Strain Rate Imaging for Noninvasive Functional Quantification of the Left Atrium: Comparative Studies in Controls and Patients With Atrial Fibrillation

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Strain rate (SR) imaging enables quantitative measurement of left ventricular (LV) function independent of cardiac translation. However, whether SR imaging is applicable for detection of left atrial (LA) dysfunction remains unknown. The purpose of this study was to assess the feasibility of measuring LA function by SR imaging, focusing on the effects of aging and LA dilatation during atrial fibrillation (AF). Echocardiographic evaluation including SR imaging was performed in 50 controls (29 males and 21 females; mean age, 41 ± 14 years) and in 27 patients with AF (15 males and 12 females; mean age, 62 ± 12 years; 8 with persistent AF and 19 with paroxysmal AF) from 3 apical views and analyzed off-line. Peak SR was measured at each LA segment (septum, lateral, posterior, anterior, and inferior), and mean peak systolic SR (SR-LAs), early diastolic SR (SR-LAe), and late diastolic SR (SR-LAa) were calculated by averaging the results for each segment. LA dimension, peak mitral and pulmonary velocities at late diastole, LA fractional shortening, and atrial filling fraction were calculated as parameters of LA function. Normal values for mean SR-LAs, SR-LAe, and SR-LAa were 3.4 ± 1.0 s⁻¹, −3.9 ± 1.7 s⁻¹, and −3.1 ± 1.0 s⁻¹, respectively, and they were successfully measured in more than 95% of the LA segments. In controls, both mean SR-LAs and mean SR-LAe correlated with age, LA dimension, and early to late diastolic mitral flow velocity ratio. Conversely, mean SR-LAa did not show significant correlation with age or parameters of LA function. In AF patients, mean SR-LAs was correlated inversely with age. The mean SR-LAs was significantly lower in persistent AF patients than in age-matched controls (1.7 ± 0.8 vs 2.9 ± 0.9 s⁻¹; P < .01). Based on our findings, we conclude that noninvasive quantification of LA function using SR imaging enables evaluation of LA dysfunction due to aging and LA dilatation.

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The left atrium (LA) functions not only as an atrial pump, but also as a reservoir and conduit to the left ventricle (LV). The hemodynamic importance of the LA for cardiac performance has been studied by using both invasive and noninvasive methods, such as M-mode, 2-dimensional, Doppler echocardiography, and an acoustic quantification technique. These indices have been applied for restored LA function after defibrillation and the maze procedure in patients with atrial fibrillation. However, quantitative assessment of LA function using invasive methods has been clinically difficult, because it requires simultaneous measurements of LA volume and pressure. Although several noninvasive methods have been used to assess LA function, major limitations, including single-plane assessment, dependence of altered left ventricular hemodynamics, and image quality, have prevented clinical application. Furthermore, peak velocities during atrial contraction on Doppler echocardiography have been used as indices of LA function, but they are only surrogate markers of LA function that represent blood flow during the atrial contraction phase, not intrinsic changes of the LA wall.

The development of novel echocardiographic techniques, such as tissue Doppler imaging (TDI), has enhanced the ability to assess regional myocardial function noninvasively. Although this technique has been used for the assessment of both regional ventricular function and regional atrial function, there remain some problems to be overcome.
including cardiac rotational motion and the tethering effect. Recently, strain rate (SR) parameters have been calculated as spatial differences in tissue Doppler velocities between 2 different myocardial regions. SR imaging enables quantitative measurement of regional function independent of cardiac rotational motion and the tethering effect.\textsuperscript{16-18} SR imaging has been applied to the detection of regional LV dysfunction,\textsuperscript{16-18} but not to the assessment of LA function. The purpose of this study was to assess the feasibility of measuring LA function using SR imaging and to define normal values of SR parameters of LA. We also compared LA function assessed by SR imaging in controls and patients with AF, focusing on the effects of aging and LA dilatation during AF.

**METHODS**

**Patient Population**

Fifty healthy subjects (29 males and 21 females, age 19-70 years) without cardiovascular disease, hypertension, or diabetes mellitus were enrolled as controls for assessing the reliability of SR parameters. None of these subjects had abnormal findings on physical examination or abnormalities on electrocardiographic or conventional echocardiographic examinations, and none were receiving cardioactive medication. A total of 27 patients with AF (15 males and 12 females, age 31-79 years), including 19 patients with paroxysmal AF (70%) and 8 patients with persistent AF (30%), were also enrolled as an AF group. Eight of the patients with paroxysmal AF had organic heart disease and/or underlying systemic disease (4 with hypertension, 2 with mild mitral stenosis, 1 with mild tricuspid regurgitation, and 1 with premature ventricular complexes). Six of the patients with paroxysmal AF exhibited LA dilatation on echocardiograms (LA dimension &gt; 40 mm). Paroxysmal AF was considered present when both AF and sinus rhythm had been documented on electrocardiograms. No regional or global systolic LV dysfunction (ejection fraction &lt; 50%), LV dilatation (LV end-diastolic diameter &gt; 55 mm), LV hypertrophy (both septal and posterior wall thickness &gt; 12 mm), or significant valvular disease was found in the controls and paroxysmal AF patients. All patients except those with persistent AF were in sinus rhythm at the time of the echocardiographic examination.

Four of the patients with persistent AF had organic heart disease (1 with hypertensive heart disease, 1 with severe mitral stenosis, 1 with severe tricuspid regurgitation, and 1 with dilated cardiomyopathy). Seven of the patients with persistent AF exhibited LA dilatation on echocardiograms. The AF duration determined from the first episode of AF was 59.3 ± 52.1 months (range, 0.5 to 180 months). Informed consent was obtained from all subjects.

**Echocardiographic Study**

A commercially available ultrasound machine (Vivid 7; General Electric Medical System, Milwaukee, Wis) equipped with a 2.5-MHz variable-frequency transducer was used for all of the echocardiographic evaluations. Standard echocardiographic views, including parasternal long-axis and apical 4-, 3-, and 2-chamber views, with the subjects in the left lateral decubitus position, were obtained in 2-dimensional and color TDI modes. The septal and posterior wall thicknesses at end diastole (mm) and LV end-diastolic dimension (mm) were determined from M-mode echocardiograms. LV volume was computed from the biplane modified Simpson’s (mL) at end diastole and end systole, and LV ejection fraction (%) was calculated by the standard method. Three LA anteroposterior dimensions were determined by M-mode: maximum at LV end systole (LA dimension [mm]), immediately before atrial systole (LDa), and minimum at the end of atrial contraction (LAdd). LA fractional shortening was calculated by the following formula:5,6 LA fractional shortening = (LDa – LAdd)/LDa × 100 (%). LV mass was calculated using the formula proposed by Levy et al\textsuperscript{19} and normalized for body surface area (LV mass index, g/m²). Each parameter was obtained from an average of 3-5 measurements.

Transmitral flow velocities were obtained by pulsed wave Doppler echocardiography, positioning a sample volume at the level of the mitral tip in an apical 4-chamber view. Mitral flow parameters measured included peak velocities during early diastole (E) and late diastole (A), their ratio (E/A ratio), and E wave deceleration time. Atrial filling fraction (%) was calculated using the ratio of the time velocity integral of the mitral A wave to the total time velocity integral of mitral inflow.6 Pulmonary venous flow velocities were measured from the apical 4-chamber view by sampling the right upper pulmonary vein and placing a sample volume of 1 cm into the pulmonary vein. Pulsed wave Doppler signals were obtained at a sweep speed of 100 mm/s. All examinations were performed using second harmonic imaging.

**Measurements of SR Imaging in the LA**

Gray-scale and color tissue Doppler images from apical 4-, 3-, and 2-chamber views were recorded, with frame rates of 80-120 frames/sec automatically changed by the sector width. Data of single cardiac cycle loops triggered to the QRS complex were saved on a magneto-optical disk (MO-4.8 GB; IMATION, Japan) and were analyzed off-line using commercial software (Echopac version PC; General Electric Medical Systems).

SR is measured from TDI data on the basis of instantaneous differences in myocardial velocities (corrected for distance −6 mm in this study) (Figure 1). A 2 × 6 mm region of interest was positioned on each mid-segment of the LA wall in 4-, 3-, and 2-chamber views and was manually tracked frame by frame to maintain its position within the LA wall. SR presents dimensionless descriptions of change in length reflecting the deformation of tissue due to applied force and is...
negative in shortening of the myocardium and positive in stretching of the myocardium. Representative SR curves in the controls and the patients with paroxysmal and persistent AF are shown in Figures 2 and 3. In the patients with persistent AF, no decrease in SR by atrial contraction was observed, but small phasic movements by the AF wave were recorded. SR-LAs, SR-LAc, SR-LAa were measured from SR curves in each of the 5 mid-LA segments (septum, lateral, posterior, anterior, and inferior), and mean SR-LAs, mean SR-LAc, and mean SR-LAa were calculated by averaging the results for each segment. Data were excluded if a smooth SR curve could not be obtained or if the angle between the scan line and LA wall was > 30 degrees, to preclude the angle dependency of these parameters. To reduce noise, data were taken from 3 consecutive heart cycles and averaged.

**Statistics Analysis**

All numeric data are expressed as mean ± standard deviation (SD). Differences in continuous variables between 2 groups were assessed using the unpaired Student t-test, and comparison among multiple groups was performed by analysis of variance with Scheffé’s post hoc test. Categorical variables were analyzed by the χ² test, with Fisher’s exact test used when appropriate. The correlation between 2 variables was tested by linear regression analysis. All data analysis was performed using commercially available statistical analysis software packages (Statview version 4.1; Abacus Concepts, Berkeley, Calif or SPSS version 9.0; SPSS Inc, Chicago, Ill). Results were considered statistically significant when the P value was < .05.

**RESULTS**

**Clinical Characteristics and Echocardiographic Findings**

The clinical characteristics and echocardiographic parameters of controls and AF patients are summarized in Table 1. Significantly larger percentages of both paroxysmal and persistent AF patients than controls were taking calcium antagonists (50% and 26%, respectively), digitalis (16% and 38%, respectively), or sodium channel blockers (25% and 58%, respectively). The percentage of paroxysmal AF patients taking angiotensin II receptor blockers (16%) was significantly larger than the percentage of controls these medications. The percentage of persistent AF patients taking beta-blockers (38%) or angiotensin-converting enzyme inhibitors (25%) was significantly larger than the percentage of controls taking these medications. There were no differences
in gender, body mass index, posterior wall thickness, LV dimensions, and LA fractional shortening among the 3 groups.

Reproducibility and Feasibility Data and Segmental Variation of SR Parameters

Of the 250 segments in the controls, 245 (98%) had adequate waveforms for assessment of SR. Of the 135 analyzed segments in AF patients, 122 (90%) had adequate waveforms for the assessment of SR. Interobserver and intraobserver variability of these variables, the entire process was repeated from the interobserver and intraobserver variability of the mean SR-LAs, SR-LAe, and SR-LAa were 0.6 ± 0.5 s⁻¹ (CV = 17.7%), 0.4 ± 0.3 s⁻¹ (CV = 16.9%), and 0.6 ± 0.5 s⁻¹ (CV = 18.7%), respectively. Significant segmental variation was found in SR-LAs and SR-LAe, but not in SR-LAa (Table 2).

Relationship of SR Parameters With Clinical and Echocardiographic Variables

In the controls, mean SR-LAs correlated inversely with age (Figure 4, A), LA dimension (Figure 4, B), and atrial filling fraction. Mean SR-LAs also correlated positively with E/A ratio. Mean SR-LAc correlated inversely with mitral E wave and E/A ratio. Mean SR-LAc also correlated positively with age, systolic blood pressure, LA dimension, mitral A wave, and atrial filling fraction (Table 3; Figure 4, C and D). In contrast, mean SR-LAa did not show significant correlation with any clinical and echocardiographic variable except body surface area (Table 3). Mean SR-LAs correlated inversely with mean SR-LAa (P = .001; r = −.45) and mean SR-LAc (P =
Table 2 Segmental variation of strain rate parameters of the left atrium in 50 controls

<table>
<thead>
<tr>
<th>Segment</th>
<th>SR-LAs (s(^{-1}))</th>
<th>SR-LAe (s(^{-1}))</th>
<th>SR-LAa (s(^{-1}))</th>
</tr>
</thead>
<tbody>
<tr>
<td>LA septum</td>
<td>2.6 ± 1.3 (49)</td>
<td>-2.6 ± 1.4 (49)</td>
<td>-2.9 ± 1.2 (49)</td>
</tr>
<tr>
<td>LA lateral</td>
<td>3.9 ± 1.5 (49)†‡</td>
<td>-4.5 ± 2.5 (49)†</td>
<td>-3.4 ± 2.0 (49)</td>
</tr>
<tr>
<td>LA posterior</td>
<td>3.5 ± 1.6 (49)</td>
<td>-5.2 ± 2.5 (49)†‡§</td>
<td>-3.2 ± 1.7 (49)</td>
</tr>
<tr>
<td>LA anterior</td>
<td>3.7 ± 2.0 (49)†‡§</td>
<td>-3.6 ± 2.5 (49)</td>
<td>-2.8 ± 1.6 (49)</td>
</tr>
<tr>
<td>LA inferior</td>
<td>3.0 ± 1.3 (49)</td>
<td>-3.5 ± 2.0 (49)</td>
<td>-3.2 ± 1.6 (49)</td>
</tr>
</tbody>
</table>

Data are presented as mean value ± SD or number of segments.
*Analysis of variance P < .01.
†P < .05 versus LA septum.
‡P < .05 versus LA inferior.
§P < .05 versus LA anterior.

Figure 4 Correlations between age and mean SR-LAs (A) and mean SR-LAe (C) in controls. Correlations between left atrial dimension (LAD) and mean SR-LAs (B) and mean SR-LAa (D) in controls. Open circles, controls; hatched circles, paroxysmal atrial fibrillation (AF) patients; solid circles, persistent AF patients.

Mean SR-Lac correlated positively with mean SR-LAa (P < .05; r = .31).

In the patients with AF, mean SR-LAs also correlated inversely with age (P < .05; r = -.51). Mean SR-Lac correlated positively with mean SR-LAa (P < .05; r = .31).

Age-matched data from 17 controls, 14 paroxysmal AF patients, and 8 persistent AF patients (age range, 50-75 years) were analyzed to eliminate the influence of aging on SR values, and then clinical and echocardiographic variables in the 3 groups were compared. Age-matched paroxysmal AF patients had a marginally lower mean SR-LAs (P = .07) than did age-matched controls, independent of LA dimension and Doppler parameters. Age-matched persistent AF patients had significantly lower mean SR-LAs than did age-matched controls (Table 4). Heart rate, systolic and diastolic blood pressures, LA dimension, septal wall thickness, and mitral E wave velocity were significantly larger in age-matched patients with persistent AF than in controls. No significant differences in mean SR-Lac were found among these age-matched groups (Table 4).

Table 3 Correlation between strain rate parameters of the left atrium and clinical characteristics and echocardiographic variables in controls

<table>
<thead>
<tr>
<th>r coefficient</th>
<th>Mean SR-LAs</th>
<th>Mean SR-LAe</th>
<th>Mean SR-LAa</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>-0.42**</td>
<td>0.66***</td>
<td>0.20</td>
</tr>
<tr>
<td>Body surface area</td>
<td>0.09</td>
<td>0.26</td>
<td>-.35*</td>
</tr>
<tr>
<td>Heart rate</td>
<td>0.14</td>
<td>0.19</td>
<td>-.24</td>
</tr>
<tr>
<td>Systolic BP</td>
<td>0.05</td>
<td>0.20*</td>
<td>-.10</td>
</tr>
<tr>
<td>LA dimension</td>
<td>-0.37**</td>
<td>0.42**</td>
<td>0.03</td>
</tr>
<tr>
<td>LV ejection fraction</td>
<td>0.11</td>
<td>0.04</td>
<td>0.11</td>
</tr>
<tr>
<td>LA fractional shortening</td>
<td>0.08</td>
<td>0.05</td>
<td>0.05</td>
</tr>
<tr>
<td>Mitral E</td>
<td>0.25</td>
<td>-0.60***</td>
<td>-0.11</td>
</tr>
<tr>
<td>Mitral A</td>
<td>-0.16</td>
<td>0.42**</td>
<td>-.06</td>
</tr>
<tr>
<td>E/A ratio</td>
<td>0.30*</td>
<td>-0.67***</td>
<td>-0.02</td>
</tr>
<tr>
<td>Deceleration time of E</td>
<td>0.15</td>
<td>0.01</td>
<td>0.04</td>
</tr>
<tr>
<td>Atrial filling fraction</td>
<td>-0.31</td>
<td>0.57***</td>
<td>0.15</td>
</tr>
<tr>
<td>Pulmonary A</td>
<td>0.04</td>
<td>-0.22</td>
<td>0.04</td>
</tr>
<tr>
<td>Pulmonary S</td>
<td>-0.04</td>
<td>0.08</td>
<td>0.25</td>
</tr>
<tr>
<td>Pulmonary D</td>
<td>0.20</td>
<td>-0.17</td>
<td>0.01</td>
</tr>
</tbody>
</table>

Abbreviations are as in Table 1.
*P < .05.
**P < .01.
***P < .001.

Comparison of SR Parameters in Controls and AF Patients

Both mean SR-LAs and mean SR-Lac in the controls were significantly higher in controls than in patients with paroxysmal AF and persistent AF. However, no significant difference in mean SR-LAa was found between the controls and the paroxysmal AF patients (Table 4).
Mean SR-LAs and mean SR-LAe tended to be lower in patients with paroxysmal AF with LA dilatation than in those with paroxysmal AF without LA dilatation, although the difference was not significant. In patients with paroxysmal and persistent AF, organic heart disease and antiarrhythmic or cardioactive medication use had no influence on systolic and diastolic SR parameters.

**DISCUSSION**

SR imaging has been proposed as a novel noninvasive technique to quantify regional LV function; however, little is known about SR imaging in the LA. To our knowledge, this is the first study attempting to quantify LA function by SR imaging. Major findings in this study are that SR imaging in the LA was successfully performed with high feasibility and modest reproducibility, and mean SR-LAs was significantly correlated with age and LA dimension and was significantly lower in patients with persistent AF than in age-matched controls.

**Assessment of LA Reservoir Function by SR Imaging**

SR imaging has been proposed as a method for assessing intrinsic myocardial function independent of cardiac rotational motion and the tethering effect.16-18 Actually, the SR curve of LA obtained in this study is very similar to previously reported curves of LA area derivative9 and LA volume derivative (dV/Δt).20 Several indices during systole as shown in volumetric examination have been considered as indicators of LA reservoir function.9,10 We focused on the initial positive movement of an SR parameter (ie, SR-LAs) during systole (Figure 2), that is, in passive stretching of the LA wall during LV systole. Thus this index could be used as an index of LA reservoir function. Mean SR-LAs decreased with advance of age. Several previous studies 21,22 have demonstrated a relationship between aging and histological changes in the LA (eg, increased fibrosis) in normal hearts. Masugata et al23 reported that fibrosis of the LA wall increases in normal hearts and that LA wall elasticity, an important factor regulating LA reservoir function, increases with aging. These findings support our results. However, Spencer et al9 reported that parameters of LA reservoir function obtained by acoustic quantification did not vary with aging. Acoustic quantification measures only changes in the area of the LA, not LA myocardium function directly. The difference in methodology may explain this discrepancy.

Interestingly, no significant difference in mean SR-LAs was found between age-matched paroxysmal AF patients and age-matched controls. This finding may be explained by the interval from the last episode of AF. Seventeen paroxysmal AF patients had a long interval (> 3 months) from the last episode of AF. Schotten et al24 reported that LA dysfunction induced by paroxysmal AF resolved completely in 2 days. Thus, decreased LA reservoir function may be detected by SR imaging shortly after the termination of AF. Barbier et al25 reported that reduced movement of the LV base during ischemia determines the LA reservoir function. However, no patients had a history of ischemic heart disease, and most of them had normal LV systolic function. The decreased mean SR-LAs in patients with persistent AF is consistent with findings of a previous invasive study4 and may reflect not only impairment of LA wall stretching, but also histological changes (eg, fibrosis and necrosis) in the LA myocardium in lone AF patients.26

**Assessment of LA Booster Pump and Conduit Function by SR Imaging**

There is no widely accepted gold-standard parameter reflecting LA contractility. Late diastolic mitral flow velocity has been used as a parameter of LA contractility.9,11 however, the assessment of late diastolic mitral flow velocity reflects only the atrioventricular pressure gradient between the LV and LA, not LA intrinsic function per se.27 LA acoustic quantification also allows assessment of LA booster function noninvasively.9,10,28 However, like all ultrasound techniques, assessment of LA function using

| Table 4 Comparison of LA strain parameters of controls and AF patients |
|-----------------------------|-----------------------------|-----------------------------|
|                            | Mean SR-LAs (s⁻¹)           | Mean SR-LAe (s⁻¹)           | Mean SR-LAs (s⁻¹)           |
| Controls                   | 3.4 ± 1.0 (n = 50)          | −3.9 ± 1.7 (n = 50)         | −3.1 ± 1.0 (n = 50)         |
| Paroxysmal AF              | 2.4 ± 1.0* (n = 19)         | −2.4 ± 1.2* (n = 19)        | −2.6 ± 1.1 (n = 19)         |
| Persistent AF              | 1.7 ± 0.8* (n = 8)          | −1.8 ± 1.1* (n = 8)         | −2.7 ± 0.8 (n = 17)         |
| Age-matched controls       | 2.9 ± 0.9 (n = 17)          | −2.6 ± 1.1 (n = 17)         | −2.5 ± 1.1 (n = 14)         |
| Age-matched paroxysmal AF  | 2.2 ± 0.7 (n = 14)          | −2.4 ± 1.2 (n = 14)         | −2.5 ± 1.1 (n = 14)         |
| Age-matched persistent AF  | 1.7 ± 0.8† (n = 8)          | −1.8 ± 1.1 (n = 8)          | −2.5 ± 1.1 (n = 14)         |

Data are presented as mean value ± SD.

*P < .05 versus controls.
†P < .01 versus age-matched controls.
this method depends on image quality. Furthermore, LA function is assessed by only single-plane, not multiplane, techniques. Recently, TDI has been applied for evaluating LA booster function. Although this method can measure LA myocardial velocity as a direct indicator of LA booster function, the effect of LV motion on LA myocardial velocity remains a major problem. These limitations may account for the discrepancy between the augmented LA booster pump parameters with aging and the lack of augmented mean LA-SRa with aging. SR-LAa is negative during LV late diastole (Figure 2), which means shortening of the LA wall; thus it could be used as an index of LA booster function. Dernellis et al demonstrated that LA pump function decreases with increasing pulmonary wedge pressure. The controls and paroxysmal AF patients had normal systolic LV function, no LV dilatation, and no LV hypertrophy and were thought to have nonelevated pulmonary wedge pressure. This may be another explanation of why mean SR-LAa did not change with aging. Conversely, our findings are supported by results of a previous study showing that LA contractility of healthy subjects assessed by M-mode echocardiography (eg, LA fractional shortening) is hardly affected by aging. No significant difference in LA fractional shortening was found in the controls and patients with paroxysmal AF. Furthermore, LA fractional shortening was correlated with mean SR-LAa in patients with paroxysmal AF. These findings suggest that LA fractional shortening is also an indicator of LA intrinsic function.

SR-LAc was assessed in a phase in which LA worked mainly as a conduit and could be used as an index of LA conduit function. Decreased LA conduit function (eg, passive emptying fraction) with aging and advancing LV diastolic dysfunction have been demonstrated and were also found in our study.

### Clinical Implications

Assessment of LA function using SR imaging may permit the detection of LA dysfunction noninvasively. Mean SR-LAs tended to be less in patients with persistent AF than in those with paroxysmal AF. This finding suggests that stretching of the LA wall is impaired during the progression from paroxysmal AF to persistent AF. After defibrillation, LA mechanical function gradually recovers, suggesting a return of LA contractility. SR imaging may allow evaluation of the recovery of LA reservoir and booster function in the very early phase. Kono et al suggested that increased pressure on the LA myocardium may lead to progressive intrinsic LA dysfunction. Thus LA dysfunction will be present even in a stage with slightly elevated LA pressure. In this stage, parameters of LA contractility assessed by conventional Doppler echocardiography are augmented; thus it will be difficult to detect LA dysfunction by this method. SR imaging may enable detection of LA dysfunction even in this stage. LA dysfunction induces thrombogenesis and intra-arterial stasis, and consequently, it may be possible to predict the risk of stroke and thromboembolism. Follow-up study is needed to determine whether LA dysfunction assessed using SR imaging is a predictor for the development of cardiac events (eg, stroke, thromboembolism, atrial arrhythmias).

### Study Limitations

The need to manually track the LA wall and reposition the region of interest on each of the 5 walls frame by frame makes using this method in a clinical setting prohibitively tedious. The manual tracking system is associated with the modest reproducibility of LA SR parameters. The reproducibility of LA SR parameters also may have been hampered by the small region of interest (2 × 6 mm). Accurate tracking requires approximately 20 minutes for training and data analysis per patient. Consequently, SR imaging in the LA as it currently stands is limited to use as a research tool, and further technical developments, such as the introduction of an automated tracking system, will be needed before it can be applied as a clinical tool. The associations between SR parameters and LA function are speculative, but invasive or histological studies in normal subjects would not be ethically acceptable. Measuring the level of atrial natriuretic peptide, a biochemical marker of increased atrial pressure, may provide information to support our findings. The small number of AF patients in each subgroup limited the power of our conclusions. Large prospective studies are needed to determine whether SR imaging can provide new important insights into LA function in AF patients.

### Conclusions

Noninvasive quantification of LA function is feasible by SR imaging. In controls, mean SR-LAs was associated with aging, LA dilatation, and LV diastolic function. The decreased mean SR-LAs in patients with persistent AF may reflect impairment of passive stretching of the LA wall. SR imaging might be useful for detecting LA dysfunction due to aging and LA dilatation.

### REFERENCES


