Introduction

In severe chronic primary mitral regurgitation (MR), the left ventricle (LV) and left atrium (LA) are subject to increased preload. Despite this, these patients may remain asymptomatic for a long time. Conventional indices of LV function are difficult to interpret in this situation, and the enhanced or normal LV ejection fraction may mask underlying ventricular dysfunction. Assessment of LA size and function may provide further information on the level of cardiac compensation with regards to the timing of surgery.

Increased LA mechanical work in chronic MR may contribute to LA failure and atrial fibrillation. LA contractile performance improves after surgery, suggesting a state of decreased atrial function prior to surgery, even in patients with apparently normal LV function. LA size also carries prognostic information in this disease. LA enlargement is accompanied by chronic inflammatory changes, cellular hypertrophy, and interstitial fibrosis that increase vulnerability to atrial fibrillation. The presence of atrial fibrillation and LA dilatation in organic MR and well-compensated LV function.

Methods

We recruited 27 patients with chronic degenerative mitral valve (MV) disease (due to MV prolapse) and moderate-to-severe MR from cardiology clinics at University Hospitals of South Manchester. Their eligibility for inclusion in this study was confirmed by estimation of the MR regurgitant fraction (calculated by pulsed wave Doppler at the MV annulus and left-ventricular outflow) which was 59 ± 14%, placing our patients clearly in the moderate-to-severe range according to American Society of Echocardiography criteria.

Conclusion

LA deformation is increased in all phases in MR. Unchanged LA EF and reduced A' may reflect the reduced contractile contribution to left ventricular filling.

Aims

To study global and regional left atrial (LA) mechanics in chronic primary mitral regurgitation (MR) with echocardiography.

Methods and results

LA volumes during reservoir, conduit, and contractile phases were measured in 27 MR patients and 25 controls. LA ejection fraction (EF) and ejection force were calculated. Reservoir (SR-R), conduit (SR-C), and contractile phase (SR-A) strain rates, and reservoir phase strain were obtained. LA volumes were higher in MR in all phases. In MR, ejection force was increased (21.5 vs. 12.3 kdynes, P = 0.001); reservoir phase strain (32.91 ± 14.26%); SR-R (2.65 ± 0.87); SR-C (−2.02 ± 0.58), and SR-A (−2.55 ± 1.31 s⁻¹) were increased (23.14 ± 7.96%, 1.62 ± 0.53, −1.29 ± 0.59, −1.98 ± 0.65 s⁻¹, in controls, respectively, P ≤ 0.004). Regional deformation correlated with corresponding volumetric parameters. Despite enhanced SR-A in MR, LA EF was unchanged (31.34 vs. 29.23%, P = ns), and LA contractile tissue velocity (A') was reduced (−5.39 ± 1.95 vs. −6.91 ± 1.80 cm/s, P = 0.006). The LA contractile contribution to left ventricular filling was significantly reduced in MR. LA deformation is increased in all phases in MR. Unchanged LA EF and reduced A' may reflect the reduced contractile contribution to left ventricular filling.

KEYWORDS

Left atrium; Tissue Doppler; Strain; Strain rate; Mitral regurgitation

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All patients were asymptomatic and had a normal or supranormal ejection fraction (EF). Exclusion criteria were a history of ischaemic heart disease, atrial fibrillation, or other sustained atrial arrhythmias, known coronary stenosis of more than 70%, past history of cardiothoracic surgery, co-existent valve lesions (except tricuspid regurgitation), haemoglobin concentration <10 g/dL, creatinine concentration more than 180 µg/L, congenital heart disease, and severe lung and/or systemic disease. Twenty-five age-matched healthy controls, with no history of cardiovascular or systemic disease, were recruited from hospital staff and a local fracture clinic, for comparison. All patients and controls gave written informed consent before entering the study, which was approved by our local Ethics Committee.

Echocardiography of left atrium

All echocardiograms were performed using a Vivid 7 system (GE Vingmed, Horten, Norway) using a 2.5 MHz probe. Parasternal long axis and apical views of the LV were recorded. Images of the LA were recorded from the parasternal long-axis view and apical windows for dimensions and volume measurements. Pulsed wave Doppler recordings at the mitral annulus level were taken for estimation of transmural blood flow during the different atrial phases. Pulsed wave Doppler at the LV outflow tract was obtained for timing of aortic valve closure.

2D colour tissue Doppler images of the atrial walls were obtained from the apical views. Sector width and depth was adjusted such that each of five atrial walls (interatrial septum, lateral, inferior, anterior, posterior) were studied individually at high frame rates. A minimum of three cardiac cycles were recorded, during an end-expiratory breath hold. Care was taken to ensure good angulation and avoid echo drop-out at the foramen ovale (in apical four chamber view) and base of left atrial appendage (in apical two chamber view).

Analysis of conventional echocardiography data

All analysis was carried out offline on data stored on compact disc, using customized software (Echopac v4.0.1 GE Vingmed Ultrasound), by one experienced operator (A.N.B.). LV wall and chamber dimensions were measured from the parasternal long-axis view. LV volumes and EF were calculated using the modified Biplane Simpson’s method. LA anteroposterior diameter was measured from the parasternal long-axis view at end-systole. LA volumes were measured using the area–length method, from the apical four and two chamber views.19 The following LA volumes were measured:

(a) Maximum LA volume (LAVmax): just prior to MV opening
(b) LA volume prior to atrial contraction (LAVp): just prior to ECG p wave
(c) Minimum LA volume (LAVmin): immediately after MV closure.

From these volumes, the following dynamic volumes were calculated:

(a) LA reservoir volume: LAVmax – LAVp
(b) LA passive emptying volume: LAVmax – LAVp
(c) LA conduit volume: LV total stroke volume – LA Reservoir Volume
(d) LA contractile volume: LAVp – LAVmin.

LA ejection fraction (LA EF) was calculated as LA contractile volume/LAVp, and expressed as a percentage. The LA ejection force,20 in units kdynes, was calculated as follows:

LA ejection force = 0.5 × ρ × mitral orifice area × A².

where ρ is the density of blood (1.06 g/cm³), and A is the velocity of the late diastolic wave of mitral inflow at the level of the MV annulus in cm/s. Where the E and A waves are partially fused, the A wave was taken as the perpendicular difference between the crossover point and the peak of the late diastolic transmitral flow. Regarding transmitral flow, velocity-time integral (VTI) of three phases was measured: early, diastasis, and late diastolic filling. Multiplying VTI with MV orifice area yielded transmitral flow for each of these phases. The contribution of the three mitral inflow phases to total LV stroke volume was calculated by dividing the VTI of each phase by the total mitral inflow VTI.21

Analysis of tissue Doppler data

Complete analysis of tissue Doppler data required ~20 min for each patient. A sample volume was placed at 2 cm above the MV annulus for each of the interatrial septal, lateral, inferior, anterior, and posterior walls. The sample volume was appropriately tracked throughout the cardiac cycle. Longitudinal atrial deformation was measured using an offset distance of 10 mm. A sample length and width of 12 and 6 cm was used, respectively. Linear drift compensation function was activated for strain curves. Gaussian averaging (40 ms) was used for smoothing of curves. Stroke was calculated relative to end diastole, which was defined as the peak of the ECG R wave.

SR was measured during the reservoir phase (SR-R), conduit phase (SR-C), and active atrial contractile phase (SR-A); peak strain at the end of the reservoir phase (at aortic valve closure) was also recorded (Figure 1). Each value is the mean of at least three measurements. Global SR and strain values were calculated by averaging values from all five sites.

Systolic (S) and diastolic mitral annular velocities (E’ and A’) were measured from five annular sites and averaged. Similarly, corresponding LA systolic and diastolic velocities, measured from the same sample volume used for strain and SR, were averaged from five atrial walls.

Figure 1 Strain rate and strain traces for the reservoir, conduit and atrial contractile phases of atrial function. Black lines represent tissue-Doppler deformation curves. Grey lines represent ECG. SR-R, reservoir phase SR; SR-C, conduit phase SR; SR-A, atrial contractile phase SR; AVC, aortic valve closure.
Statistics
Analyses were performed by SPSS for Windows release 11.0 (Chicago, IL, USA). Normally distributed variables are expressed as means ± 1 standard deviation, while non-normally distributed variables are expressed as median (interquartile range). Comparisons between groups were carried out using independent samples Student’s t-test and Mann Whitney U-test. Correlations between variables were carried out using Spearman’s Rank Correlation. Proportions were compared using the χ² test with Yates continuity correction. A significance level of <0.05 was used for all statistical tests. For measurement variability, seven MR and seven control patients were randomly selected, and variability for tissue Doppler measurements of SR-R and peak reservoir phase strain was calculated. To calculate intra-observer variability, two sets of measurements were carried out by one operator, 2 weeks apart. To calculate inter-observer variability, measurements were carried out by a second operator. Variability was estimated using the percentage variability (absolute differences between two measurements divided by the mean of the two measurements) and the repeatability co-efficient.

Results
Baseline characteristics
Baseline characteristics of the study population are shown in Table 1. Mitral regurgitant fraction and volume in the MR population were 59 ± 14.2% and 54.2 ± 20.4, respectively. LV EF, LV, and LA diameters are increased in MR compared with controls.

Left atrial volumes and global function
Figure 2 summarizes the differences in static and dynamic volumes between MR and controls. There is increased flow into the LA during the reservoir period (combined flow from the pulmonary veins and from the mitral regurgitant jet), increased flow across the MV during early (passive filling) and late diastole (contractile phase filling), and increased flow of blood from the pulmonary veins during the conduit phase in early diastole. Expressing the VTI of the early, diastasis, and late diastolic filling of the LV as a percentage of total VTI of transmural flow, the individual contribution of each phase (passive, diastasis, and active filling) to the total stroke volume of the LV can be assessed. Passive filling contribution is increased in MR: 62.27 ± 6.19% vs. 58.22 ± 4.44%, P = 0.01 while active filling contribution is decreased: 30.65 ± 7.82% in MR vs. 39.94 ± 6.09% in controls, P < 0.001. Diastasis contribution was also higher in MR compared with controls, having a median value of 7.0 (0–12.2%) in former vs. 0.0 (0.0–1.1%) in latter group, P = 0.002. Measurable diastasis flow was present in 63% of MR and 24% of controls, P = 0.005. There was a significant positive correlation between LAVp and contractile volume in both controls (R = 0.558, P = 0.005) and MR (R = 0.562, P = 0.003).

Transmitral E velocity was increased in MR while A velocity was unchanged, resulting in a significantly raised E/A ratio in MR compared with controls (Table 2). Longitudinal tissue velocities of the LA walls were averaged from five walls. LA S and E’ tissue velocity were similar in the two groups; peak A’ velocity was, however, decreased in MR.

As a measure of global contractile function, LA EF was not significantly different between controls (38.80, 29.23–44.72%) and MR (31.34, 23.02–42.76%), P = NS. However, the ejection force was significantly increased in MR (21.46, 13.43–28.32 kdynes) compared with controls (12.28, 8.59–17.71 kdynes), P = 0.001.

Regional left atrial deformation
For SR analysis, in the MR group, 30 out of a total of 135 LA segments were excluded due to echo dropout or artifact. This was roughly twice the corresponding drop-out rate in controls, in which 13 of 125 segments were excluded from analyses, P < 0.001. SR was increased during the three phases in all five walls in MR, reaching statistical significance in most walls. Reservoir strain was also increased in all walls in MR, reaching significance in inferior wall only (P < 0.01). Data for deformation values averaged from all five walls were subjected to further analysis. Average SR (SR-R) and strain were increased in the reservoir phase, while SR was also increased in conduit (SR-C) and atrial contractile phase (SR-A), in MR compared with controls (Figure 3). In addition, the relationship between atrial deformation and phasic flow was demonstrated by the presence of significant correlations between SR and corresponding dynamic volumes during the reservoir and early filling phases in the MR subgroup (Figure 4A and B). SR-A did not correlate with active contractile volume index, but showed significant direct correlations with LA EF and ejection force (Figure 4C and D).

Table 1  Baseline characteristics of the study population

<table>
<thead>
<tr>
<th>Variable</th>
<th>MR (n = 27)</th>
<th>Controls (n = 25)</th>
<th>P-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>64.7 ± 12.5</td>
<td>64.6 ± 12.0</td>
<td>NS</td>
</tr>
<tr>
<td>Gender (% males)</td>
<td>81.5</td>
<td>52.0</td>
<td>0.05</td>
</tr>
<tr>
<td>Body surface area (m²)</td>
<td>1.85 ± 0.14</td>
<td>1.85 ± 0.22</td>
<td>NS</td>
</tr>
<tr>
<td>Heart rates (bpm)</td>
<td>74 ± 10</td>
<td>70 ± 14</td>
<td>NS</td>
</tr>
<tr>
<td>Systolic blood pressure (mmHg)</td>
<td>143 ± 15</td>
<td>148 ± 21</td>
<td>NS</td>
</tr>
<tr>
<td>LV end-systolic diameter (cm)</td>
<td>3.26 ± 0.54</td>
<td>3.05 ± 0.52</td>
<td>NS</td>
</tr>
<tr>
<td>LV end-diastolic diameter (cm)</td>
<td>5.59 ± 0.77</td>
<td>4.62 ± 0.63</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LV end-diastolic volume index (mL/m²)</td>
<td>23.9 ± 7.7</td>
<td>20.1 ± 5.3</td>
<td>0.04</td>
</tr>
<tr>
<td>LV ejection fraction (%)</td>
<td>67.8 ± 6.1</td>
<td>62.0 ± 5.7</td>
<td>0.001</td>
</tr>
<tr>
<td>MV annulus area (cm²)</td>
<td>11.7 ± 2.7</td>
<td>7.5 ± 2.2</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>LA diameter index (cm/m²)</td>
<td>2.38 ± 0.47</td>
<td>1.97 ± 0.23</td>
<td>&lt;0.001</td>
</tr>
</tbody>
</table>

The bold-face values represent P < 0.05.
In MR, SR-C correlated with SR-R ($r = 0.55, P = 0.004$) and reservoir phase strain ($r = 0.58, P = 0.002$). In addition, SR-R and reservoir phase strain showed significant correlations with SR-A ($r = 0.55, P = 0.004$ and $r = 0.58, P = 0.002$, respectively) and MV annulus longitudinal systolic velocities ($r = 0.42, P = 0.032$ and $r = 0.71, P < 0.001$, respectively).

Finally, in controls, there was a significant negative correlation between age and SR-A ($r = -0.46, P = 0.019$), while SR-R, SR-C, and reservoir phase strain showed no relationship with age. Passive filling contribution to LV filling decreased with age ($r = -0.46, P = 0.02$) while active filling contribution increased with age ($r = 0.55, P = 0.005$).

<table>
<thead>
<tr>
<th>Doppler modality</th>
<th>Variable</th>
<th>MR</th>
<th>Controls</th>
<th>$P$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Transmitral flow Doppler</td>
<td>Peak E wave velocity (m/s)</td>
<td>1.14 ± 0.36</td>
<td>0.74 ± 0.13</td>
<td>&lt;0.001</td>
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<td></td>
<td>E wave deceleration time (ms)</td>
<td>182 ± 44</td>
<td>184 ± 37</td>
<td>NS</td>
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<td></td>
<td>Peak A wave velocity (m/s)</td>
<td>0.71 ± 0.14</td>
<td>0.75 ± 0.14</td>
<td>NS</td>
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<td></td>
<td>$E/A$ ratio</td>
<td>1.67 ± 0.67</td>
<td>1.00 ± 0.22</td>
<td>&lt;0.001</td>
</tr>
<tr>
<td>Left atrial tissue velocities (averaged from five walls)</td>
<td>Peak S velocity (cm/s)</td>
<td>4.43 ± 1.69</td>
<td>5.19 ± 1.31</td>
<td>0.08</td>
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<tr>
<td></td>
<td>Peak $E'$ velocity (cm/s)</td>
<td>-4.14 ± 1.56</td>
<td>-4.48 ± 1.26</td>
<td>NS</td>
</tr>
<tr>
<td></td>
<td>Peak $A'$ velocity (cm/s)</td>
<td>-5.39 ± 1.95</td>
<td>-6.91 ± 1.80</td>
<td>0.006</td>
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<tr>
<td></td>
<td>$E'/A'$ ratio</td>
<td>0.84 ± 0.21</td>
<td>0.71 ± 0.26</td>
<td>0.045</td>
</tr>
</tbody>
</table>

The bold-face values represent $P < 0.05$. 

In MR, SR-C correlated with SR-R ($r = 0.55, P = 0.004$) and reservoir phase strain ($r = 0.58, P = 0.002$). In addition, SR-R and reservoir phase strain showed significant correlations with SR-A ($r = 0.55, P = 0.004$ and $r = 0.58, P = 0.002$, respectively) and MV annulus longitudinal systolic velocities ($r = 0.42, P = 0.032$ and $r = 0.71, P < 0.001$, respectively).

Finally, in controls, there was a significant negative correlation between age and SR-A ($r = -0.46, P = 0.019$), while SR-R, SR-C, and reservoir phase strain showed no relationship with age. Passive filling contribution to LV filling decreased with age ($r = -0.46, P = 0.02$) while active filling contribution increased with age ($r = 0.55, P = 0.005$).
Reproducibility of left atrial tissue Doppler parameters

Intra-observer variability for SR-R was 11.1% (percentage variability) and 0.53 s⁻¹ (repeatability coefficient), while corresponding values for reservoir phase strain measurement were 11.8 and 8.64%. Inter-observer variability for SR-R was 18.1% (percentage variability) and 0.79 s⁻¹ (repeatability co-efficient), while corresponding values for reservoir phase strain were 18.1 and 13.09%.

Discussion

The main findings in this study are (i) static and dynamic LA volumes are increased in chronic primary MR compared with controls, (ii) longitudinal deformation indices derived from tissue Doppler echocardiography of the LA walls are enhanced in the reservoir, conduit, and active contractile phases in chronic MR, and (iii) LA deformation indices are correlated with volume changes during corresponding phases.

LA global function has three phases:²² a reservoir phase during which filling proceeds from the pulmonary veins, contemporaneous with ventricular systole; a conduit phase, characterized by passive flow from pulmonary veins down a pressure gradient initiated by LV relaxation; and an active contractile phase, during which the atrium contracts and expels blood into the LV in late diastole, LA regional function has been studied using strain gauges,²³ sonomicrometry,²⁴ and by echocardiographic colour tissue Doppler-derived velocities.¹⁶ The feasibility of measuring LA deformation during the contractile, reservoir, and conduit phases by tissue Doppler echocardiography in normal volunteers was explored by Sirbu et al.¹⁷ Due to the fact that the thickness of the LA is less than the limits of spatial resolution of this methodology, only longitudinal deformation from the apical views could be assessed. Heterogeneity in deformation magnitude and timing to peak deformation was noted in the atrial walls. Regional LA strain and SR were also found to be abnormal in myocardial infarction²⁵ and atrial fibrillation.²⁶

The left atrium in chronic mitral regurgitation: reservoir and conduit phases

Chronic MR induces LA enlargement, together with structural remodelling, chronic inflammatory changes, LA myocyte hypertrophy, interstitial fibrosis, and decreased matrix metalloproteinase expression,⁸,⁹ together with other ultrastructural abnormalities.²⁷ LA size and function also has an impact on prognosis in chronic MR. LA enlargement is predictive of symptoms in chronic MR,²⁸ is a precursor for development of atrial fibrillation,²⁹ increases the risk of cardiac death and need for surgery,¹⁴ and is a predictor of post-operative mortality.⁷ Atrial fibrillation develops in 48% of patients with medically treated degenerative MR after 10 years of follow-up, and is associated with increased morbidity and mortality, including sudden death.¹¹–¹³,³⁰ Despite the immense prognostic impact of the LA in chronic MR, it has received little attention in current guidelines concerning the management of these patients.¹⁵

The reservoir phase is essential for LV filling by storing energy during ventricular systole that is released after MV opening,³¹ a function which also depends on atrial compliance.³² This phase is also determined by left-ventricular basal systolic descent and LA contraction,³³ which is consistent with the direct correlations between LA reservoir phase deformation and MV annulus tissue Doppler S velocity in our
MR subgroup. MR causes dilatation of the LA, with an increase in the reservoir volume and in the rate of atrial filling during ventricular systole. Our patients also exhibited increased LAV_max and reservoir volumes (Figure 2), with a corresponding increase in SR-R and reservoir strain (Figure 3), possibly due to enhanced atrial compliance. The LA myocytes may also undergo hypertrophy in order to normalize wall stress. We did not find a relation between age and LA SR-R in healthy subjects, while other authors have demonstrated a negative correlation.

Passive LV filling is increased in MR. This has been attributed to an increased astroventricular gradient, decreased LV chamber stiffness and increased recoil of the LA. In our patients, this was manifest by an increased conduit function, as evidenced by increased peak transmural E wave (Table 2), conduit volume and SR-C (Figures 2 and 3). As expected, SR-C correlated with the passive emptying volume index (Figure 4B). In MR, this enhancement of conduit function may diminish with declining LV function, 

The left atrium in chronic mitral regurgitation: active contractile phase

Atrial contractile function is determined by preload, afterload, and contractility. Like the LV, the LA myocardium obeys Starling’s law, manifested as enhanced contractile function in response to increased preload or to the presence of MR. In our study, this is illustrated by the significant correlation between LA preload (LAVp) and LA contractile volume in the MR group. Reduced contractility caused by LA ischaemia, on the other hand, will result in decreased LA contractile volume despite an increase in LA preload.

Since MR is a state of enhanced preload, our patients had increased contractile volumes and SR-A. In accordance with the known preload-dependency of the contractile phase, SR-A correlated positively with reservoir phase deformation. The correlations between SR-A, and LA EF and ejection force reflect the validity of SR-A as an index of contractile function in our study (Figure 4C and D). In our study, LA EF was not significantly different from controls in MR, similar to previous findings. Also, in the MR group in our study, LA A’ velocity was decreased compared with controls, despite the enhanced preload, contractile volume, and contractile phase deformation. This may reflect the decreased LA contractile phase contribution to LV filling noted in our study, as discussed in what follows.

While passive filling contribution to total LV filling is enhanced, the LA contractile phase contributes towards a lesser proportion of LV filling in MR, which is in agreement with previous studies. The association between preload, afterload, LA contractile function, and the LA contractile contribution to LV filling is particularly complex: the presence of high LV filling pressures in MR will result in a greater atrial afterload, and thus a decreased atrial contractile contribution. Yet, an increased atrial preload, also seen in this situation, would tend to increase atrial contractile contribution. The contribution of LA contraction to LV filling is known to increase with age, but our groups were appropriately age-matched, making it unlikely for age to contribute to the differences we observed in the study. We can hypothesize that the reduced contribution of LA contraction in chronic MR may be due to a combination of increased early filling at the expense of late filling, and increased LV diastolic pressures (increased LA afterload), which is a direct consequence of MR. Ultimately, decreased LA contractility may also play a role. Progressive MR eventually results in decreased atrial contractile function.

The decreased contractile function with worsening MR is attributable to increased LA afterload (elevated LV diastolic pressures), an overwhelmed Frank-Starling mechanism, wherein the LA starts operating on the descending limb of the length-active tension curve with excessive preload, or to fibrosis and other cellular changes. Interestingly, in a group of patients with chronic primary MR, other authors have also noted an unchanged contractile fractional area change of the LA appendage together with decreased contractile phase tissue velocities of the LA appendage, compared to controls. These observations also led the authors to conclude that LA appendage function is impaired in chronic MR.

Atrial ejection force represents the force exerted by the atrium in accelerating blood into the LV. The increase in atrial ejection force in our MR patients is due to the wider MV annulus orifice in patients, as the other component of the equation, the A wave velocity, is not significantly different among between MR and controls.

Limitations

A potential error in planimetry of atrial areas is the inevitable visual extrapolation in the area of the pulmonary veins and atrial appendage on the roof and anterior wall of the LA, respectively. However, a recent study has confirmed the superior accuracy of the area-length method for LA volume, (which was employed in this study) compared with other methods. Echo dropout at the zone of the fossa ovalis (interatrial septum) and atrial appendage (anterior wall) often required moving the tissue Doppler region of interest away from or towards the mitral annulus, or abandoning strain and SR measurements altogether. The manual tracking of the region of interest to maintain its position over the thin atrial walls is tedious and time-consuming, especially when analysing a mobile and aneurysmal interatrial septum.

Based on our observations of changes in LA volumes and deformation, we attempted to speculate on changes in atrial compliance and contractility. However, a complete assessment of stress and strain relationships is necessary to describe these two parameters. For instance, in one study of patients with heart failure, LA stiffness was found to be increased despite increased reservoir volumes. Although we were unable to measure intra-atrial pressures, our conclusions are, however, supported by previous invasive studies.

We excluded patients with AF from this study. It may be interesting to see how the superimposition of AF on MR would affect LA myocardial deformation, since it is likely to further impair contractile function.

Finally, we have not assessed pulmonary venous inflow, which could potentially provide more information on LA function, due to difficulty in obtaining good pulmonary vein traces in our population using transthoracic echocardiography.
Conclusions
LA longitudinal deformation is enhanced in chronic MR, commensurate with increases in static and phasic volumes. In agreement with previous studies, we demonstrated a decrease in atrial contribution to LV filling in chronic MR. Despite a significant increase in LA ejection force, contractile volume, and contractile phase SR in MR, we found a decreased mitral annular A’ and unchanged LA EF, and conclude that these observations might reflect the decreased active contractile phase contribution to LV filling in chronic MR. Serial measurement of LA deformation may be able to detect the onset of decreasing LA compliance and contractile dysfunction that is known to occur in more advanced disease. Thus these parameters may prove to be useful in the timing of surgery prior to the onset of irreversible myocardial dysfunction.

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Disclosure from https://academic.oup.com/ehjcimaging/article-abstract/10/7/833/2396626 by guest on 04 November 2018


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