NON-INVASIVE IMAGING

Left atrial function: pathophysiology, echocardiographic assessment, and clinical applications

Monica Roşca,[1,2] Patrizio Lancellotti,[1] Bogdan A Popescu,[2] Luc A Piérard[1]

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[1]Department of Cardiology, University of Liège, CHU Sart Tilman, Liège, Belgium
[2]"Carol Davila" University of Medicine and Pharmacy, Bucharest, Romania

Correspondence to
Professor Luc A Piérard, University of Liège, CHU Sart Tilman, Liège, 4000, Belgium; lpierard@chu.ulg.ac.be

This article describes the pathophysiology of left atrial mechanical function and discusses both conventional and new echocardiographic parameters used to evaluate left atrial function. The evidence regarding the clinical usefulness of left atrial function assessment is also presented.

PATHOPHYSIOLOGY OF LEFT ATRIAL MECHANICAL FUNCTION

Atrial function, in a close interdependence with left ventricular (LV) function, plays a key role in maintaining an optimal cardiac performance. The left atrium (LA) modulates LV filling through its reservoir, conduit, and booster pump function, whereas LV function influences LA function throughout the cardiac cycle. The LA can act to increase LA pressure (in significant atrial disease) and can react to increased LV filling pressure (in significant ventricular disease). LA remodelling is related to LV remodelling[w1] and LA function has a central role in maintaining optimal cardiac output despite impaired LV relaxation and reduced LV compliance.[1] Understanding how each component of LA function is influenced by LV performance, and how each LA phasic function contributes to maintain an optimal stroke volume in normal and diseased hearts, is important for interpreting data derived from quantification of LA function.

During LV systole and isovolumic relaxation, the LA functions as a reservoir, receiving blood from the pulmonary veins and storing energy in the form of pressure. This atrial function is modulated by LV contraction, through the descent of the LV base during systole, by right ventricular systolic pressure transmitted through the pulmonary circulation, and by LA properties (ie, relaxation and chamber stiffness).[w2] During early LV diastole and diastasis, the LA functions as a conduit. Blood is transferred into the LV through the LA via a small pressure gradient during early diastole and flows passively from the pulmonary veins into the LV during diastasis. The conduit function is modulated especially by LV diastolic properties (LV relaxation and early diastolic pressures). During late LV diastole, the LA functions as a pump, the LA contraction augmenting LV stroke volume by approximately 20–30% in normal subjects and substantially more in the presence of impaired LV relaxation. LA booster pump function is modulated by LV compliance, LV end-diastolic pressure, and LA intrinsic contractility.[w3]

It has been demonstrated that the Frank–Starling mechanism is also operative in the LA. LA output increases as atrial diameter increases, which contributes to maintaining a normal stroke volume.[w4] Moreover, LA contractile function might decrease in the presence of severe LA dilation when the optimal Frank–Starling relationship is exceeded (figure 1). Thus, the study of LA function can provide additional information, incremental to LA volume measurement.

To achieve a better understanding of LA function, knowledge of the LA pressure and volume changes during the cardiac cycle is important. Beginning with mitral valve closure, blood flows into the LA through the pulmonary veins, producing an increase in volume accompanied by a continuous pressure rise ('v' wave). After the opening of the mitral valve, atrial volume begins to decrease accompanied by a parallel fall in LA pressure. During atrial diastasis, the volume remains relatively constant and atrial pressure increases. Atrial volume starts to decrease with the beginning of active atrial emptying, accompanied by a new pressure rise ('a' wave). The instantaneous changes of LA pressure and LA volume during one cardiac cycle in a normal subject are shown in the left panel of figure 2.

ECHOCARDIOGRAPHIC ASSESSMENT OF LA FUNCTION

The evaluation of volume–pressure curves is the most accurate and representative index for characterising LA mechanical function in different haemodynamic conditions.[2] The pressure–volume relationship is depicted in the right panel of figure 2. The curve forms a double loop, one corresponding to atrial filling (V loop) and the other to the passive, and subsequently, active atrial emptying (A loop). However, these relationships require combined invasive measurements which limit their use to experimental studies. On the other hand, echocardiography is a simple and widely available tool that has been increasingly

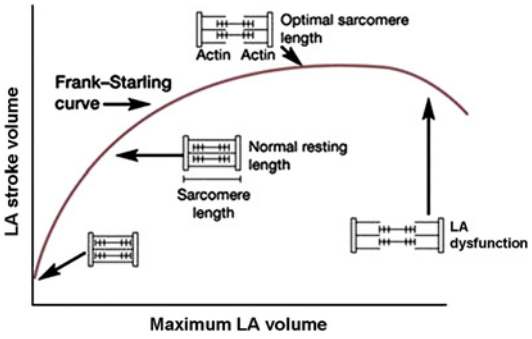

Figure 1 Frank–Starling law applied to the left atrium (LA).

used for the non-invasive assessment of LA function. This may include several parameters ranging from conventional methods based on the pulse wave (PW) Doppler evaluation of transmitral flow, pulmonary venous flow and calculation of LA phasic volumes, to newer methods based on measurements of atrial myocardial velocities or deformation (table 1).

Conventional parameters

PW Doppler derived peak A wave velocity and A wave time velocity integral (TVI) of mitral inflow can reflect LA booster pump function. Atrial fraction calculated as the ratio between the TVI of the A wave and the TVI of diastolic transmitral flow, representing the percentage of ventricular filling during atrial contraction, may also be used as a measure of LA contractile function.[w5]

$$\text{Atrial fraction} = \text{TVI}_{\text{A wave}}/\text{TVI}_{\text{mitral inflow}}$$

The main limitations of these parameters are their dependence on heart rate and loading conditions.

Systolic (S), diastolic (D), and atrial reversal (Ar) waves, measured by PW Doppler at the level of pulmonary venous flow, are determined by events that regulate phasic LA pressure and can theoretically describe the reservoir, conduit, and booster pump function of the LA. These parameters are highly dependent on LV diastolic properties.[w6] Thus, an increase in duration of the Ar wave reflects rather an increase in LV end-diastolic pressure than an increase in atrial performance, thus limiting the usefulness of this parameter as a measure of LA pump function.

Atrial ejection force, representing the force exerted by the LA to propel blood across the mitral valve into the LV during atrial systole, is another parameter proposed to describe atrial mechanical function.[3] It can be calculated as the product of the mass and acceleration of blood passing through the mitral annulus during the accelerative phase of atrial systole:

$$\text{Atrial ejection force} = \text{mass} \times \text{acceleration}$$

Mass is further defined as the product of the density of blood (ρ=1.06 g/cm^3) and the volume of blood passing through the mitral orifice during this portion of atrial ejection. Assuming that the upslope of the A wave is nearly flat and A wave acceleration is relatively constant, the above formula becomes:

$$\text{Atrial ejection force} = 0.5 \times 1.06 \times \text{mitral annulus area} \times (\text{peak A velocity})^2$$

The robustness, reproducibility, and incremental value of this parameter over LA volume assessment have not been documented.

The most common method used for the assessment of LA function is based on the measurement of LA phasic volumes: maximum volume (Vol_{max}) measured just before the opening of the mitral valve, minimal volume (Vol_{min}) measured at the closure of the mitral valve, and the volume just before the atrial contraction, measured at the onset of the P wave on the ECG (Vol_{P}). The following three indices, reflecting the phasic functions of the LA (reservoir, conduit, and booster pump) can be derived from the LA phasic volumes[w7]:

$$\text{LA expansion index} = (\text{Vol}_{\text{max}} - \text{Vol}_{\text{min}})/\text{Vol}_{\text{min}} \times 100$$

$$\text{LA passive emptying fraction} = (\text{Vol}_{\text{max}} - \text{Vol}_{\text{P}})/\text{Vol}_{\text{max}} \times 100$$

$$\text{LA active emptying fraction} = (\text{Vol}_{\text{P}} - \text{Vol}_{\text{min}})/\text{Vol}_{\text{P}} \times 100$$

Left atrial time–volume curves obtained by real-time three dimensional echocardiography (RT3DE), providing accurate information on LA size similar to cardiac magnetic resonance,[4] can also be used to estimate phasic LA functions.[w8 w9] The biplane area–length method or the biplane Simpson's method are currently recommended for the measurement of LA volumes by two dimensional echocardiography.[w10] However, in future it is likely that three dimensional echocardiography, providing more accurate and reproducible measurements, will emerge as the best and preferred method to calculate LA volumes. The main limitation of LA phasic

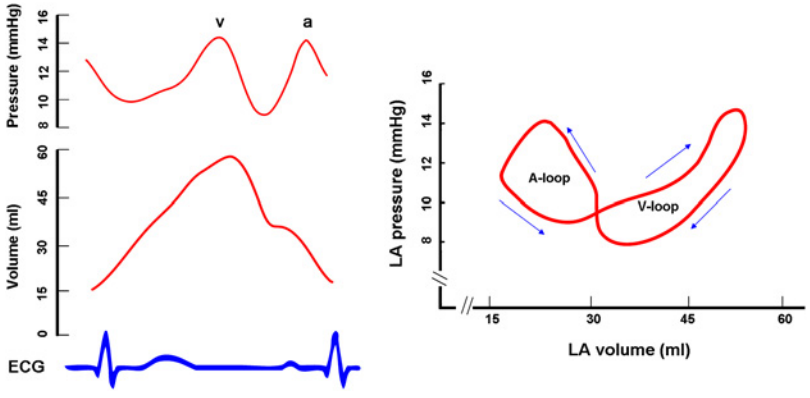

Figure 2 Instantaneous changes of left atrial pressure and left atrial volume during one cardiac cycle (left panel). Left atrial (LA) pressure–volume relationship comprising the A loop, representing the left atrial pump function, and the V loop, representing the reservoir function of the left atrium (right panel).

Table 1 Echocardiographic parameters for the assessment of left atrial function

Echocardiographic technique	Echocardiographic parameters	LA function assessed	Limitations
PW transmitral inflow	A wave velocity	Contractile	Load dependent
	A wave time velocity integral	Contractile	
	Atrial fraction	Contractile	
PW pulmonary venous flow	S, D, Ar waves velocities	Reservoir/conduit/contractile	Load dependent
2D/3D echocardiography	LA expansion index	Reservoir	Load dependent
	LA passive emptying fraction	Conduit	
	LA active emptying fraction	Contractile	
2D and PW transmitral inflow	Atrial ejection force	Contractile	Load dependent
TDI	Late diastolic mitral annular velocity (a')	Contractile	Angle dependent
	LA segmental velocities (S, e', a')	Reservoir/conduit/contractile	Influenced by translation/tethering
	LA strain	Reservoir	Time consuming
	LA strain rate (SSr, ESr, ASr)	Reservoir/conduit/contractile	Low reproducibility
	Total atrial conduction time	Electrical	
STE	LA strain	Reservoir	Influenced by image quality
	LA strain rate (SSr, ESr, ASr)	Reservoir/conduit/contractile	No validation of the software

ASr, late diastolic atrial strain rate; ESr, early diastolic atrial strain rate; LA, left atrium; PW, pulse wave; SSr, systolic atrial strain rate; STE, speckle tracking echocardiography; TDI, tissue Doppler imaging.

volume derived parameters is the inability to distinguish between the increase in LA function due to a larger amount of blood received/ejected and a real increase in intrinsic LA compliance/contractility.

Tissue Doppler imaging parameters

Tissue Doppler imaging (TDI) allows the quantification of LV longitudinal myocardial velocities, providing a relatively load independent measure of both systolic and diastolic LV function. Several studies have demonstrated that peak velocity of the mitral annulus in late diastole a', secondary to atrial contraction, can be used as a marker of atrial function.[w11] Unlike the early diastolic tissue Doppler velocities, late diastolic septal and lateral velocities (a') seem not to be significantly different.[w12] TDI can also be used for the assessment of regional LA function.[w13] The velocities recorded at the level of atrial segments adjacent to the mitral annulus are higher than the velocities of the superior segments of the atrium that are relatively fixed. However, the velocities are influenced by translation and tethering and therefore are not able to distinguish true atrial contraction from mitral annular and ventricular motion. In contrast, atrial strain and strain rate demonstrate a good site specificity and are able to describe the longitudinal shortening and lengthening of the atrium which are discordant with ventricular longitudinal motion (figure 3). Moreover, strain rate analysis, with a good temporal resolution, allows the quantification of all three components of LA function: systolic atrial strain rate (SSr) for reservoir function, early diastolic atrial strain rate (ESr) for conduit function, and late diastolic atrial strain rate (ASr) for contractile function.[5] In addition, TDI allows non-invasive quantification of atrial electromechanical delay, by measurement of the time interval from the onset of the P wave on the ECG to the peak of the a' wave on the atrial myocardial velocities curves.[6] In normal subjects, the electromechanical coupling is shortest at the level of the right atrial wall, and progressively longer at the level of the interatrial septum, and the lateral LA wall. Thus, total atrial conduction time can be non-invasively estimated as the time from the beginning of the P wave on the ECG to the peak of the

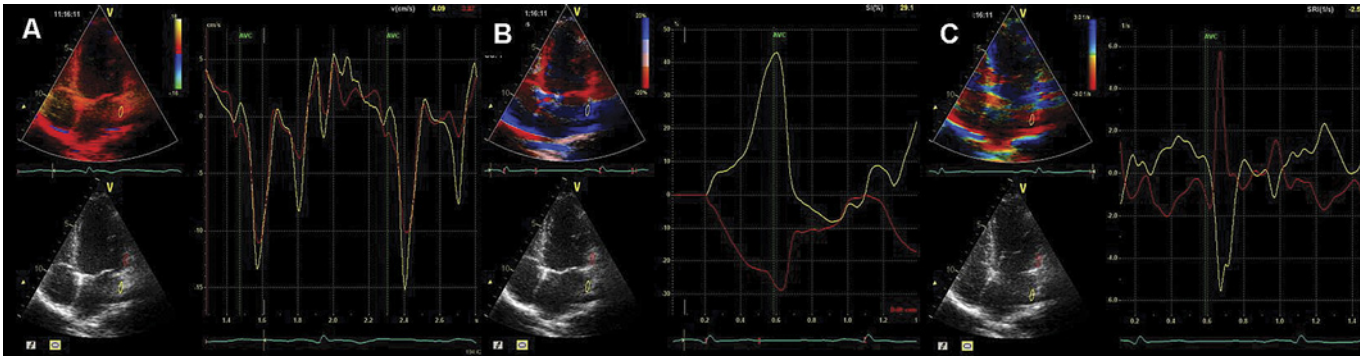

Figure 3 Myocardial velocities (A), strain (B), and strain rate (C) recorded by tissue Doppler imaging (TDI) at the level of the left ventricular (LV) lateral wall (red curves) and the left atrial (LA) lateral wall (yellow curves). The displayed curves demonstrate the limitation of measuring velocities (A) in distinguishing atrial contraction from mitral annular and ventricular motion (the curves are in the same direction) and the good site specificity of strain (B) and strain rate (C), which are able to describe the discordant movement of the atrium and the ventricle throughout the cardiac cycle (the curves are in opposite directions).

a' wave velocity recorded at the level of the lateral LA wall (figure 4).[6] Although this time interval overestimates the atrial electromechanical delay, it has demonstrated a very good correlation with total atrial conduction time measured by signal averaged ECG technique, and thus can be a suitable parameter for the identification of a potential atrial substrate vulnerable for atrial fibrillation. TDI is an angle dependent technique and wall-by-wall sampling is time consuming, limiting the use of this method in clinical practice.

Speckle tracking echocardiography parameters

Speckle tracking echocardiography (STE) is a new technique based on tracking the movement of natural acoustic markers (speckles) present on standard grey scale images. STE derived strain and strain rate parameters are relatively independent of wall tethering and loading conditions. STE has been recently proposed for the quantification of atrial function,[7 w14] allowing a comprehensive, angle independent assessment of myocardial deformation, and overcoming the limitations of TDI. Global longitudinal LA strain and strain rate parameters, such as the average deformation of all LA segments recorded in one view, can be determined (figure 5). STE reliability is influenced by image quality. Moreover, the currently available STE software was developed for LV function assessment and its use for LA function assessment has not yet been fully validated.

The feasibility of TDI and STE for the assessment of LA longitudinal deformation has been documented, and normal values for atrial deformation in different segments have been reported.[5 7 w14] The values obtained by TDI are generally higher than those obtained by STE. However, the population in the TDI studies was significantly younger than the population in the STE studies. Normal values reported for global longitudinal strain and strain rate, obtained by STE, are also different depending on the model (with 12 segments[7] or 15 segments[w14]) and type of gating used (QRS[5] or P wave[6]). The lack of standardisation is an important limitation to the widespread use of these parameters in routine clinical practice. The variability reported for STE derived parameters is lower than the variability reported for TDI parameters.

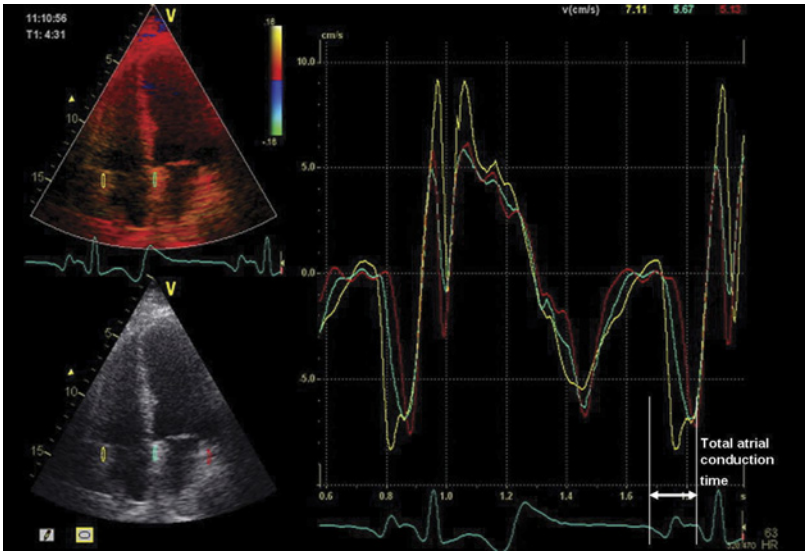

Figure 4 Left atrial myocardial velocities recorded at the level of right left atrial (LA) wall (yellow curve), interatrial septum (green curve) and lateral LA wall (red curve) in a normal subject. Time interval from the beginning of the P wave on the ECG to the peak of the a' wave (electromechanical delay) is shortest at the level of the right atrial wall, and progressively longer at the level of the interatrial septum, and the lateral LA wall. Total atrial conduction time can be estimated as the time from the beginning of the P wave on the ECG to the peak of the a' wave velocity recorded at the level of lateral LA wall.

LA FUNCTION AND AGEING

The structural changes of the LA wall with ageing, including the increase in fatty tissue, collagen and fibrosis, can lead to progressive LA remodelling. However, current data support the theory that LA dilation and impaired LA function reflect rather the clinical conditions that frequently accompany ageing than represent the effect of physiologic ageing alone. Changes in LV diastolic function with ageing are initially compensated by an increased atrial contribution to LV filling. This could be an explanation for the conflicting data regarding the effects of ageing on LA pump function. Some studies reported higher values for parameters describing LA contractile function in elderly subjects,[w13 w15] while other studies reported similar values for these parameters in young and old subjects.[4] In all these studies, the LA conduit function decreased with ageing, which can be an expression of LV diastolic dysfunction, whereas LA reservoir function remained unchanged.

CLINICAL APPLICATIONS

There is increasing evidence demonstrating the prognostic role of LA size in predicting the risk of development of atrial fibrillation, heart failure or cardiac and all cause mortality.[8] The modalities to assess LA size, its physiological determinants and clinical applications have been extensively reviewed elsewhere.[4 8 w16] On the other hand, the clinical usefulness and potential prognostic value of LA function assessment have received less attention. In patients with LV dysfunction, valvular heart disease or atrial fibrillation, the decrease in atrial function has been related to symptoms, risk of arrhythmias or outcomes. Herein we present an overview of the clinical and prognostic implications of LA dysfunction in different clinical settings (table 2).

LA function and heart failure

The contribution of LA phasic function to LV filling is dependent on LV diastolic properties.[w17] With abnormal relaxation, the relative contribution of LA contractile function to LV filling increases, whereas the conduit function decreases. As LV filling pressures progressively increase, the limits of atrial preload reserve are reached and the LA serves predominantly as a conduit.

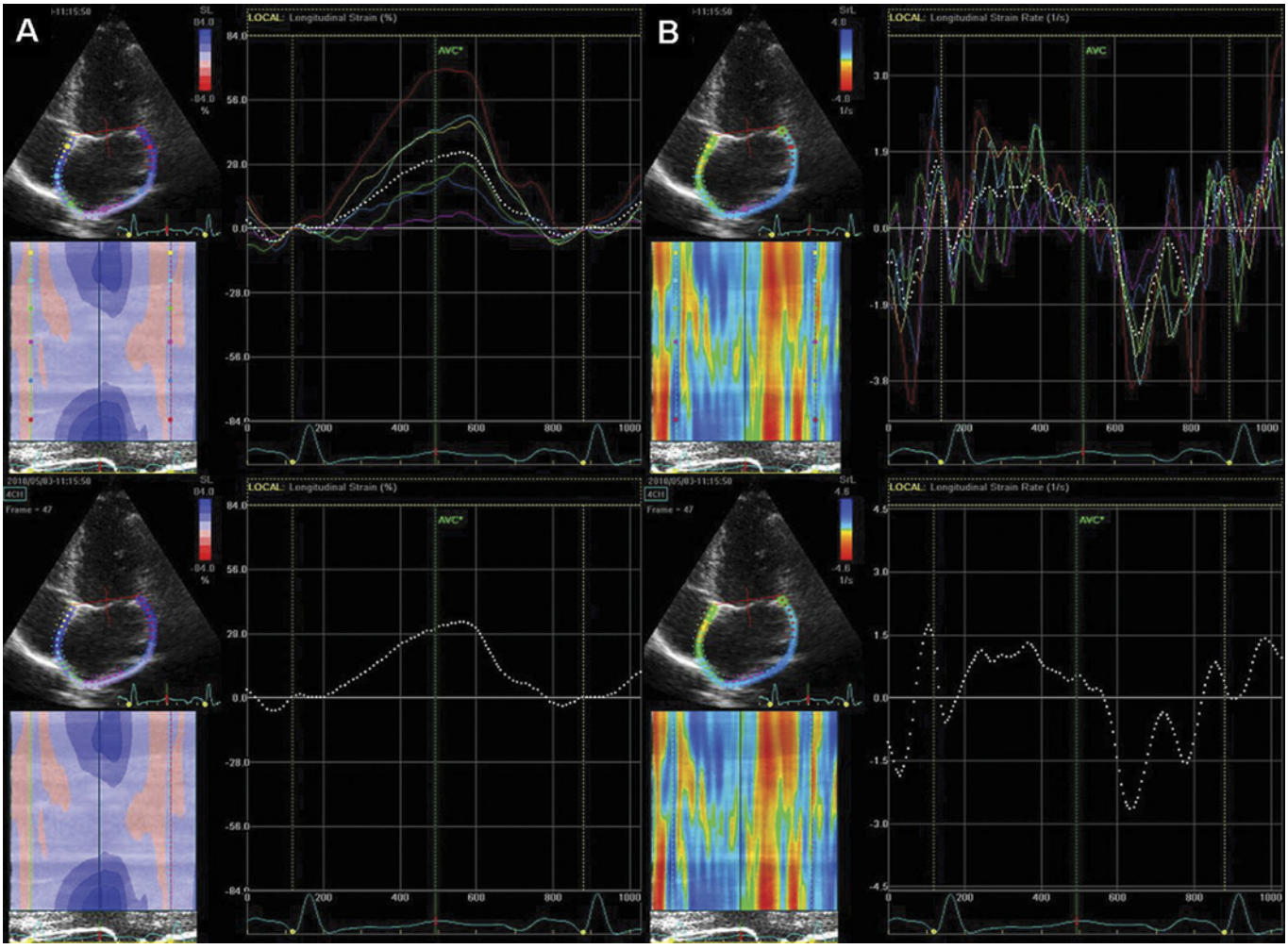

Figure 5 The assessment of longitudinal left atrial strain and strain rate by speckle tracking echocardiography. Left atrial (LA) strain (A) and strain rate (B) curves for each of the six LA segments recorded from an apical four chamber view (upper panels) and global longitudinal LA strain (A) and strain rate (B) curves (bottom panels).

In patients with mild hypertension without LV hypertrophy or LA dilation, despite the presence of normal conventional parameters of atrial function, a reduction in LA conduit volume has been demonstrated, reflected also by a reduction in early diastolic LA strain rate (ESr).[9] The authors suggested that the reduction in LA conduit function may be an early marker of LV diastolic dysfunction before the occurrence of overt LV hypertrophy and LA enlargement, and could be a risk factor for the development of atrial fibrillation or heart failure symptoms in hypertensive patients.

The potential role of LA dysfunction in the pathophysiology of heart failure with normal LV ejection fraction (HFNEF) has been recently suggested.[10] An increase in late diastolic mitral annular velocity a' during exercise has been found in normal subjects and asymptomatic hypertensive patients, but not in patients with HFNEF. Thus, hypertensive patients appear to be able to compensate for their increase in LV filling pressures during exercise by increasing LA contractile function, while patients with HFNEF are unable to benefit from this mechanism.

In patients with chronic heart failure and LV systolic dysfunction, LA contractile function—as assessed by TDI derived late diastolic mitral annulus velocity and LA strain during atrial contraction—was found to be an independent predictor of maximal workload or peak oxygen consumption during exercise.[w18] Moreover, LA contractile dysfunction represents an important prognostic marker in patients with heart failure and LV systolic disfunction.[11] A late diastolic mitral annular velocity a' <5 cm/s was the most powerful predictor of cardiac death or hospitalisation for worsening heart failure compared with clinical, haemodynamic, and other echocardiographic variables.

LA function in cardiomyopathies

The theory of a generalised myopathic process affecting both ventricular and atrial myocardium in cardiomyopathies is generally accepted. For similar LA volumes and LV pressure–volume curves, LA contractile function as assessed by LA active emptying fraction was significantly lower in patients with idiopathic dilated cardiomyopathy than in patients with aortic stenosis.[w19] LA systolic dysfunction in idiopathic dilated cardiomyopathy

Table 2 Potential clinical role of left atrial function assessment in different pathologies

Clinical settings	Echocardiographic technique	LA function involved	Parameters	Clinical role
Mild hypertension[9]	2D, TDI	Conduit	Decrease of conduit volume and early diastolic LA strain rate (ESr)	Early identification of LV diastolic dysfunction
Heart failure with normal LV ejection fraction[10]	TDI	Contractile	Lack of increase of late diastolic mitral annular velocity (A′) during exercise	Identifies patients with heart failure and normal LV ejection fraction
LV systolic dysfunction[w18]	TDI	Contractile	Late diastolic mitral annulus velocity and LA strain during atrial contraction	Predict maximal workload or peak oxygen consumption during exercise
LV systolic dysfunction[11]	TDI	Contractile	Late diastolic mitral annular velocity (a′) <5 cm/s	Predicts cardiac death and hospitalisation for worsening heart failure
Idiopathic dilated cardiomyopathy[12]	2D	Contractile	Increase in left atrial active emptying fraction after inotropic stimulation	Related to peak oxygen consumption
Hypertrophic cardiomyopathy[13]	TDI and STE	Reservoir	2D atrial strain <10.8%	Differentiates hypertrophic cardiomyopathy from other types of LV hypertrophy
Hypertrophic cardiomyopathy[14]	STE	Contractile	Late diastolic LA strain rate (ASr) < −0.92/s	Related to presence of heart failure symptoms
Mitral stenosis[w20]	TDI	Reservoir	LA systolic strain rate (SSr) < −1.69/s	Predictor of events
Mitral regurgitation[w22]	3D	Contractile	LA emptying fraction	Correlated with pulmonary artery pressure
Atrial fibrillation[15]	VVI	Reservoir	LA strain	Related to atrial fibrosis assessed by MRI
Atrial fibrillation[16]	TDI	Reservoir	LA systolic strain rate (SSr) >1.80/s	Predicts maintenance of sinus rhythm after cardioversion
General population[17]	TDI	Electrical	Total atrial conduction time >190 ms	Predicts new onset atrial fibrillation
Acute myocardial infarction[w23]	TDI	Electrical	Total atrial conduction time >127 ms	Predicts new onset atrial fibrillation

ASr, late diastolic atrial strain rate; ESr, early diastolic atrial strain rate; LA, left atrium; LV, left ventricle; SSr, systolic atrial strain rate; STE, speckle tracking echocardiography; TDI, tissue Doppler imaging; VVI, vector velocity imaging.

was not entirely explained by the degree of LA dilatation, LA tension or LV filling pressures, suggesting that LA myopathy might be involved. The degree of LA dysfunction has been related to exercise capacity in these patients.[12] In patients with non-ischaemic dilated cardiomyopathy, LA active emptying fraction increased after inotropic stimulation in patients with peak oxygen consumption >14 ml/kg/min, and decreased in patients with peak oxygen consumption <14 ml/kg/min.

LA longitudinal function is significantly reduced in patients with hypertrophic cardiomyopathy in comparison with other types of LV hypertrophy.[13] A decrease in LA reservoir function, as assessed by two dimensional atrial strain, with a cut-off value of 10.8%, had an added value in differentiating hypertrophic cardiomyopathy from other types of LV hypertrophy. The reduction in LA function, as assessed by two dimensional strain, has also been related to the presence of heart failure symptoms in hypertrophic cardiomyopathy.[14] When compared with control subjects, the asymptomatic and symptomatic patients with hypertrophic cardiomyopathy had a progressive reduction in LA longitudinal strain and strain rate parameters, reflecting significantly lower values for the reservoir, conduit, and contractile LA function. Moreover, LA contractile function was the only independent correlate of symptomatic status in multivariate analysis. A late diastolic LA strain rate (ASr) less than −0.92/s identified symptomatic patients with hypertrophic cardiomyopathy with 75% sensitivity and 83% specificity.[14]

LA function in valvular heart disease

In patients with mitral stenosis, LA compliance measured invasively proved to be an important physiological determinant of systolic pulmonary artery pressure.[18] Patients with lower LA compliance had higher systolic pulmonary artery pressures both at rest and during exercise, and were more symptomatic. Moreover, these patients were at high risk of underestimation of disease severity as commonly assessed by the pressure half-time method. LA reservoir function, assessed by systolic LA strain rate, emerged as the best predictor of events in patients with mitral stenosis, in a multivariate analysis that also included age, LA volume, and mitral valve area.[w20] A cut-off value of 1.69/s for LA peak systolic strain rate (SSr) predicted events with a sensitivity of 88% and a specificity of 80.6%.[w35]

All three components of LA function are reduced in patients with severe aortic stenosis, and LA contractile dysfunction is related to aortic stenosis severity.[19] Moreover, in aortic stenosis, LA volume derived function has been found to be poorly correlated with LA strain, suggesting that changes in LA function do not parallel changes in LA size and that new parameters could be more sensitive for detecting LA dysfunction in this setting.[w21] The impact of reduction in LA active function on the clinical status requires further studies.

In a study of LA function assessed by RT3DE, patients with degenerative mitral regurgitation and high systolic pulmonary artery pressure had lower values of LA contractile function, as assessed by analysis of the LA time–volume curve derived from RT3DE.[w22] Effective regurgitant orifice area, LA contractile function, E/e′ ratio, and lateral a′ velocity were independently correlated with pulmonary artery pressure.[w22] Thus, pulmonary hypertension in chronic mitral regurgitation may depend not only on mitral regurgitation severity, but also on LA function.

Left atrial function: key points

- An accurate and thorough assessment of left atrial (LA) function by echocardiography, although difficult, is currently possible.
- The conventional parameters used for the assessment of atrial function are load dependent and require a skilful acquisition technique and calculations that are not routinely performed.
- The newer parameters derived from tissue Doppler imaging or speckle tracking echocardiography are reproducible and more sensitive than conventional measurements in identifying early changes in LA function.
- A comprehensive evaluation of LA function may permit early detection of subclinical disease, and could refine risk stratification or guide therapy.
- The extent of LA remodelling reversibility with medical treatment, and the impact of such changes on outcomes, need further studies.

LA function and atrial fibrillation

Atrial fibrillation is another condition commonly associated with LA remodelling, including structural and functional changes. Recently, LA wall fibrosis assessed by delayed enhancement magnetic resonance was related to LA function impairment, as assessed by LA strain and strain rate, and both were related to the risk of atrial fibrillation.[15] Patients with persistent atrial fibrillation had extensive fibrosis and lower values of LA strain, whereas patients with paroxysmal atrial fibrillation had mild fibrosis and higher values of LA strain. Non-invasive imaging of LA fibrosis by LA deformation measurement may be helpful in predicting the risk of atrial fibrillation and guiding therapeutic strategies.

A prolonged atrial conduction time, measured with TDI, may predict the development of new-onset atrial fibrillation in the general population.[17] Total atrial conduction time >190 ms has been proposed as a tool in identifying patients at risk and in guiding therapeutic strategies to prevent the occurrence of atrial fibrillation or the development of its complications.

Total atrial conduction time, estimated by TDI, also independently predicted new-onset atrial fibrillation after acute myocardial infarction, providing an incremental prognostic value to traditional clinical and echocardiographic parameters.[w23] Receiver operating characteristic curve analysis demonstrated that a cut-off value of 127 ms had a sensitivity of 89% and a specificity of 74% for the prediction of new-onset atrial fibrillation in these patients.

During atrial fibrillation, not only is the booster pump function lost, but the reservoir and conduit functions are also impaired. Moreover, the LA reservoir function is lower in patients with recurrence of atrial fibrillation after cardioversion than in those with maintenance of sinus rhythm. An LA inferior wall systolic strain rate >1.80/s before cardioversion predicted with 92% sensitivity and 79% specificity the maintenance of sinus rhythm after cardioversion.[16]

Atrial function improves progressively after successful cardioversion, with a maximum at 1 month. Unlike late diastolic atrial myocardial velocity a', late diastolic LA strain rate (ASr) did not completely normalise at 6 months, suggesting a persisting degree of atrial dysfunction.[20] Moreover, although the amplitude parameters improved, the timing parameters such as total atrial conduction time remained unchanged after cardioversion. The identification and quantification of this persisting LA dysfunction could be useful in guiding therapy, suggesting the need for a longer term use of antiarrhythmic treatment in patients with significant persistent LA dysfunction. A progressive reduction in LA volume has also been described after catheter ablation of atrial fibrillation. A recent meta-analysis found a significant decrease in LA size after successful radiofrequency catheter ablation, but without an improvement in LA function, as assessed by LA phasic volumes.[w24] The new tissue Doppler derived parameters identified a parallel improvement in LA function and in both LV systolic and diastolic function in patients who remained in sinus rhythm after ablation of atrial fibrillation.[w25]

SUMMARY

The assessment of LA function provides important pathophysiological information and may be of clinical value in different settings. The accurate assessment of LA function by echocardiography has significantly improved in recent years. Differentiating intrinsic atrial dysfunction from changes in LA function secondary to LV dysfunction seems to be more accessible using the new echocardiographic techniques. The newer parameters derived from TDI or STE are less load dependent and have higher sensitivity in assessing LA function than conventional parameters. However, the lack of standardisation limits the routine use of these

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parameters in current clinical practice. Preliminary studies suggest that evaluation of atrial function using newer echocardiographic techniques may have important clinical implications in predicting symptom development or risk of arrhythmias in different conditions. The extent of LA remodelling reversibility with medical treatment and the impact of such changes on outcomes require further studies.

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Monica Rosca, Patrizio Lancellotti, Bogdan A Popescu, et al.

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