Perspective

Standard transthoracic echocardiography and transesophageal echocardiography views of mitral pathology that every surgeon should know

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The mitral valve is the most commonly diseased heart valve and the prevalence of mitral valve disease increases proportionally with age. Echocardiography is the primary diagnostic imaging modality used in the assessment of patients with mitral valve disease. It is a noninvasive method which provides accurate anatomic and functional information regarding the mitral valve and can identify the mechanism of mitral valve pathology. This is especially useful as it may guide surgical repair. This is increasingly relevant given the growing trend of patients undergoing mitral valve repair. Collaboration between cardiac surgeons and echocardiographers is critical in the evaluation of mitral valve disease and for identification of complex valvular lesions that require advanced surgical skill to repair. This article will provide an overview of transthoracic and transesophageal assessment of common mitral valve pathology that aims to aid surgical decision making.

Keywords: Mitral valve; echocardiography; surgery; mitral stenosis (MS); mitral regurgitation (MR)

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Introduction

Mitral valve disease is one of the most common valvular pathologies. As the population ages, the prevalence of significant mitral valve disease is increasing and expected to rise exponentially (1). Accordingly, the number of patients undergoing mitral valve surgery is also increasing (2). Echocardiography has become invaluable in the clinical management of mitral valve disease in the context of pre-surgical and post-surgical assessment. It is a non-invasive, readily accessible tool, which yields accurate information about the functional anatomy of the mitral valve and mechanisms of mitral valve disease. Systematic echocardiographic assessment of the various anatomical components of the mitral valve also provides information which guides decision-making regarding the timing and type of intervention needed. Finally, a number of echocardiographic parameters have been identified as predictors of the likely success of surgical intervention (3), which provides valuable prognostic information in regard to surgical outcomes. This enables an appropriate pre-operative assessment of risk. The aim of this review is to outline some of the standard echocardiographic features, which may be relevant to the surgical management of patients with mitral valve disease.

Overview of mitral valve anatomy

The mitral valve can essentially be divided into three main anatomical components: (I) annulus; (II) leaflets (anterior and posterior); and (III) subvalvular structures (chordae tendineae and papillary muscles). The mitral annulus is part of the fibrous skeleton of the heart and is composed of a fibromuscular ring situated between the left atrium and ventricle. The mitral valve leaflets attach to this annulus. The normal annulus is elliptical in shape, with an annular area between 5-11 cm^2 (mean, 7 cm^2), and has a saddle-

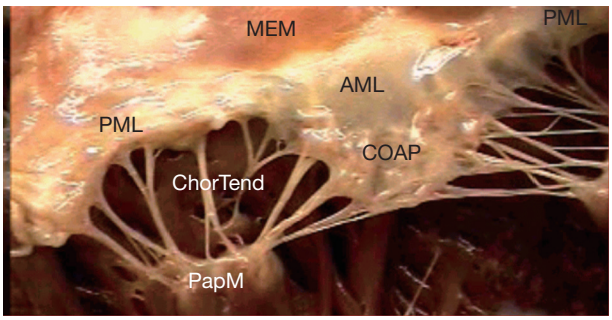

Figure 1 Fresh mitral valve demonstrating the AML and PML, ChorTend and associated PapM into which the chordae tendinae insert. The MEM and COAP of the leaflets are also shown. AML, anterior leaflets; PML, posterior leaflets; ChorTend, chordae tendinae; PapM, papillary muscle; MEM, the membranous zone; COAP, coaptation zone.

shaped configuration (4-6). The annulus can be divided into anterior and posterior segments based on insertion of the corresponding anterior and posterior leaflets. It undergoes dynamic changes in shape during the cardiac cycle. It increases in size at the end of systole and reaches its maximum dimension at the end of diastole (7), while becoming less circular in systole than in diastole (5). The dynamic change in the size and shape of the mitral annulus is significant as it facilitates the opening motion and coaptation of the leaflets. Annular contraction occurs during systole resulting in narrowing of the valve's orifice, which encourages leaflet apposition in combination with the apical force on commissural areas by the papillary muscles (8). The anterior part of the annulus is, however, relatively immobile compared to the posterior annulus. Hence, dynamic changes in the mitral annulus are likely due to movement of the posterior annulus. Pathological annular dilatation predominantly arises due to an increase in posterior annular circumference. This increases the anteroposterior valve dimension, thereby pulling the leaflets apart and compromising leaflet apposition (8).

The anterior and posterior leaflets of the mitral valve are similar in composition and thickness (approximately 1 mm) but have subtle morphological and functional differences. Both leaflets are essentially anchored to the annulus at their base. They have two distinct zones: the membranous zone, which forms the base of the leaflets and has a smooth and translucent appearance, and the coaptation zone, where the numerous chordae tendineae fuse. The coaptation zone is the rougher, more nodular and thicker portion of the leaflet (see *Figure 1*). The anterior leaflet is trapezoidal in shape, devoid of any well-defined indentations, extends vertically and is taller in height than the posterior leaflet. Via its attachment to one third of the circumference of the annulus, the anterior leaflet is also anchored to the fibrous portion of the annulus, which is more resistant to pathologic dilatation. As the cardiac fibrous skeleton becomes discontinuous away from the trigones along the mitral annulus, the posterior leaflet does not insert directly into the fibrous portion of the annulus. Instead, it inserts 2 mm from the fibrous portion of the annulus via a very thin band of connective tissue, predisposing this segment to dilatation and calcification (9). The posterior leaflet also typically has two identifiable clefts, which allow the leaflet to open fully during diastole. These clefts are used as anatomical landmarks to divide the posterior leaflet into three distinct segments, identified as P1, P2 and P3 (10). As the anterior leaflet is devoid of any prominent clefts, its division into the three segments A1, A2 and A3 is arbitrary and based on the adjacent segments of the posterior leaflet. The middle segment of the posterior leaflet, P2, typically has variable thickness and greater redundancy with the impact of greater systolic pressure, hence is more prone to prolapse and lesions (11,12). The distinct areas at which the annular attachments of the anterior and posterior leaflets come together are defined as the commissures and may represent a few millimeters of tissue or a well-developed leaflet segment. During systole, the margins of the anterior and posterior leaflets oppose over several millimeters at the coaptation zone. This zone is significant as its length can be a critical determinant of valve competency under several physiologic conditions. Normal mitral leaflets on two-dimensional echocardiography have the appearance of thin, translucent and highly mobile structures, which demonstrate maximum mobility at the leaflet tips (see *Figure 2*). Overall, the anterior mitral leaflet typically exhibits greater mobility in a number of views.

The subvalvular apparatus can be divided into the chordae tendineae and the papillary muscles. The chordae tendineae are filament-like structures of connective fibrous tissue that join the ventricular surface and free border of the leaflets to the fibrous heads of the papillary muscles and the posterior wall of the left ventricle. These form the leaflet suspension system that ultimately determines the position and tension on the leaflets at the end of systole. Marginal or "primary" chordae insert on the free margin of the leaflets and serve to prevent marginal prolapse and to align the coaptation zone. Intermediate or "secondary" chordae insert onto the ventricular face of the leaflets. These prevent the valve

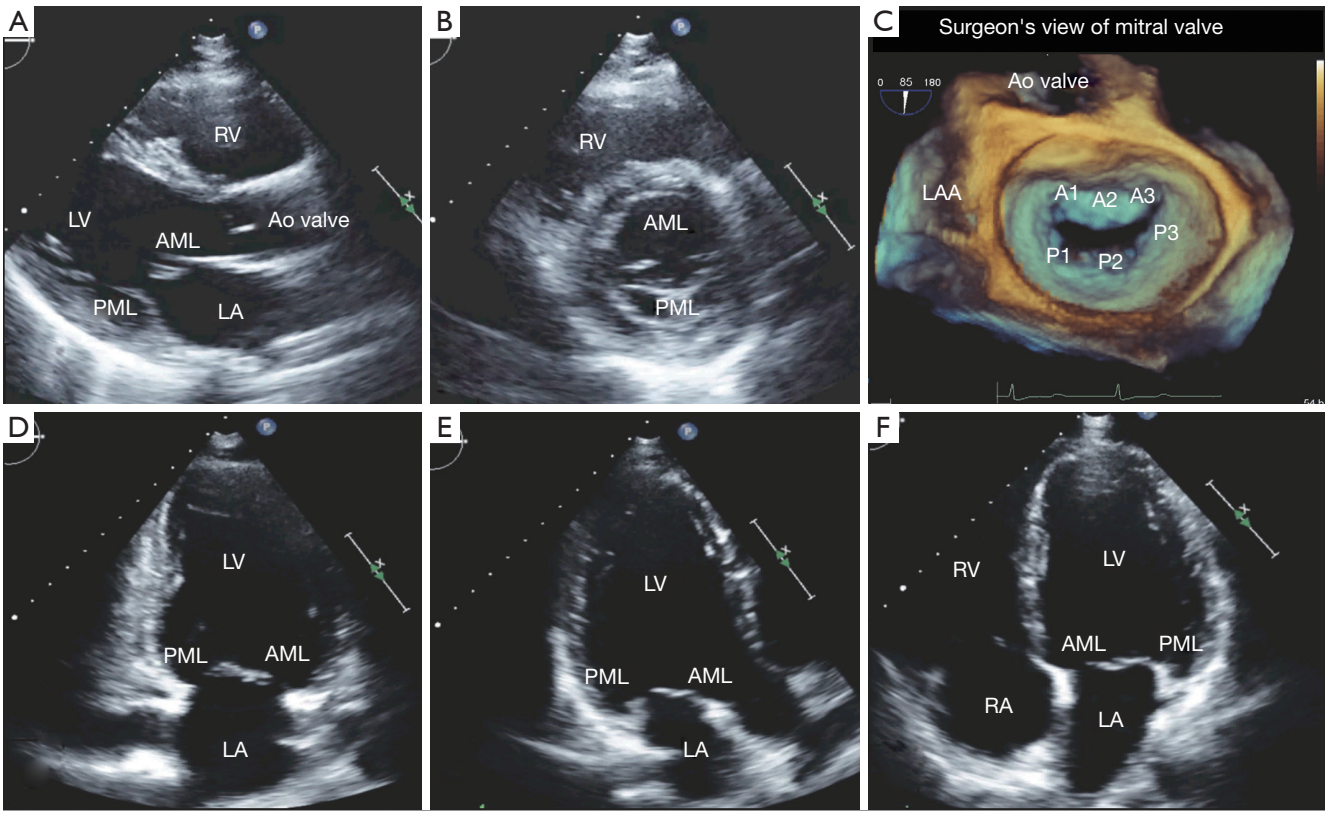

Figure 2 Common transthoracic echocardiographic views used to visualize the mitral valve in a healthy subject: (A) parasternal long axis; (B) parasternal short axis; (D) apical 4-chamber; (E) apical 2-chamber; (F) apical 3-chamber; and transesophageal three-dimensional reconstruction of the mitral valve *en face* view with the scallops and (C) surrounding structures labelled in the surgeon's view. Ao valve, aortic valve; AML, anterior mitral leaflet; PML, posterior mitral leaflet; LV, left ventricle; LAA, left atrial appendage; LA, left atrium; RA, right atrium; RV, right ventricle.

leaflet from developing excess tension and thus billowing by distributing tension across the ventricular surface of the leaflets. In addition, they may be important in maintaining dynamic ventricular shape and function due to their contribution to ventricular-valve continuity (13,14). Finally the basal or "tertiary" chordae are only associated with the posterior leaflet and connect the base of the leaflet and the posterior mitral annulus to the papillary muscles (*Figure 3*).

The papillary muscles arise from the area between the apical and middle thirds of the left ventricular free wall and are divided into two groups: the anterolateral and the posteromedial papillary muscles (15). The anterolateral papillary muscle is the larger of the two and has a single body but two heads (anterior and posterior heads). It is supplied by the first obtuse marginal branch of the circumflex artery and the first diagonal branch of the anterior descending artery. On the other hand, the posteromedial papillary muscle is smaller in size, has two bodies and three heads (anterior, intermediate and posterior heads). It is supplied by the posterior descending artery, a branch of the right coronary artery in 90% of cases and of the circumflex artery in the other 10% when it is much more vulnerable to ischemia (16). The attachment of the papillary muscles to the lateral wall of the left ventricle also means that the ventricle itself is an important functional component of the mitral valve. Hence any change in ventricular geometry that affects position of either papillary muscle can change the axial relationship of the chordae and leaflets and result in poor coaptation (8).

Mitral valve pathology and the key echocardiographic features

Due to the complex nature of mitral valve anatomy,

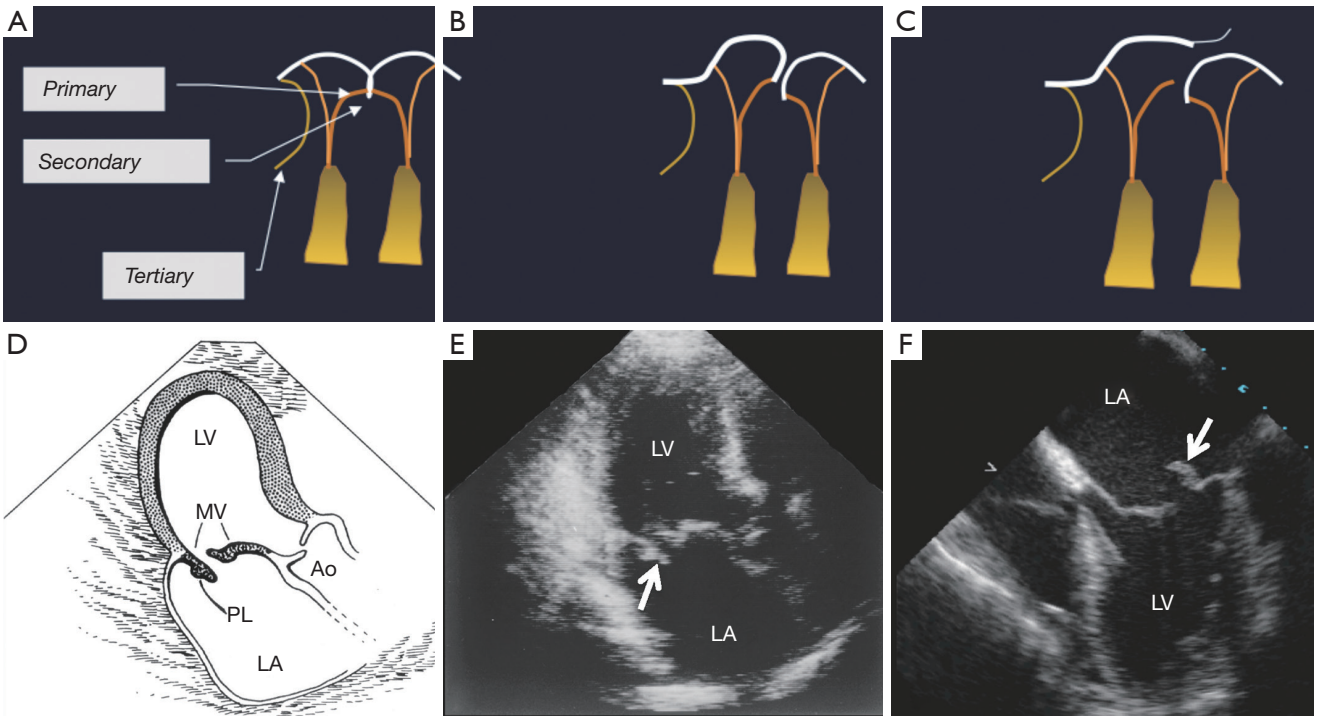

Figure 3 (A) Schematic diagram demonstrating relationship of the chordae tendineae and the mitral valve leaflets; (B) elongation of the chordae leads to prolapse of the segment or leaflet; (C) and eventual rupture of a chord results in a flail leaflet; (D-F) a flail leaflet can be identified by the loss of convex shape of the leaflet with "eversion" of the normal convex shape of the leaflet scallop as in the case of the posterior leaflet. (E) is an apical long axis view on TTE and (F) is a 4-chamber view on TEE (0°). The white arrow indicates the prolapsed segment of the posterior leaflet. Ao, aorta; LA, left atrium; LV, left ventricle; PL, posterior leaflet; TTE, transthoracic echocardiography; TEE, transesophageal echocardiography.

coordination between all its independent anatomical components is required in order for the valve to function normally. Hence the echocardiographic assessment of the mitral valve should include a systematic segmental evaluation of its individual anatomical components as well as the valve as an entire unit in order to precisely identify specific pathologic abnormalities in valve structure and function. This requires integration of information obtained from both transthoracic and transesophageal echocardiography (TEE). Views and measurements enabled by transthoracic echocardiography (TTE), such as the mitral valve orifice area, and Doppler imaging, have been validated for a range of mitral valve pathologies. TEE is generally regarded as the standard of care for the surgical assessment of mitral valve disease. Currently, a comprehensive echocardiographic assessment should integrate information from multiple two-dimensional multi-planar tomographic views with and without color Doppler and three-dimensional imaging, if available. Echocardiography is also utilized to assess the success of a surgical procedure immediately post-operatively and for subsequent follow-up.

Mitral valve dysfunction can arise from pathology at any one of the multiple anatomical levels. Essentially, mitral valve pathology requiring surgical intervention can be broadly classified into either mitral regurgitation (MR) or mitral stenosis (MS). MR is by far more common in developed countries. Functional MR occurs in up to 50% of patients who present with systolic heart failure. The most common type of degenerative mitral valve disease is mitral valve prolapse, which occurs in 1-3% of the population in the United States. Conversely, rheumatic MS is the least common pathology affecting the mitral valve in the United States, accounting for less than 1% of cardiac diagnoses, although more frequently seen in developing nations (17).

Mitral stenosis (MS)

The two most common etiologies of MS are rheumatic

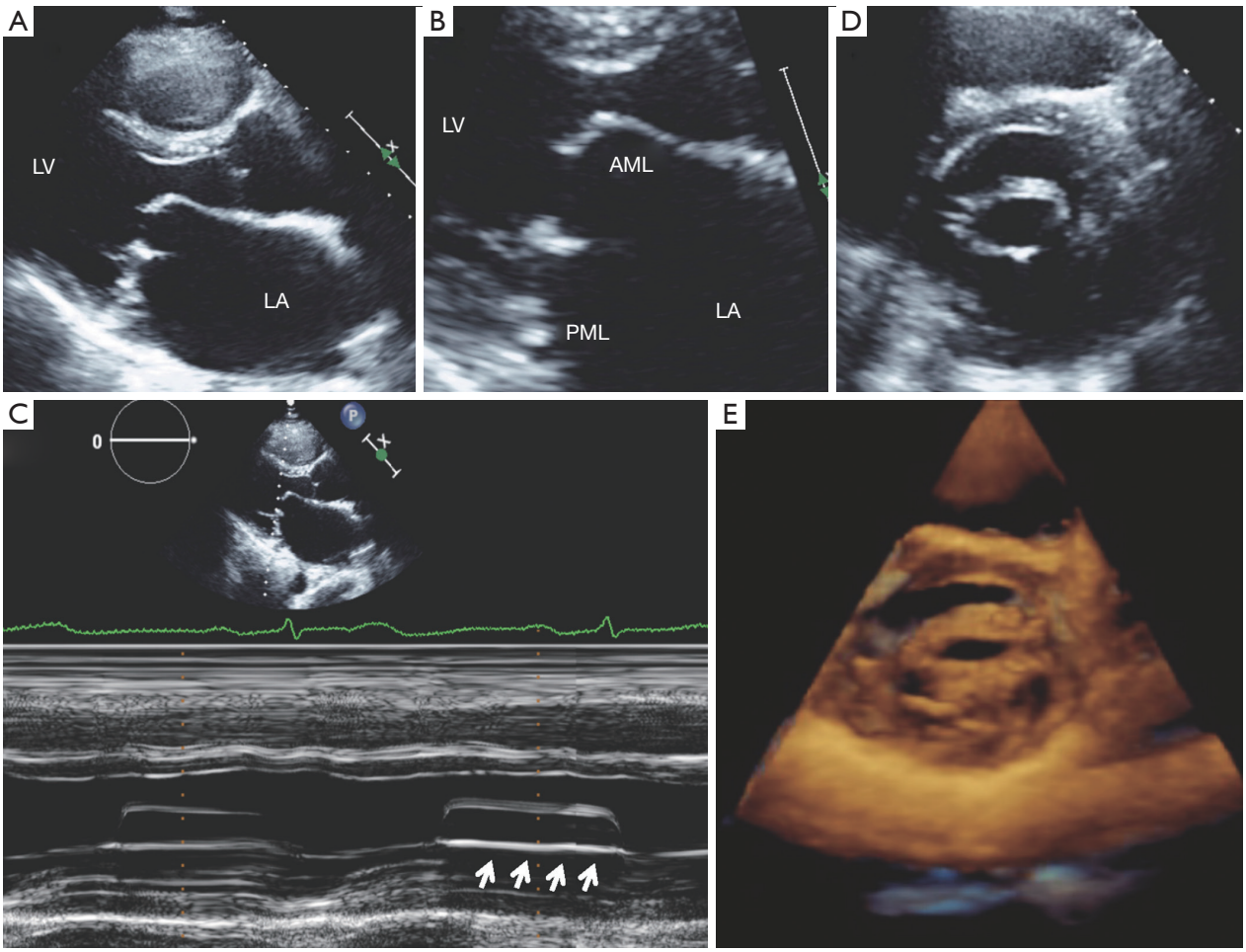

Figure 4 Rheumatic mitral valve disease. (A,B) Parasternal long axis view demonstrating the "hockey stick" appearance of the anterior mitral leaflet; (C) M-mode imaging of the mitral valve demonstrating anterior movement of the posterior valve in parallel to the anterior leaflet rather than a posterior motion (indicated by the white arrows; (D) showing fusion/calcification of the commissures and the mitral leaflet tips but not the body of the leaflets; (E) parasternal short axis view at the level of the mitral valve and 3D real-time full volume image of the mitral valve. LA, left atrium; LV, left ventricle; AML, anterior mitral leaflet; PML, posterior mitral leaflet.

MS and degenerative (calcific) MS. The main mechanism of rheumatic MS is commissural fusion. Other anatomic lesions such as chordal shortening, chordal fusion, leaflet thickening and superimposed calcification may restrict leaflet motion by causing progressive narrowing of the mitral valve orifice at the level of the leaflet tips. Key echocardiographic features include valve thickening (typically the posterior leaflet is thickened and restricted), restricted valve opening, anterior leaflet doming and fusion of the leaflets at the commissures. Additionally, leaflet thickening in rheumatic MS is typically most pronounced at the tips with relative sparing of the mid portion, which gives rise to the characteristic "hockey-stick" appearance of the leaflets. These changes are best appreciated on two-dimensional echocardiography in the parasternal long and short views as well as the apical views (see *Figure 4A,B*). M-mode imaging, which is typically performed in the parasternal long and short axis views, demonstrates a distortion of the pattern to one that is distinct for rheumatic MS, which involves anterior movement of the posterior valve in parallel to the anterior leaflet rather than a posterior motion (see *Figure 4C*). Real time three-dimensional echocardiography also allows visualization of the mitral valve *en face* and can be performed via transthoracic or TEE (see *Figure 4D,E*). Assessment of stenosis severity and the presence of complications of MS should also be performed.

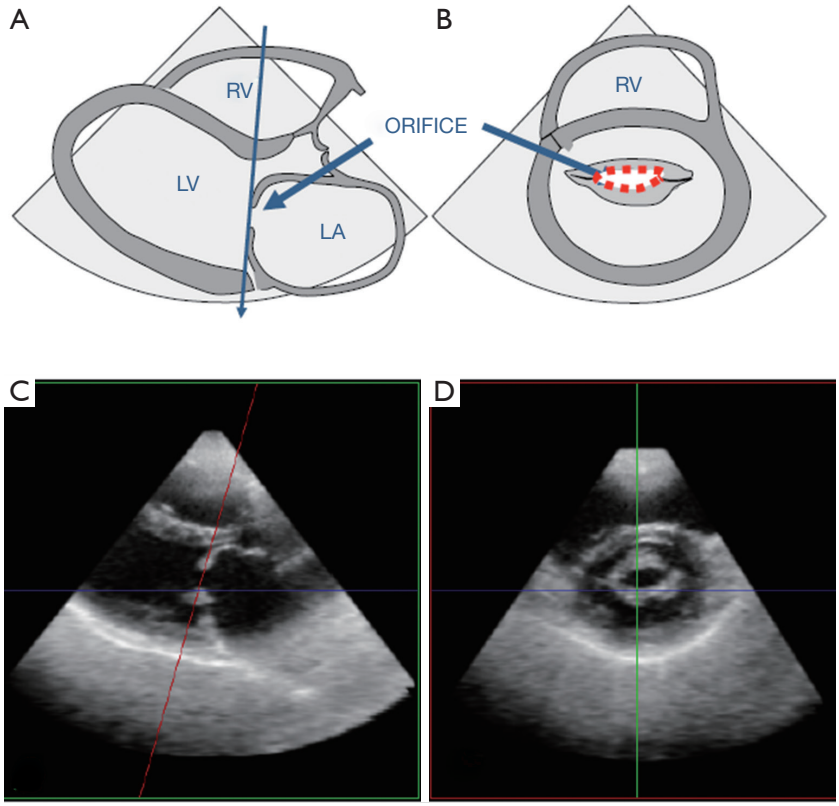

Figure 5 Measuring mitral orifice area in rheumatic mitral valve disease. Note the funnel shape of the mitral valve. Care is taken to ensure that the parasternal short axis view is at the level of (A) the mitral valve tips and (B) the red dotted line demonstrates the mitral orifice area for planimetry. RV, right ventricle; LA, left atrium; LV, left ventricle.

There are five validated methods for the assessment of severity of MS but the most common methods include measurement of the mitral orifice area by direct planimetry (see *Figure 5*) and assessment of gradients across the mitral valve by Doppler imaging or by the pressure half-time method (see *Figure 6*). Planimetry of the mitral valve is the current gold standard for the assessment of mitral valve orifice area as it is based on direct visualization of the mitral valve orifice and not limited by hemodynamic loading conditions. It has been shown to have excellent correlation with direct sizing at surgery but requires care so that planimetry is performed at the level of the leaflet tips (see *Figure 5*) (18,19). The Doppler-derived mean transmitral gradient is easily measured and is a useful tool to quantify severity. It is also reproducible and correlates well with invasive measurements. The gradient is assessed by the application of the simplified Bernoulli equation: $\Delta\text{Pressure} = 4v^2$, where v is the transmitral velocity obtained from continuous-wave Doppler interrogation of the mitral flow in the 4-chamber view. This view allows for parallel alignment of the ultrasound beam and mitral inflow. The mean gradient is obtained from the average of digitized instantaneous gradients enveloped in the continuous-wave Doppler profile. Presently, integrated software automatically calculates and displays the peak and mean gradients (*Figure 6A*). Color Doppler is also useful in identifying eccentric diastolic mitral jets. These are often encountered in cases of severe deformity of the valvular and subvalvular apparatus and assist in guiding the placement of the Doppler beam through the highest flow velocity zone (18).

The appearance of calcific MS differs markedly from rheumatic MS. Calcific MS is commonly seen in association with hypertension, atherosclerotic disease, increased age and chronic kidney disease. In the case of non-rheumatic MS, there may be leaflet thickening and restriction of leaflet mobility but the commissures are rarely fused. Calcific MS results from annular calcification, which becomes heavy and encroaches onto the valve leaflets, resulting in

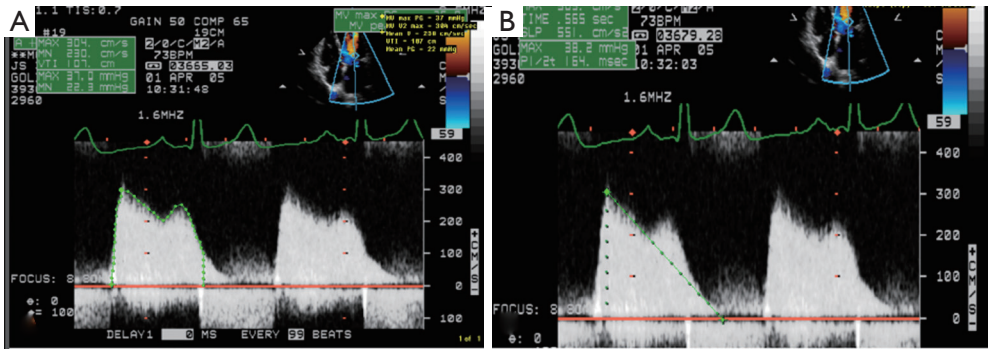

Figure 6 Assessing severity of MS. (A) Continuous-wave Doppler profiles are used to obtain gradients across the mitral valve. Pressure half-time (P1/2) is the time it takes for the pressure gradient across the mitral valve to decrease by half. P1/2 is inversely related to mitral valve area; (B) the longer it takes for the pressure gradient to drop to half its value, the greater the degree of stenosis. MS, mitral stenosis.

thickening and/or calcification of the leaflets and a decrease in the anatomic orifice area. Calcification can also extend into the myocardium of the left ventricle and beneath the endocardial surface of the posterior leaflet (20). In calcific MS, the smallest mitral orifice area is at the base of the mitral leaflets rather than at the leaflet tips such as in rheumatic MS. On two-dimensional echocardiography, mitral annular calcification appears as echogenic material within the annular region and is best visualized on the parasternal long and short axis views (*Figure* 7). Apart from the morphological changes, the methods for evaluating stenosis severity are also different since many of the 2D and Doppler-derived techniques used for the assessment of gradients across the mitral valve cannot be reliably applied to calcific MS. This is due to the pattern of valve deformation and coexisting hemodynamic abnormalities. Planimetry of the mitral orifice area is difficult to perform in calcific MS since stenosis originates from heavy annular calcification that extends to the leaflets. Hence, the limiting orifice cannot be readily appreciated in short axis (21-23).

Mitral regurgitation (MR)

MR is the most common valvular disease with moderate or severe MR found in 1.7% of the general population, 6.4% of patients aged 65-74 years and 9.3% of those >75 years (1,24). Rheumatic heart disease is the most frequent cause of MR in developing countries, however degenerative or myxomatous mitral valve disease is the leading cause of MR in developed countries. Other disease entities such as ischemic heart disease and valvular damage from bacterial endocarditis also contribute as significant causes of MR (25). The approach to the assessment of MR needs to include assessment of valve dysfunction, lesions and the mechanism underlying the pathology, which is important for surgical decision-making. Although there are a number of classification schemes for MR, one commonly used scheme is that proposed by Alain Carpentier, which is based on the closing motion of the leaflets. It comprises: (I) type I dysfunction, which involves normal leaflet motion but severe annular dilatation resulting in a central regurgitant jet or perforation of one of the leaflets; (II) type II dysfunction, encompassing excessive leaflet motion secondary to pathologic elongation or rupture of the chordae tendineae and prolapse of the leaflet or a segment of the leaflet resulting in a regurgitant jet that is directed to the opposite side of the affected leaflet; and (III) type III dysfunction, which involves restricted leaflet motion due to retraction of the subvalvular apparatus. Two subtypes of type III dysfunction are possible: type IIIa, which is apical displacement of the leaflets and restricted leaflet motion during diastole and systole (tethering of the valve), frequently seen in rheumatic disease, inflammatory processes or papillary muscle displacement (from ischemic remodeling or dilated cardiomyopathy); and type IIIb, which is restricted leaflet motion predominantly during systole resulting in a regurgitant jet typically directed to the same side as the affected leaflet (10).

Mitral valve prolapse is primarily diagnosed by echocardiography and is defined as the systolic displacement of one or both mitral leaflets into the left atrium of greater than 2 mm (*Figure 8A*). It can typically occur with or without MR and in patients with evidence of leaflet thickening on echocardiography and myxomatous changes

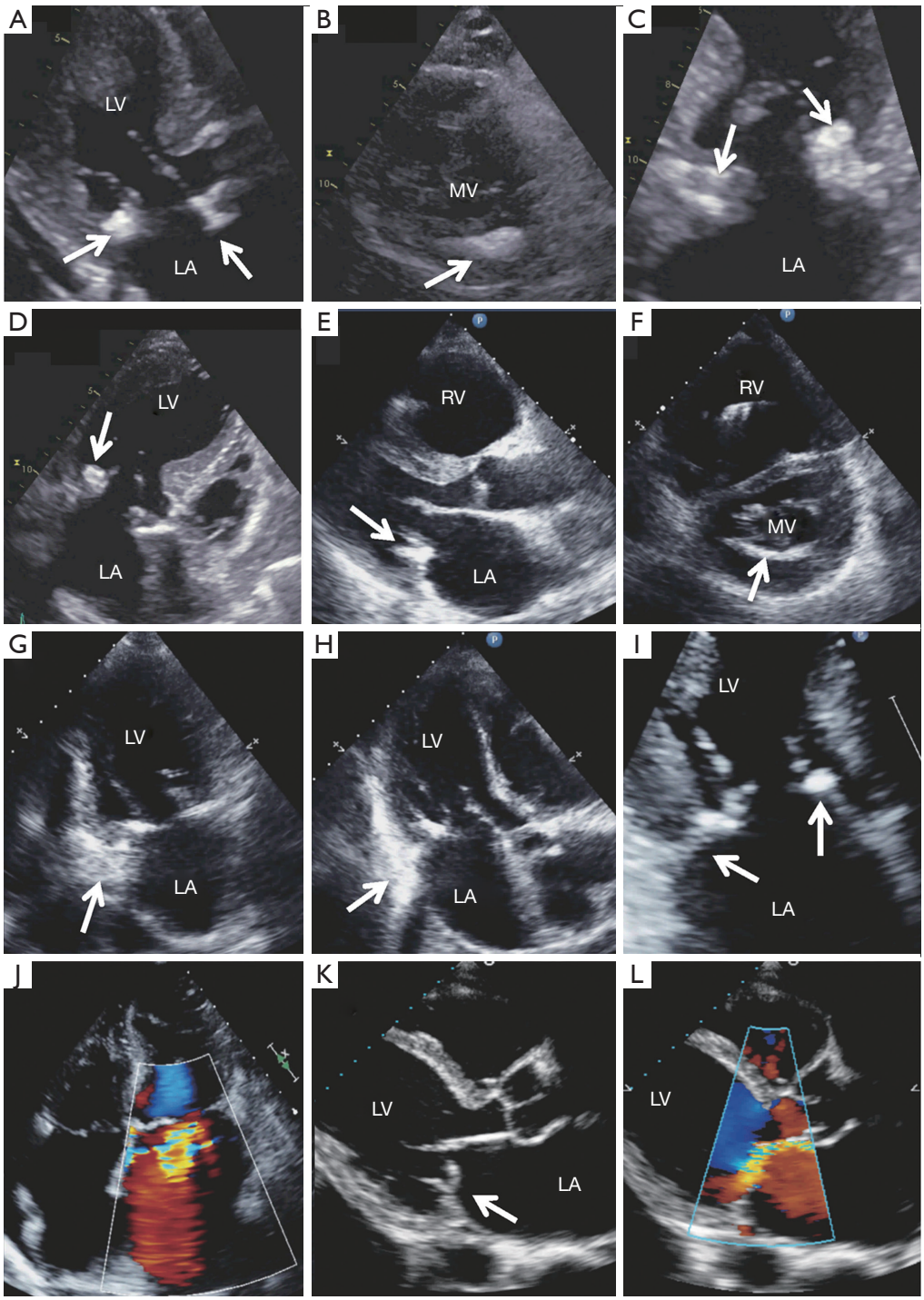

Figure 7 Four different examples of patients with mitral annular calcification demonstrating the range of calcification which can occur. Example 1 (A-D) demonstrates significant calcification of the annulus with the posterior annulus having significantly greater calcification than the anterior annulus; example 2 (E-H) demonstrates mitral annular calcification and calcification of the posterior leaflet with restriction of posterior leaflet motion; example 3 (I and J) and example 4 (K and L) demonstrate different degrees of calcification of the mitral valve leaflets and underlying chordae resulting in restriction of leaflet motion and MR as demonstrated by color Doppler (J and L); (A and E) Parasternal long axis; (B and F) parasternal short axis; (C and G) apical 4-chamber and (D and F) apical 3-chamber views. The areas of calcification are highlighted by the white arrows. LA, left atrium; LV, left ventricle; MV, mitral valve; MR, mitral regurgitation.

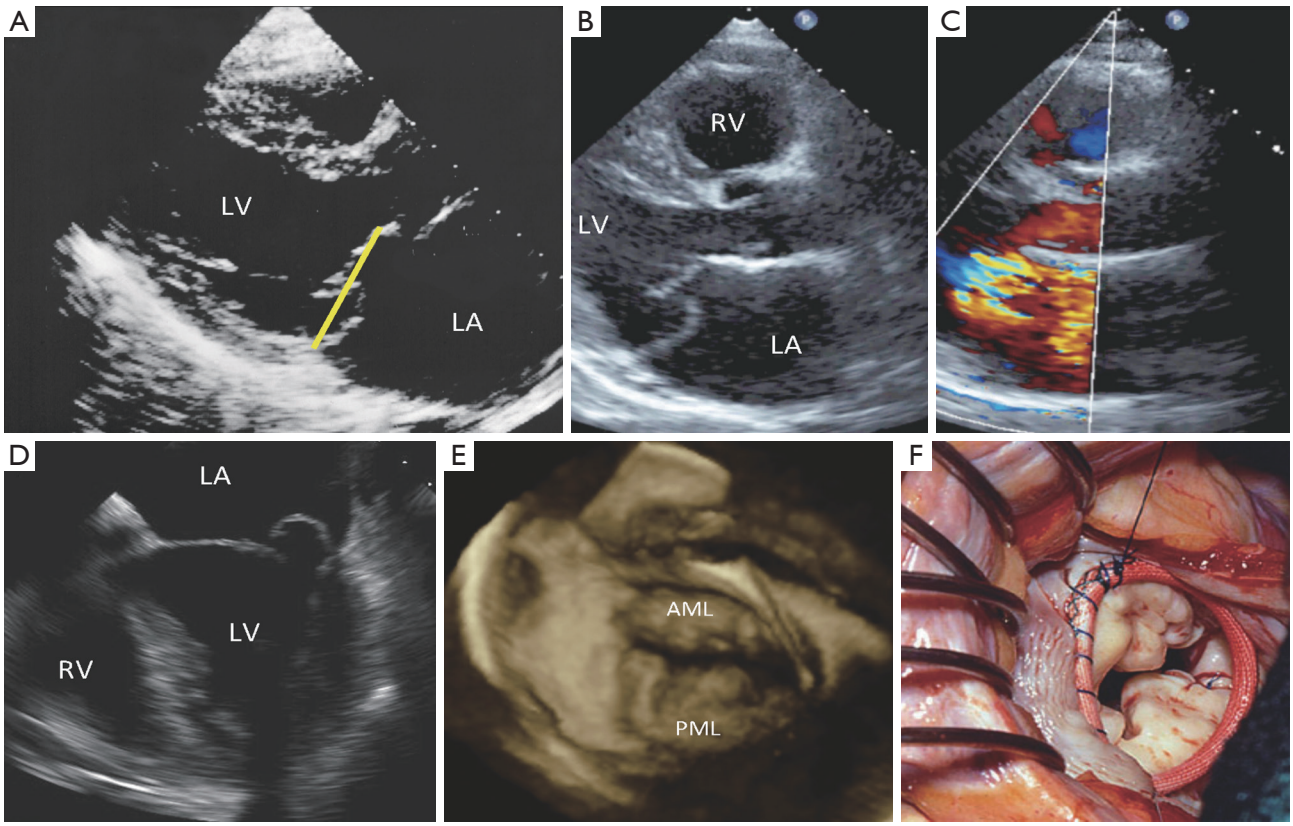

Figure 8 (A) Assessment for prolapse of the mitral leaflets of the mitral valve, which is defined as prolapse of the leaflet into the left atrium of greater than 2 mm; (B) parasternal long axis view demonstrating prolapse of the posterior leaflet; (C) with significant MR by color Doppler. In the operating room, TEE is used to help confirm and localize the prolapsed portion, which in this example is of the posterior leaflet; (E) transesophageal 3D reconstruction of the mitral valve performed in the operating room, with the valve viewed through the left atrium (surgeon's view), showing BD with the prolapsed segment seen along the lateral scallop of the posterior leaflet; (F) mitral valve repair in BD-resection of redundant leaflet tissue and insertion of a mitral ring. LA, left atrium, LV, left ventricle, RV, right ventricle; MR, mitral regurgitation; TEE, transesophageal echocardiography; BD, Barlow's disease.

found on pathoanatomy. Degenerative disease of the mitral valve is typically a result of one of two opposing clinical entities: fibroelastic deficiency (FD) and Barlow's disease (BD). Unfortunately, it is difficult to distinguish between these entities. Differentiation between the two is crucial as lesions due to BD are complex and frequently require expert surgical skill compared lesions due to FD, which are easier to repair (26-28). FD is the more common of the two and results from loss of mechanical integrity due to abnormalities of connective tissue structure or function. This typically results in localized or unisegmental prolapse or a flail leaflet (*Figure 3*). It is typically seen in patients in their sixth decade with a short, acute history of MR, likely due to rupture of a single mitral chord (26,29). On the other hand, BD results from an abnormal accumulation of mucopolysaccharides in the leaflets and chordae. This results in thick, bulky, redundant billowing leaflets and elongated chordae, which in turn results in prolapse of the leaflet(s) (*Figure 8*). This condition is typical in a younger female demographic and is usually relatively stable until the fourth decade (26,29). In regard to echocardiographic features, the key findings in BD are leaflets that have diffuse, complex lesions with prolapse and myxomatous degeneration of many segments in one or both leaflets. This may be due to excessive leaflet tissue, leaflet thickening, distension, elongation and thickening and/or the rupture of several chordae tendineae. These patients with BD also frequently have a dilated annulus and various degrees of annular and subvalvular apparatus calcification, particularly at the posterior face of the annulus and the posteromedial

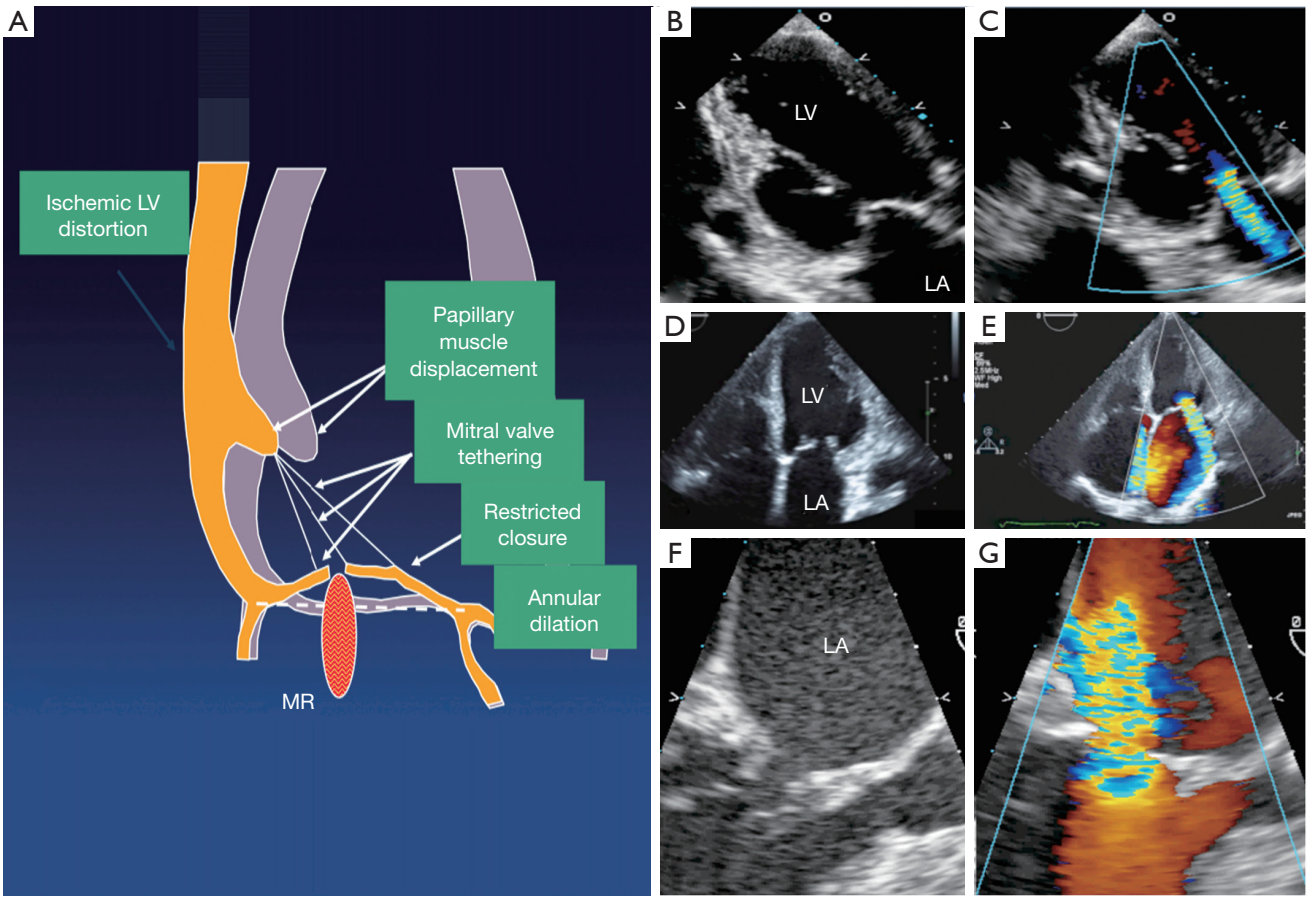

Figure 9 (A) Schematic diagram demonstrating the mechanism underlying ischemic MR; (B-E) examples of MR secondary to ischemic heart disease where tethering of the posterior leaflet results in MR; degenerative changes can develop in combination with ischemic MR as demonstrated in (F) and result in significant MR (G). LA, left atrium; LV, left ventricle; MR, mitral regurgitation.

papillary muscle (30,31). This is in contrast to other conditions that also cause MR such as rheumatic disease, infective endocarditis or FD (8,32-34). Patients with FD typically have chordal rupture due to the progressive weakening and elongation of the chordae tendineae, usually involving the mid-segment of the posterior leaflet (*Figure 3*). The leaflets and the segments are usually completely normal with no change in height, size or tissue properties (35-38). However, it should be noted that chronic FD can occasionally result in distension of the prolapsing segment and demonstration of myxomatous characteristics (39). The key to distinguishing FD from other entities is exhaustive analysis of segments contiguous to the one that has prolapsed (40).

Ischemic heart disease is currently responsible for approximately 20% of patients with MR. MR due to ischemic heart disease can arise as a direct complication of a myocardial infarct, e.g., due to papillary muscle dysfunction or rupture, or as a consequence of left ventricular remodeling secondary to a myocardial infarct (41). Remodeling of the left ventricle results in apical and inferior displacement of the papillary muscles and tethering of the mitral leaflets, failure of coaptation and hence MR (*Figure 9*) (42,43). This is of particular significance in patients who have had a posterior infarction since remodeling may result in an asymmetric restriction of the posterior leaflet in systole (44,45). In contrast, patients with a dilated cardiomyopathy or anterior and posterior infarctions typically have both leaflets restricted (46).

Conclusions

In conclusion, echocardiography plays an important role in the diagnosis and assessment of mitral valve pathology in

the context of surgical management. Key echocardiographic features associated with particular disease entities enable the use of echocardiographic aids in the diagnosis, follow-up and subsequent clinical management of patients with mitral valve disease. The information obtained from a thorough echocardiographic assessment plays a significant role in the clinical decision-making process as to whether there should be surgical intervention and, if so, the necessary expertise needed. The availability of newer echocardiographic technology such as three-dimensional echocardiography has also improved our assessment of complex mitral valve lesions, allowing for improved pre-operative risk stratification. Precise follow-up of changes in geometry, volumes and hemodynamics is possible and this facilitates appropriate surgical intervention for patients.

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Footnote

Conflicts of Interest: The authors have no conflict of interest to declare.

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