FOCUS ISSUE ON SECONDARY (FUNCTIONAL) MITRAL REGURGITATION

STATE-OF-THE-ART REVIEW

Basic Principles of the Echocardiographic Evaluation of Mitral Regurgitation

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ABSTRACT

Mitral regurgitation (MR) is a common form of valvular heart disease that is associated with significant morbidity and mortality. Treatment decisions are completely dependent on accurate diagnosis of both mechanism and severity of MR, which can be challenging and is often done incorrectly. Transthoracic echocardiography is the most commonly used imaging test for MR; transesophageal echocardiography is often needed to better define morphology and MR severity, and is essential for guiding transcatheter therapies for MR. Multidetector computed tomography has become the standard to assess whether transcatheter valve replacement is an option because of its ability to assess valve sizing, access, and potential left ventricular outflow tract obstruction. Finally, cine cardiac magnetic resonance has been recommended by recent guidelines to quantify MR severity when the distinction between moderate and severe MR is indeterminate by echocardiography. This paper focuses on the main questions to be answered by imaging techniques and illustrates some common tips, tricks, and pitfalls in the assessment of MR. (J Am Coll Cardiol Img 2021;14:843-53) © 2021 by the American College of Cardiology Foundation.

itral regurgitation (MR) is a common form of valvular heart disease that is associated with significant morbidity and mortality. Several guidelines and consensus documents have been published addressing all aspects of MR, including evaluation and treatment (1-4). Treatment decisions are completely dependent on accurate diagnosis of both mechanism and severity of MR, which can be challenging and is often done incorrectly (5). Transthoracic echocardiography (TTE) is the most commonly used imaging test for MR; transesophageal echocardiography (TEE) is often needed to better define morphology and MR severity, and is essential for guiding transcatheter therapies for MR. Multidetector computed tomography has become the standard to assess whether transcatheter valve replacement is an option because of its ability to assess valve sizing, access, and potential left ventricular (LV) outflow tract (LVOT) obstruction. Finally, cine magnetic resonance (CMR) imaging has been recommended by recent guidelines to quantify MR severity when echocardiographic assessment is uncertain or when echocardiographic and clinical findings are discordant. This paper is not intended as a comprehensive review of imaging for MR, but rather as a supplement to existing guidelines. We focus briefly on the main questions to be answered by imaging techniques (Central Illustration, Table 1) and illustrate some common tips, tricks, and pitfalls in the assessment of MR.

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ABBREVIATIONS AND ACRONYMS

3D = 3-dimensional

- AF = atrial fibrillation
- CMR = cine magnetic resonance

CW = continuous wave

- **EROA** = effective regurgitant orifice area
- LA = left atrial/atrium
- LV = left ventricle/ventricular

LVEF = left ventricular ejection fraction

LVOT = left ventricular outflow tract

MR = mitral regurgitation

PISA = proximal isovelocity

surface area

RF = regurgitant fraction

RV = right ventricle/ventricular RVol = regurgitant volume

TEE = transesophageal echocardiography

TTE = transthoracic echocardiography

WHAT IS THE MECHANISM OF MR?

Mild MR by color Doppler is common in normal, healthy individuals with a structurally normal mitral valve apparatus. This is partly due to the sensitivity of color Doppler to detect low-velocity signals from flow that cannot be seen by conventional methods such as angiography. When more-than-mild MR is suspected on the basis of color Doppler, and/or the mitral apparatus is structurally abnormal, it is important to accurately determine the mechanism of MR. involves determination This of the morphology of the mitral apparatus (leaflets, annulus, subvalvular apparatus, and supporting myocardium) as well as their motion (Carpentier classification [1-4]). When both morphology and motion are combined, it is usuallv straightforward to determine whether MR is primary, secondary, or mixed. Primary MR refers to structural abnormalities of the leaflets themselves or the subvalvular apparatus (i.e., ruptured chordae). Secondary MR is most commonly caused by LV dysfunction, which in turn causes reduced closing force on the leaflets combined with systolic tethering of the leaflets into the LV, either by global LV dilation and dysfunction or by focal wall motion abnormalities with preserved global LV function (i.e., inferobasal akinesis or dyskinesis). LV dyssynchrony due to bundle branch block or right ventricular (RV) pacing can also cause secondary MR. Another cause of secondary MR is pure mitral annular dilation due to left atrial dilation in chronic atrial fibrillation (AF) or restrictive cardiomyopathy. This is known as atrial functional MR (6). Finally, it is possible to have multiple mechanisms of MR with some features of primary and some features of secondary MR (mixed MR), especially in elderly patients with fibrocalcific changes in the valve. However, there is usually a dominant mechanism that becomes the target for treatment. A subtype of myxomatous mitral valve disease that is increasingly recognized is mitral annular disjunction, where the mitral annulus separates from the basal myocardium around part of the annulus and has been associated with serious arrhythmias and LV fibrosis by CMR (7).



TABLE 1 Main Questions to Be Answered by Cardiovascular Imaging		
	Parameters	Modality
What is the mechanism of MR?	Leaflet morphology Leaflet motion (Carpentier type) Subvalvular involvement Annulus (dilation, calcium) LV size and function LA size and function	TTE TEE MDCT CMR
Is the MR severe?	Quantitative (EROA, RV, RF) Qualitative (multiple)	TTE TEE CMR
What is the effect of MR on LV and LA size and LA/pulmonary venous pressure?	LV diameters, volumes, LVEF, GLS, fibrosis LA volume, pressure Estimated PA systolic pressure	TTE TEE CMR MDCT Catheterization
Are there anatomic features that support or preclude a given surgical or transcatheter approach?	Annulus size Leaflet length/thickening Subvalvular pathology Mitral annular calcium Potential for LVOT obstruction Others (device-specific)	TTE TEE MDCT CMR
CMR = cine magnetic resonance; EROA = effective regurgitant orifice area; GLS = global longitudinal strain; LA = left atrium; LV = left ventricle; LVEF = left ventricular ejection fraction; LVOT = left ventricular outflow tract; MDCT = multidetector computed tomography; PA = pulmonary artery; RF = regurgitant fraction; RV = regurgitant volume; TTE = transthoracic echocardiography; TEE = transesophageal echocardiography.		

IS THE MR SEVERE?

Ideally, MR would be measured by quantitative parameters along a continuous scale. Such parameters would include effective regurgitant orifice area (EROA), regurgitant volume (RVol), and regurgitant fraction (RF), which is the percentage of MR volume relative to total LV stroke volume. These values interact in complex ways. For example, a given value of EROA could have a larger or smaller RVol depending on the driving velocity of flow across the valve and the duration of MR, which is often not holosystolic. A given RVol could have a different RF depending on the size and systolic function of the LV. This has recently been highlighted in secondary MR where a given value of EROA or RVol can be disproportionately severe or even not severe depending on the LV end-diastolic volume and LV ejection fraction (LVEF) (8,9). Unfortunately, the precision and reproducibility of these quantitative parameters are limiting factors. Accordingly, all guidelines recommend using them together and with other qualitative parameters to define MR severity as mild, moderate, or severe. This helps minimize the well-known measurement errors for each parameter and helps ensure that the totality of the data is internally consistent. When TTE data are internally inconsistent or conflict with the clinical presentation, it is recommended to perform either TEE or CMR. TEE is usually very good at defining the mechanism of MR. It can often result in better assessment of severity but can also underestimate MR severity due to the effects of sedation or anesthesia. CMR is generally thought to provide more reproducible quantitative measurements of RVol and RF, as well as LV volumes and LVEF, with obvious



Parasternal long-axis view showing the anterior leaflet (AML) **(blue arrow)** overriding the posterior leaflet (PML) **(yellow arrow)** at end-systole. This is often misinterpreted as mitral valve prolapse. However, the AML never moves superiorly to the mitral annulus **(dashed blue line)** into the left atrium (LA). Instead, both leaflets are tethered into the left ventricle (LV) throughout systole, as can be seen clearly in Video 1. The **blue arrow** points to mid-AML tenting by a secondary chord, known as the "seagull sign." MR = mitral regurgitation.



limitations in irregular rhythms such as AF or frequent ectopy.

WHAT ARE THE CONSEQUENCES OF MR ON THE LV, LEFT ATRIUM, PULMONARY CIRCULATION?

Primary MR imposes a pure volume overload on the left heart. As a result, it would be expected that chronic severe MR should result in dilation of the LV and left atrium (LA), and increased LA and pulmonary venous pressure. In acute MR, the LV and LA might not be dilated, but LA pressure and pulmonary pressures should be significantly elevated. If all of these are normal, severe MR is very unlikely. Primary MR can be dynamic, but a severe morphological leaflet abnormality (i.e., flail leaflet) suggests severe MR. Secondary MR is more difficult to evaluate because leaflet morphology is normal and dilation of the LV and LA, or elevation of LA and pulmonary venous pressures, could be due to LV systolic and/or diastolic dysfunction and/or AF. Determination of severity of secondary MR is best done after optimal treatment of underlying LV dysfunction with neurohormonal antagonists, coronary revascularization, and cardiac resynchronization therapy when appropriate, and

restoration of sinus rhythm. In certain circumstances, a stiff LA (post-ablation, infiltrative cardiomyopathy) could have high pressures in the setting of moderate or even mild MR. For example, elevated LA V waves have been reported in the absence of MR in patients with heart failure and preserved LVEF.

IF AN INTERVENTION IS WARRANTED, WHETHER SURGICAL OR TRANSCATHETER, ARE THERE ANATOMIC FEATURES THAT SUPPORT OR PRECLUDE ANY GIVEN APPROACH?

A detailed description of this evolving field is beyond the scope of this paper. However, once the mechanism of MR is defined accurately, and MR is determined to be severe, detailed evaluation of 3-dimensional (3D) TEE and multidetector computed tomography is often needed to assess whether a given device/procedure/approach is anatomically suitable/ feasible/safe in a given patient. For surgical valve repair or replacement, this may involve determining whether a minimally invasive approach and cannulation are feasible and whether the tricuspid valve needs to also be addressed. For transcatheter



approaches, the imaging requirements are devicespecific and evolving. Moreover, these procedures are guided by intraprocedural TEE, so it is important that the pre-procedural TEE demonstrates that image quality is sufficient to perform the procedure. For transcatheter edge-to-edge leaflet repair, 3D planimetry of the mitral valve area is required to be sure that there is room to place the device without causing mitral stenosis (initial mitral valve area \geq 4.0 cm²). Unfortunately, this is rarely done in clinical practice, and patients often have to undergo repeat TEE.

WHAT IS THE EVIDENCE UNDERLYING THE PRECEDING QUESTIONS?

Guidelines for treatment of primary and secondary MR have been published (1-3). Briefly, the following pertain to native valve mitral regurgitation:

- Surgery is indicated for severe primary degenerative MR (flail leaflet or prolapse). It can be done by a minimally invasive approach; replacement should generally be avoided.
- Transcatheter edge-to-edge repair with the Mitra-Clip device (Abbott Vascular, Santa Clara, California) is indicated for symptomatic patients with severe (3+ or 4+) primary degenerative MR who are at prohibitive risk for surgical repair, and for patients with severe (3+ or 4+) secondary MR that persists after optimization of medical therapy (including revascularization or cardiac resynchronization) and have LVEF 20% to 50% with an LV end-systolic diameter <70 mm.
 - There is no evidence that surgery for secondary MR improves mortality, although a randomized trial against optimal medical therapy has not been done.



• Mitral valve replacement is usually needed when the leaflets are restricted in both systole and diastole (Carpentier IIIA leaflet motion) (e.g., rheumatic or radiation heart disease). MitraClip is usually contraindicated in such patients because it would cause mitral stenosis. Transcatheter mitral valve replacement devices are under investigation but are often limited by concerns of LVOT obstruction.

• Atrial functional MR is ideally treated by annuloplasty, but no good data exist for surgical or percutaneous intervention. This problem is increasingly recognized, leading to the consideration that





controlling AF earlier in the course of disease could potentially prevent the development of severe MR (or tricuspid regurgitation).

TIPS, TRICKS, AND PITFALLS IN ASSESSING MR

1. Turn off the color Doppler and visual the entire mitral apparatus in multiple views at high frame rates (>50 Hz), as recommended by imaging guidelines (3,4). It is difficult to properly determine the mechanism of MR when standard views

are missing or the underlying anatomy is not clearly seen because of the color Doppler overlay.

2. Anterior leaflet override in secondary MR (pseudoprolapse). **Figure 1** shows a parasternal long-axis view from a patient with nonischemic dilated cardiomyopathy. The posterior leaflet is severely restricted/tethered, and the anterior leaflet overrides it with an obvious gap. This is not prolapse because the anterior leaflet never moves superior to the annulus. It is tented into the LV, and the posterior leaflet points to the apex. This is a common finding in secondary MR and is often



Pulsed Doppler recordings of pulmonary vein velocities (top) and mitral inflow patterns (bottom) are helpful in mitral regurgitation (MR). Normal pulmonary vein pattern and A-wave dominant mitral inflow are not typical of severe MR (left). Systolic flow reversal in more than 1 pulmonary vein and an "E" velocity \geq 1.2 m/s are typical of severe MR.

misinterpreted as mitral valve prolapse or mixed etiology MR. This is pure secondary MR.

3. Nonholosystolic MR is common and often leads to overestimating MR severity when using singleframe measurements such as EROA by proximal isovelocity surface area (PISA), vena contracta width, or vena contracta area (10). Even when holosystolic, EROA is often highly variable, so peak EROA (single frame showing the largest proximal flow convergence zone) may overestimate MR. **Figure 2** (top panel) shows late systolic MR in an asymptomatic patient with mitral valve prolapse, normal LA and LV volumes, and normal estimated PA systolic pressure. Despite a calculated EROA of 0.3 cm², this is mild MR. Note that there is no MR at all in early or mid-systole. **Figure 2** (bottom panel) shows biphasic MR, a common finding in secondary MR due to LV dysfunction. Note the large jet in early systole, near disappearance of MR in mid-systole (when LV closing force is maximal), and a smaller jet in late systole. Continuous-wave (CW) Doppler gain should not be turned up to eliminate the biphasic signal (a common mistake by sonographers). Biphasic or early systolic MR is common in secondary MR and often leads to overestimation of MR severity. When the MR is "all or nothing" (as in the upper panel in **Figure 2**), one can use the velocity time interval of the dense CW Doppler signal to convert EROA to RV with some

Volumetric methods can be used to calculate MR RVol as shown in this figure. The **left panels** show LV end-diastolic **(top)** and end-systolic **(bottom)** volumes by biplane method of disks with a total LV stroke volume of 78 ml. The **top right** is forward stroke volume of 71 ml by the LVOT pulsed Doppler method (assuming no significant aortic regurgitation). Note that there is some spectral broadening in the signal that can overestimate forward stroke volume. By this method, MR Rvol is 7 ml, significantly lower than the 46 ml derived by the PISA method **(bottom right)**. Volumetric methods can be used but often underestimate MR severity because 2-dimensional (2D) echocardiography tends to underestimate LV volumes. CMR uses a similar method but is less likely to underestimate LV volumes (see text). 3D echo can also provide larger, more accurate LV volumes if image quality is good. LVOT = left ventricular outflow tract; RV = regurgitant volume; other abbreviations as in **Figures 2, 3, and 6**.

confidence, but when the EROA is continuously changing (as in the bottom panel in Figure 2), this approach is less supported.

4. Pay attention to the CW Doppler velocity of the MR jet. The most important determinant of jet size (assuming consistent and appropriate machine settings) is jet momentum flux (11), which is flow • v or EROA • v^2 , where v = velocity. Typical MR peak velocities are roughly 5 m/s, but can exceed 6 m/s when LV pressure is high (aortic stenosis, LVOT obstruction, severe hypertension) and lead to large color Doppler jets when MR is not severe. **Figure 3** shows an example of similar-appearing MR jets by color Doppler with different peak velocities and MR severity. The peak

velocity is divided into the PISA-derived peak regurgitant flow rate to calculate EROA. Thus, for a similar (or in this case, a smaller) flow rate, a high peak velocity indicates smaller EROA. The peak velocity can also be used to estimate LA pressure, as shown in **Figure 3**. A dense holosystolic MR jet with a triangular pattern suggests significant MR. Hypodense, biphasic, or nonholosystolic MR jets are usually not severe MR.

5. Consider the simplified PISA formula (Figure 4). Unless MR is massive, PISA works best when the color Doppler baseline is shifted in the same direction as the MR jet to a value that best identifies a hemisphere. The hemisphere radius is then used to calculate EROA as $2\pi r^2 \cdot aliasing velocity \div$ FIGURE 9 Modified Apical 2-Chamber View Showing 2 Separate MR Jets at the Center of Valve

Modified apical 2-chamber view showing 2 separate MR jets at the center of valve (between anterior and posterior middle scallops). The 2 jets have separate PISAs (arrows) in the LV, but merge into a single jet in the LA. This view (and the similar TEE bicommissural view) help identify the number and location of MR jets. TEE = transesophageal echocardiography; other abbreviations as in Figures 1 and 2.

peak velocity of the MR jet. Although this calculation is easily done in contemporary reporting packages, a simplified approach may allow "onthe-fly" estimation, particularly during interventional guidance. Assuming a typical LV-to-LA systolic pressure gradient of 100 mm Hg (corresponding to a peak velocity of 5 m/s), a baseline shift to 40 cm/s simplifies the formula $EROA = r^2/2$ (12). Thus, a radius of 8 mm would be a EROA 0.32 cm², which is moderately severe (3+) MR, whereas 9 mm pushes it to 0.40 cm², on the threshold of severe (4+). That small measurement differences lead to classification changes emphasizes the need for care in measurement, using the zoom function to maximize the number of pixels in the radius, and carefully assessing the underlying valve structure (toggling color on and off) to ensure the proper valve orifice level is identified. Using this simplified form of PISA means that an aliasing radius $\geq 8 \text{ mm would}$ usually be severe MR, unless the jet velocity was very high (as noted in the preceding text) or MR was nonholosystolic.

6. Beware small errors in PISA radius measurement. Figure 5 shows the difference in calculated EROA and RV when PISA radius is off by 1 mm. In this example, EROA is roughly 30% higher at a radius of 8 versus 7 mm. Thus, small errors in radius measurement can result in misclassification of MR severity. PISA can also be influenced contour flattening near the orifice or a nonplanar angle, which requires angle correction. Although PISA EROA and RV are good starting points for grading MR, they are prone to errors and thus should be corroborated by other findings.

- 7. Beware noncircular orifice or multiple orifices. Figure 6 shows an example of a markedly elliptical orifice in secondary MR. The calculated PISA EROA was 0.24 cm², suggesting moderate MR. However, the PISA assumption of a relatively round orifice is not met. The vena contracta area by 3D reconstruction is 0.57 cm², consistent with severe MR. When separate orifices exist, PISA EROAs or vena contracta areas can theoretically be added, but validation studies are lacking. Also, the PISAs may appear separate or merged depending on the alias velocity or direction of baseline shift. 3D vena contracta areas may overestimate MR severity due to the color Doppler blooming effect, inclusion of low-velocity eddies in the measurement, and nonplanarity of the orifice.
- 8. MR is dynamic (especially secondary MR). Video 1 shows parasternal long-axis and apical 4-chamber views from a patient presenting with severe heart failure symptoms and a known ischemic cardiomyopathy. He was also in AF with a rapid ventricular rate. Videos at left show severe MR. He was admitted to the hospital for intravenous diuretic agents and inotropes, and cardioversion. Three days later, after a 10-kg diuresis, his MR was mild and has remained so on proper doses of neurohormonal therapy. Both echocardiograms were performed with the same instrument and settings; the first was in atrial fibrillation, the second in sinus rhythm.
- 9. Adjunctive echocardiographic findings that support or contradict severe MR. Figure 7 shows examples of mitral inflow patterns and pulmonary vein flow patterns in MR. Normal estimated RV systolic pressure (not shown) would be uncommon in severe MR. These findings are very useful in avoiding the common problem of overestimation of MR.
- 10. Volumetric calculations are difficult. Calculation of MR RV as the difference between biplane Simpson or 3D LV total stroke volume and pulsed Doppler-derived forward stroke volume from the LVOT can be done. Similarly, total stroke

volume could be calculated by pulsed Doppler technique at the mitral annulus. Figure 8 shows an example of a volumetric calculation that underestimates MR severity (same patient as Figure 6), probably due to the known tendency of 2D TTE to underestimate LV volumes. In addition, the total error of the method is the root sum square of the errors of each individual measurement, which magnifies the absolute error, and the fact that 1 large quantity (LVOT stroke volume) is subtracted from another (LV stroke volume), which greatly increases the relative error. Therefore, volumetric calculation by echocardiography should be used with caution; CMR offers more reproducible volumetric calculation of MR severity.

11. The proximal convergence of the MR jet, which is used for PISA calculations, is also excellent for localizing the origin and direction of jets, critical to ascertaining both the mechanism and reparability of the pathology. In particular, the parasternal short-axis (mitral level) and apical 2-chamber views (**Figure 9**) provide guidance as to where along the commissural closure line the jet arises. Similar localization can be done by TEE, using the intercommissural view (around 60°) to guide a cross-plane to intersect each of the

HIGHLIGHTS

- What are the main questions to be answered by cardiac imaging?
- What is the underlying evidence base?
- What are the major tips, tricks, and pitfalls in assessing mitral regurgitation?

posterior leaflet scallops in turn without and with color. Localization is based on where a proximal convergence zone is seen, not on where the jet is seen in the LA, because it may have come from out of plane.

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APPENDIX For a supplemental video, please see the online version of this paper.

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