Assessment of Mitral Valve Adaptation With Gated Cardiac Computed Tomography

Validation With Three-Dimensional Echocardiography and Mechanistic Insight to Functional Mitral Regurgitation

Jonathan Beaudoin, MD; Wai-Ee Thai, MD; Bryan Wai, MD; Mark D. Handschumacher, BS; Robert A. Levine, MD; Quynh A. Truong, MD, MPH

Background—Mitral valve (MV) enlargement is a compensatory mechanism capable of preventing functional mitral regurgitation (FMR) in dilated ventricles. Total leaflet area and its relation with closure area measured by 3-dimensional (3D) echocardiography have been related to FMR. Whether these parameters can be assessed with other imaging modalities is not known. Our objectives are to compare cardiac computed tomography (CT)–based measurements of MV leaflets with 3D echocardiography and determine the relationship of these metrics to the presence of FMR.

Methods and Results—We used 2 cohorts of patients who had cardiac CT to measure MV total leaflet, closure, and annulus areas. In cohort 1 (26 patients), we validated these CT metrics to 3D echocardiography. In cohort 2 (66 patients), we assessed the relation of MV size with the presence of FMR in 3 populations: heart failure with FMR, heart failure without FMR, and normal controls. Cardiac CT and 3D echocardiography produced similar results for total leaflet (R²=0.97), closure (R²=0.89), and annulus areas (R²=0.84). MV size was the largest in heart failure without FMR compared with controls and patients with FMR (9.1±1.7 versus 7.5±1.0 versus 8.1±0.9 cm²/m²; P<0.01). Patients with FMR had reduced ratios of total leaflet to closure areas and total leaflet to annulus areas when compared with patients without FMR (P<0.01).

Conclusions—MV size measured by CT is comparable with 3D echocardiography. MV enlargement in cardiomyopathy suggests leaflet adaptation. Patients with FMR have inadequate adaptation as reflected by decreased ratios of leaflet area and areas determined by ventricle size (annulus and closure areas). These measurements provide additional insight into the mechanism of FMR. (Circ Cardiovasc Imaging. 2013;6:784-789.)

Key Words: 3-dimensional echocardiography • cardiac computed tomography • mitral valve insufficiency

Mitrval regurgitation (MR) is a common and morbid condition that represents a major health burden. MR can result from mitral leaflet disease (organic MR) or secondary to left ventricle (LV) remodeling (functional MR [FMR]). The latter is the complex result of global or local (typically after inferoposterior infarction) LV remodeling with papillary muscle displacement and mitral annulus dilatation. These LV geometric alterations can be isolated or coexist, with secondary mitral apparatus distortion and incomplete closure of the mitral valve (MV). Understanding the mechanisms underlying MR is of highest importance to choose the appropriate therapy, and echocardiography is currently the central tool used for that purpose. The complex 3-dimensional (3D) shape of the mitral apparatus makes its evaluation challenging by 2-dimensional (2D) planes, generating a potential role for 3D imaging. Recently, echocardiography-based 3D reconstructions of the mitral leaflets provided new insight into the mechanisms of FMR, in particular by demonstrating the capacity of adaptation of the mitral leaflets, which can actively enlarge in response to mechanical stretch, therefore compensating for LV dilatation and preventing MR. This compensatory enlargement can explain, at least in part, why some patients have variable severity of MR despite similar degree of LV remodeling.

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MV reconstruction algorithms allowing total leaflet area measurement have been validated with 3D echocardiography. However, transthoracic ultrasound can be limited in patients with poor echocardiographic windows, and transesophageal echocardiography is invasive. For that reason, whether or not these 3D parameters can be obtained from other noninvasive imaging modality needs to be evaluated. Cardiac computed tomography (CT) is a growing imaging modality, and the recent CT scanners are able to acquire 3D data sets with excellent spatial resolution and enhanced temporal resolution. Our first aim is to validate the 3D measurements obtained by contrast-enhanced cardiac CT with those obtained by 3D echocardiography. To assess the role of these measurements
to explore MV changes in heart failure and their potential contribution to FMR, we subsequently measured CT-derived MV leaflet area in patients with heart failure with and without FMR as well as normal controls.

**Methods**

**Validation Study Between CT and 3D Echo**

In cohort 1, we searched our institution echocardiography and radiology databases for patients who had both a 3D echocardiographic study (transesophageal or transthoracic) and a retrospectively gated cardiac CT between January 2006 and June 2011. Patients were considered if they had both studies within a period of 6 months. Patients with a delay >48 hours between the 2 examinations were enrolled if their clinical condition was stable and if serial standard 2D echocardiograms were available to show stable cavity dimensions and systolic and valve functions at both time points. Patients with history of previous mitral surgery or insufficient image quality on 1 or both imaging modality were excluded. In addition to LV volume and function, 2D and 3D echocardiography images were used to evaluate the presence, mechanism, and severity of MR, which was graded by 1 observer as trace, mild, moderate, or severe by integrating color Doppler jet area and vena contracta width.15,19

**Assessment of MV Adaptation in Heart Failure With CT**

In cohort 2, we identified 3 groups of patients who had a retrospectively gated cardiac CT and a standard 2D echocardiography for MR quantification on the same day: (1) FMR group consisted of patients with heart failure as defined by LV ejection fraction <50% and at least moderate MR, (2) patients without significant MR (mild or less) but with similar LV size and function compared with the FMR group were identified (no-FMR group), and (3) a group consisting of normal patients with no known cardiac abnormality.

**Imaging Protocols**

**Cardiac CT Acquisition**

CT scans were performed with contrast and retrospective-gating with tube current modulation. The scanners used were a 128-slice dual-source scanner (Siemens Definition, 2×64×0.6 mm detectors, gantry rotation time 330 ms, temporal resolution 83 ms), and a 64-slice single source scanner (Siemens Sensation, 1×32×0.6 mm detectors, gantry rotation time 330 ms, temporal resolution 83 ms), and a 64-slice single source scanner (Siemens Sensation, 1×32×0.6 mm detectors, gantry rotation time 330 ms, temporal resolution 83 ms). For nonnodal source CT scanners (70% of the examinations), intravenous beta-blocker was given immediately before the examination as needed to achieve heart rate <65 beats per minute.

**3D Echocardiography Acquisition**

Full volume data sets were acquired during 4 to 7 heart beats with a Philips iE33 scanner. The 3D data sets were obtained from the apical window (transsthoracic echocardiogram) using an X3-1 matrix-array transducer (Philips) or a midesophageal view centered on the MV (transesophageal echocardiogram) using an X7-2t matrix-array transducer (Philips).

**Image Analysis**

Three-dimensional data sets from the CT and 3D echocardiography were imported and processed in dedicated software (Omni4D, MD Handschumacher). Observer 1 performed the reconstructions and measurements on all patients once. For interobserver and intraobserver reproducibility, Observer 2 performed the reconstructions and measurements in 10 randomly selected patients, and Observer 1 repeated this process in 10 patients 1 month later, respectively. Total leaflet area has to be measured in diastole, when there is no tension on the leaflets (up to 15% increase in mitral surface has been shown during systole)20,21 and no coaptation surface. On both data sets, the best diastolic frame was selected (mid to late diastole), and the mitral leaflets were traced in multiple planes and reconstructed to obtain the total leaflet area as described previously21 (Figure 1). The closure area, representing the minimal surface that needs to be covered by the leaflets to prevent MR, was traced in midsystole (Figure 2). The annulus area (projected on its least-square plane) was also measured in midsystole. The ratios of total leaflet area to annulus area and of total leaflet area to closure area were then calculated. A reduction in these ratios (representing a proportionally smaller valve compared with LV and annulus size) has been previously associated with the presence of FMR.25 Other FMR key parameters such as tenting area, tenting volume, and tethering distances (distance between each papillary muscle and contralateral annulus) were measured. End-systolic and end-diastolic LV volumes were measured from the CT data sets by the Simpson method of disks. The study was approved by our institutional review committee, and the subjects gave informed consent.

**Statistical Analysis**

Continuous variables were expressed as mean±SD or median with interquartile range as appropriate. Categorical variables were expressed as percentages. We compared the differences between 2 tests using Student t tests or Wilcoxon rank-sum test for continuous variables and χ2 or Fisher exact test for categorical variables, as appropriate. All volumetric and area measurements were indexed for body surface area. For the validation part of this study, interobserver and intraobserver agreements of CT total leaflet area, closure area, and annulus area were assessed using a single measure, 2-way random effect intraclass correlation coefficient model. The paired t test was used to determine the significance for mean absolute and relative differences. We used linear regression and Bland–Altman analysis to compare CT and 3D echocardiography measurements of total leaflet area, closure area, and annulus area of the MV. For MV characterization with CT in heart failure (cohort 2), the significance of differences among the 3 groups (normal, FMR, and no FMR) was determined using ANOVA test. A 2-sided P value <0.05 was considered to indicate statistical significance for all tests. Statistical analysis was performed with Stata/IC 11.2 (StataCorp LP, TX).

**Figure 1. Total leaflet area measurement.** Anterior and posterior mitral leaflets are traced in multiple planes on computed tomography (CT) data sets (A), allowing for 3-dimensional (3D) reconstruction of the entire leaflet surface (B). The same method was used on 3D echo data sets (C and D). E, Reconstructed mitral valve viewed from the left atrium obtained from the CT data set.
Results

Validation Study Between CT and 3D Echo

A total of 32 patients were initially assessed for validation between CT and 3D echo. Six patients were excluded for poor image quality (all had poor apical echocardiographic window with noninterpretable 3D echo data sets; 1 patient also had insufficient image quality on CT). Therefore, 26 patients with CT and 3D echo (22 transthoracic, 4 transesophageal) were included in this analysis. Table 1 shows the patient demographics. Median time between the 2 examinations was 46 (20–179) days. Seven patients had normal valve and LV size and function, 5 had intrinsic MV disease (2 MV prolapse, 1 mitral cleft, 1 leaflet perforation, and 1 parachute MV), and 14 patients had evidence of cardiomyopathy (5 with FMR, 9 without).

Intraobserver and interobserver reproducibility of CT measurements of total leaflet area, closure area, and annulus area are displayed in Table 2. Correlations of MV total leaflet area, annulus area, and closed leaflet area between CT and 3D echocardiography are illustrated in Figure 3. There was excellent correlation between both modalities for open leaflet area ($R^2=0.97$; $P<0.001$; SEE, 1.18), with an average absolute difference between 3D echocardiography and CT of 0.9±0.8 cm$^2$ (relative difference: 5.6±4.3%). Good correlation was also found for the closure area ($R^2=0.84$; $P<0.01$; SEE, 2.24), average absolute difference of 1.5±1.7 cm$^2$ (average relative difference: 11.2±8.4%), as well as annulus area ($R^2=0.89$; $P<0.01$; SEE, 1.44), average absolute difference of 1.1±1.3 cm$^2$ (average relative difference: 10±9%). There was no difference in agreement with 3D echocardiography for patients scanned with single source (n=8) versus Dual-Source CT (n=18; $P=0.75$ for total leaflet area, 0.84 for closure area, and 0.85 for annulus area).

Measurements of tenting area, tenting volume, and tethering distances also yielded comparable results with both modalities ($R^2=0.85$ for tenting area and tenting volume, $R^2=0.90$ and 0.84 for anterolateral and posteromedial tethering distances, respectively). Similar results to CT were seen whether the 3D echo was transthoracic or transesophageal ($P=0.50$ for leaflet closure, and annulus areas). In this validation cohort, patients with cardiomyopathy had larger valve size than normal patients (8.7 versus 7.0 cm$^2$/m$^2$; $P=0.02$ by CT; similar results with 3D echo). Patients with FMR had decreased ratios of total leaflet area to closure area (1.1±0.1 versus 1.4±0.1; $P=0.002$) and total leaflet area to annulus area (1.4±0.1 versus 1.7±0.1; $P=0.006$).

Assessment of MV Adaptation in Heart Failure With CT

A total of 68 patients who had a retrospectively gated cardiac CT and a standard 2D echocardiography on the same day were studied; 2 patients were excluded for CT artifacts caused by rapid or irregular rhythm. Sixty-six patients are therefore included and divided into 3 groups: 25 patients with heart failure with FMR, 25 patients with heart failure without FMR, and 16 normals. Both FMR and no-FMR groups had significantly increased LV volumes and annulus and closure areas as well as decreased LV ejection fraction compared with controls (Table 3). These 2 groups were similar for age, sex, prevalence of ischemic versus nonischemic cardiomyopathy, LV volumes, and MV, mitral valve.

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### Table 1. Characteristics of Cohort 1

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age</td>
<td>55±18</td>
</tr>
<tr>
<td>Women, n (%)</td>
<td>18 (69)</td>
</tr>
<tr>
<td>Body mass index, kg/m$^2$</td>
<td>24.8±4.4</td>
</tr>
<tr>
<td>Median time between echo and CT, d</td>
<td>46 (20–179)</td>
</tr>
<tr>
<td>Body surface area, m$^2$</td>
<td>1.8±0.2</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>56±15</td>
</tr>
<tr>
<td>Groupe, n (%)</td>
<td>7 (27)</td>
</tr>
<tr>
<td>Normal</td>
<td>5 (19)</td>
</tr>
<tr>
<td>Cardiomyopathy with functional MR</td>
<td>9 (35)</td>
</tr>
<tr>
<td>Cardiomyopathy without functional MR</td>
<td>5 (19)</td>
</tr>
<tr>
<td>Organic MV disease</td>
<td>5 (19)</td>
</tr>
</tbody>
</table>

CT indicates computed tomography; LV, left ventricle; MR, mitral regurgitation; and MV, mitral valve.

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### Table 2. Interobserver and Intraobserver Reproducibility of CT Mitral Valve Metrics

<table>
<thead>
<tr>
<th>Metric</th>
<th>Interobserver</th>
<th>Intraobserver</th>
<th>Average Difference, cm$^2$</th>
<th>Relative Difference, %</th>
<th>ICC</th>
<th>$P$ Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Total leaflet area</td>
<td>0.6</td>
<td>0.6</td>
<td>4.4</td>
<td>0.974</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Closure area</td>
<td>0.9</td>
<td>0.5</td>
<td>7.6</td>
<td>0.922</td>
<td>&lt;0.01</td>
<td></td>
</tr>
<tr>
<td>Annulus area</td>
<td>0.7</td>
<td>0.5</td>
<td>6.3</td>
<td>0.944</td>
<td>&lt;0.01</td>
<td></td>
</tr>
</tbody>
</table>

CT indicates computed tomography; and ICC, intraclass correlation coefficient.

*All $P$ values are nonsignificant ($>0.05$).
and LV function. Total leaflet area index derived from CT was significantly larger in the no-FMR group compared with both normal and patients with FMR (9.1±1.7 versus 7.5±1.0 versus 8.1±0.9 cm²/m² for no-FMR, normal, and FMR groups, respectively; P<0.01). In patients without FMR, MV enlargement was proportional to LV and annular dilatation, maintaining a normal ratio of total leaflet area to closure area (1.4±0.1 versus 1.3±0.1; P=0.14) and total leaflet area to annulus area (1.7±0.1 versus 1.7±0.2; P=1.00). In contrast, both ratios were reduced in the FMR group (total leaflet area to closure area: 1.1±0.1 versus 1.4±0.1; P<0.01; total leaflet area to annulus area: 1.4±0.2 versus 1.7±0.2; P<0.01). These reduced ratios indicate inadequate leaflet enlargement to compensate for LV dilatation in patients with FMR (Figure 4). In this cohort of patients of similar LV size and function, the only variables significantly different between patients with and without FMR were total leaflet area (P=0.03), ratio of leaflet area to closure area (<0.01), and ratio of leaflet area to annulus area (P<0.01). The average radiation dose for all patients was 12.6±4.9 mSv.

**Discussion**

In this study, we show that detailed reconstruction of the MV leaflets is possible by cardiac CT, with excellent intraobserver and interobserver variability and correlation with 3D echocardiography in patients with either normal or abnormal (organic or functional regurgitation) MV. Both interobserver variability and correlation with 3D ultrasound were better for total leaflet area (measured in diastole) than for annulus and closure area (midsystolic measurements). This might be related to the use of current tube modulation algorithm with subsequent increased noise in systole. Finally, we also show that in the setting of cardiomyopathy and dilated LV, total leaflet area increases, suggesting compensatory enlargement of the MV. Patients without FMR have typically larger MV that stays proportional to LV size (as reflected by preserved total leaflet area to closure area ratio). Conversely, patients with FMR have insufficient MV enlargement to match the LV dilatation. These findings are in accordance with what was previously shown with 3D echocardiography and animal studies, suggesting that MV size is not fixed but rather can enlarge and adapt to prevent MR in patients with abnormal LV function.15–17

The classical definition and evaluation of FMR relies only on LV and annulus changes, overlooking the importance of MV size and adaptation, which should be considered as one of the most important variables. FMR genesis should not be linked to LV remodeling alone but rather seen as a mismatch between LV remodeling and MV adaptation. An interesting finding is that some, but not all, patients demonstrate adequate MV enlargement, suggesting that there might be processes to limit this adaptation. The capacity

**Table 3. Clinical and CT Characteristics in Normal and Cardiomyopathy Patients (Cohort 2)**

<table>
<thead>
<tr>
<th></th>
<th>Normal (n=16)</th>
<th>FMR (n=25)</th>
<th>No FMR (n=25)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Clinical parameters</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Age</td>
<td>47±17</td>
<td>66±17*</td>
<td>60±13*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Women, n (%)</td>
<td>10 (63)</td>
<td>8 (32)</td>
<td>7 (28)</td>
<td>0.09</td>
</tr>
<tr>
<td>Body mass index</td>
<td>28±7</td>
<td>27±5</td>
<td>27±4</td>
<td>0.56</td>
</tr>
<tr>
<td>Ischemic cardiomyopathy, n (%)</td>
<td>0</td>
<td>16 (64)*</td>
<td>12 (48)*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>CT LV volume and function</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>LVEDVi, mL/m²</td>
<td>65±13</td>
<td>105±30*</td>
<td>115±37*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LVESVi, mL/m²</td>
<td>25±10</td>
<td>71±26*</td>
<td>75±29*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>LV ejection fraction, %</td>
<td>63±8</td>
<td>29±15*</td>
<td>34±12*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td><strong>CT mitral valve metrics</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Distance PPM-annulus, mm</td>
<td>40±6</td>
<td>45±5*</td>
<td>45±6*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Distance LPM-annulus, mm</td>
<td>37±5</td>
<td>41±3*</td>
<td>43±4*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Total leaflet area, cm²</td>
<td>14.0±2.8</td>
<td>15.7±2.2*</td>
<td>18.3±4.3†</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Total leaflet area index, cm²/m²</td>
<td>7.5±1.0</td>
<td>8.1±0.9</td>
<td>9.1±1.7†</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Closure area, cm²</td>
<td>9.9±2.0</td>
<td>13.9±1.9*</td>
<td>13.6±3.2*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Closure area index, cm²/m²</td>
<td>5.3±0.7</td>
<td>7.2±0.7*</td>
<td>6.7±1.4*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Annulus area, cm²</td>
<td>8.4±1.7</td>
<td>11.3±1.5*</td>
<td>11.1±2.6*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Annulus area index, cm²/m²</td>
<td>4.5±0.6</td>
<td>5.9±0.6*</td>
<td>5.5±1.1*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Tenting area, cm²</td>
<td>1.4±0.6</td>
<td>2.9±1.0*</td>
<td>2.7±1.2*</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Tenting volume, cm³</td>
<td>1.5±0.6</td>
<td>3.7±1.5*</td>
<td>3.2±2.0*</td>
<td>&lt;0.01</td>
</tr>
</tbody>
</table>

CT indicates computed tomography; FMR, functional mitral regurgitation; LPM, lateral papillary muscle; LV, left ventricle; LVEDVi, left ventricle end-diastolic volume index; LVESVi, left ventricle end-systolic volume index; and PPM, posterior papillary muscle.

*P<0.05 vs normal group.
†P<0.05 vs FMR group.
to measure changes in total leaflet area in targeted populations will be important to explore the underlying mechanisms and limiting factors of MV compensatory enlargement. The systolic closure and annulus areas are also helpful: these parameters can be influenced by either annular or ventricular changes, and therefore represent the sum of LV alterations affecting MV closure. The adequacy of adaptation can be evaluated by comparing the total leaflet area with closure area ratio, lower ratios representing insufficient valve compensation and more FMR.

Cardiac CT is a promising tool for evaluation of MV heart disease, especially in patients with poor echocardiographic windows. It can adequately evaluate the anatomic structure of the valve, presence of calcium, or leaflet thickening, and identify patients with organic valve disease. Valve deformation and tethering in FMR have also been evaluated. Moreover, it can accurately evaluate the repercussion of MR, such as cavity dilatation, including left atrial volume. Cardiac CT has shown encouraging early results to assess MR severity, although its performance in this area is still currently limited: comparison between right and left stroke volumes to determine regurgitant volume and fraction can be useful, but rely on the assumptions that there is no shunt and other valves are competent. Direct planimetry of regurgitant orifice area has been described with variable results compared with other quantitative parameters, but this method is inherently limited by the dynamic nature of MR throughout systole and might not be practicable for patients with complex and multiple regurgitant orifices. The use of 3D parameters such as closure, annulus, and total leaflet areas can add a comprehensive evaluation of the MV in the setting of cardiomyopathy, taking into account the impact of LV geometry changes on the valve as well as the leaflet adaptive response to these changes. Although echocardiography remains the modality of choice to assess MV, we show here that cardiac CT can provide reliable and advanced information on valve size and geometry; therefore, this modality can be considered in patients with limited echocardiographic windows. Cardiac CT can also add useful information on MR mechanism and valve geometry in patients who have CT for coronary artery evaluation.

**Conclusions**

Cardiac CT can be used to provide comprehensive and detailed reconstructions of the MV. These measurements are helpful to understand why some patients have different severity of FMR despite similar LV dilatation. Further clinical and experimental studies looking at mechanisms leading to or limiting MV compensatory enlargement are needed.

**Limitations**

Despite the majority (88%) of the retrospectively gated CT studies were performed with ECG-tube modulation, we found excellent reproducibility and correlation between CT and 3D echo. In the validation study, some patients had delays between 3D echo and CT that could affect the results. However, all patients with heart failure had their CT and standard 2D echo for MR quantification the same day. Cardiac CT represents a potential risk from ionizing radiation; however, the radiation doses are expected to be consistently lower in the near future as the technology evolves. Advantages of cardiac CT compared with either 2D or 3D echo are less operator dependent, no issues with adequate acquisition windows, and better spatial resolution. Because patients with and without FMR were selected on the basis of similar LV size and function, the present study cannot assess the relative contributions of other key parameters of FMR such as annulus size, contraction, or tethering distances. This selection can also explain the lack of significant difference in tenting volume and area between patients with and without FMR. However, our study design allows us to explore the key question of why patients with similar LV remodeling have variable MR severity. Although the use of 3D-derived areas, such as total leaflet area and mitral closure area, is helpful in understanding the mechanisms of MV disease and in finding new biological phenomenon such as MV adaptation, the role of 3D-derived areas in routine clinical practice needs to be defined.

**Disclosures**

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**References**


CLINICAL PERSPECTIVE

Functional mitral regurgitation is a common and morbid complication of ischemic and nonischemic cardiomyopathies. Its mechanisms have been attributed to left ventricle and mitral annulus remodeling, whereas the mitral leaflets have been typically considered normal and only passively involved. However, recent data suggest that leaflet tissue is able to actively enlarge in response to mechanical stretch. This suggests that functional mitral regurgitation should not be viewed only as a disease of the left ventricle, but rather also as a mismatch between the remodeling ventricle and compensatory leaflet enlargement. Because valve size is dynamic and has the potential to become a therapeutic target, it is important to develop tools to assess this variable in addition to left ventricular parameters when studying this disease. In this article, we show that mitral leaflet size, geometry, and their relation to left ventricular and annular parameters can also be assessed reliably with cardiac computed tomography. We also use cardiac computed tomography to demonstrate that despite similar left ventricle size and function, some patients have variable leaflet compensation, leading to different degree of mitral regurgitation. Three-dimensional imaging will play a key role in future studies, trying to find the mechanisms and factors underlying valve adaptation.
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