Introduction

For long, the estimated ejection fraction (EF) of left ventricle (LV) determined by echocardiography has been used to predict cardiovascular outcomes. Recent evidence highlights the importance of left atrial (LA) volume with regards to prediction of cardiovascular outcomes. The LA volume has been compared to the “glycated hemoglobin of diabetes mellitus”, as it is a reflection of long-standing hemodynamic condition. This review aims to discuss the parameters used to determine LA phasic function, methodology of LA volume estimation and its usefulness in clinical practice.

Mechanical Function of Left Atrium

The mechanical function of LA has been traditionally described in three phases within the cardiac cycle: the ‘reservoir’, the ‘conduit’ and the ‘contractile’ machinery. During ventricular systole and isovolumic relaxation, the LA functions as a ‘reservoir’ receiving blood from pulmonary veins. The early phase of ventricular diastole sets the stage for LA operating as a ‘conduit’ for blood passing from the pulmonary veins into the LV. This is followed by atrial ‘contraction’ during which the LV stroke volume is augmented by approximately twenty percent.¹

Various LA volumes have been used to describe LA phasic function.
1. Reservoir volume, is calculated as the difference between maximal and minimum LA volumes. Maximal LA volume occurs at ventricular end-systole just before the opening mitral valve; while minimum LA volume occurs at end-diastole, just before closure of mitral valve.
2. Conduit volume, is calculated as the difference between maximum and pre-atrial contraction LA volume.
3. Contractile volume, is calculated as the difference between minimum and pre-atrial contraction LA volume.

The relative contribution of LA phasic function to LV filling is dependent upon the diastolic properties of LV. In subjects with normal diastolic function, the relative contribution of the reservoir, conduit and contractile function of the LA to the filling of LV is approximately 40%, 35% and 25% respectively.² As LV relaxation gradually worsens, the relative contribution of LA reservoir and contractile function increases while conduit function decreases. But with advanced diastolic dysfunction the LA serves predominantly as a conduit.

Assessment of LA size

LA is not a symmetrically shaped three-dimensional (3D) structure. Furthermore, LA enlargement may not occur in a uniform fashion. Therefore anteroposterior measurement of LA by M-mode echocardiography is likely to be an insensitive assessment of any change in LA size. In contrast, LA volume by two-dimensional (2D) or 3D echocardiography provides a more accurate and reproducible estimate of LA size as compared to magnetic resonance imaging (MRI) and cine-computerised tomography (CT). The LA size is measured at the ventricular end-systole when the LA chamber is at its greatest dimension. It is imperative to avoid foreshortening of the LA for computing LA volume. The confluence of the pulmonary veins and LA appendage should be excluded, when performing planimetry.³ For assessment of left atrial ‘reservoir’, ‘conduit’ and ‘contractile’ function, LA volumes should be measured at specified phases of the cardiac cycle. They are a) end-systolic frame just before mitral valve opening ; b) end-diastolic frame just before mitral

Abstract

Prediction of cardiovascular outcomes by non-invasive techniques remains a priority in patients with cardiovascular disease. The left ventricular ejection fraction, which is a parameter of systolic function has been the most sought after index for a long time. The importance of diastolic function has come to the foreground. Left atrial volume, is an index of long-standing diastolic function. This review discusses the methodology of estimation of left atrial volume and its clinical implications.

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Fig. 2: LA volume by 3-dimensional echocardiography

valve closure; c) last frame just before mitral valve reopening i.e. pre-atrial contraction. Echocardiographic assessment of LA volume is done by Simpson’s method, area–length method and real time 3D echocardiography.

**Simpson's method:** Estimation of LA volume by Simpson’s method of disc is well validated and recommended by the American Society of Echocardiography (ASE) guidelines3 (Figure 1A).

**Biplane area-length method:** Orthogonal apical views, apical four and two-chamber views are obtained for determination of LA area and length. The length is determined from the middle of the plane of mitral annulus to posterior wall. Left atrial volume is calculated on the basis of the algorithm \(0.85 \times A_1 \times A_2 \div L\); where \(A_1\) and \(A_2\) are the areas of LA in four and two chamber views and \(L\) is the shortest of the lengths obtained from the orthogonal views and indexed to body surface area.3 This method has also been recommended by the ASE guidelines for estimation of LA volume (Figure 1B).

**Real time 3D echocardiography:**4 Full volume 3-dimensional images are obtained with the matrix array transducer. Zoom function gain adjustments are used to clarify the endocardial border. The three-dimensional dataset is transferred to a Q-LAB system for offline analysis. For calculation of LA volume, a semiautomated tracing of the LA endocardial border is performed by marking five atrial points: the anterior, inferior, lateral, septal mitral annuli and the LA apex (Figure 2). Modifications are made to correct automatic tracings if necessary.

### Reference Values
Normal indexed LA volume has been determined in several studies involving several hundred patients using the preferred biplane technique. Most trial results indicate a value of 22 ± 6 ml/m² as the normal range and the same is recognized by ASE.3

### Determinants of LA size

1. **Left ventricular filling pressures:** The increase in LA volume is a reflection of elevated LV filling pressures in the absence of congenital heart disease, mitral valve or primary atrial pathology. In a non-compliant LV, as the LA is exposed to the pressures of the LV during diastole, LA pressure rises to maintain adequate LV filling.5 It is the increase in LA wall tension which leads to chamber dilatation. LA volume is therefore an expression of the chronicity of exposure to abnormal filling pressures.6 Thus LA volume reflects an average of LV filling pressures over time. It is thus useful for monitoring long-term hemodynamic control.

2. **Anthropometry and age:** LA size should be indexed to body surface area as body size is a major determinant of LA size. It is the variation in body size which accounts for gender difference in LA size.7 The left atrial volume index (LAVI) is independent of age from childhood onwards.8 The age related LA enlargement is a reflection of the pathophysiologic changes that accompany advancing age rather than a consequence of chronologic aging.

3. **Atrial fibrillation:** It is difficult to establish a causal relationship between atrial fibrillation and LA volume. Structural alterations in LA, may be related to the underlying pathophysiology or solely to the arrhythmia itself. Data from experimental animal studies do lend evidence to the fact that atrial arrhythmias induce structural remodeling.9

4. **Volume overload:** Chronic volume overload associated with large shunts, valvular regurgitation and high output states including athletic heart10 can contribute to LA enlargement. However myocardial relaxation physiology is usually normal as compared to abnormal myocyte relaxation seen in pressure overload situations.

### Prediction of Cardiovascular Outcomes
Atrial fibrillation (AF) is a serious cardiac arrhythmia associated with increased morbidity and mortality. Data from the Framingham11 and Cardiovascular Health Study12 have incriminated an increased anteroposterior LA diameter as the harbinger of AF. It has been confirmed that LA volume represents a superior measure over LA diameter for predicting outcomes inclusive of AF. The prognostic information provided is incremental to clinical risk factors. Increased LA volume is also a predictor of stroke and death. An indexed LA volume of ≥32 ml/m² is associated with an increased risk of stroke independent of age and other clinical risk factors for cerebrovascular disease.13 An increased LA volume is also the predictor of first stroke in elderly who are in sinus rhythm and without any history of ischemic neurological events, AF or valvular heart disease.

LA volume is the barometer of LV filing pressure and reflects the burden of diastolic dysfunction. Because a large number of individuals with LV dysfunction are in a pre-clinical phase of the disease, methods to quantify the risk of progression to symptomatic heart failure would be clinically useful. LA volume ≥ 32 ml/m² is associated with increased incidence of heart failure which is independent of age, myocardial infarction, diabetes mellitus, hypertension, LV hypertrophy and mitral inflow velocities.14,15 Even in subjects with a normal ejection fraction, an increment in LA volume is observed from baseline to the diagnosis of heart failure.

LAVI is a predictor of survival after acute myocardial infarction. An exponential increase in mortality with increasing LA volume has been documented.16 Moreover, the prognostic information is incremental to clinical data and standard...
echocardiographic measures of LV systolic and diastolic function.

There is evidence that LAVI ≤ 28 ml/m² is strongly predictive of normal stress echocardiogram. Although a robust data in this perspective is awaited, it holds promise to provide a simple means of identifying patients with low ischemic risk. LA volume is intimately related to LV mass/hypertrophy, systolic and diastolic dysfunction. The incremental value of each parameter for the prediction of death is expected to diminish when considering others. But LAVI derives its importance in providing incremental value in predicting mortality.

Limitations

The major limitation in any imaging modality is its image quality. The atria are located in the far field of apical views. The image quality of LA is therefore not optimal. Modification of gain settings may not improve image quality as increase in gain will further reduce LA lumen size. Inadvertent planimetry of a foreshortened LA would introduce considerable errors in volume estimation. Therefore it is necessary that maximal LA size be obtained during volume estimation. These pitfalls can not be negated by 3-D imaging owing to decreased resolution thus making LA trace problematic.

Certain questions need to be answered. Does regression of LA size with therapy translate into improved outcomes? What is the natural history of LA remodeling? Future studies are warranted to further our understanding.

Conclusion

LA volume is a valuable tool for clinical and prognostic implications. It should be routinely incorporated in clinical practice. It is an evolving science and more data is required to understand the natural history of LA remodeling and the impact on outcomes after LA size reduction.

References