

Modeling left atrial volume, shape, and contraction patterns in normal subjects by cardiac magnetic resonance imaging

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ABSTRACT

Background: Left atrial three-dimensional shape and contraction patterns are not well described. We quantified the LA using three-dimensional cardiac MRI (CMR) in a group of normal subjects.

Methods: Three-dimensional vectors were used to quantitate atrial shape and contraction using a geometric model as a three-dimensional prolate ellipsoid. Atrial area and length at end-systole and end-diastole were made in the horizontal long axis (HLA) and vertical long axis (VLA) planes. Biplane area-length products and the orthogonal LA long axis vector comprised 3 orthogonal vector lengths composed of axis measures for shape and volume calculations at end-diastole and end-systole. Vector fractional shortening in 3 dimensions was calculated for each 3-space orthogonal vector. Echocardiograms were used for comparison.

Results: The normal LA is an oblate ellipsoid with significantly longer HLA short axis than the vertical VLA short axis ($p < 0.001$). LA contraction in the long axis dimension is smaller than both HLA and VLA short axis dimensional changes ($p < 0.001$). Linear correlations between LAEDV vs. LASV and LAESV vs. LAEF were highly significant.

Conclusions: This dimensional analysis quantitates normal left atrial shape for the first time, modeled as a prolate 3-D ellipsoid. LA contractile functions and derives mostly from contraction in the HLA and VLA short axis directions. Though LA end-diastolic volume is considered the marker of left atrial health or disease, this notion should be reconsidered in view of LA static and functional modeling in 3 dimensions.

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1. Introduction

The left atrium (LA) serves as a reservoir for receiving pulmonary venous flow during ventricular systole when the mitral valve is closed. Much is known about LA volume from prior echocardiographic studies. LA volume provides important insight for pathologic states since increased LA size and dysfunction usually result from left ventricular (LV) dysfunction [1,2]. LA volume change correlates with pathologic conditions such as congestive heart failure, atrial fibrillation [3,4], and valvular heart disease [5].

2. Methods

Although many reports have examined LA volume and function in health and disease, few studies have quantitated these features in 3 dimensions [6]. More extensive description of LA volume, shape, and contraction pattern could prove useful for early detection of cardiac disease. We therefore evaluated the LA in normal subjects by CMR imaging to characterize LA size, shape, and contraction dynamics. We also compared the results with LA volume obtained from 2-D echocardiography.

Abbreviations: LA, left atrium; LV, left ventricle; ES, end-systole; ED, end-diastole; MRI, magnetic resonance imaging; CT, computed tomography; HLA, horizontal long axis; VLA, vertical long axis; EF, ejection fraction; BSA, body surface area.

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2.1. Study population

A cohort of normal cardiac MRI scans was selected from patients undergoing evaluation at the Minneapolis Heart Institute under IRB approval. Scans were eligible for inclusion in the study if they were interpreted as completely normal by an expert CMR clinician.

2.2. CMR images

Functional EKG-gated cardiac MRIs were used for all cardiac measurements. CMR sequences contained 25 equal interval images in the cardiac cycle, from LA end-systole (ES) to the following LA ES. Measurements for ES were obtained at phases where LA volume was visually minimal, and LA end diastolic (ED) measurements were made just prior to mitral valve opening where LA volume was visually largest. Digital measurements were made using Vitrea Softread® software (Vital Images, Minnetonka MN).

2.3. Calculation of LA three-dimensional structure vectors

Published guidelines for CMR evaluation of LA size are similar to those of the American Society of Echocardiography using biplane area-length methods. The LA was modeled as a three-dimensional prolate ellipsoid with 3 orthogonal vector radii as follows. In standard CMR HLA and VLA planes, the left atrial chamber was manually planimeted for area and the left atrial long axis (LA) diameter from the mitral valve annulus to the posterior wall was measured. The HLA and VLA long axes in orthogonal planes are an identical anatomic dimension, which is the distance from the mitral valve annulus to the posterior atrial wall. These HLA and VLA long axis measurements were averaged $(HLA + VLA)/2$ to obtain a representative LA long axis dimension. Atrial area in both HLA and VLA planes was planimeted manually, using care to avoid pulmonary veins.

2.4. The LA prolate ellipsoid model

Three orthogonal radii are required to calculate the volume of a prolate ellipsoid. These dimensions are summarized as follows:

1. LA long axis length: $r_1 = \frac{\text{Major HLA} + \text{Major VLA diameter}}{2}$
2. HLA minor axis length: $r_2 = \frac{\text{Area HLA}}{\pi \left(\frac{\text{Average diameter}}{2}\right)}$
3. VLA Minor axis length: $r_3 = \frac{\text{Area VLA}}{\pi \left(\frac{\text{Average diameter}}{2}\right)}$

A three-dimensional LA “contraction vector” was defined as the 3-space vector whose respective unit components were the differences between diastolic and systolic vectors. A contraction ratio in each dimension was thus $\frac{\text{ED Radius} - \text{ES Radius}}{\text{ED Radius}}$.

2.5. Comparison with 2-D echocardiography

Echocardiographic records were obtained from subjects in this study. When available, LA volume was recorded for comparison with the MRI LA calculation. Statistical correlation and Bland–Altman analysis was performed by standard technique.

3. Results

3.1. Demographics

Demographic characteristics for 244 normal subjects are shown in Table 1. Subjects were 62% male, and as expected, all had normal left ventricular function.

3.2. Left atrial volumes, dimensions, and contraction patterns

Mean LA end-diastolic and end-systolic volumes, LA stroke volume, and LA ejection fraction are shown in Table 2. The mean left atrial HLA short axis and the VLA short axis were both shorter than the LA long axis ($p < 0.05$) (Tables 3a and 3b).

The contraction ratios in orthogonal dimensions were as follows. The HLA contraction ratio (0.17 ± 0.07) was significantly smaller than both the VLA (0.26 ± 0.10) and the LA long axis (0.26 ± 0.10). The greatest left atrial systolic contraction thus occurred equally in both the HLA and VLA short axis dimensions.

Linear regression between the contraction ratio in the LA long axis dimension and the LA EF had an r^2 of 0.48, indicating a moderate correlation. Linear regression of VLA short axis dimension contraction and LA EF was found to have a similar correlation and $r^2 = 0.43$. Regression analysis applied to the LA contraction ratio versus LA EF found no correlation ($r^2 = 0.004$).

A multivariate stepwise regression was created to model and predict left atrial ejection fraction based on contraction in the VLA and HLA short axis dimensions. This model produced the equation $\text{EF} = 58.1(\text{HLA}\%) + 52.6(\text{VLA}\%) + 25.9$. Regression results of this model versus actual, measured EF resulted in $r^2 = 0.81$, indicating accurate predictive value for this model in anatomic size, shape, and function. This analysis confirmed the observation that LA contraction in the LA long axis direction has only a minor contribution to LA contractile function since HLA and VLA contraction alone account for the majority of LA EF in normal subjects.

Fig. 1 shows the relationship between LAEDV (LA end-diastolic volume) and LASV (LA stroke volume), and a strong positive correlation is evident. Fig. 2 shows the relationship between LAESV and LAEF. A strong inverse relationship is seen here. Both of these graphs also indicate a large distribution of LA size in the normal population.

Table 1
Baseline clinical characteristics of 244 normal subjects (mean \pm SD).

Number of subjects (n)	244
Age mean (years)	42.4 \pm 17.8
Male (%)	62
LV EF (%)	67.0 \pm 7.4

Table 2
Left atrial volume and function data of 244 normal subjects (mean \pm SD).

LA ED volume (mL)	81 \pm 24
LA ES volume (mL)	38 \pm 15
LA stroke volume (mL)	43 \pm 13
LA EF (%)	54 \pm 10

Table 3a
LA diastolic dimensions (mean \pm SD), n = 244.

HLA short axis (cm)	VLA short axis (cm)	LA long axis (cm)	p value
2.55 \pm 0.38	2.62 \pm 0.40	2.86 \pm 0.46	$p < 0.001$ HLA or VLA short axis vs. LA long axis $p = \text{ns}$ HLA vs. VLA short axis

Table 3b
LA contraction ratios (mean \pm SD).

HLA short axis (cm)	VLA short axis (cm)	LA long axis (cm)	p value
0.17 \pm 0.07	0.26 \pm 0.10	0.26 \pm 0.10	$p < 0.001$, HLA short axis vs. both VLA long axis and LA long axis

Importantly, LA stroke volume rises proportionally to LA volume in normals.

3.3. Echocardiographic comparison

Two-dimensional echocardiogram report data were available in 78 of the 244 subjects. The correlation coefficient for LA volume by CMR vs. echocardiography was 0.41. A Bland–Altman plot for CMR diastolic volume compared with echocardiography is shown in Fig. 3. The data show very wide limits of agreement (LOA) and a large number of widely disparate readings, indicating an overall poor correlation between these two methods of LA volume determination.

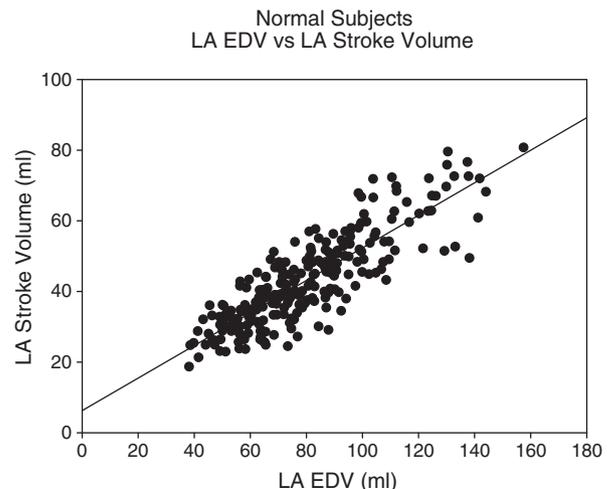


Fig. 1. Plot of LAEDV vs. LASV in 244 normal subjects. A strong positive association is evident.

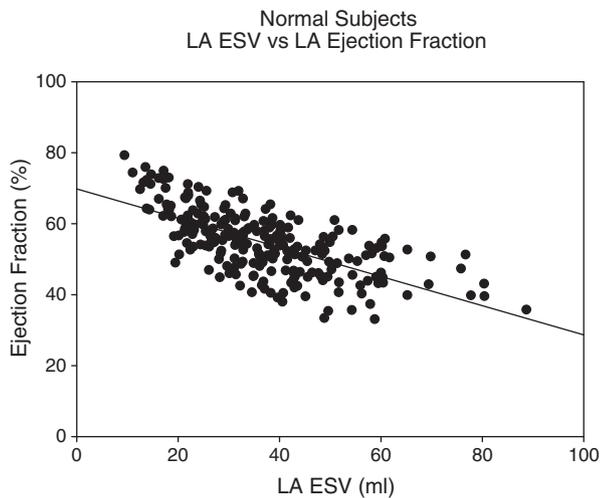


Fig. 2. Plot of LAESV and LAEF in normals. A strong inverse relationship is seen here.

4. Discussion

Left atrial shape has not been well described in health and disease. Loperfido et al. [7,8], using echocardiography, reported that the LA long axis is the largest LA dimension and that the VLA short axis dimension is the smallest. These results differ slightly from our results. We found that at LA end-diastole, the LA long axis is largest, and that VLA and HLA short axes are comparable in size. These differences may have occurred for several reasons. The Loperfido et al. data were obtained from fewer subjects, and the echocardiographic views used older echocardiographic equipment, where the LA can be difficult to visualize in its entirety. Cardiac MRI by comparison is a three-dimensional technology that easily visualizes the entire LA including the posterior wall.

Our model showed no statistical relationship between orthogonal LA dimensions since in correlation comparison, the LA orthogonal dimensions had r^2 values less than 0.1. The Loperfido et al. study described LA shapes and suggested that orthogonal dimensions cannot be accurately predicted from knowledge of one or even two of the other dimensions. A significant correlation was found between LAEF and HLA/VLA short axes. This suggests the majority of LA pump function in normals occurs by contraction in the VLA and HLA short axis directions. This is consistent with the observation that long axis contraction was smallest of the three dimensional contraction vectors.

Comparison of CMR and echocardiographic quantification of LA volume showed relatively poor correlation between these imaging modalities by Bland–Altman analysis. This is consistent with current understanding that CMR is a “gold standard” for cardiac chamber volume analysis. Echocardiographic accuracy is less due to incomplete ability to visualize the entire LA chamber and also due to less spatial resolution for detecting myocardial-chamber borders.

In our study, there was a significant variability in atrial size, and all had normal function as measured by both LAEF and LASV. The

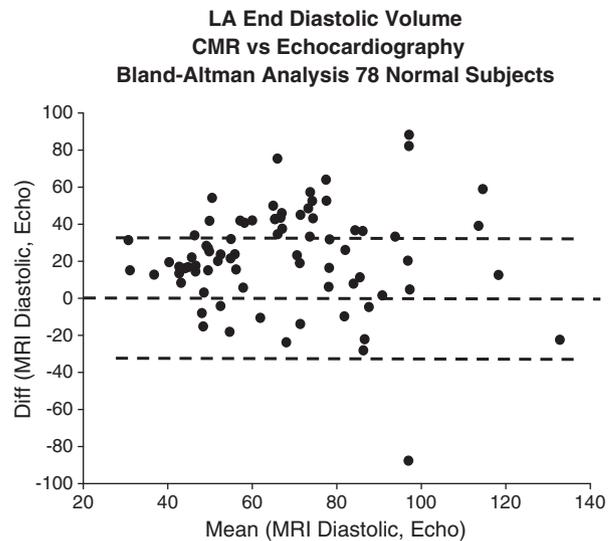


Fig. 3. Bland Altman plot for LA diastolic volume between CMR and echocardiography. Center dotted line is the mean, while ± 2 SD lines are shown above and below the mean. A moderate association is evident.

traditional view that LAEDV alone should be the standard for evaluation of left atrial health should be reconsidered based on our data. Future study should be directed toward understanding size and function relationships in disease states to better understand the relationship between size and function and whether such interactions can detect pathologic states earlier than currently possible.

References

- [1] Ng J, Villuendas R, Cokic I, et al. Autonomic remodeling in the left atrium and pulmonary veins in heart failure: creation of a dynamic substrate for atrial fibrillation. *Circ Arrhythm Electrophysiol* 2011;4:388–96.
- [2] Shen YQ, Wang LM, Che L, Song HM, Zhang QP. Relationship of left heart size and left ventricular mass with exercise capacity in chronic heart failure. *Chin Med J (Engl)* 2011;124:2485–9.
- [3] Zhuang J, Wang Y, Tang K, et al. Association between left atrial size and atrial fibrillation recurrence after single circumferential pulmonary vein isolation: A systematic review and meta-analysis of observational studies. *Europace* 2012 May;14(5):638–45.
- [4] Stahrenberg R, Edelmann F, Haase B, et al. Transthoracic echocardiography to rule out paroxysmal atrial fibrillation as a cause of stroke or transient ischemic attack. *Stroke* 2011;42:3643–5.
- [5] Vaturi M, Hadar T, Yedidya I, et al. The association of left atrial volume with exercise capacity in patients with chronic severe mitral regurgitation. *Isr Med Assoc J* 2010;12:150–3.
- [6] Blume GG, McLeod CJ, Barnes ME, et al. Left atrial function: physiology, assessment, and clinical implications. *Eur J Echocardiogr* 2011;12:421–30.
- [7] Loperfido F, Digaetano A, Santarelli P, et al. The evaluation of left and right ventricular hypertrophy in combined ventricular overload by electrocardiography: relationship with the echocardiographic data. *J Electrocardiol* 1982;15:327–34.
- [8] Loperfido F, Pennestri F, Digaetano A, et al. Assessment of left atrial dimensions by cross sectional echocardiography in patients with mitral valve disease. *Br Heart J* 1983;50:570–8.