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Echocardiographic Parameters Associated With Asymmetrical Structural Remodelling In Patients With Or Without Atrial Fibrillation

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Abstract

Objectives: Left atrial (LA) dilation can evolve into asymmetrical remodeling. The aim of this study was to determine the echocardiographic parameters associated with LA asymmetric structural remodeling (ASR) in patients with and without nonvalvular atrial fibrillation (AF).

Methods and results: A total of 170 patients with a dilated LA were prospectively enrolled. ASR was defined as an atrium shape that is no longer ellipsoidal (LA basal dimension measured at the junction between the pulmonary vein and atrium greater than the mitral annular dimension). Symmetric structural remodeling (SSR) was defined as all other cases. Echocardiographic parameters of LA function and left ventricular diastolic function, measured by pulsed-wave Doppler and Tissue Doppler Imaging, were analyzed to identify the parameters associated with ASR. The mean age of the patients was 67 ± 11 years. Forty-one percent had a stable sinus rhythm (SR), and 59% had AF. LA-ASR was detected in 66% of the patients: 55% with AF and 45% with SR (p=0.002). The mean LA-ASR and LA-SSR volume indexes were 49 ± 14 ml/m² and 29 ± 13 ml/m², respectively (p<0.001). LA systolic myocardial velocity (p=0.036) and peak systolic pulmonary venous flow velocity (p=0.033) were the parameters best associated with ASR. The sensitivity and specificity of both parameters, based on ROC curve analysis, were 77 and 70%, respectively. The AUC was 0.765 (95% CI: 0.662-0.849, p=0.0001).

Conclusion: LA dilation is associated with a great number of asymmetrical structural remodeling. Echocardiographic parameters that reflect LA reservoir function are best associated with asymmetrical remodeling.

Introduction

Left atrial (LA) size, depending mostly on LA morphology and shape, is an important predictor of cardiovascular outcome that provides diagnostic and prognostic information regarding atrial fibrillation (AF) and other cardiovascular diseases like hypertension, coronary heart disease and left ventricular (LV) dysfunction.[1] Although the mitral annulus, mitral-aortic and inter-atrial septum are not very susceptible to dilation due to their relatively fixed position while the rest of the atrial myocardium (i.e., the junction and antrum of the pulmonary veins) is prone to morphological changes by enlargement, mostly in the superior-inferior and medial-lateral dimensions because of the lack of fibrous components.[2,3] LA dilation can result in alterations in LA geometry and shape associated with symmetric (SSR) or asymmetric structural remodeling (ASR).[1,4] The two recommended parameters to evaluate LA size are the LA volume index and the LA area, which are markers of structural remodeling, regardless of electrical remodeling.[1] Therefore, because of the alteration in shape and morphology, the assessment of LA volume by the classic ellipsoid formula might not be appropriate in patients with asymmetric dilation.[2] Computed tomography, ventriculography and magnetic resonance imaging, considered the gold standard methods for LA size assessment, showed both good agreement with each other and a tendency to underestimate LA volume in comparison with echocardiographic measurements due to asymmetric dilation.[1,2,4] Moreover, the frequency of ASR in patients with or without AF and the relationship with LA size and function is unclear. Therefore, the aim of our study was to assess the frequency of ASR in patients with dilated LA with and without AF and to identify echocardiographic parameters of LA function and LV diastolic function, by pulsed-wave Doppler (PW) and tissue Doppler Imaging (TDI) associated with ASR.

Key Words:

Atrial Fibrillation, Left Atrium, Structural Remodeling, Reservoir Function.

Disclosures:
None.

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Methods

Patient Selection

The patients were consecutively and prospectively enrolled in the study between November 2010 and June 2011. We diagnosed and classified AF according with the current guidelines.[5] The inclusion criteria were patients older than age 18, with or without nonvalvular

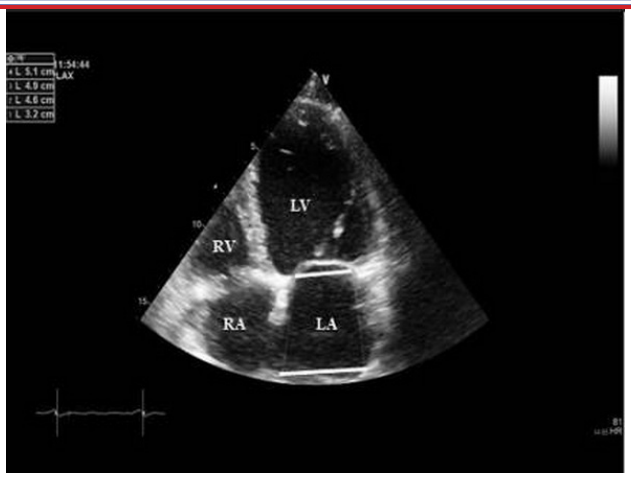

Figure 1: **Definition of left atrium asymmetric structural remodeling.**

(LA=left atrium, LV= left ventricle, RA=right atrium and RV=right ventricle).

AF, and LA area greater than 20 cm^2 (according to the current definition of atrium dilation).[1] Exclusion criteria were patients who refused to participate in the study, with a poor echocardiographic window, or those with LA dilation secondary to valvular heart disease, valvular prosthesis, acute coronary syndrome, ischemic heart disease with severe septal kinetics disorder, pulmonary artery systolic pressure ≥ 35 mmHg, congenital heart disease, constrictive pericarditis, amyloidosis, atrioventricular and intraventricular conduction disorder, cardiac pacing, or neoplasia. The Ethics Committee of the hospital approved this study and, all patients gave written informed consent.

Echocardiography

All echocardiographic measurements were performed on patients in the left lateral decubitus position during expiratory apnea, according to the current guidelines of the European Association of Echocardiography and the American Society of Echocardiography.[6,7] LA-ASR was defined as a LA shape that was not ellipsoidal anymore (basal dimension measured at the junction between the pulmonary vein and atrium greater than the mitral annular dimension, as shown in fig. 1). SSR was defined as all other cases. The following parameters were measured: peak early diastolic mitral inflow velocity (E), peak late diastolic mitral inflow velocity (A), mid-diastolic mitral annular velocity (L), early diastolic mitral annular velocity (Em), late diastolic mitral annular velocity (Am), systolic mitral annular velocity (Sm), isovolumetric relaxation time (IVRT), E-wave deceleration time (EDT), mitral A-wave duration (Amdur), atrial reverse flow duration (Ardur), peak systolic pulmonary venous flow velocity (S), peak diastolic pulmonary venous flow velocity (D), peak systolic LA myocardial velocity (Sa), early diastolic LA myocardial velocity (Ea), and late diastolic LA myocardial velocity (Aa). LA function was assessed using the Sa, Ea and Aa parameters obtained by TDI on the LA lateral walls (fig. 2a), as described in the literature.[8,9] Moreover, PW parameters were measured on the right superior pulmonary vein (fig. 2b). According to previous studies on LA deformation properties, systolic myocardial velocity (Sa) reflects atrial reservoir function, and the early (Ea) and late (Aa) diastolic myocardial velocities reflect conduit and booster pump function, respectively.[10,11,12] Each final value of the PW and TDI parameters resulted from the average of three to five measurements at a sweep speed of 100 mm/s, with mean heart rate between 60 and 80 beats/min. These echocardiographic parameters assessing LA and LV diastolic function were used to identify LA-ASR markers. Additionally, two-dimensional transthoracic echocardiography was used to assess the following parameters: LA anterior-posterior diameter, LA area and volume, LV end-diastolic and end-systolic diameter, LV inter-ventricular septal dimensions and posterior wall dimensions, LV ejection fraction (EF) with Simpson's method, and LV mass with the linear method.[1] LA volume index was measured with the ellipsoid biplane area-length formula in all patients, regardless of SSR or ASR. All measurements were performed by the same operator (MF) on GE Vivid 9 (GE Healthcare) or Philips iE33 machines (Philips Medical Systems), with 3.5 MHz transducers.

Statistical Analysis

Statistical analysis was performed with SPSS 15.0 (SPSS Inc., Chicago, IL, USA) and MedCalc (Mariakerke, Belgium) software. Data were expressed as frequency distributions, simple percentages, mean values and standard deviations. The correlation between two variables was tested by linear regression analysis. The variables with a p-value lower than 0.05 in univariate analysis were entered into the multivariate model for stepwise regression analysis (the anterograde and retrograde LR and Wald tests). The area under the curve (AUC) was calculated as a quantitative measure of the predictive value. A p-value <0.05 was considered statistically significant.

Results

A total of 170 patients were included: 70 (41%) with stable sinus rhythm (SR) without history of nonvalvular AF, and 100 (59%) with AF (24% paroxysmal, 15% persistent and 61% permanent). The mean age of the patients was 67 ± 11 years, the mean body mass index was 28 ± 5 kg/cm^2, 61% were men, 74% had hypertension, 33% had coronary heart disease and 19% had diabetes mellitus. The mean ventricular frequency was 72 ± 10 per min. The mean LV mass in patients with ASR and SSR was 150 ± 38% g/m^2 (95% CI: 139-161) and 130 ± 43 g/m^2 (95% CI: 122-138) g/m^2, respectively (p=0.002). The mean LV EF in patients with ASR and SSR was 49 ± 15% (95% CI: 45-53) and 51 ± 12% (95% CI: 49-53), respectively (p=0.67)). LA-ASR was detected in 112 (66%) of the patients: 62 patients had AF (55%) and 50 patients had stable SR (45%) (p=0.002).

LA Volume Index Depending On The Type Of LA Remodeling

The mean LA-ASR and LA-SSR volume indexes, assessed by ellipsoid biplane area-length formula in all patients irrespective of

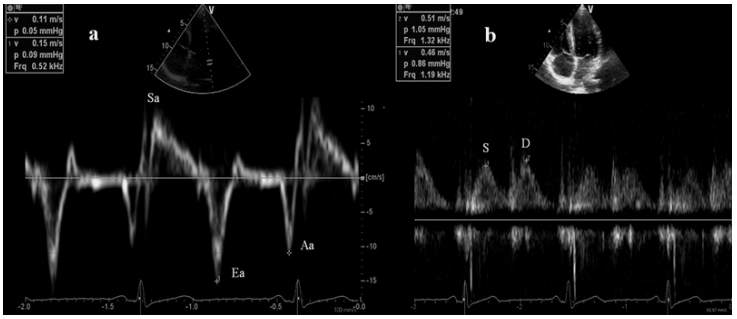

Figure 2: **Assessment of LA function (a) and pulmonary vein flow (b) by TDI respectively PW: Sa, Ea and Aa as index of LA reservoir, conduit and booster pump function, respectively**

(Aa=left atrium late diastolic myocardial velocity, D=peak pulmonary diastolic flow velocity, Ea=left atrium early diastolic myocardial velocity, S=peak pulmonary systolic flow velocity and Sa= left atrium systolic myocardial velocity).

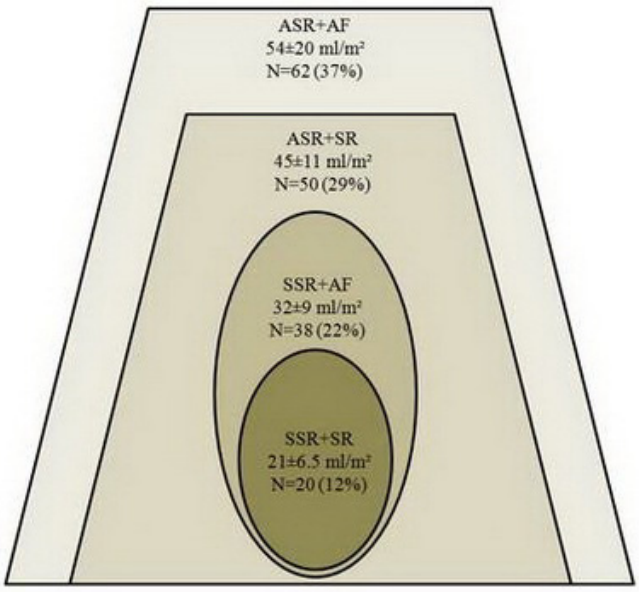

Figure 3: **Repartition of patients included in the study according to LA volume, type of LA remodelling and presence of AF or SR.**

(SSR+SR=patients with symmetrical structural remodelling and sinus rhythm, SSR+AF= patients with asymmetrical structural remodelling and atrial fibrillation, ASR+SR= patients with asymmetrical structural remodelling and sinus rhythm, ASR+AF= patients with asymmetrical structural remodelling and atrial fibrillation).

the presence of AF or SR, were 49 ± 14 ml/m² and 29 ± 13 ml/m², respectively (p<0.001); they were well correlated (r=0.567, p<0.001). The mean LA volume index in patients with ASR+SR (patients with LA-ASR and SR) vs. ASR+AF (patients with LA-ASR and AF) was 45 ± 11 ml/m² (n=50, 95% CI: 41.8-48) vs. 54 ± 20 ml/m² (n=62, 95% CI: 49-59.4) (p<0.001). The mean LA volume index in patients with SSR+SR (patients with LA-SSR and SR) vs. SSR+AF (patients with LA-SSR and AF) was 21 ± 6.5 ml/m² (n=20, 95% CI: 17.7-23.8) vs. 32 ± 9 ml/m² (n=38, 95% CI: 28.9-34.8) (p<0.001). The relationship between the degrees of LA dilation assessed by LA volume index, depending on the type of LA remodelling in patients with or without AF included in the study, is shown in Fig. 3. In patients with mild (area of 20-30 cm², n=140) and moderate (area of 31-40 cm², n=27) LA dilation the ASR/SSR ratio was 1.7 and 2.8, respectively (p=0.001). Only three patients had LA area greater than 40 cm² (severe LA dilation). Hypertension (r=0.772 vs. r=0.677, p<0.001), coronary heart disease (r=0.607 vs. r=0.567, p<0.001), left ventricular hypertrophy (r=0.747 vs. r=0.657, p<0.001) and diabetes mellitus (r=0.307 vs. r=0.276, p=0.005) correlated better with LA-ASR than LA-SSR.

Echocardiographic Parameters Associated With LA Asymmetrical Structural Remodeling

LA regional function could be assessed in 87% of patients (with TDI parameters: Sa, Ea and Aa) and pulmonary venous flow parameters in 81% of patients (with PW parameters: S and D). Mid-diastolic mitral annular velocity was identifying in 16 patients (9.4%). The TDI and PW echocardiographic measurements are summarized in table 1. The parameters depending on atrial contraction (LA booster pump function assessed by A, Am and Aa) were assessed only in patients with SR (n=70) and in patients with AF and SR at the time of inclusion in this study (n=24). The mean Sa in patients with ASR was significantly lower in patients with AF than SR (6.6 ± 2.4 cm/s, 95% CI: 5.9-7.2 vs. 7.6 ± 1.9 cm/s, 95% CI: 7.1-8.2; p=0.001). Similarly, the mean S in patients with ASR was significantly lower in AF than SR (36 ± 14 cm/s, 95% CI: 31.8-39.7 vs. 42 ± 12 cm/s, 95% CI: 38.6-44.7; p=0.005). On the contrary, there was no significant difference between these parameters in AF vs. SR in patients with SSR. The TDI and PW echocardiographic parameters associated with ASR by univariate analysis were: L, Amdur, Am-Ar, S, D, Em, Sm and Sa (all p<0.005). Multivariate logistic regression revealed LA systolic myocardial velocity (Sa; p=0.036) and peak systolic pulmonary venous flow velocity (S, p=0.033) as the best TDI and PW echocardiographic parameters associated with LA-ASR, with a power prediction of 91.8% (95% CI, p=0.0001). When correcting for the number of patients (90% CI), ASR prediction improved to 99.1%, but the statistical significance decreased slightly (p=0.073). The sensitivity and specificity of both parameters based on ROC curve analysis were 77 and 70%, respectively (fig. 4); the AUC was 0.765 (95% CI: 0.662-0.849, p=0.0001). Using the two echocardiographic parameters, we created an equation for the assessment of LA-ASR probability: (ez/(1+ez), where z = - 0.511 - 0.062 x (S) + 0.536 x (Sa), and developed a scale to estimate ASR probability in patients with LA dilation, regardless of electrical remodeling (fig. 5). For example, using this scale, a patient with S value of 40 cm/s and Sa value of 11 cm/s would have a probability of association with asymmetric remodeling of 95%.

Discussion

Asymmetric Structural Remodeling Of Dilated Atria: Relationship With Left Atrial Volume And Electrical Remodeling

We analyzed the frequency and the parameters independently associated with LA-ASR in dilated LA using echocardiographic parameters of LA function and LV diastolic function regardless of electrical remodeling. LA dilation reflects the severity and chronicity of diastolic dysfunction, as well as the magnitude of LA pressure elevation.[1] In diastole, the pulmonary veins "check and measure" the LV end-diastolic pressure during mitral valve opening. Then,

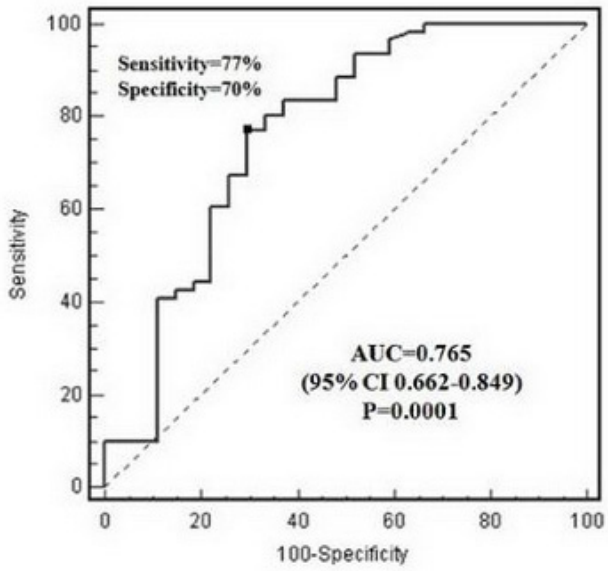

Figure 4: **ROC curve for prediction of the left atrium asymmetrical structural remodeling using peak pulmonary vein flow systolic velocity (S) and peak systolic atrial myocardial velocity (Sa).**

(AUC=area under curve; CI=confidential interval)

Sa /S	4	5	6	7	8	9	10	11	12	13	14
25	52	64	76	84	90	94	96	97	98.7	99.2	99.5
30	44	58	70	80	87	92	95	97	98.3	99	99.4
35	36	50	63	74	83	89	94	96	98	98.6	99.2
40	30	42	56	68	78	86	91	95	97	98	98.9
45	24	35	48	61	72	82	88	93	96	98	98.5
50	19	28	40	54	66	77	85	90	94	96	98
55	14	22	33	45	59	71	80	87	92	95	97
60	11	17	27	38	52	64	75	84	90	93	96
65	8	13	20	31	43	57	69	79	86	91	95
70	6	10	16	24	36	49	62	73	82	89	93
75	5	8	13	20	29	41	55	67	78	86	91

Figure 5: **Prediction scale of LA-ASR using the best echocardiographic parameters associated with LA-ASR: S**

(peak pulmonary vein flow systolic velocity) value (rows) and Sa (peak systolic atrial myocardial velocity) value (columns).

depending on the LV filling pressure, the left atrium adapts by its dilation and by changing function (from reservoir during ventricular systole, to conduit during early ventricular diastole, and to booster pump during late ventricular diastole). According to the Frank Starling law, a greater LA volume is associated with a greater LA reservoir function and increased LV filling in patients with SR.

In our study, LA volume was significantly greater in patients with ASR than SSR and AF patients than SR patients (Fig. 3). More patients with ASR, as compared to SSR, had AF (22% vs. 37%). But patients with AF did not significantly present more ASR than patients with SR. However, in statistics, absence of evidence is not evidence of absence.[13] Thus, although in this study patients with AF did not have significantly higher ASR than those with SSR, this does not provide evidence for the absence of a relationship between AF and ASR. However, in the present study, it is difficult to say that ASR and AF were related. Further (longitudinal) studies should be performed to test this hypothesis. The ASR/SSR ratio increased with LA dilation. Thus, LA enlargement was associated with ASR frequency. Therefore, these results indicate that (1) a more dilated atria is more often remodeled into an asymmetric structure, and (2) asymmetric dilation (mainly on the LA posterior wall) occurs more often in larger atria.

LA structural remodeling is related to LA electrical remodeling, and both contribute to the development and perpetuation of AF.[14] The assessment of LA volume in patients with ASR+SR and ASR+AF vs. SSR+SR and SSR+AF revealed that ASR+SR is associated with greater LA volume than SSR+AF (45 ± 11 ml vs. 32 ± 9 ml; p<0.001), probably because of functional remodeling and/or fibrosis apparition before electrical remodeling. This result indicates that the type of LA dilation (ASR or SSR) might be more important for LA structural remodeling than the LA electrical remodeling or only LA volume assessment. AF might be associated with ASR; the pulmonary vein would become arrhythmogenic because of LA pressure and activation of stretch-gated channels, secondary to LA enlargement mostly on the posterior wall.[2,14] However, this is only a hypothesis, which is difficult to sustain.

LA volume index was calculated with the ellipsoid biplane area-length formula in all patients, regardless of SSR or ASR. Even if LA dilation did not follow the ellipsoidal shape of a classical LA resulting in asymmetrical enlargement, in clinical practice, the ellipsoid biplane area-length formula could be still applied to assess LA volume because LA-ASR and LA-SSR volume were well correlated (r=0.567, p<0.001).

Echocardiographic Parameters Associated With Asymmetrical Structural Remodeling In Dilated Atria

The association between LA functional remodeling and AF seems stronger than between LA structural remodeling and AF.[15] A reduced LA reservoir function could be associated with a higher risk of atrial arrhythmia.[16] Indeed, ASR is not only related to electrical remodeling, but also to LA reservoir function. In patients with ASR, the mean Sa and S parameters were significantly lower, especially in patients with AF. LA reservoir function depends on the degree of LA dilation. Apparition of LA fibrosis decreases LA reservoir capacity. In patients with LA-SSR, these parameters were not significantly different between patients with AF and patients with SR, even if LA volume was significantly greater. Both Sa and S echocardiographic parameters reflect LA reservoir function. In this study, the parameters that reflected LA reservoir function were best associated with ASR. It has also been shown that LA reservoir function could predict first AF or flutter in persons > or = 65 years of age and AF recurrence after catheter ablation.[16,17] Clearly, asymmetric structural remodeling, electrical remodeling and functional remodeling seem to be all related to LA dilatation.

Clinical Implications Of Early Appropriate Assessment Of Asymmetric Structural Remodeling

In the literature, few papers focused on asymmetrical LA dilatation and its clinical significance. However, it is well known that a dilated LA might not have an ellipsoidal shape as a normal LA, because of asymmetrical enlargement. EAE guidelines for assessing LA recommended to check for the presence of asymmetric LA remodeling in dilated atria and also indicated that an increase in LA volume could be associated with adverse cardiovascular outcomes.[1,18] Therefore, the appropriate assessment of LA size is very important. We show in the present study that in a dilated LA, the dimension measured at the junction between the pulmonary vein and atrium could be greater than the mitral annular dimension, and thus in these cases, LA is not ellipsoidal anymore, and LA volume might be underestimated. Clinically, we think that it is important to identify LA-ASR and perhaps to be able to predict it, because LA dilatation and LA-ASR are related. Moreover, it is probably not appropriate to use an ellipsoidal formula to assess LA volume in those atria. This asymmetrical remodeling of the LA could also be a possible explanation for the well-accepted underestimation of LA volume by echocardiography as compared to MRI, computed tomography or ventriculography.[3,4] In clinical practice, an underestimation of LA size could lead to the inappropriate evaluation of LA volume, and therefore of adverse cardiovascular outcomes. For example, LA volume index is known to be an important parameter in the algorithm for the diagnostic of chronic heart failure with preserved ejection fraction (with a cut-off value of 34 ml/m2). An underestimation of the real LA volume could lead to a misdiagnosis of chronic heart failure with preserved ejection fraction in a large group of patients in

Table 1: Echocardiographic measurement by PW and TDI for all patients included in the study

Echocardiographic parameters	SSR			ASR		
	AF	SR	P value	AF	SR	P value
E (cm/s)	79±23	65±21	0.007	83±28	69±18	0.003
A (cm/s)*	57±21	69±22	NS	74±25	78±25	NS
E/A*	1.2±0.5	1±0.4	NS	1±0.43	0.96±0.44	NS
Sm (cm/s)	5.4±1.1	6.4±1.4	0.01	6.4±1.3	6.3±1.1	NS
Em (cm/s)	8.3±2.4	7.7±2.3	NS	9±2	8±2	<0.001
Am (cm/s)*	8.7±1.6	7.8±2.3	NS	8.8±0.7	8.7±1.7	NS
E/Em	10±3.2	8±2	0.035	9.4±3.6	9.1±2.6	NS
L (cm/s)	35±3	0	NA	26±4	27±9	<0.001
IVRT (ms)	98±17	99±24	NS	102±25	94±29	NS
TDE (ms)	194±74	203±50	NS	186±62	203±56	NS
Amdur (ms)*	132±6	132±8	NS	135±13	131±11	0.05
Ardur (ms)*	106±11	117±8	0.009	110±12	115±12	NS
Am–Ar (ms)*	26±10	15±5	0.006	25±7	17±6	<0.001
S (cm/s)	41±11	39±12	NS	36±14	42±12	0.005
D (cm/s)	41±16	36±14	NS	39±11	33±11	0.003
Sa (cm/s)	6.5±2.6	6.4±2.1	NS	6.6±2.4	7.6±1.9	0.001
Ea (cm/s)	8.6±2.7	7.4±1.9	NS	7.7±2.2	6.9±2.4	NS
Aa (cm/s)*	9.5±2.8	8.3±4.2	NS	9.2±4.4	10.4±3.3	NS

* Measurements were possible only in patients in SR (n=70) or known with AF but in SR at the time of inclusion (n=24).
A=peak late diastolic transmitral flow velocity; Aa=left atrium late diastolic myocardial velocity; AF=atrial fibrillation; Am=late diastolic mitral annular velocity; Amdur=mitral A wave duration; Ardur=atrial reflux duration; ASR=asymmetric structural remodeling; D=peak pulmonary diastolic flow velocity; E=peak early diastolic transmitral flow velocity; Ea=left atrium early diastolic myocardial velocity; Em=early diastolic mitral annular velocity; IVRT=isovolumetric relaxation time; L=mid-diastolic mitral annular velocity; NS=non-significant; PW= Pulse Doppler wave; S=peak pulmonary systolic flow velocity; Sa=left atrium systolic myocardial velocity; Sm= systolic mitral annular velocity; SR=sinus rhythm; SSR=symmetric structural remodeling; TDE=deceleration time of E wave; TDI=Tissue Doppler Imaging.

daily practice.

Early detection of LA-ASR and appropriate assessment of LA volume by non-invasive techniques like echocardiography could be very important, in terms of diagnostic and prognostic implications, because it could help correctly assess LA size and function in a very large group of patients. In our daily clinical practice, these echocardiographic parameters could be used in patients with dilated atria for early identification of LA-ASR and for appropriate assessment of structural remodeling before the occurrence of electrical remodeling. The treatment of patients with LA dilation with angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists may (1) induce reverse structural remodeling, (2) reduce AF progression from paroxysmal to persistent and (3) reduce the number of relapses.[19,20] Thus, early treatment onset with angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonist's therapy could be critical in patients with LA dilation without ASR. It is recently shown that earlier treatment with renin-angiotensin system inhibitors may improve abnormal LA-left ventricle interaction even in asymptomatic patients with left ventricle diastolic and systolic dysfunction.[21] However subtle LA and left ventricle dysfunction with individual cardiovascular risk factors are more aggravated with the comorbid conditions in asymptomatic patients.[22] In addition, it seems that LA dyssynchrony documented by three-dimensional echocardiography, could maintained despite early LA structural reverse remodeling observed 3 months after pulmonary vein isolation.[23] These results may affect medical therapy after successful pulmonary vein isolation. But in these specific group of patients late gadolinium enhancement MRI can identify better LA wall structural remodeling and stratify patients who are likely or not to benefit from ablation therapy.[24]

In conclusion, we think that it is important not only to quantify correctly LA size but also to make an appropriate identification of asymmetrical dilatation and to assess LA functional remodeling, because LA enlargement, asymmetrical and functional remodeling are related, asymmetrical remodeling frequency increase with the degree of LA dilatation, and because of risk to misdiagnosis important comorbidities like chronic heart failure with preserved ejection fraction in a large group of patients of our daily practice.

Study Limitations

LA area was used as inclusion criteria. LA volume is the preferred parameter recommended for the assessment of LA size according to the EAE recommendations. However, LA area is one of the parameters that can be used for the assessment of LA size. LA area is included in the ellipsoid biplane area-length formula. Therefore, as expected, LA area and LA volume assessed with the ellipsoid biplane area-length formula, were well correlated ($r=0.837$, $p<0.001$). We think that in clinical practice, the assessment of LA area, which is well correlated with LA volume, is easier and more rapid than that of LA volume. The echocardiographic parameters of pulmonary venous flow and LA function could not be accurately measured in all patients. It has been shown that the technique is feasible in only 80% of cases.[6] The assessment of regional LA function by TDI is angle-dependent, and LA walls are relatively thin. The measurement of TDI parameters is a technique that is currently used in clinical practice but not for LA function. Myocardial velocity is influenced by translation and tethering, and therefore, it is difficult to distinguish true shortening and lengthening of the atrium from mitral annular and ventricular motion. We carefully adjusted the beam on the

lateral wall and gain settings to avoid aliasing and to allow reliable measurement of tissue velocity. Thus, an alternative for LA function measurement that would have been more appropriate is LA strain and strain rate. However, it is a less-accessible, emerging technique that is not yet widely used. Inter-vendor variability is still an issue for this new echocardiographic method.

Conclusion

LA dilation was associated with a great number of asymmetrical structural remodeling in this specific group of patients. Echocardiographic parameters that reflect LA reservoir function are best associated with asymmetrical remodeling. In practice, the type of LA dilation, which is related to functional remodeling, might be the most important parameter to correctly assess LA structural remodeling.

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