Left atrial function

Aliakbar Arvandi MD

Abstract

The left atrium (LA) is a left posterior cardiac chamber which is located adjacent to the esophagus. It is separated from the right atrium by the inter-atrial septum and connected to the left ventricle by the mitral valve apparatus. Left and right pulmonary veins carry oxygenated blood to the LA during the cardiac cycle. During ventricular systole, the LA functions mainly as a receiving chamber while during diastole after the opening of the mitral valve it empties the blood into the left ventricle. Healthy LA function is crucial to maintain normal diastolic and systolic function, and it changes in a variety of disease states, including hypertension and coronary artery disease. Left atrial size and function can be evaluated by multimodality imaging, including echocardiography, cardiac computed tomography, and cardiac magnetic resonance imaging, and are important prognostic factors in some cardiovascular diseases.

Keywords: Left atrium, left atrial function, left atrial size, left atrial strain, speckle tracking, atrial fibrillation, pulmonary veins.

The left atrium (LA) is anatomically located as a left posterior cardiac chamber that serves as a conduit between the pulmonary veins and the left ventricle (LV). Although anatomical variation is not uncommon, usually four pulmonary veins (left upper and lower and right upper and lower) are connected to the posterior wall of the LA. The connection between the LA and LV is through the mitral valve apparatus. The interatrial septum separates the two atria, but communication between the left and right atria by a patent foramen ovale occurs in up to 25% of the population.

The LA is filled during systole and reaches its maximum volume at the end of isovolumetric relaxation right before the opening of the mitral valve. Subsequently its volume starts to decrease by the opening of the mitral valve and reaches its smallest diameter at the end of diastole after atrial contraction. Normal phasic emptying during diastole has an important role in determining LV stroke volume, meaning that most of the LV preload and the final stroke volume depend on passive filling during the earlier part of diastole. In normal conditions, atrial contraction contributes 15-30% of the LV stroke volume. The LA appendage has an important role in the contraction phase of LA. It is trabeculated compared to the rest of the atrium, and in conditions like atrial fibrillation when the atrium loses effective contraction, it is the main site for thrombus formation. Due to the close proximity of the LA to the esophagus, transesophageal echocardiography (TEE) has proven to be an excellent method to detect LA thrombus. The changes in LA size during the cardiac cycle can be measured by M-Mode and two dimensional (2-D) echocardiography (see Figures1-A and 1-B).

Corresponding author: **Aliakbar Arvandi**
Contact Information: Aliakbar.arvandi@ttuhsc.edu
DOI: 10.12746/swrccc2017.0517.225

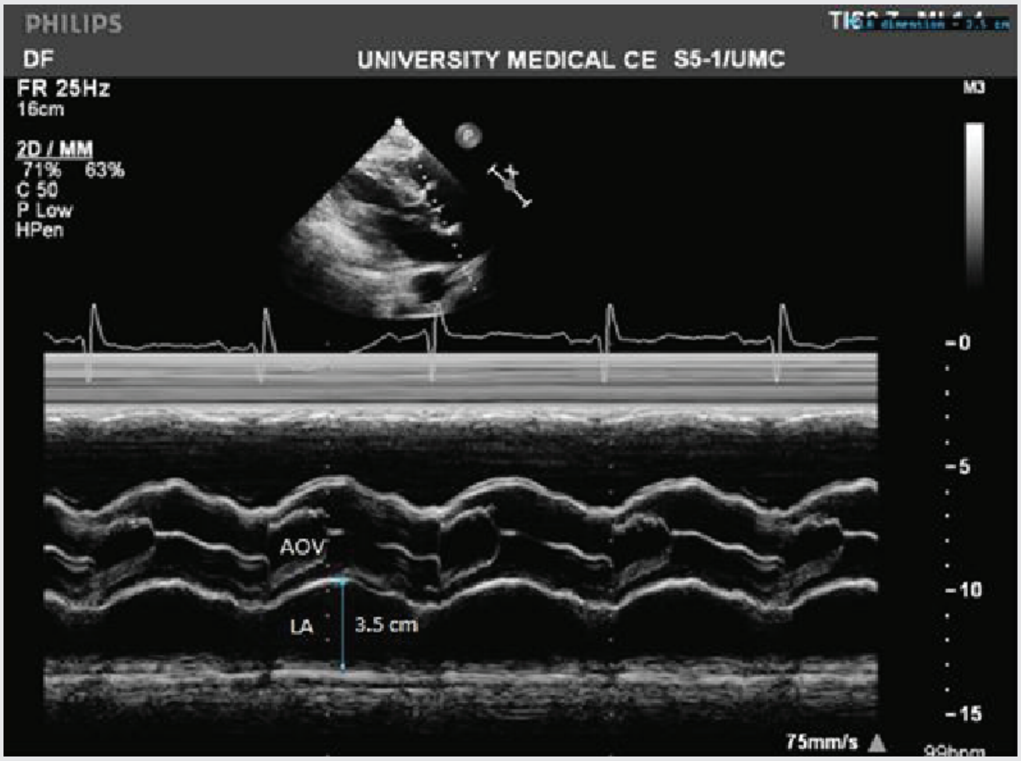

Figure 1-A. LA size measured by 2-D guided M-Mode echocardiography in parasternal long axis view
LA size is measured at its maximum diameter at end systole. In this case it is 3.5 cm which is normal. This image was obtained 12/1/2016 from the echocardiography department at University Medical Center, Lubbock, Texas. LA- left atrium, AOV-aortic valve.

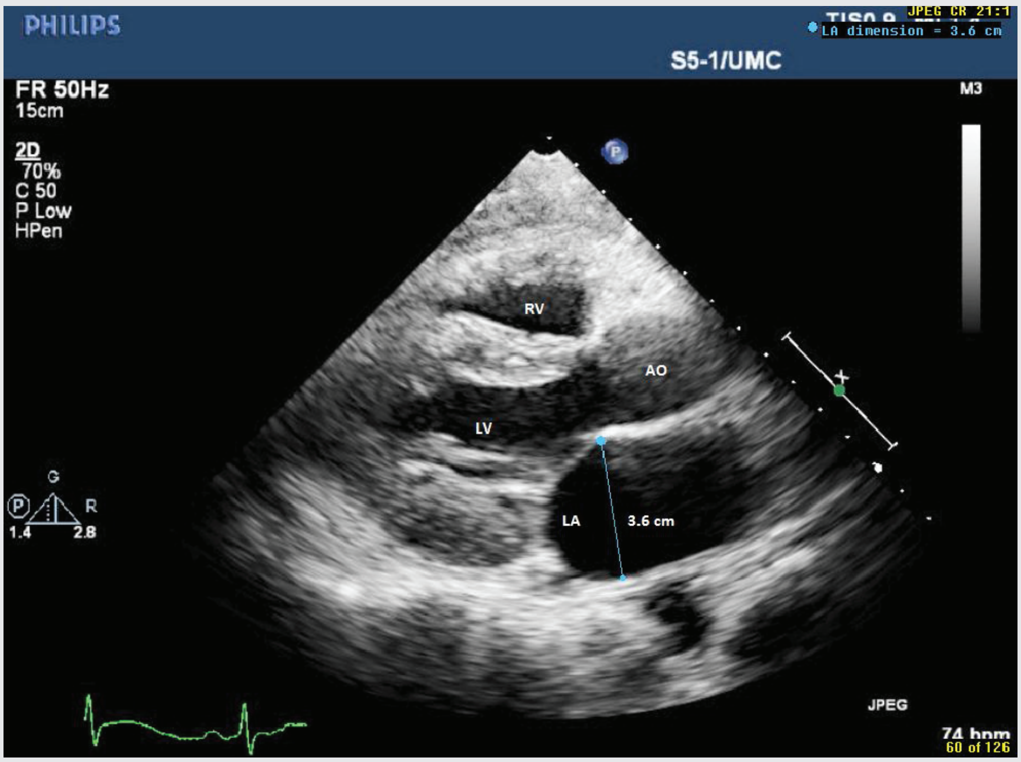

Figure 1-B. LA size measured by 2-D echocardiography in parasternal long axis view
LA size is measured at its maximum diameter at end systole. In this case it is 3.6 cm which is normal. This image was obtained 12/1/2016 from the echocardiography department at University Medical Center, Lubbock, Texas. Ao- aorta, LA- left atrium, LV- left ventricle, RV- right ventricle.

The normal LA dimension by M-Mode or 2-D in the parasternal long axis view is 4.0 cm or less. More accurate and reliable evaluation of LA size is done by measuring LA volume index (LAVI) by 2-D echocardiography using disk summation algorithm, similar to that being used to measure LV volume (LAVI <34 ml/m^2 is considered normal) (Figure 2).

The LA has an important role in LV end diastolic volume, LV filling pressure, LV stroke volume, and cardiac output. Left atrial function is affected by LV diastolic and systolic function. Increasing stretch in the LA wall causes secretion of natriuretic peptide, and through interaction with the renin-angiotensin-aldosterone system, the LA has an important role in the volume and hemodynamic status of the cardiovascular system. In conditions with abnormal LV diastolic or systolic function when LV filling pressure is elevated, the LA contribution to LV filling is also affected. In these conditions effective LA-LV coupling and LA contraction have an even more important role in maintaining adequate cardiac output.

In conditions with elevated LV filling pressure, the LA afterload is increased, and the LA undergoes remodeling with increasing size and hypertrophy. In normal conditions, LV filling mostly occurs during the first third of diastole followed by atrial contraction at the end of diastole. The contribution of LA contraction to LV filling increases in some conditions. For example, in patients with hypertension, coronary artery disease, and hypertrophic cardiomyopathy, LA afterload increases due to abnormal relaxation and elevated LV filling pressure. The impact of hypertension on LA size and function has been studied by Eshoo et al.[1] In this study even mild hypertension was associated with decreased early diastolic filling of the LV and resulted in increased late diastolic filling by LA contraction. This

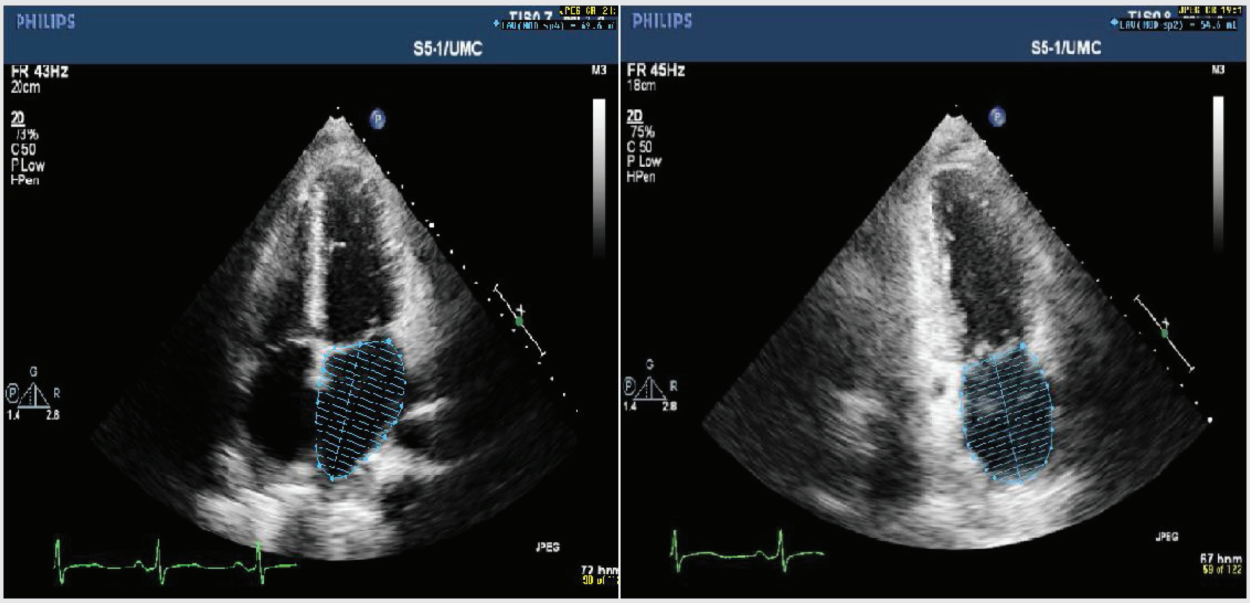

Figure 2. Measurement of LA volume by 2-D imaging
LA volume is measured using the disk summation algorithm, similar to that used to measure LV volume. LA size is traced and measured in apical two chamber (A2C) and apical four chamber (A4C) views at the end systole when LA is at its maximum size. LA volume then calculated and divided by body surface area to obtain the LA volume index (Normal < 34 ml/m^2). In this case it is calculated at 30.5 ml/m^2 which is in the normal range. This image was obtained 12/1/2016 from the echocardiography department at University Medical Center, Lubbock, Texas.

pattern of abnormal LV filling might be the mechanism which explains LA enlargement. In hypertrophic cardiomyopathy the LV function depends on healthy LA mechanics, and the function of these two chambers are related and affected by each other.[2]

In atrial fibrillation with ineffective LA contraction, cardiac output may be reduced and pulmonary capillary wedge pressure increased, causing symptoms of congestive heart failure, especially in disorders in which LV filling is already impaired. Atrial fibrillation has a negative effect on LA function which deteriorates over time. Left atrial contractile function improves after pulmonary vein isolation if there is a lower incidence of atrial fibrillation afterward. Left atrial volume and size are often studied to predict the recurrence of atrial fibrillation after ablation.[3]

Multimodality imaging, including echocardiography (2-D and 3-D), cardiac computed tomography (CT), and cardiac magnetic resonance imaging (MRI), has been used to evaluate LA size and function.[4] Cardiac CT is commonly used to study LA before performing pulmonary vein isolation. For example, Wolf et al evaluated L A function by cardiac CT before LA radiofrequency catheter ablation.[5] In this study cardiac CT provided good information about LA size and LA function before ablation and had very low interobserver variability.

Among all imaging modalities echocardiography is still one of the fastest, easiest, and most cost effective with great temporal resolution. Three dimensional echocardiography is used to evaluate LA volume change in real time to study the LA Frank Starling relationship.[6] Left atrial volume and size are commonly measured in echocardiography in daily practice (see Figure 2) to evaluate patients for LV diastolic dysfunction, and LA volume as an indicator of LV diastolic dysfunction has been studied.[7]

Strain imaging is also used to evaluate LA function. Myocardial deformation or strain is defined by the relative change in diameter compared to the original length of the fiber, and the strain rate (SR) is determined by the instantaneous rate by which this change occurs. Left ventricular strain imaging can be accomplished by 2-D speckle tracking while doing echocardiography. Thomas and Popovic have reviewed the clinical application of LV strain imaging using tissue Doppler derived and speckle tracked derived strain imaging for evaluation of regional myocardial function.[8] Abnormal LV strain (> -20) can be used to determine subclinical systolic dysfunction. Left ventricular strain imaging with 2-D speckle tracking is done frequently in many labs to evaluate patients for subclinical LV systolic dysfunction when overall LV ejection fraction is preserved, especially in condition like amyloidosis and hypertrophic cardiomyopathy. Left atrial strain measured by two-dimensional speckle tracking also provides a tool to evaluate LA function.[9]

In summary, the LA is a chamber that maintains connection between pulmonary veins and LV, functions as a receiving chamber, and has an important role in cardiovascular hemodynamics by providing a contractile function at the end of diastole. Left ventricular and LA function are closely related and dependent on each other, and LA function is a crucial factor in determining LV volume, filling pressure, and stroke volume. Its interaction with neuro-hormonal pathway also determines the body's volume status, cardiac output, and perfusion pressures. Left atria function and size are easily measured with echocardiography.

Article citation: Arvandi A. Left atrial function. The Southwest Respiratory and Critical Care Chronicles 2017; 5(17): 1-8.
From: Department of Internal Medicine, Texas Tech University Health Sciences Center, Lubbock, TX
Submitted: 10/2/16
Accepted: 12/4/2016
Reviewer: Scott Shurmur MD
Conflict of interest: none

References

1. Eshoo S, Ross DL, Thomas L. Impact of mild hypertension on left atrial size and function. Circ Cardiovasc Imaging 2009; 2:93–9.
2. Shin MS, Fukuda S, Song JM, et al. Relationship between left atrial and left ventricular function in hypertrophic cardiomyopathy: a real-time 3-dimensional echocardiographic study. J Am Soc Echocardiogr 2006; 19:796–801.
3. Helms AS, West JJ, Patel A, et al. Relation of left atrial volume from three-dimensional computed tomography to atrial fibrillation recurrence following ablation. Am J Cardiol 2009; 103:989 –93.
4. Wen Z, Zhang Z, Yu W, Fan Z, Du J, Lv B. Assessing the left atrial phasic volume and function with dual-source CT: comparison with 3T MRI. Int J Cardiovasc Imaging 2010; 26 Suppl 1:83–92.
5. Wolf F, Ourednicek P, Loewe C, et al. Evaluation of left atrial function by multidetector computed tomography before left atrial radiofrequency catheter ablation: comparison of a manual and automated 3D volume segmentation method. Eur J Radiol 2010; 75:e141– 6.
6. Anwar AM, Geleijnse ML, Soliman OI, Nemes A, ten Cate FJ. Left atrial Frank-Starling law assessed by real time, three-dimensional echocardiographic left atrial volume changes. Heart 2007; 93:1393–7
7. Tsang TS, Barnes ME, Gersh BJ, Bailey KR, Seward JB. Left atrial volume as a morphophysiologic expression of left ventricular diastolic dysfunction and relation to cardiovascular risk burden. Am J Cardiol 2002; 90:1284-9.
8. Thomas JD, Popovic ZB. Assessment of left ventricular function by cardiac ultrasound. J Am Coll Cardiol 2006; 48:2012–25.
9. Saraiva RM, Demirkol S, Buakhamsri A, et al. Left atrial strain measured by two-dimensional speckle tracking represents a new tool to evaluate left atrial function. J Am Soc Echocardiogr 2010; 23:172– 80.