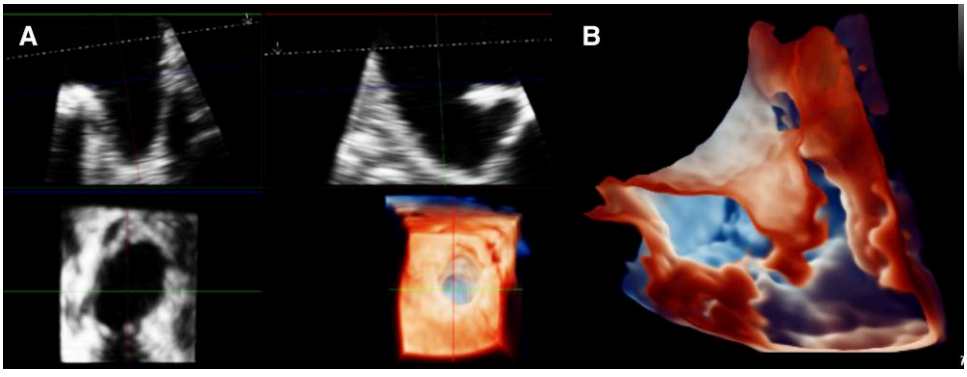


Figure 7 (*A*) Zoom-mode acquisition and multiplane display of LAA morphology by cropping and re-orienting the 3D data set with 3D photo-realistic rendering. (*B*) Glass view of the LAA.

CMR is an alternative to TOE or CCT in centres having adequate experience in LAA image acquisition and interpretation, depending on the resources.

Finally, CMR (T1-weighted or cine SSFP images) can accurately quantify the volume and area of pericardial adipose tissue.[44] A recent meta-analysis showed that LA epicardial adipose tissue (EAT) thickness was a strong parameter associated with the risk of AF recurrences after catheter ablation.[45]

CMR may suffer from artefacts in case of AF with high heart rate and devices (particularly intracardiac defibrillator).

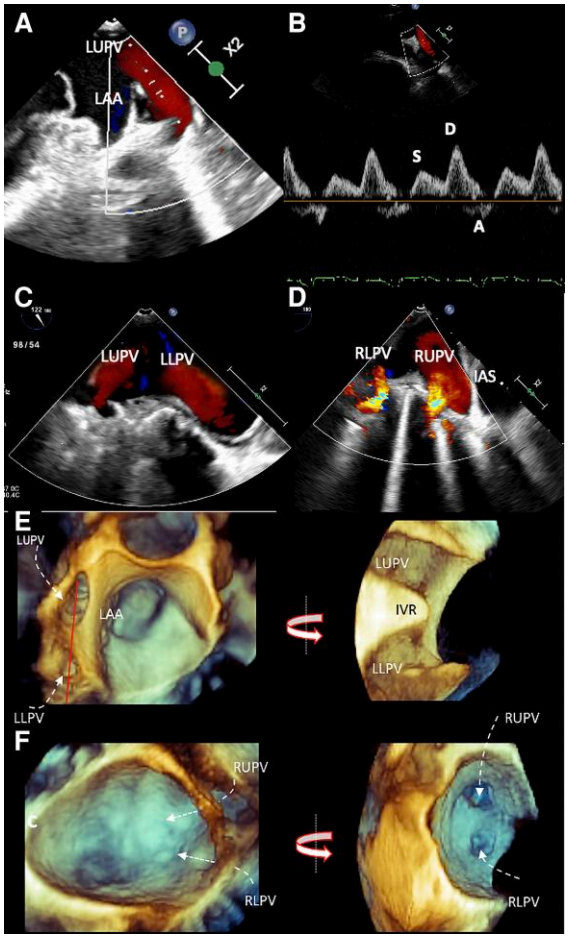


Figure 8 PVs by TOE. (*A*) Left upper PV, 60°, colour Doppler; (*B*) left upper PV, pulsed-wave Doppler; (*C*) left upper and lower PVs, 120°, colour Doppler; (*D*) right upper and lower PVs, 0° clockwise rotation; (*E*, *F*) the left and right PVs, 3D-rendered image.

Assessment of function

LA function can be assessed with CMR by computing LAEF from measurements of maximum and minimum volumes and phasic LA volume–time curves from the cine images (*Figure 10B*) (*Table 4*).[34,46,47] Recent technology enabled LA strain quantification by automated feature tracking methods from two- and four-chamber cine images (*Figure 10D*). A recent meta-analysis showed that feature tracking vendor matters for the heterogeneity of measurements rather than the CMR vendor, sex, and age.[48] The pooled mean values of LA phasic strain are presented in *Table 5*. There are promising advances in measuring peak velocity and vorticity by 4D flow imaging paving the way for the assessment of atrial haemodynamics[46,49] (*Figure 10F*).

CCT

Due to its excellent spatial resolution, CCT plays a pivotal role in defining the morphology of the LA, LAA, PVs, and function of the LA and in guiding electro-anatomical mapping. For evaluating LA, PVs, LAA morphology, and epicardial fat, a single arterial phase acquisition with ≥64-slice CCT with ECG triggering is required. Prospective ECG triggering is used in patients with sinus rhythm and low heart rate whereas retrospective ECG triggering is appropriate in patients with high and non-stable heart rates.[50] In patients who are in AF during the scan, acquisition is more challenging, but the introduction of more recent technology allows adequate image quality even during AF.[51,52] *Figure 11* shows a typical 3D LA reconstruction with CCT. An additional delayed scan after contrast injection is mandatory for ruling out LAA thrombus.[53] While the time to delayed images varied in studies, most were acquired 30–180 s after the initial images.

Assessment of morphology

CCT has a higher spatial resolution than CMR and is a well-established technique to evaluate LA and LAA morphology and volume and PV patterns, to rule out LAA thrombus, and to detect peri-atrial adipose tissue.

CCT systematically detects higher LA volumes compared to 2DE and CMR because of several reasons.[54] First, due to higher temporal resolution, the proper image reconstruction windows for

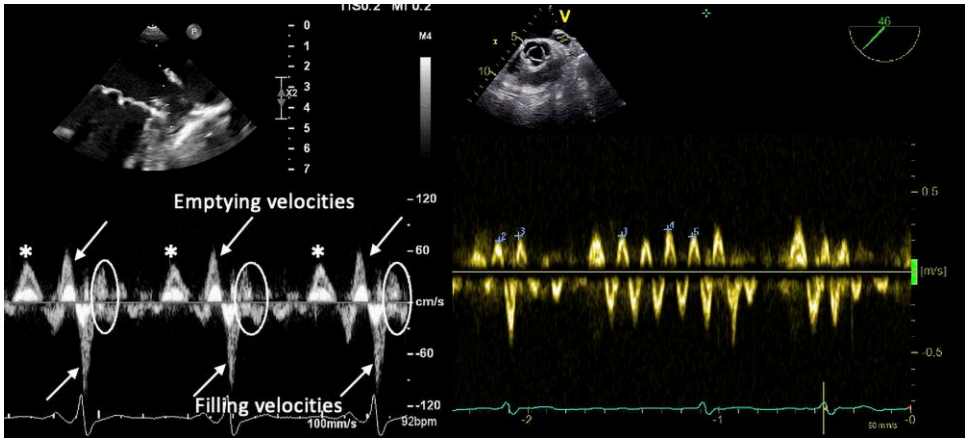


Figure 9 Normal quadriphasic wave pattern of LAA flow. Early diastolic emptying (asterisk), systolic reflection (circles) waves.

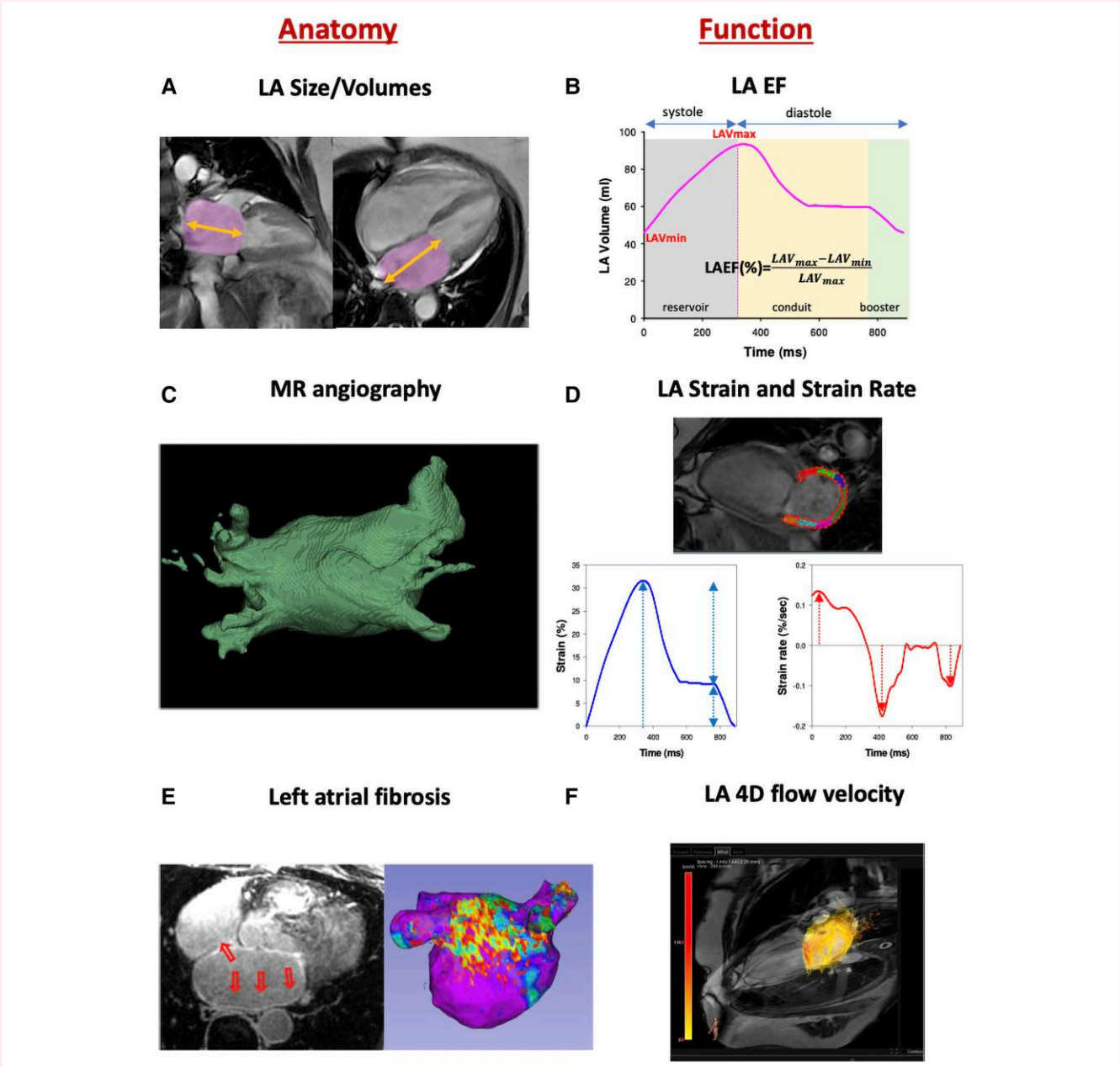


Figure 10 LA by CMR. (*A*) Area–length method from two- and four-chamber cine images. (*B*) Computation of reservoir, conduit, and contractile function and emptying fraction. (*C*) 3D anatomy by contrast or non-contrast-enhanced magnetic resonance angiography (MRA). (*D*) LA strain and strain rate. (*E*) LA fibrosis (arrows) by LGE and the computation of 3D maps of atrial fibrosis. (*F*) The myocardial blood flow distribution and velocity by 4D phase contrast.

accurate measurement of LA volume could be more achievable with 2D echo or CMR rather than CCT. Second, image noise during systolic phase may contribute to the overestimation of LA volume by CCT. Third, bolus injection of high-volume iodine contrast agent at high-rate infusion or drugs usually used for CCT scan such as β-blockers could modify LA morphology transiently, partly by incorporating more PV volume. *Table 6* represents the normal reference values of LA volumes by gender from 569 healthy subjects undergoing 320-detector CCT as a part of the Copenhagen General Population Study.[55]

CCT is now recognized as a good alternative for detecting LAA thrombus. Romero *et al.*[53] described a diagnostic accuracy of 94% of CCT vs. TOE to rule out LAA thrombus with 41% positive predictive value, because incomplete opacification of the LAA is common in patients with AF mimicking thrombus in acute phase scans. The positive predictive value increased to 92% with an overall diagnostic accuracy of 99% if delayed contrast imaging (venous phase) is added to arterial phase acquisition (*Figure 12*). CCT also clearly differentiates LAA morphologies as cactus, chicken wing, windsock, or cauliflower pattern.[56]

Contrast-enhanced CCT has potential for tissue characterization. Distribution of hypoattenuation is one way by which CT can identify myocardial fibrosis.[57] Also, new methods to perform extracellular volume quantification using CT are emerging.[58] CCT is the preferred

Table 4 Reference values of LA by CMR[34]

	Men		Women	
	Mean ± SD	Lower–upper limits	Mean ± SD	Lower–upper limits
Bi-plane LAVImax (mL/m^2)	38 ± 11	17–59	39 ± 11	17–61
Bi-plane LAVImin (mL/m^2)	14 ± 5	3–24	13 ± 5	4–23
Bi-plane LA EF (%)	62 ± 8	46–77	63 ± 8	48–78
Simpson LAVImax (mL/m^2)	41 ± 8	24–57	44 ± 8	28–60
Simpson LAVImin (mL/m^2)	19 ± 5	9–28	19 ± 4	11–27
Simpson LA EF (%)	54 ± 8	38–70	57 ± 6	45–69

Abbreviation: SD, standard deviation.

Table 5 LA strain reference values by CMR feature tracking[48]

	Mean	95% CI
LAS reservoir %	34.9	29.6–40.2
LAS conduit %	−21.3	−16.6 to −26.1
LAS contraction %	−14.3	−11.8 to −16.8

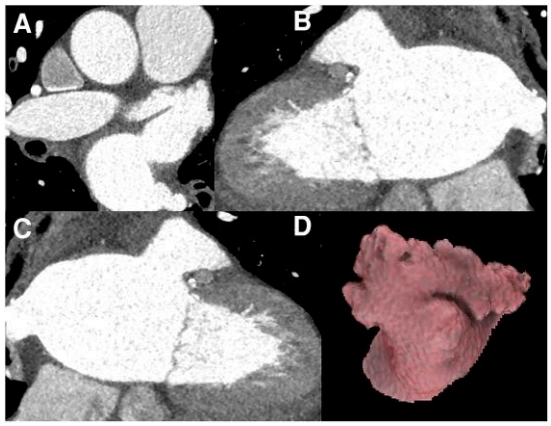


Figure 11 LAA. (*A*) Axial, *B*) sagittal oblique, *C*) coronal oblique views, and *D*) 3D volume-rendered image.

method to assess the PVs, although no differences are described between CCT and CMR in terms of diagnostic accuracy of PV pattern[51,54] (*Figure 13*).

CCT is accurate and reproducible to assess epicardial fat tissue (EFT) by either manual or semi-automated volume quantification. This latter algorithm defines all structures with contrast attenuation ranging between −195 and −45 HU as adipose tissue.[59] A volumetric quantification is advised rather than area measurements.[59] CCT also allows visualization of the peri-atrial anatomy for safe guidance of procedures and helps to avoid phrenic nerve injury or atrio-oesophageal fistula.[60,61]

Assessment of function

With ECG-triggered acquisition, functional series can be obtained in order to perform LA strain quantification. However, currently, data regarding LA strain assessment using functional CCT images are scarce. LA strain is usually obtained by tracing endocardial LA borders on multiple apical two-chamber views excluding LAA and PVs. Szilveszter *et al.*[62] demonstrated excellent intra-observer reproducibility for this approach with an intraclass correlation of 0.95, a correlation coefficient of 0.87, and 5.6 points of underestimation as compared to 2D echo. Yet, relatively low temporal resolution and radiation exposure limit the use of CCT for LA strain quantification in clinical practice.

Advice table 4 Use of CMR and CT

CMR is the gold standard for the quantification of peri-atrial adipose tissue volume.	
CCT is advised for assessing the anatomy of PVs.	
CMR and CCT are alternatives to echocardiography for the assessment of LA, LAA morphology, volume, and thrombus.	
Using the same imaging modality is strongly advised for intra- and inter-subject comparisons.	
CMR can be used as an alternative to CCT for assessing the anatomy of PVs.	
CMR with automated feature tracking can be used for LA strain quantification.	
CMR with LGE can be used for visualization of LA wall fibrosis with good-quality imaging, by the experts.	

Nuclear imaging

Molecular positron emission tomography (PET) imaging as a tool to assess atrial inflammation and fibrosis is in early stages. Increased atrial 18F-FDG uptake has been shown in patients with AF and in those with sarcoidosis as a predictor of subsequent AF.[63,64] More recently, 68Ga-fibroblast activation protein inhibitor (FAPI-PET) was used to detect increased fibroblast activation in the atria of patients with AF or after PV isolation.[65,66] Atrial activity is rarely reported but seems to be clinically relevant. Oncologic patients frequently have atrial uptake on their PET/CT which is also linked to the risk of AF and prothrombotic state with cardio-oncologic consequences.[64] Further investigations are awaited to consolidate clinical implications.

Table 6 Reference values (mean and 2.5–97.5%) for LAVI by CCT[55]

Age group	Men				Women			
	40–50	50–60	60–70	>70	40–50	50–60	60–70	>70
LAVImax (mL/m^2)	76 (47–108)	81 (53–118)	83 (44–114)	92 (67–154)	63 (43–91)	67 (49–96)	71 (49–102)	78 (55–124)
LAVImin (mL/m^2)	38 (25–54)	41 (27–58)	43 (21–63)	49 (36–74)	36 (25–50)	39 (28–54)	42 (30–56)	48 (38–77)

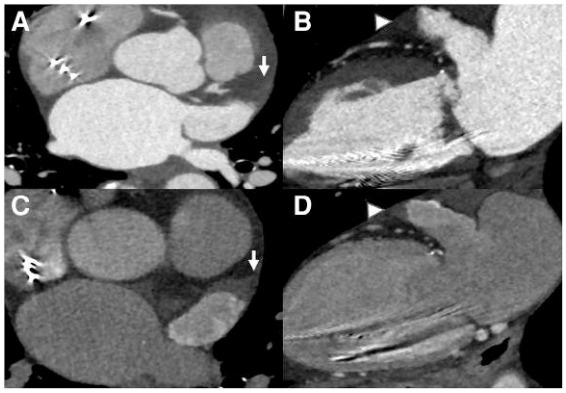


Figure 12 LAA thrombus. Arterial phase CCT scans (*A*, axial; *B*, oblique parasagittal three-chamber views) with endocavitary filling defect at the apex of the LAA, persisting into the late phase of imaging, 15 s later (*C–D*), consistent with thrombus (arrows).

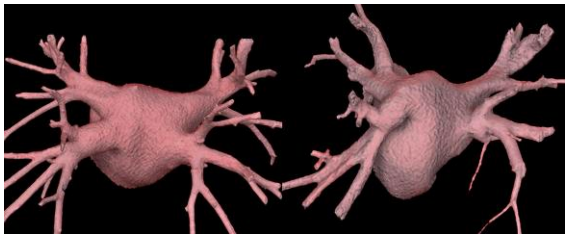


Figure 13 3D volume-rendered CT images of the LA. (*left*) Normal anatomy: four PVs draining into the superior posterior surface of LA. (*right*) A common anatomical variant characterized by the two left PVs draining into a common trunk.

Integration of LA imaging into diagnostic and prognostic evaluation

HF

Asymptomatic HF

The development of overt HF is preceded by HF risk factors with or without abnormal cardiac structure and function.[67] While LV structural (e.g. LV hypertrophy) or functional parameters [reduced EF or global longitudinal strain (GLS)] are classically used to define HF regardless of symptoms, evidence of atriopathy also seems to be a marker of HF. Thus, LA size (maximum and minimum volume) and function (LAEF, LASr, and LASct) might likely be incorporated into the definition of HF as an objective evidence of functional and structural abnormality. For example, in 1802 participants in the Dallas Heart Study, increasing left atrial volume index (LAVI) and decreasing LAEF were associated with hypertension and elevated brain natriuretic peptide (BNP), LAVI was most strongly associated with LVEDVi, and LAEF was associated with left ventricular ejection fraction (LVEF) and morphology. Reduced LAEF was independently associated with 8-year mortality in the general population and provided incremental prognostic utility to clinical risk factors, LV mass, and LVEF.[68] The availability of LA strain has made the detection of cardiopathy more feasible. In an analysis of 112 subjects with incident HF and 112 case–controls from the Multi-Ethnic Study of Atherosclerosis (MESA), atrial changes were present in CMR images obtained 8 years previously despite asymptomatic status. Specifically, subjects who developed HF had larger baseline LA volume index (40 ± 13 vs. 33 ± 10 mm^3/m^2, $P < 0.001$) and lower peak longitudinal LA strain (25 ± 11 vs. $38 \pm 16\%$, $P < 0.001$) years before. In fact, LASr was associated with incident HF independent of clinical risk factors, LV mass, and natriuretic peptides.[69]

HF with preserved ejection fraction and DD

During ventricular diastole, LA is exposed to the pressure of LV. With LV DD, LA pressure rises to maintain adequate LV filling, which in turn leads to dilatation and stretching of the LA wall. LAVI >34 mL/m^2 is one of the criteria for the diagnosis of DD.[70] However, LAVI has limitations during the early phases of DD.[71] In addition, LAVI increases with age and is modified by the percentage of age-predicted O_2 consumption (cardiorespiratory fitness).[72] Assessment of LASr in addition to the European Association of Cardiovascular Imaging (EACVI)/American Society of Echocardiography (ASE) criteria has been proposed for improving the diagnostic precision of the DD algorithm by decreasing the indeterminate cases.[73] LASr, unlike traditional parameters, deteriorates progressively with the severity of DD. Thresholds of LASr were proposed to separate normal from Grade 1 to 3 (35%), Grade 1 from 2 to 3 (24%), and Grade 3 from 1 to 2 DD (19%), respectively.[74]

While LAVI takes place in the algorithm of estimating LV filling pressure, LASr does not.[70,75] However, replacing a missing parameter in this algorithm with LASr (cut-off <18%) has been shown to facilitate the detection of increased filling pressure.[75] LASr is more sensitive in detecting elevated filling pressures even when LAVI is normal during sinus rhythm.[73,76] LASr <18% is associated with increased LV filling pressure (PCWP ≥15 mmHg) particularly if LVEF is <50%.[77]

Enlarged LAVI, reduced LASr, and GLS together with LV hypertrophy are supportive for the diagnosis of HF with preserved ejection fraction (HFpEF).[67,75] In the absence of a specific aetiology, recognition of DD and estimation of LV filling pressure are crucial for the diagnosis of HFpEF. Of note, the PARAMOUNT trial showed that LA phasic function and LASr are decreased independently of LAVI and the history of AF in HFpEF.[78] In the substudy of TOPCAT, LASr was associated with HF hospitalization and related to both LV systolic and diastolic function.[79] Although LASr is not cited among the diagnostic criteria of HFpEF,

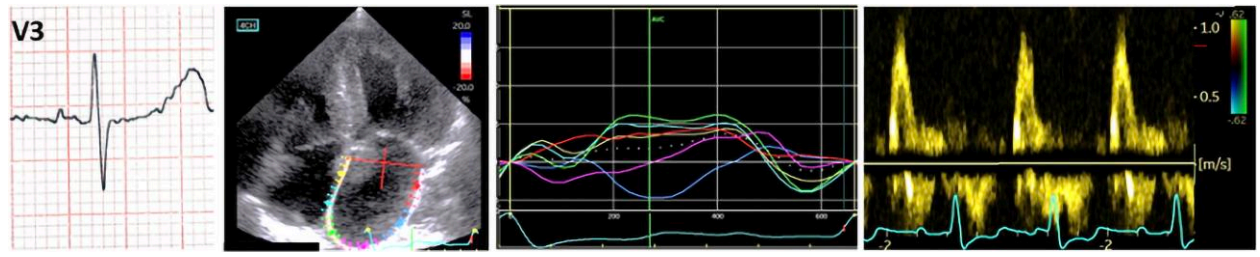


Figure 14 Atrial electromechanical dissociation in CA. Poor LA strain, absent LA contraction strain, and A wave, despite sinus rhythm.

abnormal LASr is associated with dyspnoea, NYHA class, and HF hospitalization and is a useful adjunct to the evaluation of DD and estimation of LV filling pressure algorithms in indeterminate cases.[73,79]

HF with mildly reduced and reduced EF

LA enlargement in HF with mildly reduced EF (HFmrEF) and HF with reduced EF (HFrEF) is associated with adverse cardiovascular events.[80] However, the impact of LASr on outcomes, in these patients, has been less studied. The best relationship between LASr and filling pressure is found in patients with reduced systolic function.[77,81] HFmrEF (EF = 41–49%) by definition needs the presence of symptoms and/or signs of HF. The presence of increased LAVI, elevated natriuretic peptides, and evidence of structural heart disease make the diagnosis more likely, but are not mandatory for diagnosis.[82]

Ischemic heart disease

Patients with ischaemic heart disease or after myocardial infarction (MI) make up the largest Stage B HF group. Accordingly, LA function and remodelling could be a marker of abnormal cardiac function with a diagnostic value. Additionally, LA function has been shown to predict HF hospitalizations after MI[83] and was incremental to LAVI.[84] A recent large CMR study showed that LAEF is independently associated with increased mortality in patients with ischaemic cardiopathy (LVEF <50%) even after adjusting for infarct size and MR severity.[85] LASr, assessed within 48 h of acute MI, was associated with the composite outcome of death and HF[86] and provided incremental value to LAVI in patients treated with percutaneous coronary interventions.[87] Data from multicentre prospective CMR studies [AIDA STEMI (NCT00712101) and TATORT NSTEMI (NCT01612312)] also showed that LASr (cut-off of 18.8%) is an independent predictor of outcome and incremental to LVEF, GLS, microvascular obstruction, and infarct size.[88] LAVI predicted morbidity and mortality after acute MI as well.[89,90] However, LA dilatation reflects a chronic process therefore may not be an ideal marker shortly after an acute MI in contrast to the indices of LA function that correlate more strongly to LV filling pressure after acute MI. Additionally, reduced LASr was shown to predict an increased risk of new-onset AF after coronary artery bypass graft surgery.[91]

Athlete's heart

LA dilatation is triggered by the increase in preload during athletic training as an adaptive mechanism.[92,93] Age, type of sport, and duration and intensity of training influence the degree of atrial remodelling. LAVI is associated with higher cardiorespiratory fitness and maximal oxygen consumption during exercise in both men and women.[94] A systematic review including 7189 elite athletes and 1375 controls described increased LAVI in athletes with an upper limit of normal 35.8 mL/m^2 compared to <34 mL/m^2 in the general population.[95] Spencer *et al.*[96] reported LAVI exceeding 48 mL/m^2 in 40% of male and 32% of female athletes. Importantly, there is a balanced adaptation with global remodelling in both atria and ventricles. Despite LA enlargement, *E*/*e*′ remains normal by means of increased LA and LV compliances and bradycardia and maintains LA pressure within normal range.[96] Conflicting evidence from relatively small cohorts exists about reversal of LA dilation with detraining.[93,97] In athletes, LASr is either preserved or mildly reduced (39%; 95% CI, 38–41%) compared to untrained controls,[92] and LA active emptying is lower in athletes (17%; 95% CI, 16–19%). Athletic atrial remodelling seems to be dependent on the intensity of training.[98,99] Adaptation of phasic volume changes during exercise enables distinction between physiological and pathological atrial remodelling.[100] Moderate exercise appears to protect against AF, whereas strenuous exercise increases the risk of AF which is postulated to be mediated by atrial dilatation, vagal tone, exercise-related adrenergic stimulation, and augmented LA pressure during exercise.[101,102] Increasing intensity and duration of athletic training leads to atrial enlargement and reduced atrial strain; only subtle further changes occur with AF. Therefore, prediction of AF in athletes by LA volume and strain is challenging as evidenced by conflicting results.[103–105]

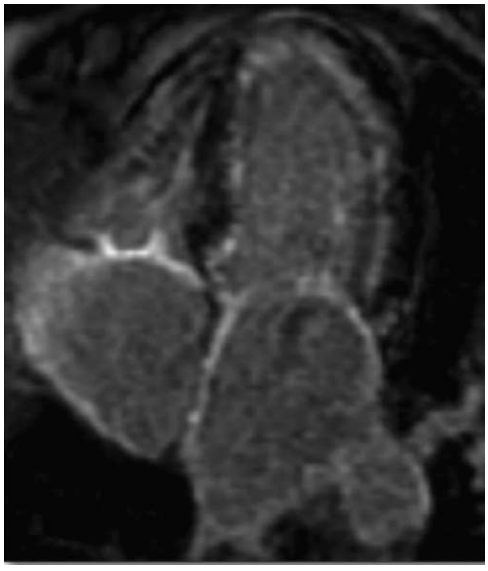


Figure 15 Diffuse LGE on the LA wall in CA.

HCM

LA remodelling is promoted by impaired LV filling and raised LA filling pressures in HCM. HCM may also cause direct LA cardiopathy as

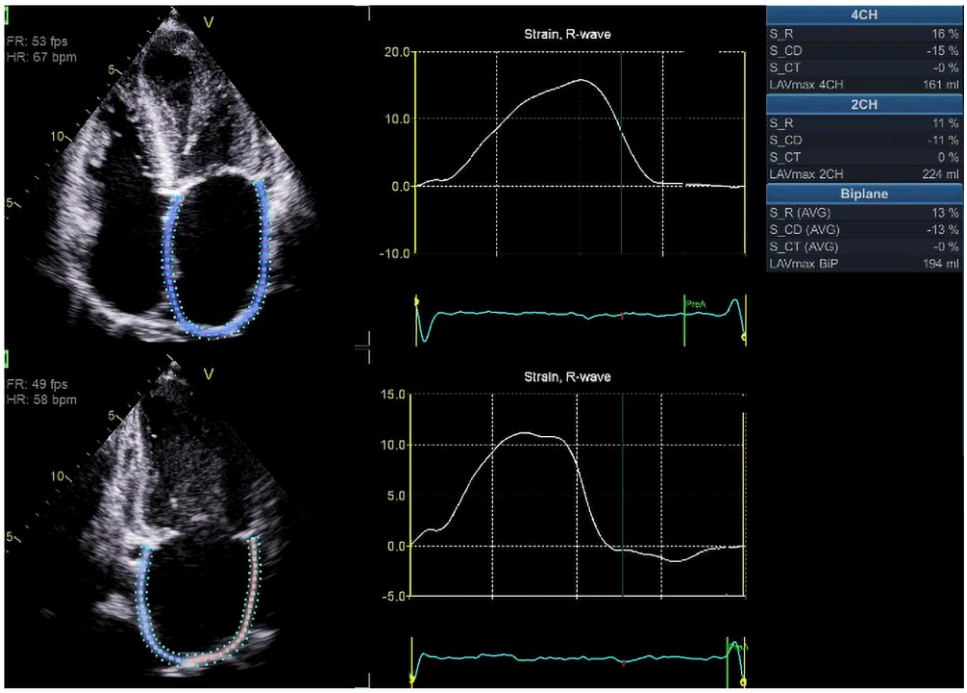


Figure 16 LA strain in AF. Note the lack of contraction; only the reservoir strain can be quantified which is significantly reduced.

evidenced by reduced passive and active LA emptying in the preclinical stage with positive genotype but without evident LV hypertrophy.[106] LA imaging and identification of AF risk are important because HCM is associated with a five-fold higher risk of AF incidence as compared with the general population and an increased rate of cardioembolism.[107] Increased LA volume, reduced atrial EF, and reduced LASr have been found to predict incident AF in the HCM populations.[108,109] A high burden of atrial LGE on CMR was reported in patients with HCM and AF.[110] Adverse LA remodelling in HCM has been shown to be a marker of poor outcome.[111,112] LA diameter is a component of the sudden cardiac death risk scoring system in HCM patients as validated in 2014.[113] The utility of more novel LA metrics has not been tested in identifying sudden cardiac death risk in large cohorts. Treatment of HCM is associated with LA structural and functional changes. Hegde *et al.*[114] documented reductions in LA volumes and improvement in LV diastolic function and natriuretic peptide levels after treatment with mavacamten. Finally, regarding the controversy of exercise training in HCM, a similar LAVI increase was observed with competitive exercise in athletes with and without HCM.[115]

CA

Both primary LA cardiopathy from amyloid accumulation-mediated damage and secondary involvement due to increased LV filling pressure, MR, and AF occur in CA.[116] Amyloid infiltration typically increases the thickness and stiffness of the atrial wall and IAS preventing excessive dilatation. Consequently, deformation-based parameters become more relevant than LA size for risk stratification in this population. Poor LASr and poor or absent LASct are typical findings in CA.[117] The increase in LA stiffness can be estimated by the ratio *E*/*e*′/LASr.[28] During ventricular systole, the LA acts as a non-distensible (stiff) reservoir causing increased LA pressures and reducing the energy stored in the walls which affects the conduit phase. Finally, a lack of atrial mechanical contraction can be observed in a proportion of the patients

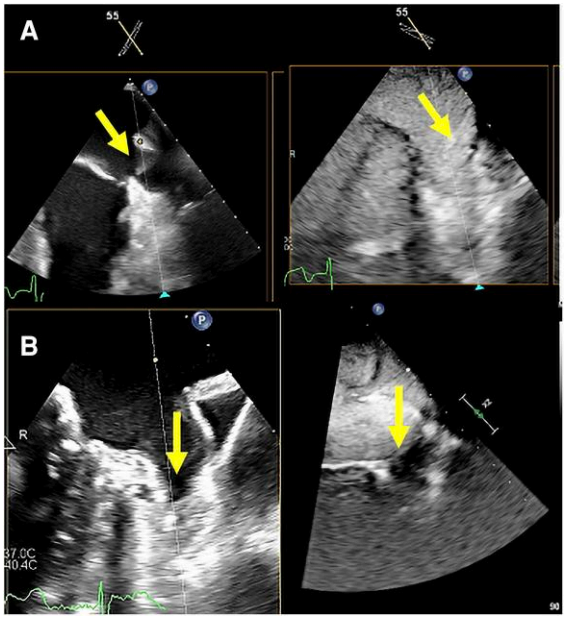
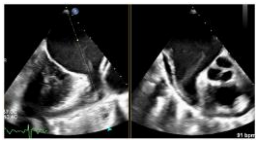
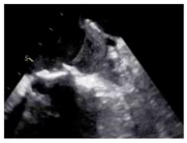
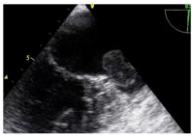


Figure 17 Ultrasound contrast for LAA opacification: (*A*) artefact mimicking thrombus, washing out with contrast, (*B*) thrombus producing a filling defect with contrast.

despite sinus rhythm, i.e. atrial electromechanical dissociation as a distinct feature and poor feature and poor prognosticator in CA (*Figure 14*).[116] LA strain is independently associated with high thrombotic risk in patients with CA.[118] LGE due to amyloid deposition or fibrosis in the LA wall can be detected by CMR[42] (*Figure 15*) and is

Table 7 Clinical characteristics of SEC, sludge, and thrombus[56]

	SEC	Sludge	Thrombus
Prevalence	 ≈50%	 1–14%	 13%
Echocardiographic characteristics	Smoke-like echogenicity with variable density. Grade 1: minimal dynamic echogenicity in the LAA or sparely distributed in the LA; transient during cardiac cycle; Grade 2, swirling pattern with similar distribution to Grade 1; Grade 3, constantly detectable dense swirling pattern in the LAA that spills into the LA with less dense intensity; Grade 4, very slow swirling dense smoke-like echoes in the LAA, extending with similar density into the LA. Full, opacification with contrast, no filing defect with colour Doppler[a]	Echo density with viscid gelatinous features but without a solid component. Opacification with swirling contrast, no filling defect with colour Doppler[a]	Echo dense mass with margins and motion distinct from the atrial wall. Filling defect with colour Doppler[a], echo-free area with contrast
Thromboembolic risk	↑	↑↑	↑↑↑

[a]Low Nyquist limit, 25–35 cm/s.

Table 8 Imaging markers of cardioembolic risk

TTE	TOE	CMR	CCT
LA volumes	LAA emptying velocity SEC in LA, LAA	LA volumes	LA volumes
LA strain	Sludge/thrombus in LA or LAA	LA fibrosis	SEC in LA, LAA
SEC in LA	LAA non-chicken wing morphology	LA strain	LA or LAA thrombus
Thrombus in LA	PFO	LA 4D flow	LAA non-chicken wing morphology

associated with reduced LA function.[119–122] LA cardiopathy holds diagnostic[122] and prognostic significance for all-cause mortality and increased risk of AF development and cardioembolic events in CA.[118,122]

MR

LA dilation is an adaptive response to volume overload in patients with progressive MR.[123,124] Furthermore, enlarged LAVI identifies individuals at increased risk of mortality, independent of the severity of MR or AF.[125] The 2021 European Society of Cardiology (ESC)/European Association of Cardiothoracic Surgery (EACTS) Guidelines for the management of valvular heart diseases recommend early surgical mitral valve repair in low-risk asymptomatic patients with severe primary MR when LAVI ≥60 mL/m^2 or LA diameter ≥55 mm.[123] In addition to LA dilation, reduced LASr has been independently associated with all-cause mortality in patients with significant primary and secondary MR and has shown incremental prognostic value over LAVI and LV GLS.[126,127] LA fibrosis that occurs in the process of MR also reduces LASr.[128] The impact of mitral valve repair on the reversibility of LA fibrosis is currently investigated by LGE CMR (NCT05345730).[41] In atrial functional MR, which occurs most commonly in the setting of chronic HFpEF or AF, LA dilatation is deemed to be the main driver of MR through annular dilatation.[129] LA reverse remodelling after mitral valve repair is a favourable prognosticator but depends on several factors including pre-operative LAVI, MR severity, post-operative trans-mitral pressure gradient,[130] and intrinsic atrial cardiopathy. Recently, bi-leaflet prolapse was found to be associated with reduced LA function regardless of MR severity, suggesting a primary cardiopathy in these patients.[131]

MS

The pressure overload in MS promotes excessive dilatation of the LA with decreasing deformability, compliance, and contraction. In rheumatic MS, rheumatic atrial cardiopathy further exacerbates LA enlargement leading to one of the largest LAs observed in humans. The assessment of LA remodelling in MS includes LA size, EF, emptying fraction, PV flow patterns, LAA function, and LA deformation. From a clinical standpoint, LA compliance rather than the size is instrumental for mitigating pulmonary hypertension and increasing stroke volume downstream, whereby modulating symptoms in MS.[132] LA reservoir function, quantified by longitudinal strain, reflects LA compliance and is a function of pure MS in young subjects. Of note, concomitantly reduced LV compliance would affect LA compliance, LASr, conduit, and pump strain in the elderly[75] which is typical for degenerative MS with mitral annular calcification. LA dilatation and reduced LASr predict symptoms, hospitalizations, valve intervention, recurrence of functional tricuspid regurgitation after tricuspid valvuloplasty,

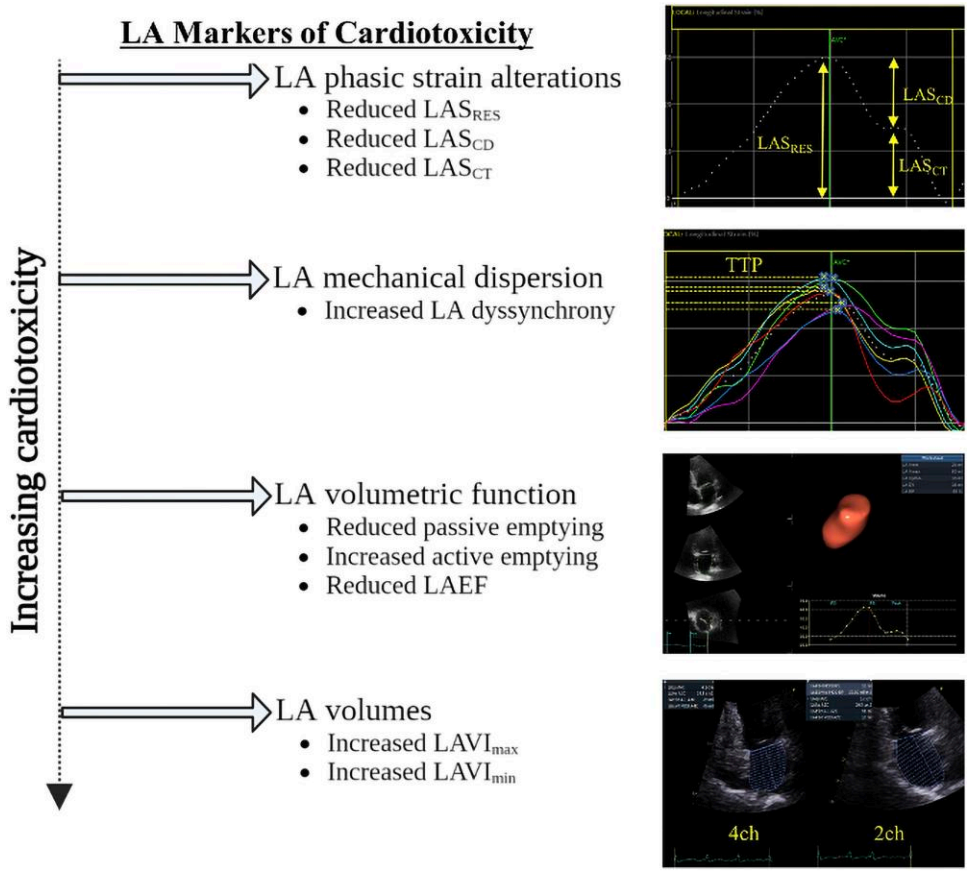


Figure 18 LA markers of cardiotoxicity.

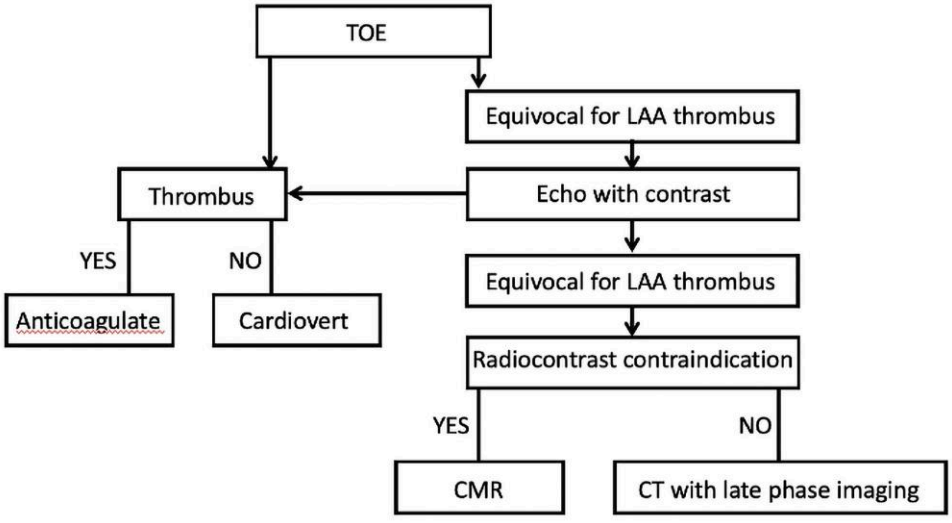


Figure 19 Imaging algorithm for direct-current cardioversion.

and AF and thromboembolic complications.[133–140] Anticoagulation with vitamin K antagonists should be considered if LAVI exceeds 60 mL/m^2 in patients with rheumatic MS even in sinus rhythm according to the 2021 ESC/EACTS Guidelines for the management of valvular heart disease.[123] Importantly, LASr and LA compliance were shown to improve following balloon mitral valvuloplasty, and this improvement translates into functional capacity.[141,142] Similarly, LAA contraction was shown to improve after balloon mitral valvuloplasty.[143]

AF

Atrial contraction is abolished during AF. Atrial volume increases, while LA reservoir function decreases (*Figure 16*). LA remodelling and fibrosis

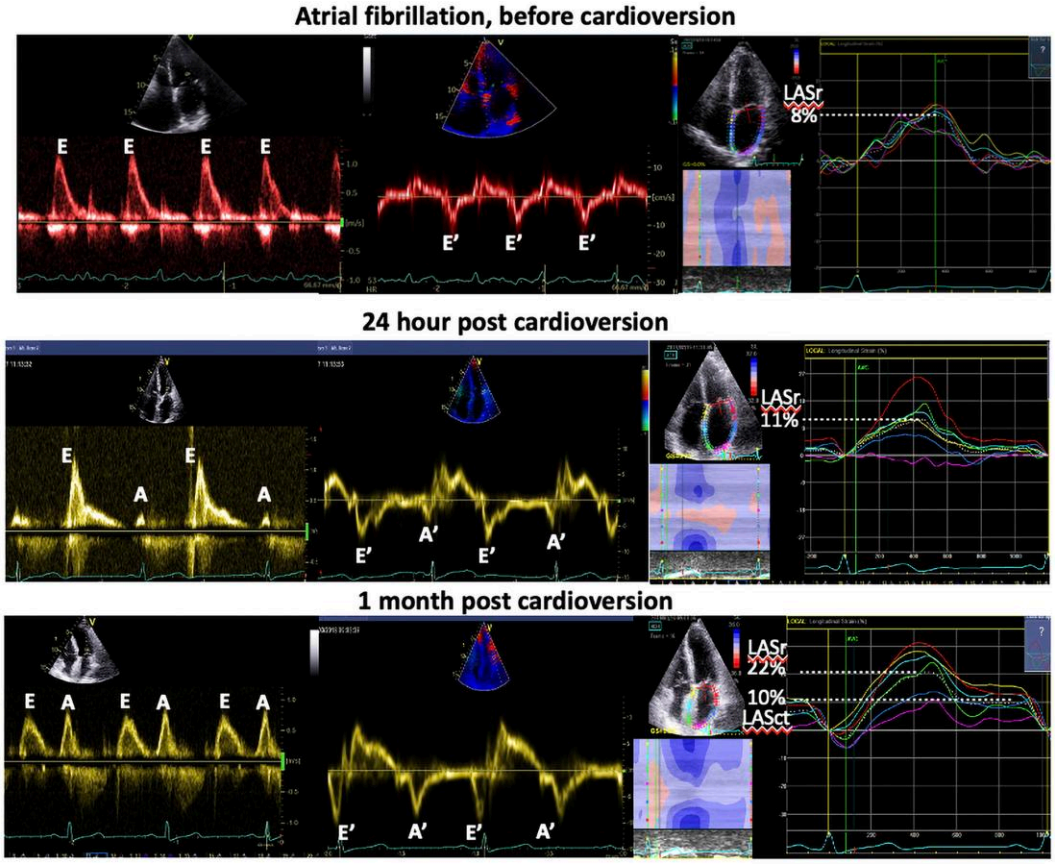


Figure 20 LA function before, early after, and late after cardioversion. Note the regeneration of LA contraction (A, a′, LASct).

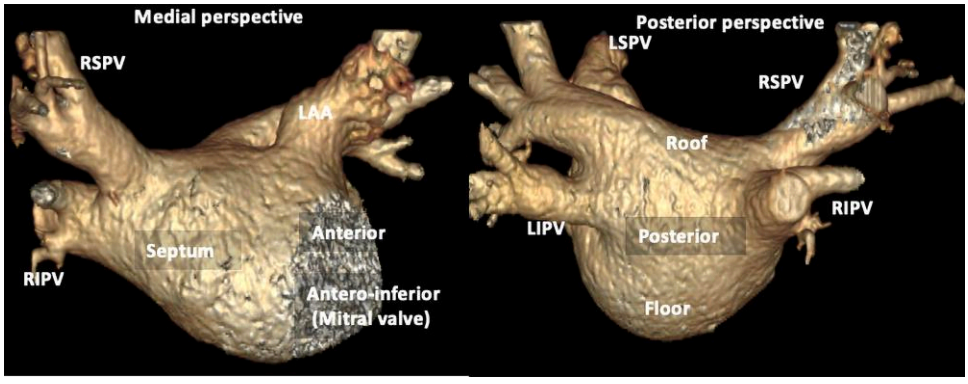


Figure 21 Demonstration of different parts of LA from a 3D volume-rendered reconstruction by contrast CCT performed for the assessment of PVs prior to PV isolation.

contribute to the perpetuation of AF.[144,145] Dilatation of the LA predicts the development of AF in the elderly and its predictive value is incremental to linear measurements.[146] Likewise, LA enlargement predicts AF recurrence after radiofrequency ablation or cardioversion.[147] Total atrial conduction time which is the time interval from the onset of P wave to atrial contraction (tissue Doppler *a'*) has been considered as a marker of LA fibrosis and shown to predict AF recurrence in patients following rhythm control strategy.[148]

LASr is an early marker of altered structure and impaired function.[149] In AF, measurements are averaged from five consecutive cycles. Only LASr can be computed during AF.[24,25] LASr predicted AF development in patients at risk in the general population,[150] in patients undergoing cardiac surgery, and in patients after a first stroke while in sinus rhythm.[133,151,152] Hence, LASr has the potential to improve risk stratification for AF development and monitoring strategies.[153] From a prognostic standpoint, improvement in LASr is associated with a higher rate of sinus rhythm maintenance after cardioversion or ablation.[154–156] Furthermore, an inverse relationship has been shown between LA strain and the extent of fibrosis measured by LGE CMR.[157] Of note, for diagnostic and prognostic purposes, in other diseases such as HFpEF and MR, LA enlargement and LASr should be interpreted with caution in the presence of AF.

Several studies showed that larger amounts of epicardial fat were associated with an increased risk of AF.[158] Moreover, Wong *et al.*[159] showed a more robust association of AF with epicardial fat as compared to abdominal or overall adiposity.

Thromboembolism

Visualization of thrombus

Thrombi can be found free-floating or attached to the LA wall, in-transit across the patent foramen ovale (PFO), on prosthetic materials (valves, occluders), or in the LAA. LAA is the most common location of thrombi (98%) in non-valvular AF because of stasis in the blinded pouch.[160,161]

TOE is the most frequently used tool to diagnose thrombus (sensitivity 93–100% and specificity 99%) in the LAA.[162] The use of ultrasound contrast agents improves the diagnostic accuracy of TOE for detecting thrombus (*Figure 17*).[163] False-positive results are frequently due to the misdiagnosis of pectinate muscles as thrombi. Differential diagnosis also includes masses with a potential for embolization such as fibroelastomas, tumours, or vegetations. Contrast-enhanced CMR is the method of choice to make the differential diagnosis of intracardiac masses.[164]

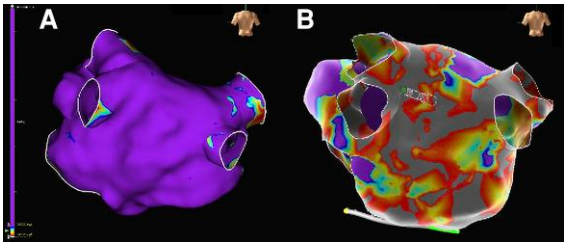


Figure 22 Electro-anatomical colour-coded voltage map (violet voltage >0.5 mV). (*A*) Normal voltage of the posterior wall. (*B*) Low-voltage areas and scar (grey colour) in native atrial cardiopathy.

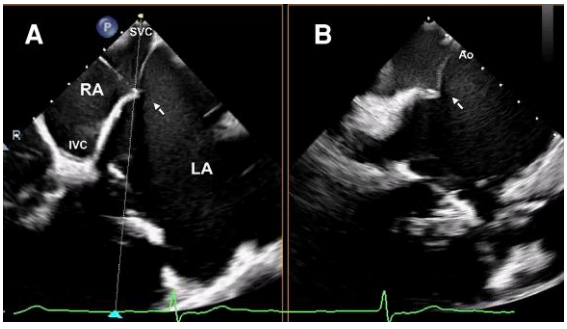


Figure 24 Trans-septal access with 3D ICE catheter using 2D X-plane imaging. A) the inter-atrial septum (IAS) is imaged in the superior and inferior orientation with the ICE catheter tip retroflexed. B) X-plane showing the anterior (Ao) and posterior. SVC- Superior vena cava; IVC = Inferior Vena Cava.

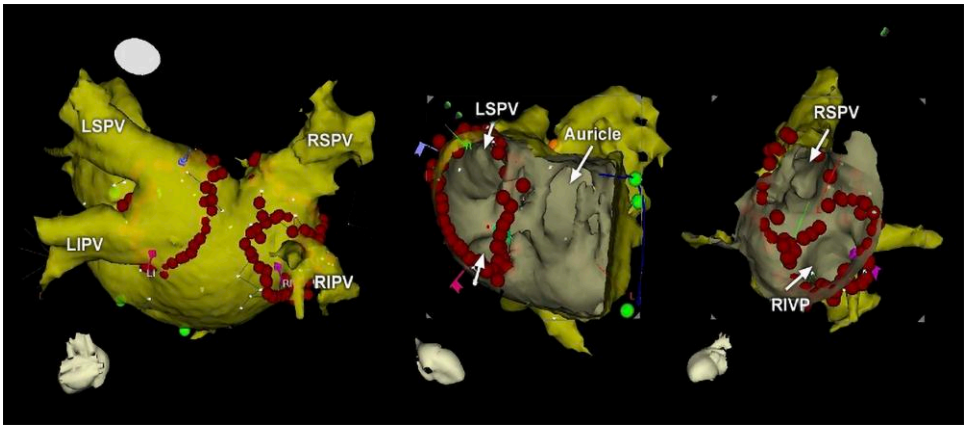


Figure 23 Integration of pre-acquired CCT reconstruction with electro-anatomical map used to guide PV isolation. The circle tags indicate radiofrequency energy delivery sites at the PV ostia. LSPV, left superior PV; LIPV, left inferior PV; RSPV, right superior PV; RIPV, right inferior PV.

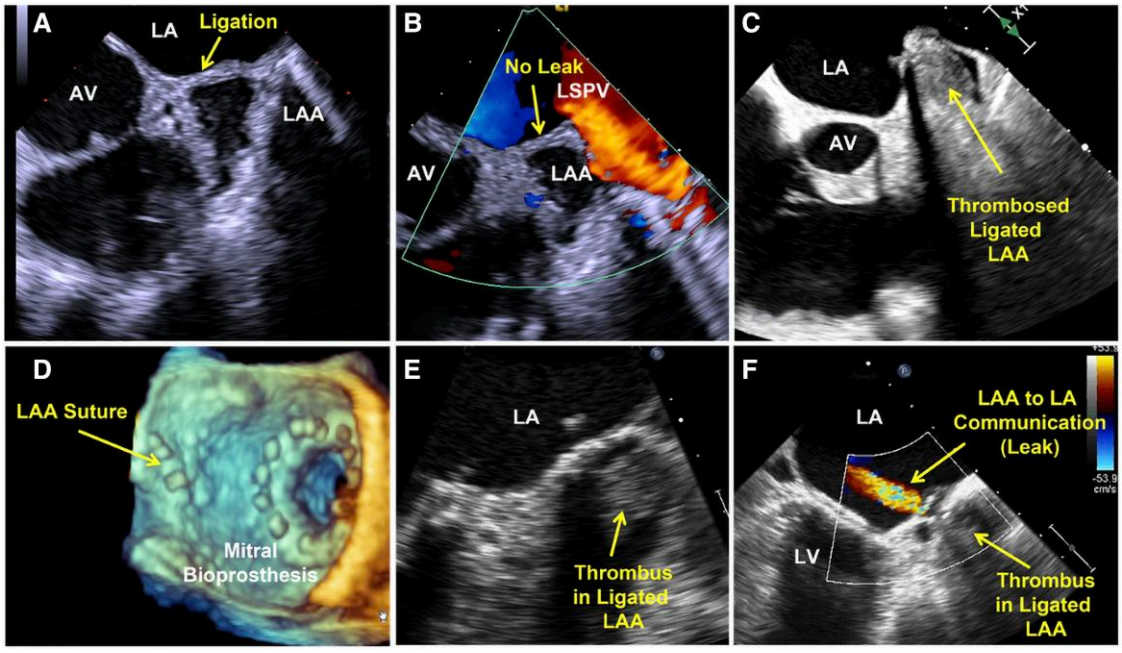


Figure 25 Surgically ligated LAA. A,B) surgically ligated LAA with no residual leak, C) subsequent tissue ingrowth and thrombus formation inside the ligated LAA, D) suture line, E) thrombus inside an incompletely ligated LAA, F) the residual LAA to LA communication.

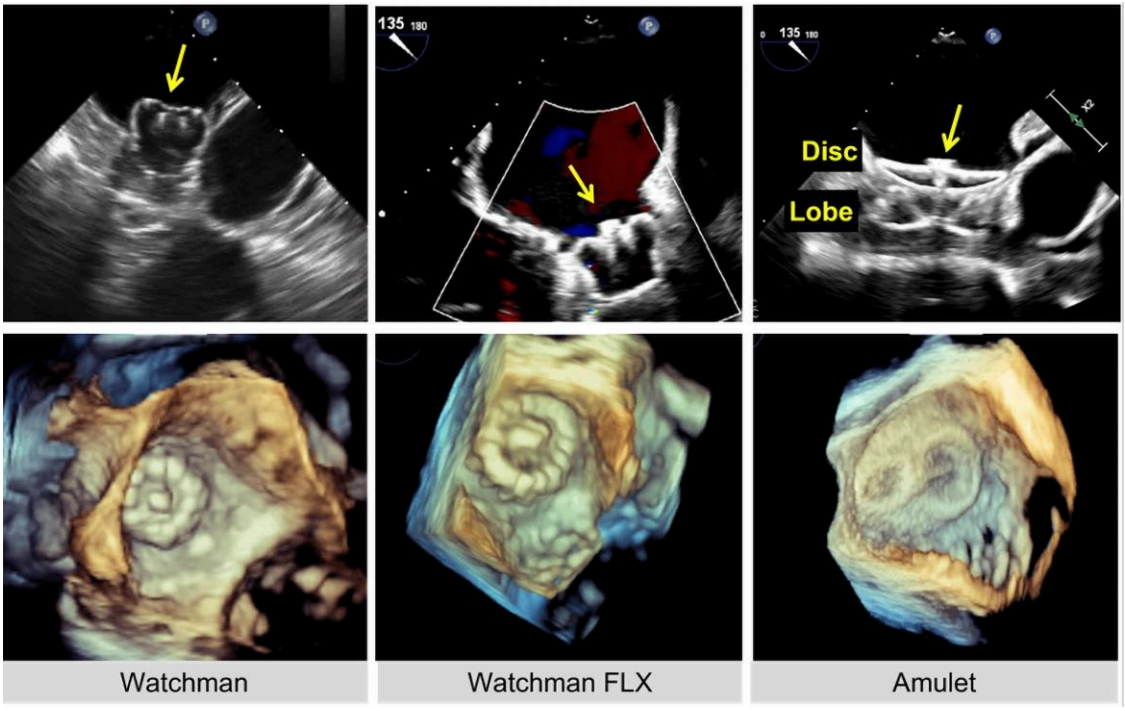


Figure 26 LAA closure devices correctly deployed seen by 2D saggital and 3D en face views.

CCT and CMR with dedicated protocols that ensure full replenishment of the LAA by contrast (see CMR and CCT) are also highly diagnostic (99%) for detecting LAA thrombus (*Figure 12*).[53,165–168] However, CCT and CMR are less well studied for detecting circulatory stasis within the LAA.[166,168]

Cardioembolic risk assessment

Imaging provides comprehensive information about thromboembolic risk: mitral valve prostheses, PFO, atrial septal defect (ASD), LA dilatation, loss of LA contraction, thrombus, and spontaneous echocardiographic contrast (SEC) are risk factors that are detected by imaging, for cardioembolic events. SEC is a marker of stagnation associated with low flow velocities in the LAA (<20 cm/s) and a precursor of thrombus.[169] The presence of dense SEC is a strong risk factor for thromboembolic events.[170–172] In terms of severity, SEC is graded semi-quantitatively[173] (*Table 7*). On the other hand, sludge gives the impression of impending thrombus and is associated with thromboembolism and all-cause mortality.[174] Following cardioversion, LAA and LA mechanical contractile dysfunction or 'stunning' develops in 38–80% of cases, with the formation of SEC.[175] Importantly, SEC does not disappear with anticoagulation.

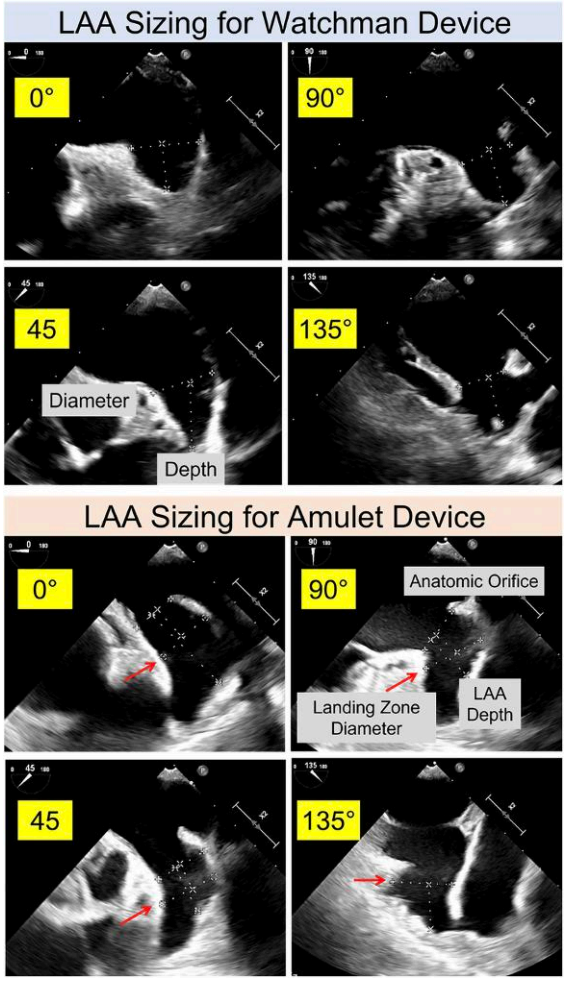


Figure 27 LAA Sizing by 2D TOE at 4 angles, 45 degrees apart (0, 45, 90 and 135°).

The size of LAA (>34 cm^3), the number of lobes ≥3, a large 8non-chicken wing (cauliflower in particular) morphology, and low-velocity flow are considered risk factors for thrombus formation.[176–178] The risk of thrombus development in the LAA increases with velocities ≤55 cm/s.[176,177] Emptying velocity <20 cm/s is specifically associated with LAA thrombus formation.[32] The ratio of LVEF/LAVI <1.5 showed 100% sensitivity for predicting the presence of LAA thrombus in patients with non-valvular AF.[179,180]

AF associated with MS, HCM, CA, or CHA_2DS_2-VA score >1 is an indication of anticoagulation[181] (vitamin K antagonists for moderate-to-severe MS). However, the temporal dissociation between the AF episodes and embolic stroke,[182,183] rhythm control strategies that failed to reduce the risk of stroke, suggested the possible impact of LA cardiopathy and metabolic factors to thrombogenic substrate.[184] Furthermore, LA dilatation[185,186] reduced deformation,[187–189] fibrosis,[190,191] and other atrial cardiopathy markers were shown to be associated with cardioembolic risk regardless of AF.[192–194] LA fibrosis and LA strain were also shown to provide incremental risk prediction over CHA_2DS_2-VASc score and LA volume.[187,195] The extent of LA fibrosis on LGE CMR was associated with LAA thrombus, stroke, and incident AF.[196,197] Likewise, patients with reduced LA strain had a higher incidence of LAA dysfunction and LAA thrombus in non-valvular AF.[198] Ongoing studies are further exploring the relationship between atrial fibrosis and stroke (NCT03830983).[199] Various markers of cardioembolic risk can be obtained from different imaging modalities (*Table 8*).

Chemotherapy-related cardiotoxicity

This section mainly focuses on the most commonly encountered anthracycline and trastuzumab toxicity. LA dilation was reported as an indicator of cardiotoxicity.[200–205] Alterations in LA function without changes in volume,[206–209] reduced passive emptying with increased active emptying,[205,206] and reduced LA emptying fraction by 3DE[210] and CMR[202] have been reported. Also, reduced LASr and conduit strain,[206–208] decreased LA contractile strain, and prolonged mechanical dispersion have been noted

Advice table 5 Multi-modality imaging of LA and LAA for disease-oriented purposes

Heart failure	
Assessment of LASr is advised as an adjunct to EACVI/ASE criteria of DD.[70,73]	
Both LASr and LAVI quantification is advised whenever HFpEF is suspected or established.	
Quantification of LASr and LAVI is advised as part of the assessment of functional and structural cardiac abnormality (previously designated as Stage B HF).	
Ischaemic heart disease	
Assessment of LAVI and LASr may be appropriate for risk stratification in acute and chronic ischaemic heart disease independently of LV ejection fraction.	
Athlete's heart	
It is advised to assess LA enlargement as an adaptative mechanism in athletes.	
LA size or strain is not suitable for differentiating adaptive from pathological remodelling in athletes.	
Hypertrophic cardiomyopathy	
Assessment of LA cardiopathy (dilatation, dysfunction, and fibrosis) is integral to the evaluation of HCM as an adjunct to diagnosis and prognostication.	

Cardiac amyloidosis

Assessment of LA cardiopathy (dilatation, dysfunction, and fibrosis) is advised for the diagnosis and prognostication of CA

Identification of atrial electromechanical dissociation is a distinct clinical phenotype indicating poor prognosis in CA

Mitral regurgitation

In primary MR, it is appropriate to evaluate LA size for making decision about mitral valve intervention.

It is appropriate to evaluate LA dilatation, annular dilatation, and altered annular contraction for the diagnosis of atrial functional MR.

Mitral stenosis

Quantification of LA function and remodelling complements the haemodynamic assessment of MS and prognostication.

Atrial fibrillation

Assessment of LAVI and LASr is advised to improve prediction of the risk of AF development, persistence, and recurrence during sinus rhythm in subjects with CHA_2DS_2-VA score >1 (excluding gender), moderate-to-severe rheumatic MS, HCM, and CA.

Cut-offs for abnormal LA size and function are not valid in the presence of AF for diagnostic and prognostic purposes.

Thromboembolism

The use of ultrasound contrast agents is advised to increase the accuracy of TOE as needed to rule out cardiac thrombus.

TOE and delayed contrast imaging with CCT or CMR with inversion time myocardial delayed enhancement are appropriate to rule out LA/LAA thrombus.[181,216]

Assessment of LA cardiopathy by volume, strain, and LAA dysfunction is advised as risk modifiers during sinus rhythm.

Anticoagulation within the therapeutic range should not be interrupted before TOE.

LA cardiopathy alone is not considered a valid argument for anticoagulation.

(*Figure 18*).[211] LA function parameters are sensitive markers and usually precede LA volume increase. They correlate with LV DD and AF generation. Of note, Ibrutinib-related AF is a well-known cardiotoxic complication for which LASr was found as an important predictor.[212,213] Cut-offs for LA cardiotoxicity are not standardized, with studies using absolute LASr <35% or <10th percentile, ≥10–15% reduction from baseline or comparison to controls. Yet, the prognostic utility of LASr in predicting cardiotoxicity remains debatable.[214,215]

Peri-procedural imaging of LA and LAA for outcome optimization

Cardioversion

The Assessment of Cardioversion Using TOE (ACUTE) multi-centre trial was the first randomized prospective study that introduced TOE-guided cardioversion into clinical practice.[217,218] Today, the TOE-guided approach is the common strategy when there is a sub-therapeutic or interrupted anticoagulation over the last 3 weeks to expedite cardioversion[181,219–222] (*Figure 19*). Atrial stunning is maximum immediately after cardioversion and improves typically over 4 weeks (*Figure 20*).[223] It is important not to stop the anticoagulation during atrial stunning. Of note, full recovery of mechanical function may extend beyond 3 months in almost 50% of the patients, depending on the duration of AF, atrial size, and structural heart disease.[224]

Peri-AF ablation

Depending on experience and resources, CCT and ICE are used in clinical practice for visualizing LA/LAA thrombus before catheter ablation as alternatives to TOE. The following groups are considered high risk for thromboembolic complications: (i) patients with CHA_2DS_2-VA >1 but have not received therapeutic anticoagulation during the last 3 weeks; (ii) patients having CA, HCM, and rheumatic moderate-to-severe MS, despite therapeutic anticoagulation within the last 3 weeks; and (3) patients with a history of thrombus despite therapeutic anticoagulation.[181,225] Imaging should be close in time to the procedure, ideally within 48 h. The anatomic modelling of PVs before AF ablation may impact the ablation approach and can be accurately performed by CCT or CMR with contrast.[216,225,226] Of note, there is less data with CMR for PV modelling. Therefore, CCT is more frequently used than CMR for clinical practice (*Figure 21*). Imaging for PV stenosis post-ablation is only required if patients are symptomatic.[225]

Electroanatomic mapping (EAM) systems serve to obtain real-time anatomical information from the LA, LAA, and PVs and identify low-voltage areas that may be additional targets for AF ablation beyond PV isolation (*Figure 22*).[227,228] Preprocedural CCT images loaded and merged with the real-time mapping system can facilitate anatomic reconstruction, pre-acquired delayed enhancement CMR sequences may assist with the identification of scar extent and location[39,228–230] (*Figure 23*). Although integration of pre-acquired CCT or CMR images was thought to decrease the fluoroscopy duration, previous randomized studies and a new meta-analysis demonstrated that integration of pre-acquired CCT or CMR images does not improve outcomes from AF ablation.[216,225,226,231]

Although among patients with AF undergoing catheter ablation, fibrosis estimated by delayed enhancement was found to be associated with the likelihood of recurrent arrhythmia in the Determinant of Successful Radiofrequency Catheter Ablation of Atrial Fibrillation (DECAAF) study,[232] the DECAAF-2 study did not demonstrate the benefit of adding CMR-guided fibrosis ablation to PV isolation for preventing atrial arrhythmia recurrence.[39] CMR may allow visualization of completeness of post-ablation lesions which is relevant for the recurrence of AF.[233,234]

CCT is the preferred imaging modality to assess PV stenosis after PV isolation.[235]

Lately, ICE has become a useful guide for AF ablation procedures. ICE enables visualization of PV ostia, the position of catheters and guides trans-septal puncture, thereby increasing the safety of the procedure (*Figure 24*).[236–238]

Imaging is key to diagnosing complications of the procedures. Pericardial effusion is the most common complication of AF ablation (0.4–1.3%).[225] Echocardiography should be readily available to make the diagnosis. Echocardiography is also the first step for assessing when valvular damage is suspected. Thrombotic complications and PV stenosis are diagnosed with CCT or CMR with contrast while atrio-oesophageal fistula is best diagnosed by chest CT with contrast.[216,239,240] Further work-up for the management of complications is described in the European Heart Rhythm Association (EHRA)/EACVI document on cardiac imaging in electrophysiology.[216]

LAA closure

It is advised to exclude LAA thrombus before percutaneous LAA closure in every patient. Surgical exclusion requires less guidance with

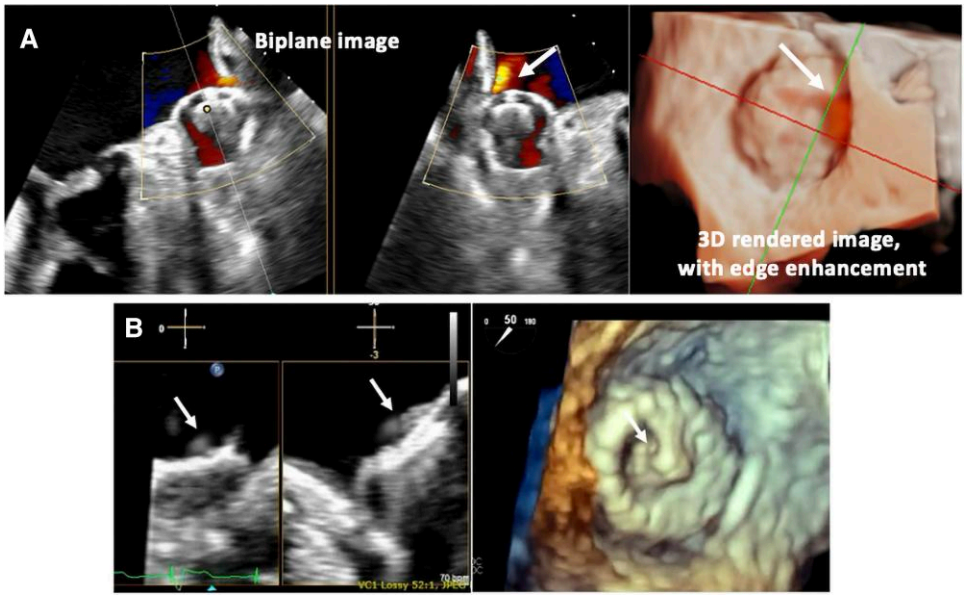


Figure 28 A) Peri-device leak by color Doppler (arrows), B) Thrombus on the occluder (arrows).

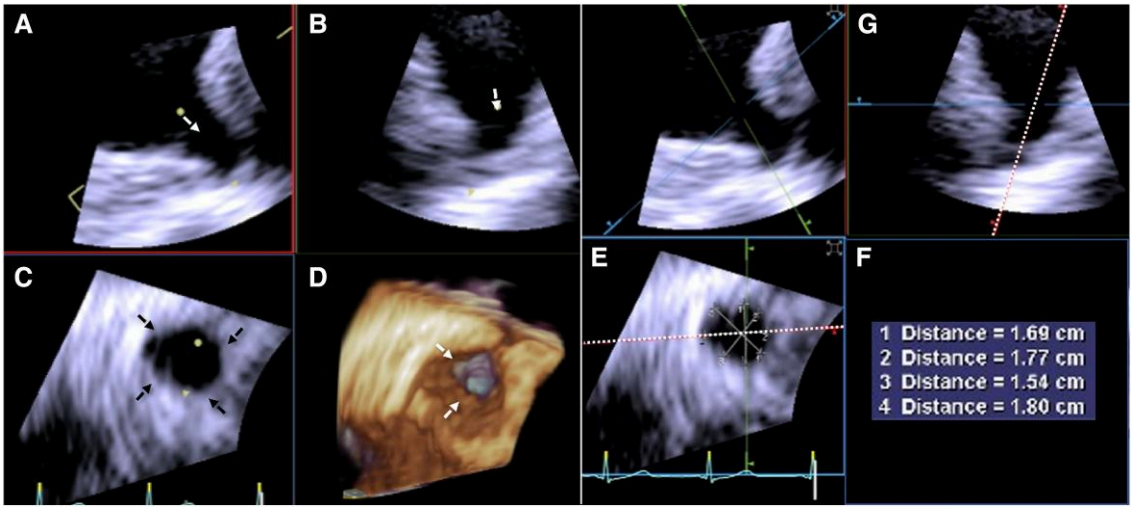


Figure 29 Imaging the LAA with the AcuNav 3D ICE catheter in the LA. (*A*, *B*) Long-axis orientations equivalent to 45 and 135° TOE views, respectively. (*C*) Short-axis view of the LAA ostia. These 2D multiplanar reconstructions are generated from the volume data shown in D. (*E*) Measurements of the ostial size in the four standard orientations of conventional TOE guidance. (*F*) Additional measurements obtained from E. (*G*) Depth of LAA (dotted line).

imaging, but post-surgical evaluation is important because it may be incomplete and may paradoxically increase the risk of thromboembolism[241] (*Figure 25A–F*). Various transcatheter closure devices are in use with specific appearances (*Figure 26*).[242–244] Measurements for device sizing are performed either by TOE or CCT. Device sizing aims to determine the maximum landing zone diameter at the level of the left circumflex artery (*Figure 27*). Sizing by CCT offers higher spatial resolution and tends to be 2–3 mm larger than TOE.[245] CCT can also be useful for selecting trans-septal puncture site.[246] Intraprocedural guidance utilizes a combination of fluoroscopy with either TOE or ICE. The goal is to achieve a complete occlusion of the LAA orifice without peri-device leak (PDL). The size of a PDL is measured at its narrowest diameter (vena contracta) by colour Doppler using a Nyquist limit of ~35 cm/s. Recent studies have shown that irrespective of the size, patients with PDLs have a higher incidence of thromboembolic complications compared with those without PDL.[247,248] A device surveillance at 45–90

Advice table 6 Peri-procedural use of multi-modality imaging

Cardioversion	
Imaging to rule out cardiac thrombus before cardioversion is advised in indications defined by the 2024 ESC guidelines for the management of AF[a].[181]	
Transcatheter and surgical procedures	
Cardiac imaging prior to AF ablation in high-risk patients may be useful (see text).	
Guidance with TOE or ICE is advised for transcatheter LAA occlusion.	
CCT is the preferred modality for assessing PV patterns before AF ablation and PV stenosis post-ablation, in symptomatic patients.	
Guidance with ICE increases the safety and decreases the duration of AF ablation.[237,238]	
Echocardiography should be readily available in the catheter laboratory.	
Chest CT with contrast is advised when there is suspicion of atrio-oesophageal fistula after AF ablation.	
The utility of assessing LA wall fibrosis with CMR before and after AF ablation is uncertain.	
TOE should be avoided if there is suspicion of atrio-oesophageal fistula.	

[a](i) To expedite cardioversion in non-anticoagulated subjects with AF ≥24 h and CHA_2DS_2-VA >1, (ii) if anticoagulation has been suboptimal within the last 3 weeks without interruption, and (iii) after 4 weeks of anticoagulation if a thrombus was initially detected.

days by TOE is advised to verify device stability, erosion, and complete occlusion without PDL and to rule out thrombus that is associated with unfavourable outcomes[245] (*Figure 28*). Patients with PDLs may either require anticoagulation or undergo additional transcatheter closure procedures.

ICE is frequently used during LAA device closure.[249] LAA views are optimally obtained when the ICE catheter is positioned in the right ventricle or the pulmonary artery. However, invasive nature and high costs of the technique limit its use (*Figure 29*).

Future perspectives

Developments in molecular imaging are promising to explore the inflammation and fibrotic process associated with atrial cardiopathy. Computational fluid dynamics simulations enable comprehensive blood flow pattern analysis in the LA, LAA, and PVs helping to explore the thrombogenic milieu.[250] Nevertheless, some simple but important gaps in evidence restrict the widespread clinical use of imaging for the assessment of LA cardiopathy. LA cardiopathy for risk stratification in patients having severe aortic stenosis,[251] the impact of assessing LA remodelling on outcomes, diagnostic and prognostic cut-offs of LA remodelling specific to diseases, and imaging modalities are awaited.

Conclusions

Consistent evidence and uniform expert consensus favour assessing LA cardiopathy and LAA by multi-modality imaging as an indispensable adjunct to patient management. The major gaps in evidence include the demonstration of the game-changing impact of multi-modality imaging for improving the outcomes. Further evidence from randomized studies is awaited to integrate multi-modality imaging of LA and LAA into clinical decision-making algorithms of the guidelines for patient management.

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Data availability

The data underlying this article are provided by the EACVI by permission. Data will be shared on request to the corresponding author with permission of the EACVI.

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