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Intraoperative Evaluation of Mitral Stenosis by Transesophageal Echocardiography

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A 58-year-old woman with chronic renal failure, atrial fibrillation (AF), and mitral stenosis (MS) undergoing urgent bowel resection. Intraoperative transesophageal echocardiography (TEE) is requested to determine the significance of her MS and to guide hemodynamic management. What measurements or evidence would facilitate grading? What other consequences of MS should be evaluated by TEE?

PATHOPHYSIOLOGY

MS is defined as reduction in the mitral valve (MV) orifice area because of pathologic leaflet changes. Congenital MS is rare, resulting from abnormalities of the MV or subvalvular apparatus. In adults, MS is caused by either rheumatic or degenerative disease. Rheumatic MS is caused by autoimmune cross-reactivity between group A B-hemolytic streptococcus antigens and endothelial tissue, resulting in inflammatory scarring of MV tissue. Degenerative MS causes annular calcification extending into the leaflets. Its incidence increases with age and in patients with renal failure, disorders of calcium metabolism, or after radiation.¹ The distinct mechanisms of rheumatic and degenerative MS influence the emphasis of the TEE examination with respect to the structural differences, associated lesions, and reliability of echocardiographic modalities for severity grading.

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QUALITATIVE ASSESSMENT (2D AND 3D)

The MV apparatus can be evaluated at the midesophageal level at different omniplane angles (4-chamber view at 0–10°, mitral commissural view at 50–70°, 2-chamber view at 80–100°, and long-axis view at 120–140°). The transgastric basal short-axis view shows commissural pathology, whereas the 2-chamber view displays the subvalvular apparatus. Three-dimensional (3D) imaging provides en face views of the MV from the left atrial (LA) or left ventricular (LV) perspective, demonstrating the relationship of the MV apparatus with adjacent structures.

Rheumatic MS manifests as commissural fusion, chordal shortening, leaflet thickening, and reduced leaflet mobility. The posterior leaflet is more often affected than the anterior.² The disease involves predominantly the leaflet tips with relative sparing of the mitral annulus and leaflet bases. These result in the characteristic “hockey-stick” leaflet appearance (from the LA, a concave appearance of the leaflets in diastole) and minimal leaflet tip motion between opening and closing. The smallest MV area is at the leaflet tips (Fig. 1; Supplemental Digital Content 1, Video, <http://links.lww.com/AA/B406>). In degenerative MS, calcification of the MV annulus extends progressively into the leaflet tips (also posterior > anterior), resulting in reduced diastolic excursion with the smallest orifice area at the base of the MV leaflets (Fig. 2; Supplemental Digital Content 1, Video, <http://links.lww.com/AA/B406>).¹ The subvalvular apparatus also may be calcified.

Several qualitative 2D scoring systems describe the MV morphology. The Wilkins score characterizes leaflet mobility, valve thickness, subvalvular thickening, and calcification. It was devised to determine suitability for balloon valvuloplasty and postprocedural mitral regurgitation (MR) risk³ but also communicates the varied locations and manifestations of MV pathology (Table 1). The Carpentier functional classification also describes mixed MV disease (stenosis and regurgitation) as type IIIa with leaflet restriction throughout the cardiac cycle. MS graded as severe by echocardiography is now further classified as symptomatic (stage D) or asymptomatic (stage C), to guide disease management.⁴

QUANTITATIVE ASSESSMENT

MS severity is determined using estimation of transmitral pressure gradient estimation by Doppler and measurements or calculations of mitral valve area (MVA) (Table 2). The normal MVA is 4 to 5 cm². MVA of >1.5, 1.5 to 1, and <1 cm² correspond to mild, moderate, and severe stenosis, respectively.

MVA by Planimetry

Planimetry by 2D imaging measures the MVA by tracing the MV orifice outline in mid-diastole in the transgastric basal short-axis view (Fig. 3A). It is a direct, anatomic measurement; therefore, it is less dependent on flow conditions⁵ and chamber compliance. Tachycardia may cause underestimation of MVA by planimetry because of incomplete opening during short cardiac cycles. Small adjustments in probe position and probe flexion or simultaneous orthogonal imaging may be needed to find the smallest orifice of the MV. In degenerative MS, calcific acoustic shadowing artifact may obscure the leaflets, making

planimetry unreliable. In addition, the narrowest orifice may not be at the level of the leaflet tips.

Three-dimensional imaging may overcome these limitations by allowing simultaneous display of 3 orthogonal views and improve identification of the narrowest orifice (Fig. 3B).⁶ In severely distorted valves, even the flexibility of 3D imaging may not allow accurate planimetry. In 3D imaging, more so than in 2D, echodense objects can appear falsely large or thickened (blooming artifact), particularly with high gain settings. This would result in planimetric orifice area underestimation. Acquisition at low frame rate may prevent capture of the true mid-diastolic frame. Adding color flow Doppler (CFD) to 3D imaging may help locate the narrowest orifice area and allow functional orifice area measurement (Fig. 3C). The functional orifice area and the anatomic orifice area are not technically interchangeable. In stenotic valves, the functional orifice, which is measured by using CFD, occurs slightly distal to the anatomic orifice at the point where blood flow contracts and achieves maximal velocity. Therefore, the functional area is slightly smaller than the anatomic area. Although planimetry measures the anatomic MVA, planimetry by 3D with CFD and MVA calculations by pressure half-time (PHT), proximal isovelocity surface area (PISA) method, and continuity equation assess functional MVA. All methods correlate well with surgical findings.⁷ Three-dimensional with CFD also is limited by lower temporal resolution, CFD gain settings, and color artifacts.

Pressure Gradient

Transmitral pressure gradients are measured using continuous wave Doppler (CWD) of diastolic transmitral flow in any midesophageal view that allows optimal alignment of the Doppler beam and flow (within 15° of parallel). Visualization of flow direction using CFD may help ensure adequate alignment, especially in severe MV apparatus deformation where flow may be eccentric. Mean pressure gradients are used because peak velocities (and so also peak gradients) are more variable, being influenced by LA compliance and LV diastolic relaxation (Fig. 4A). Mean gradients of <5 mm Hg, 5 to 10 mm Hg, and >10 mm Hg correspond to mild, moderate, and severe stenosis, respectively. These cutoffs are based on heart rates of 60 to 80 bpm in sinus rhythm.⁸

Caution must be exercised in AF, tachycardia, or with high or low transmitral flow. AF results in beat-to-beat variation in flow. Therefore, mean pressure gradients should be averaged over 5 beats. Decreased diastolic filling time in tachycardia results in elevation of the LA pressure and the transmitral mean gradient. High transmitral flow because of hyperdynamic states (e.g., sepsis, hepatic failure) or severe MR will increase the mean gradient⁹ and cause overestimation of MS severity. Conversely, low cardiac output causes underestimation of MS severity. The mean pressure gradient also is sensitive to alterations in atrioventricular compliance, as seen in patients with diastolic dysfunction. Coexistent diastolic dysfunction, likely in older patients or those with ischemic heart disease, hypertension, LV hypertrophy, or aortic stenosis (AS) (factors also associated with degenerative MS), decreases the early diastolic transmitral gradient and underestimates MS severity.¹⁰

MVA Calculation

Three echocardiographic methods are used to estimate MVA: PHT, continuity method, and PISA. MVA can be derived invasively by the Gorlin formula, using the catheterization-derived cardiac output and transvalvular mean pressure gradient. Although all echocardiographic methods have shown good correlation with invasive MVA calculations and direct surgical measurements,⁷ each has inherent limitations in specific clinical situations.

MVA by PHT—The PHT represents the time elapsed as the peak diastolic transmitral pressure gradient decays to half its peak value. On the velocity scale, PHT is the time necessary for the peak transmitral velocity ($V_{\max}$) to decrease to $V_{\max}/2$. PHT measurement is performed by tracing the transmitral flow slope from peak velocity to at least the point on the Doppler spectrum where velocity is two thirds of the peak velocity (Fig. 4B). Therefore, PHT measurement is inaccurate in situations of abbreviated diastolic filling such as sinus tachycardia or short cardiac cycles in AF. Also, fusion of the transmitral E and A waves in tachycardia prevents accurate measurement. If the mitral inflow Doppler tracing is bimodal and nonlinear, the deceleration slope should not be traced from the early part (representing initial, early diastolic decline in pressure gradient) but rather using extrapolation of the linear, midportion of the profile (Fig. 4C).¹¹

MS results in slower decay of the pressure gradient between the LA and the LV and a prolonged PHT. PHT can be used in a simple, empirically derived formula to estimate the MVA: $MVA = 220/PHT$ (milliseconds) (Table 2).¹² The constant 220 is directly proportional to the product of the net compliance of the LA and LV and with the square root of the initial transmitral pressure gradient. Net chamber compliance and pressure gradient change in opposite directions, thus offsetting their effect on PHT. However, this assumption may not be valid with acute changes in chamber compliance.¹³

PHT is relatively unaffected by MR¹² and rate-controlled AF.¹⁴ With AF, measurements should be made and averaged across 5 cardiac cycles, and tracing of mitral flow from short diastoles should be avoided.⁸ PHT measurements can be unreliable with tachycardia. With increasing heart rate, the PHT method overestimates MVA.⁹ Significant aortic regurgitation (AR) contributes to diastolic LV filling and causes significant shortening of PHT with MVA overestimation.¹⁵ This effect is highly dependent on chamber compliance and on the severity of MS, which may explain conflicting results in other studies regarding the impact of AR on PHT.¹³ Coexistent diastolic dysfunction, especially in patients with degenerative MS and concomitant AS, results in a shortened PHT and MVA overestimation⁸; PHT should not be used in these patients.

PHT is related to deceleration time (DT); $PHT = 0.29 \times DT$, where DT is the time taken for the peak early diastolic velocity to decrease to zero. If the early diastolic transmitral flow velocity does not decrease to zero, the decay slope should be extrapolated to the zero baseline (Fig. 4B). MVA is calculated using $MVA (cm^2) = 759/DT$ (milliseconds).

MVA by Continuity Equation—The continuity method is based on the conservation of mass. Transmitral stroke volume (SV) is equal to the SV through the left or right ventricular

(RV) outflow tracts (LVOT or RVOT). For example, $MV\ SV = LVOT\ SV$; therefore, $MVA = (LVOT\ CSA \times LVOT\ VTI) / (MV\ VTI)$, where VTI = velocity time integral and CSA = cross-sectional area. LVOT or RVOT VTI is measured using pulse wave Doppler with the sample volume placed approximately 5 mm proximal to the aortic or pulmonary valves, respectively. The LVOT and RVOT diameter also should be measured at this proximal point. Diameter measurements are made carefully because these values are squared and can exaggerate error. MV VTI should be measured using CWD to capture the velocity profile where the orifice is smallest, with beam alignment optimization as performed for pressure gradient measurement.

The continuity equation is less dependent on cardiac output than the pressure gradient. A major limitation of the continuity equation is that it cannot be used if there are significant intracardiac shunts or regurgitant jets at measurement points or anywhere between them. AR or pulmonary regurgitation results in overestimation of MVA, whereas MR results in underestimation of MVA. Also, because measurements are made in several different imaging planes and cardiac cycles, irregular rhythms and variable loading conditions during acquisition invalidate the assumption of equivalent beat-to-beat flow.

MVA by PISA—The PISA method depends on flow acceleration; as flow accelerates toward a narrow orifice, it forms hemispheric “shells” of increasing velocity and decreasing radius. Each hemisphere is considered an isovelocity surface. Calculation of MVA by PISA is an application of the principle of continuity of flow; peak flow rate across the area of any hemispheric shell must equal peak flow rate through the MV orifice. Because the MV orifice is the narrowest point in the path of blood flow, it corresponds to the location of maximal diastolic flow velocity (V_{max}) as measured by CWD through the MV (Fig. 5A); applying these variables to the continuity equation yields the MVA: $MVA = (2 \pi R^2 \times V_a) / V_{max}$, where R = radius of the hemispheric shell, V_a = velocity at the level of the shell or aliasing velocity (Fig. 5B; Supplemental Digital Content 2, Video, <http://links.lww.com/AA/B407>). This formula assumes flow acceleration toward a flat, narrow orifice. In MS, however, the MV leaflets form a funnel at an angle α , which constrains the isovelocity shell to an arc of α degrees. Therefore, the MVA formula should account for the angle correction: $MVA = (2 \pi R^2 \times V_a) / V_{max} \times \alpha / 180$.

The PISA method is not confounded by chronic or acute changes in LA pressure and LV compliance.¹⁶ Being independent of reference valve flow, PISA is not affected by significant MR or AR. Loading conditions also are less likely to influence the calculations because the acquisition of all formula components can be accomplished in a couple of beats. There are, however, several limitations to this method. Measurement of the α angle is not automated and is most often estimated (introducing error). Accurate MVA calculation also is dependent on accurate measurement of the isovelocity surface radius because squaring this value exaggerates error. The PISA changes shape as the V_a changes. PISA is flatter at high velocities and more upright at lower velocities. A hemispheric shape is usually achieved with V_a set between 30 and 40 cm/s. The PISA model also is based on the assumption of hemispheric flow convergence toward the stenotic MV orifice and may not be true with an elliptical orifice such as in rheumatic MS. Some of these limitations may be overcome by

the measurement of the PISA from a 3D data set with CFD, but this requires postprocessing software.

TEE EVALUATION FOR CONSEQUENCES OF MS

TEE for MS should include evaluation of other structural and functional changes associated with impaired transmitral flow (Table 3). Patients with LA enlargement may have or develop AF. Hemodynamically significant stenosis and sluggish LA blood flow predispose to thrombus formation. TEE in these patients may reveal spontaneous echo contrast (SEC) in the LA. Positive and negative predictive values for thrombus with SEC are 44% to 48% and 82% to 93%, respectively,^{17,18} so SEC should prompt thorough evaluation of LA appendage and chamber walls for thrombus.

Patients with MS also may have LV dysfunction. LV contractility is difficult to assess because ejection can be reduced by impaired LV preload and elevated afterload (low cardiac output–induced vasoconstriction). Reduced LV contractility has been reported in patients with aggressive inflammation secondary to rheumatic MS.¹⁹ Both diastolic dysfunction and MS can result in increased LA pressure and pulmonary congestion. A very short isovolumic relaxation time (<70 milliseconds) and systolic blunting of pulse wave Doppler pulmonary venous flow (systolic velocity < diastolic velocity) are both correlated with increased left atrial pressure (LAP). Unfortunately, the relative contribution of MS versus diastolic dysfunction to increased LAP is difficult to distinguish by TEE but is important to consider for 2 reasons: (1) diastolic dysfunction also impairs LV filling and may lower the mean transmitral gradient, causing underestimation of MS severity and (2) severe diastolic dysfunction may not improve after MS intervention and may impact long-term patient outcome. Unfortunately, MS confounds most echocardiographic modalities of diastolic assessment (pulmonary venous Doppler, mitral inflow Doppler, and annular tissue Doppler). Because patients with isolated MS normally have low to normal LV end-diastolic pressure versus diastolic dysfunction, in which LV end-diastolic pressure is high, direct LV pressure measurements during cardiac catheterization can help distinguish between the 2.

Other valves may be affected in rheumatic or degenerative MS. MR may be present with either etiology.^{20,21} Infective endocarditis, AR, and tricuspid regurgitation (TR) are associated with rheumatic MS,²² whereas calcific AS is more common with degenerative disease. In combined MS and AS, pressure gradients may lead to inaccurate severity grading because MS may falsely lower gradients by imposing a low-flow state across the aortic valve.

Finally, increased LAP because of MS may precipitate pulmonary congestion and result in vascular remodeling, RV impairment, RV dilation, and tricuspid annular dilation leading to functional TR. Pulmonary artery systolic pressure (PASP) can be estimated using CWD of a TR jet to estimate the systolic peak gradient between the RV and the right atrium. RV systolic pressure (mm Hg) is calculated as the sum of right atrial pressure and the peak gradient associated with the TR jet. In the absence of significant RVOT or pulmonary valve gradients, RV systolic pressure approximates PASP. PASP <30 mm Hg correlates with mild MS and >50 mm Hg correlates with severe. However, a wide range of PASP can be present

for a given valve area.⁸ TR repair at the time of surgery for rheumatic MS deserves special consideration. The higher burden of pulmonary hypertension and subtle rheumatic involvement of the tricuspid valve apparatus make these patients more prone to progression of TR after MV surgery.²³ The incidence of echocardiographically significant late TR after MV replacement has been found to be as high as 68% in patients with rheumatic MS.²⁴

MS poses significant challenges during surgery, depending on the etiology of the disease. Significant mitral annulus calcification seen in patients with degenerative MS may contribute to paravalvular leaks or cardiac rupture at the atrioventricular groove or LV free wall after MV replacement. In patients with rheumatic MS, valve repair with complex techniques such as leaflet enlargement and commissurotomy is possible, although more controversial because of the progressive nature of the rheumatic disease.²⁵ In summary, echocardiographic grading of disease severity is important for management decision making; the strengths and limitations of each quantitative method, along with supportive evidence, should be carefully considered.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Teaching Points

- The smallest MVA is at the leaflet base in degenerative MS and at the leaflet tips in rheumatic MS. Two-dimensional or 3D planimetry provides direct measurement. CFD-facilitated planimetry measures the functional MVA.
- MVA is calculated by PHT ($MVA = 220/PHT$). The PHT method is limited by LV hypertrophy, significant AS, severe AR, and tachycardia.
- MVA can be calculated by PISA and by continuity equation. The PISA method is limited mostly by geometric assumptions and potential technical errors. The continuity equation method is limited by variations in beat-to-beat filling, as well as the presence of intracardiac shunts or regurgitant jets at the measurement points or anywhere between them.
- The mean pressure gradient facilitates MS grading. It must be averaged over 5 beats in AF and does not correlate well with severity in the setting of low or high transmitral flows or in the presence of tachycardia.

Figure 1.

Rheumatic mitral stenosis. A, Midesophageal long-axis view zoomed on the mitral valve (MV), showing the thickened leaflets, acute angle of left ventricular inflow across the MV, with the narrowest orifice at the leaflet tips, and “hockey-stick” appearance of the anterior leaflet of the MV. B, MV leaflets with commissural fusion seen in the transgastric basal short-axis view. C, Three-dimensional data set from the same patient with en face view of the MV as seen from the left atrium. This view demonstrates well the presence of commissural fusion.

Figure 2.

Degenerative (calcific) mitral stenosis. A, Midesophageal 4-chamber view showing extensive calcification of the mitral annulus. Because of acoustic shadowing, the structures in the far field cannot be visualized well. B, Three-dimensional data set with en face view of the mitral valve as seen from the left atrium. This view demonstrates the presence of calcifications in the mitral annulus with relative sparing of the leaflets.

Figure 3.

Diastolic mitral valve orifice area measurement by 3 planimetry methods in the same patient. A, Two-dimensional transgastric basal short-axis view of the mitral leaflets. B, Three-dimensional color flow Doppler quantification in multiplanar view with similar plane alignment and measurement of the color flow area during diastole as an approximation of the functional mitral valve orifice area. Note that 2D planimetry has overestimated the valve area relative to the other methods consistent with prior studies. C, Three-dimensional quantification in multiplanar view after alignment of red, green, and blue planes to accomplish orthogonal viewing planes at the tips of the MV leaflets (approximated by the narrowest part of the color Doppler jet), with area measurement in the perpendicular plane (blue).

Figure 4.

A, Pressure gradient in a patient with severe mitral stenosis (mean gradient = 33 mm Hg). Fusion of the E and A waves in this image prevents accurate tracing of the pressure half-time (PHT) slope. B, Tracing of the deceleration slope; PHT measured at 271 ms, with the calculated mitral valve area (MVA) of 0.8 cm². C, Example of bimodal, nonlinear Doppler tracing. The PHT has been measured (green line) by extrapolating the mid-diastolic deceleration slope (PHT = 290 ms, MVA = 0.75 cm²). An incorrect measurement using the initial, high velocity part of the tracing (red dashed line) would give a falsely short PHT and overestimate valve area (PHT = 279 ms, MVA = 0.79 cm²).

Figure 5.

Proximal isovelocity surface area (PISA) method for calculation of mitral valve orifice area.

A, Midesophageal (ME) 4-chamber view with continuous wave Doppler across the mitral valve. B, ME 4-chamber view with color flow Doppler with scale and baseline adjusted to minimize the negative aliasing velocity to demonstrate a highly visible, isovelocity hemispheric “shell”. α° = angle of correction for mitral leaflets; MVA = mitral valve area; R = radius of isovelocity hemisphere; V_a = aliasing velocity of color flow Doppler; $V_{\max}$ = maximum velocity of diastolic continuous wave Doppler.

Table 1**Mitral Stenosis Qualitative Wilkins Score**

Score	Leaflet mobility	Valve thickening	Calcification	Subvalvular thickening
1	Highly mobile	Near normal (4–5 mm)	Single area of brightness	Minimal chordal thickening
2	Reduced mobility	Thickened tips	Scattered areas at leaflet margins	Chordal thickening up to one-third
3	Basal leaflet motion only	Entire leaflet thickened (5–8 mm)	Brightness extends to midleaflets	Distal third of chordae thickening
4	Minimal motion	Marked leaflet thickening (>8–10 mm)	Extensive leaflet brightness	Extensive thickening to pap muscle

The mitral valve morphology is considered favorable if the mitral echocardiographic score is ≤ 8.

Wilkins score = Leaflet Mobility + Valve Thickening + Calcification + Subvalvular Thickening.

Table 2**Methods for Mitral Stenosis Orifice Area Assessment**

Method	Area calculation	Advantages	Limitations
Planimetry	Direct measurement	Independent of loading conditions and chamber compliance	Calcification/dropout, particularly in degenerative MS Imaging plane alignment
Pressure half-time	$MVA = 220/PHT$	Simple	Abnormal loading conditions, impaired LV compliance Aortic regurgitation, ASD, VSD Atrial tachycardia
Continuity equation	$MVA = (LVOT\ CSA \times LVOT\ VTI)/(MV\ VTI)$	Relatively independent of CO and abnormal loading conditions	Valvular regurgitation Intracardiac shunts Irregular rhythms
PISA	$MVA = 2 \pi R^2 \times \alpha / 180 \times V_a / V_{max}$	Independent of CO, loading conditions, regurgitant lesions	Technically challenging Irregular rhythms Geometric assumptions

α = mitral valve leaflet angle; ASD = atrial septal defect; CO = cardiac output; CSA = cross-sectional area; LV = left ventricle; LVOT = left ventricular outflow tract; MS = mitral stenosis; MV = mitral valve; MVA = mitral valve area; PHT = pressure half-time; PISA = proximal isovelocity surface area; R = radius of the isovelocity hemispheric shell; V_a = shell aliasing flow velocity; V_{max} = maximum diastolic flow velocity; VSD = ventricular septal defect; VTI = velocity time integral.

Table 3**Summary Comparison of Rheumatic and Degenerative Mitral Stenosis**

	Rheumatic mitral stenosis	Degenerative mitral stenosis
Prevalence with age	Any age >5 y	Increasing with advanced age
Pathophysiology	Autoimmune cross-reactivity with Strep A antigen; inflammatory tissue damage	Degenerative (age) or disordered calcium metabolism
Qualitative assessment	Chordal shortening	Annular calcification
	Leaflet thickening	Sparing of leaflet tips and subvalvular apparatus unless advanced
	Reduced leaflet mobility (posterior > anterior)	Calcific shadowing
	Commissural fusion (3D TEE)	
Quantitative assessment	2D planimetry	2D planimetry—may be unreliable
	3D planimetry; pressure gradients	3D planimetry; pressure gradients—may be unreliable
	MVA by PHT	MVA by PHT—may be unreliable
	MVA by continuity equation	MVA by continuity equation
	MVA by PISA	MVA by PISA
Associated findings	Aortic regurgitation tricuspid regurgitation (functional)	Aortic stenosis; LVH; hypertension
	Infective endocarditis	Diastolic dysfunction

2D = two-dimensional; 3D = three-dimensional; LVH = left ventricular hypertrophy; MVA = mitral valve area; PHT = pressure half-time; PISA = proximal isovelocity surface area; TEE = transesophageal echocardiography.