Degenerative Mitral Valve Regurgitation: Surgical Echocardiography

David H. Adams, MD, Anelechi C. Anyanwu, MD, Lissa Sugeng, MD, MPH, and Roberto M. Lang, MD

Corresponding author
David H. Adams, MD
Cardiothoracic Surgery, Mount Sinai Medical Center, 1190 Fifth Avenue, New York, NY 10029, USA.
E-mail: david.adams@mountsinai.org

Current Cardiology Reports 2008, **10:**226–232
Current Medicine Group LLC ISSN 1523-3782

Echocardiography is an essential tool in managing patients with mitral regurgitation. It allows identification and precise summation of anatomic lesions that lead to valve dysfunction. In this article, we highlight important aspects of mitral valve anatomy and pathophysiology as they pertain to surgical repair and discuss the critical role of echocardiography in surgical planning and assessment. Better understanding of surgical anatomy of the mitral valve and systematic segmental valve analysis by echocardiographers will allow easy identification of complex valve lesions or dysfunctions that require advanced surgical skill, techniques, or expertise to affect a repair. By triggering referral of such patients to "reference" mitral valve surgeons and by providing information that enables the surgeons to adequately plan a repair preoperatively, echocardiographers will help eliminate the scenario in which mitral valve replacements are performed because the surgeons discover intraoperatively that it is beyond their skill to repair a valve.

Introduction

Because of its complex three-dimensional (3D) anatomy, the mitral valve is often viewed as the most "complex" of the four human heart valves, and its function can be affected at multiple anatomical levels. It is critical that the cardiovascular imager has a thorough understanding of mitral valve anatomy in normal and diseased states. This article seeks to provide the reader with a primer of mitral valve surgical anatomy. Mitral valve repair is an underused technique in most heart centers today. The cardiologist performing an echocardiogram is in a prime position to provide information to the surgeon, as well as to the patient and referring physician, that will positively impact the chances of a successful and durable repair.

Mitral Valve Anatomy

The mitral valve is a "one-way door" occupying the left atrioventricular groove, ensuring unidirectional diastolic flow of blood from the left atrium into the relaxed left ventricle. During systole, a precise and complex interaction of anatomical components and ventricular pressure results in mitral valve closure. From the surgical and imaging standpoint, mitral valve anatomy should be evaluated systematically according to these components and then as a unit to precisely identify the lesions (the pathologic abnormalities in valve structure) and dysfunctions (the alteration in closure mechanism that leads to mitral regurgitation), a concept introduced by Carpentier [1] more than two decades ago.

The annulus

The mitral annulus is a fibromuscular ring situated between the left atrium and ventricle to which the anterior and posterior mitral valve leaflets attach. The opening motion of the leaflets hinges at their insertion site. The mitral annulus is usually divided segmentally by the insertion of the corresponding leaflet (anterior and posterior annulus) or less frequently by anatomical relationship (septal and lateral annulus). From a surgical perspective, it is useful to consider the hands of a clock when describing locations on the annulus, in which 12 o'clock is the midpoint of the anterior annulus (this replicates the surgeon's intraoperative view of the valve). The anterior mitral annulus is fibrous in nature, and through the left and right fibrous trigones is in continuity with the heart's fibrous skeleton, and the aortic-mitral curtain. Implications of this include relative resistance to pathologic dilatation and vulnerability of this portion of the valve to "skip" or direct extension lesions from aortic valve endocarditis. The cardiac fibrous skeleton becomes discontinuous as it proceeds away from the trigones along the mitral annulus, explaining why the posterior portion of the mitral annulus is more prone to pathologic dilatation. Annular dilatation

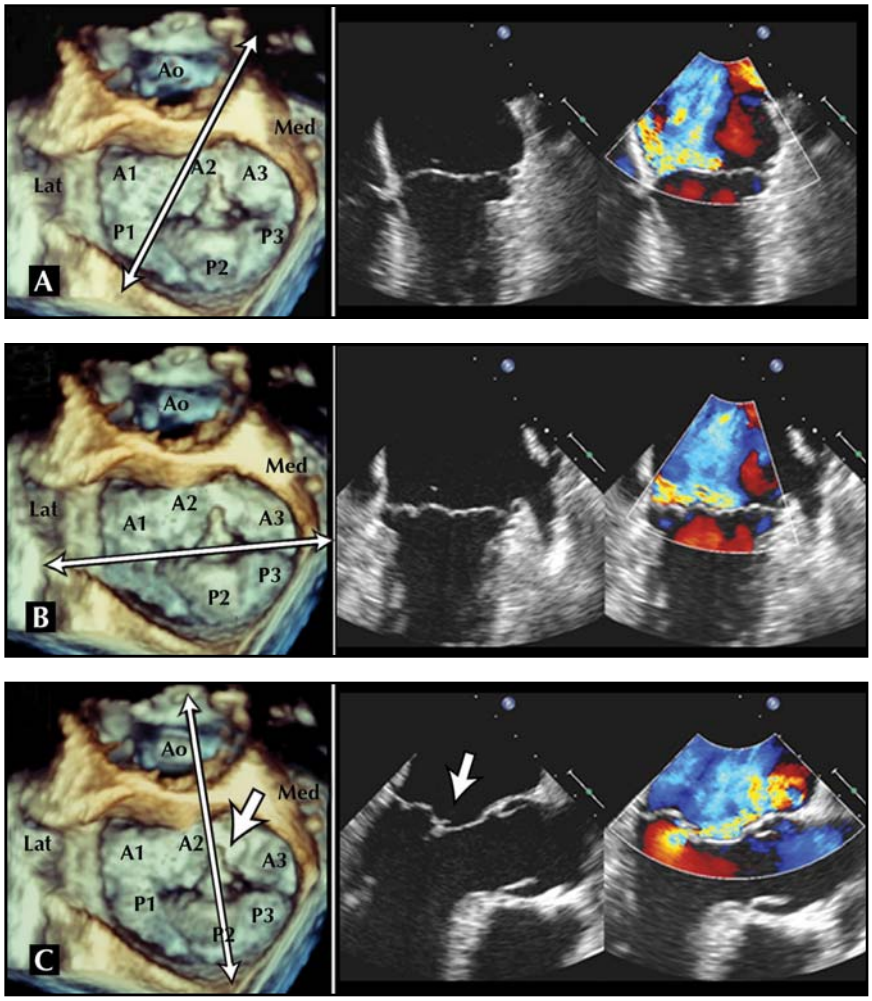

Figure 1. Example of a patient with a type II – P2 degenerative mitral valve disease due to fibroelastic deficiency. **A,** Four-chamber view obtained at 0° depicting A2 on the left and P2 on the right. **B,** Two-chamber view obtained by rotating the transducer to 90° to visualize P3 on the left and the three scallops of the anterior mitral valve leaflet on the right. **C,** Long-axis view obtained by further rotating the transducer to 120° to view the flail P2 segment (*small arrow*) on the left and A2 on the right. The direction of the color jet in the long-axis view (bottom left) is away from the side of the lesion. The images in the left column correspond to the surgical mitral valve image (as visualized from the left atrial perspective) obtained using real-time three-dimensional matrix-array transesophageal echocardiography (TEE) volume rendering. The *long arrows* represent the approximate cut planes from which the respective two-dimensional TEE images were obtained. A1, A2, A3—scallops of the anterior mitral valve; Ao—aorta; Lat—lateral; Med—medial; P1, P2, P3—scallops of the posterior mitral valve.

manifests primarily as an increase in posterior annular circumference, which has the effect of increasing the anterior-posterior valve dimension, thereby pulling the valve leaflets apart and compromising leaflet apposition. The normal mitral annulus has a 3D saddle shape. During systole, annular contraction occurs, narrowing the valve orifice and encouraging leaflet apposition, and the commissural areas are pulled apically by the papillary muscles, accentuating the anterior mitral saddle and accommodating the bulging aortic root.

Leaflets and commissures

The posterior leaflet has a greater length but a shorter height than the anterior leaflet and attaches to the posterior three fifths of the annulus and typically has two identifiable clefts or indentations. Carpentier [1] ascribed a useful nomenclature to "segment" the leaflet based on these indentations (Fig. 1). The middle posterior leaflet segment is designated as P2, whereas the adjacent anterior and posterior segments are designated as P1 and P3, respectively. The anterior leaflet has a shorter base but is taller in height than the posterior leaflet and occupies about two thirds of the orifice area of the valve during systole. The anterior leaflet margin does not have well-defined indentations, but the opposing segments to the posterior scallops are designated as A1, A2, and A3. The areas in which the two leaflets insert next to one another are designated as the anterolateral (or anterior) and posteromedial (or posterior) commissures. These commissures define the distinct areas at which the annular attachments of the anterior and posterior leaflets come together, and may represent a few millimeters of tissue or present as a well-developed leaflet segment. During systole, the margins of the two leaflets oppose for several millimeters; the length of this "coaptation zone" is a critical determinant of valve competency under several physiologic conditions.

Chordae, papillary muscles, and the ventricle

The chordae tendineae make up the leaflet suspension system that ultimately determines the position and tension on the leaflets at end systole. Chordae originate from the fibrous heads of the papillary muscles and may be classified according to their insertion site on the leaflet. Marginal or "primary" chordae insert on the free margin of the leaflets and prevent marginal prolapse and also align the coaptation zone. Intermediate or "secondary chordae" insert on the ventricular

surface of the body of the leaflets and primarily prevent billowing and distribute tension on leaflet tissue. They may also play a role in dynamic ventricular shape and function due to their contribution to ventricular-valve continuity [2,3]. Basal or "tertiary chordae" connect the posterior leaflet base and mitral annulus to the papillary muscle.

Two papillary muscles arise from the area between the apical and middle thirds of the left ventricular free wall: the anterolateral papillary muscle is often composed of an anterior head and a posterior head, whereas the posteromedial papillary muscle often has an anterior, intermediate, and posterior head [4]. Each papillary muscle provides chordae to both leaflets, and the axial relationship of the chordae prevents chordal abrasion or dyssynchrony. The anterolateral papillary muscle blood supply may originate from one or more left coronary artery branches. The left circumflex or right coronary artery (depending on dominance) provides the blood supply to the posteromedial papillary muscle. Because of its single vascular supply, this papillary muscle is particularly prone to injury from myocardial infarction. The attachment of the papillary muscles to the lateral wall of the left ventricle means that the ventricle itself is also an important functional component of the mitral valve. Any change in ventricular geometry that affects position of either papillary muscle can change the axial relationship of the chordae and leaflets, resulting in poor coaptation.

The Pathophysiologic Triad of Mitral Valve Disease

Carpentier et al. [1,5] were the first to emphasize the importance of making a distinction between the disease causing mitral regurgitation, the lesions resulting from the disease, and the valvular dysfunction that results, terming this the "pathophysiologic triad" of mitral valve disease. Carpentier and colleagues [1,5] classified mitral valve dysfunctions based on the closing motion of the margin of the leaflets in relation to an imaginary line representing the level of the annular plane. Type I dysfunction signified normal leaflet motion, type II dysfunction implied excess motion of the leaflet margin above the plane of the annulus, and type III dysfunction equated to restricted leaflet motion resulting in coaptation below the annular plane. Type III was later divided, with type IIIa designating restricted leaflet motion in systole and diastole (ie, restricted opening and closure), and type IIIb implying restricted leaflet motion that occurs only in systole (ie, restricted closure only) [6].

Any degenerative mitral valve regurgitation can be classified on the basis of this pathophysiologic triad (etiology, lesion, and dysfunction). In type I dysfunction, the lesion would be a pure degenerative annular dilatation that can occur, for example, in patients with chronic atrial fibrillation. The lesions most commonly associated with type II dysfunction are chordal elongation or rupture, which occur across the spectrum of degenerative mitral valve disease (Fig. 2). The typical lesions seen in type IIIa dysfunction, which may occur in conjunction with type II dysfunction in degenerative patients with Barlow's disease, are chordal or papillary retraction, fusion, and thickening, usually involving the anterior subvalvular apparatus. Type IIIb dysfunction is the result of ventricular remodeling, with the primary lesion being leaflet tethering due to papillary muscle displacement. It may coexist with prolapse (type II dysfunction) in degenerative patients with long-standing severe regurgitation that has resulted in pronounced left ventricular dilatation. Associated annular dilatation is a common finding in patients with chronic degenerative mitral regurgitation, but the classification of dysfunction should differentiate the primary lesion causing the regurgitation (eg, chordal rupture) from secondary lesions (typically annular dilatation).

Special distinction of the pathophysiologic triad is important in degenerative mitral valve disease. Specifically, a distinction between Barlow's disease and fibroelastic deficiency should be sought by the echocardiographer. Although both cause leaflet prolapse, there are key distinguishing features between these two degenerative diseases [7•]. Barlow's disease is typically diagnosed in young adulthood, and patients are often under long follow-up before developing an indication for surgery, often during the fourth or fifth decade of life. The most common lesions are chordal elongation, tall leaflets with excess tissue and very large annular size (Fig. 2B). Calcification can occur in the annulus or anterior papillary muscle. Billowing of the leaflet (in which the body of the leaflet rises above the annular plane due to excess tissue and elongation of intermediary chords) should be differentiated from prolapse of the margin (ie, flail), with the latter used to differentiate single from bileaflet involvement (Fig. 2B). The timing of the regurgitant jet can help differentiate the lesions in Barlow's disease (if the regurgitant jet occurs in mid-to-late systole, it is usually due to chordal elongation as opposed to chordal rupture, which tends to cause holosystolic regurgitation). In contrast, fibroelastic deficiency results from a connective tissue deficiency rather than an excess of myxomatous tissue. Leaflets are generally thin and translucent, and chordae are thin, sparse, and deficient. Patients most commonly present in the sixth decade of life with relatively short histories of mitral valve disease and severe holosystolic type II regurgitation that results from a ruptured chord or chords, usually involving a single segment of the valve (Fig. 2A). Although the affected segment may demonstrate myxomatous changes in the chronic setting, the remainder of the valve is small with normal leaflet height. Figure 2 illustrates the distinction (lesion and dysfunction) between these two forms of degenerative disease.

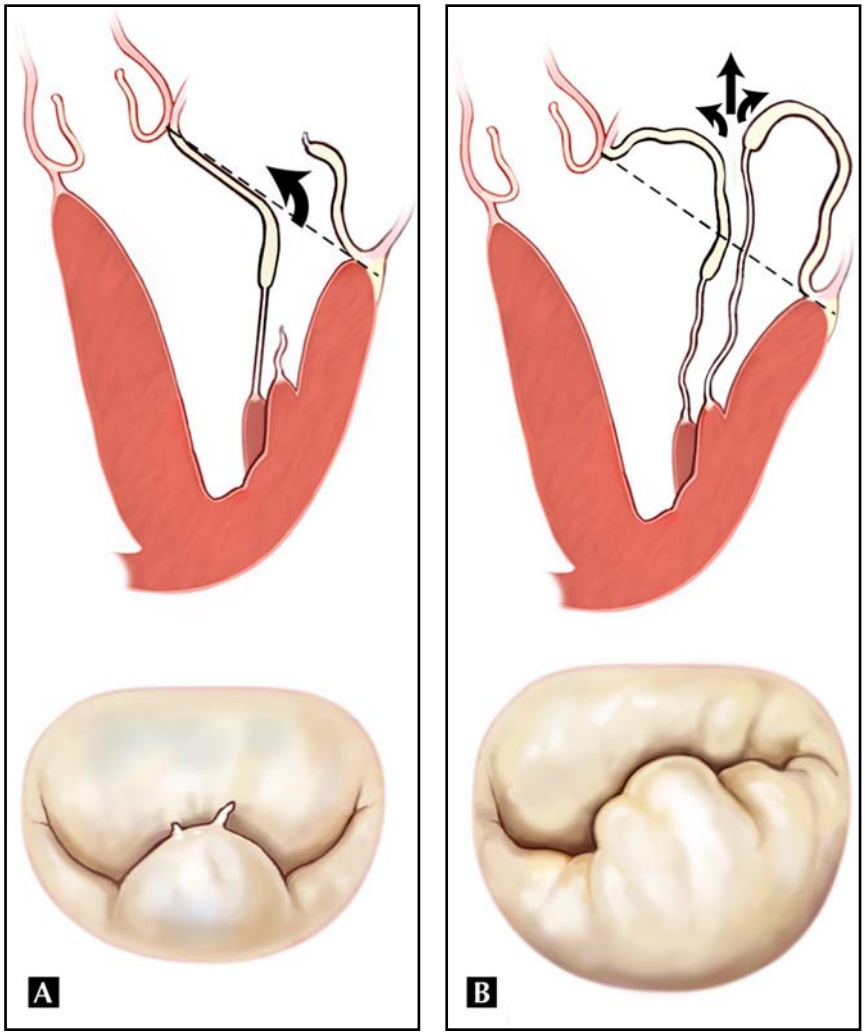

Figure 2. Echocardiographic differentiation of Carpentier type II dysfunction. **A,** *Upper panel* shows chordal rupture due to fibroelastic deficiency with a flail leaflet segment but otherwise normal-sized leaflets resulting in posterior leaflet prolapse. The corresponding surgical view is shown in the *lower panel*. **B,** *Upper panel* shows chordal elongation resulting in posterior leaflet prolapse: leaflets are tall with excess leaflet tissue, typical of Barlow's disease; note that although the body of the anterior leaflet billows above the annular plane, the margin of the anterior leaflet does not prolapse. These tall leaflets place the patient at risk for systolic anterior motion after a repair. The corresponding surgical view is shown in the *lower panel*. *Arrows* indicate the regurgitant jet. *Dotted lines* represent the annular plane.

Echocardiographic Analysis of the Surgical Anatomy

Preoperative assessment

An accurate segmental analysis is the optimal approach to performing a thorough echocardiographic assessment of the mitral valve affected by a disease process. In such a manner, the mitral valve's various anatomical components can be assessed individually in a systematic manner, which will allow identification of all lesions and resulting dysfunctions and help determine the etiology in most circumstances. Transthoracic echocardiography is initially used and in some cases may provide a sufficiently thorough mitral valve assessment. However, transesophageal echocardiography (TEE) is currently the most valuable modality for mitral valve imaging and is generally regarded as a standard of care for the surgical assessment of mitral valve disease. When using two-dimensional TEE to diagnose mitral valve pathology, or judge the success of mitral valve repair, the operator needs to obtain multiple two-dimensional multiplanar tomographic views with and without color Doppler to fully characterize the mitral valve.

Assessing severity of mitral valve regurgitation

A prerequisite for undertaking surgery for isolated mitral valve regurgitation is the demonstration of severe regurgitation. Although severe regurgitation is demonstrable by qualitative assessment of the Doppler color flow, quantitative estimation of regurgitation is preferable because it is more accurate and robust and lends itself to standardization and reproducibility [8]. Quantitative assessment of mitral regurgitation avoids the subjective interpretation that may arise from qualitative assessments of a color flow. Quantitative echocardiographic variables, such as the effective regurgitant orifice (ERO) and the regurgitant volume, can be used to stratify asymptomatic patients according to risk of cardiovascular events (eg, it has been shown that patients with ERO > 40 mm^2 have a better survival with mitral repair compared with watchful waiting) [9••]. A definition of severe regurgitation should be based on quantitative and anatomic criteria; such criteria are detailed in published guidelines [8].

A semiquantitative grading scale of 0 to 4+ is often used and offers the clinicians and researchers the simplicity

of describing regurgitation as a simple integer. However, definitions of these grades do vary among different echocardiographers, and several patients graded as 2+ or 3+ mitral regurgitation will actually be found to meet criteria for severe regurgitation if measured quantitatively [10]. Also, from a research perspective, it should be emphasized that mitral regurgitation grade is not a quantitative variable and should not be presented or analyzed as such, as is often the case [11•].

Examination of the mitral valve using two-dimensional TEE

The systematic examination consists of four standard mid-esophageal views (four-chamber, bicommissural, two-chamber, and long-axis views) and the transgastric basal short-axis view. The classification of the mitral valve scallops in any given plane (Fig. 1) may vary among patients according to individual anatomy. Accordingly, orientation to internal landmarks (ie, mitral valve commissures) is paramount to enhance the accuracy of the scallop's diagnosis.

Mid-esophageal four-chamber view

This view is obtained by advancing and slightly retroflexing the endoscope tip to the mid-esophageal position (~ 30–35 cm from the incisors) to direct the imaging sector toward the left ventricular apex. With the transducer at 0°, the imaging sector transects the coaptation line perpendicularly, showing both commissures (posteromedial and anterolateral commissures) and the middle scallop of the anterior and posterior mitral valve leaflets (A2 and P2). With the transducer slightly rotated to 20°, the imaging plane transects the coaptation line more obliquely, passing through A2, A1/P1.

Mid-esophageal bicommissural view

This view is obtained by rotating the probe to 55° from the mid-esophageal four-chamber view. The imaging plane passes through the intercommissural plane, transecting both leaflets to view both commissures, the medial scallop (P3) on the left, the middle region of the anterior leaflet (A2) in the center, and the lateral scallop of the posterior leaflet (P1) on the right. In this view, both papillary muscles are usually visible, whereas the left atrial appendage is not. This view helps identify the regurgitating segment. A regurgitation jet arising from the left coaptation point indicates involvement of P3/A3, whereas a jet arising from the right suggests involvement of A1/P1. The height of the P1 and P3 segments should be measured in this view. This height measurement is important in predicting complexity of mitral repair—where the height of either segment is more than 1.5 cm, there is a preponderance toward postoperative systolic anterior motion of the anterior leaflet (SAM) [12–14]; thus, such a finding should trigger the cardiologist to refer the patient to a mitral repair subspecialist because a successful repair may require advanced techniques (eg, sliding leaflet plasty).

Occurrence of postoperative SAM after attempted mitral repair remains a prevalent reason in nonspecialist settings for intraoperative change to mitral valve replacement in patients scheduled for degenerative mitral valve repair. However, using advanced surgical techniques, these valves are easily repairable, without occurrence of SAM, by mitral subspecialists. Such unnecessary replacements can be minimized by echocardiographic measurement of posterior leaflet height and referral to a mitral repair reference center if the P1 or P3 height is greater than 1.5 cm.

Mid-esophageal two-chamber view

This view is obtained by further electronically rotating the transducer to an angle of 90°. In this view, P3 is on the left of the image, and all three segments of the anterior leaflet (A1, A2, A3) are on the right. This provides a good view of the entire anterior mitral valve leaflet and the posteromedial (P3/A3) coaptation segments.

Mid-esophageal long-axis view

This view is obtained by further rotating the transducer to 120°. In this view, the operator should visualize the mitral and aortic valves but neither papillary muscle. The imaging plane cuts the coaptation line perpendicularly, transecting P2 (on the left) and A2 (on the right). This view is relevant because the cut plane transects P2, the scallop most frequently affected in degenerative disease.

Transgastric basal short-axis view

This view is obtained from the fundus of the stomach (35–40 cm from the incisors) by retroflexing the probe. This view displays all six segments of the mitral valve, with P3 being the closest to the apex of the sector. The anterolateral commissure is the one farthest away from the transducer. This view is comparatively difficult to obtain in a consistent manner.

Examination of the mitral valve using real-time 3D TEE

The recent development of a 3D fully sampled matrix-array TEE (3D-MTEE) transducer (Philips Medical Systems, Andover, MA) allows real-time acquisition and online display of (3D) images of the mitral valve and ventricle. To obtain the surgical view of the mitral valve (ie, the view the surgeon has when positioned on the patient's right side when examining the mitral valve through the opened left atrium), the valve is best imaged in the 3D zoom mode. This mode displays a small, magnified pyramidal volume of the mitral valve, which may vary from a 20° × 20° up to 90° × 90° depending on the density setting, resulting in superior quality volume rendered images of mitral valve apparatus, including the anterior and posterior leaflets, annulus, commissures, and subvalvular structures (Fig. 1). Similar to the previously used 3D TEE acquisition methods, mitral valve displays from atrial and ventricular perspectives are unique to 3D imaging; however, what distinguishes 3D-MTEE from rotational 3D acquisition is the consistency of superb

quality of the mitral valve, devoid of rotational artifacts. It is anticipated that with the ability of real-time acquisition, online adjustments of rendering, and cropping capabilities, this modality will be used routinely in the perioperative planning of mitral valve surgery.

Post-Repair Assessment

Immediately after mitral valve repair, several important aspects of the final valve anatomy must be addressed by the imager. The most critical aspect of postoperative valve analysis is to confirm the absence of significant residual mitral regurgitation. The depth of coaptation should be documented and be at least 5 mm in a two-dimensional long-axis view to ensure adequacy of coaptation. It is important to perform a segmental assessment to rule out regurgitation along any aspect of the coaptation surface, including the commissures. If a residual leak is identified, the lesions responsible and the residual dysfunction should be sought. Common causes of a residual leak include uncorrected segmental prolapse or restriction, a residual restricted leaflet indentation, an incorrectly sized or positioned ring that distorts the coaptation zone, a perforation of the leaflet from an annuloplasty suture, or a defect in a leaflet closure line. Any significant degree of mitral valve regurgitation (other than trivial to mild regurgitation) should prompt a return to cardiopulmonary bypass and valve re-exploration to correct residual or new defects. This strategy is imperative to avoid a heightened risk of reoperation in the patient early during follow-up, as there is a correlation between residual mitral regurgitation and early reoperation.

SAM is almost unique to mitral valve repair, but rarely also occurs in a primary setting, and results from a mismatch of annular septolateral dimension and residual combined leaflet height. Typically, the margin of the anterior leaflet is displaced into the outflow tract, causing a significant outflow tract gradient and varying degrees of mitral insufficiency. The best echocardiographic view to interrogate this is the mid-esophageal long-axis view. In some circumstances, volume loading and increasing the ventricular afterload repositions the anterior leaflet, minimizing the outflow tract gradient and valve regurgitation. If such maneuvers are not successful, valve re-exploration with placement of a larger ring, leaflet height shortening, or even valve replacement is required.

The Echocardiographer's Role in Mitral Surgery

Several aspects of the echocardiographic study aid the surgical assessment of mitral valve regurgitation. Pre-referral echocardiographic assessment plays a pivotal role in directing patients, cardiologists, and surgeons toward the optimum therapy. Beyond defining the degree of mitral regurgitation and its impact on left ventricular function and dimensions, a careful echocardiographic assessment can also provide a "road map for the repair strategy" and appropriate matching of surgical expertise to degenerative valve complexity. It is becoming increasingly clear that multisegment disease with excess tissue, typical of Barlow's disease, is most appropriately referred to "mitral super-specialists" to optimize the repair rate in this most complex subgroup of degenerative mitral valve regurgitation [15•,16]. The echocardiogram has a critical role in the management algorithm for degenerative mitral disease because it defines parameters for guideline intervention and also serves as the gatekeeper for the patient's access to appropriate level surgical expertise required for a mitral valve repair. Many patients receive unnecessary valve replacements because the echocardiogram has not been considered in surgical referral and operative planning. Given that current national repair rates generally approximate less than 60% of operated patients, despite guideline recommendations for valve reconstruction in all degenerative valves when feasible [17•], it is clear that use of preoperative echocardiography to match cases to the appropriate level of surgical expertise required to effect a repair is being underused. By systematic documentation of segmental echocardiographic findings, it is easy to predict whether the repair would be simple or one that requires specialized surgical techniques. For example, whereas an isolated P2 prolapse secondary to a chordal rupture from fibroelastic deficiency can be repaired with high probability by most cardiac surgeons, a posterior leaflet prolapse with tall leaflets secondary to Barlow's disease can only be consistently repaired in a mitral valve reference center [16]. The intraoperative control of the mitral procedure is no less important a role. The echocardiographer provides a clear demonstration of the lesions and dysfunctions that must be confirmed by surgical analysis in the operating room and also "controls" the immediate result of the valve repair, guiding the surgeon to accept the result or return to cardiopulmonary bypass to correct a residual concern. Through precise classification of mitral valve disease before valve repair, the echocardiographer will also facilitate robust outcomes studies that will clarify the long-term efficacy of specific reconstructive procedures and the risk factors for recurrence of mitral valve regurgitation, as the outcome of repair procedures is related to the original lesions and dysfunction treated [11•,18•].

Conclusions

Systematic echocardiographic assessment and documentation is essential for the triage of patients to medical or surgical therapy and should guide the decision on referral strategy to a general cardiac surgeon or a mitral valve reference surgeon. An accurate interpretation of the echocardiogram- and echocardiography-guided surgical referral should eliminate the scenario in which surgeons "discover" in the operating room that it is beyond their ability or understanding to repair a particular valve,

thus forcing an unnecessary valve replacement. Detailed echocardiographic assessment and use of these data to guide clinical decision making and surgical referral will help maximize mitral valve repair rates by facilitating preoperative surgical repair planning, as well as ensuring appropriate referral of patients to surgeons most likely to achieve a repair for the specific conditions of etiology, lesions, and dysfunctions seen on echocardiography. Although our group is among the first to emphasize this critical role of echocardiography in expertise-based referral of patients for surgical repair, we believe that future guidelines will endorse this approach as critical to maximizing the likelihood of a durable repair in the individual patient and also as the strategy most likely to make mechanical valve replacement for degenerative disease a rare and historical procedure.

Disclosures

Dr. David H. Adams is a consultant and inventor, and receives royalties from Edwards Lifesciences. Dr. Lissa Sugeng is on the speaker's bureau for Philips Medical Systems and receives software support from TomTec Imaging Systems. Dr. Roberto M. Lang is on the advisory board of Philips Medical Systems and receives grant support from TomTec Imaging Systems and Point Biomedical Corp.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- • Of importance
- •• Of major importance

1. Carpentier A: **Cardiac valve surgery—the "French correction".** *J Thorac Cardiovasc Surg* 1983, **86:**323–337.
2. Rodriguez F, Langer F, Harrington KB, et al.: **Importance of mitral valve second-order chordae for left ventricular geometry, wall thickening mechanics, and global systolic function.** *Circulation* 2004, **110:**II115–II122.
3. Rodriguez F, Langer F, Harrington KB, et al.: **Effect of cutting second-order chordae on in-vivo anterior mitral leaflet compound curvature.** *J Heart Valve Dis* 2005, **14:**592–601.
4. Dreyfus GD, Bahrami T, Alayle N, et al.: **Repair of anterior leaflet prolapse by papillary muscle repositioning: a new surgical option.** *Ann Thorac Surg* 2001, **71:**1464–1470.
5. Carpentier A, Chauvaud S, Fabiani JN, et al.: **Reconstructive surgery of mitral valve incompetence: ten-year appraisal.** *J Thorac Cardiovasc Surg* 1980, **79:**338–348.
6. Carpentier AF, Lessana A, Relland JY, et al.: **The "physio-ring": an advanced concept in mitral valve annuloplasty.** *Ann Thorac Surg* 1995, **60:**1177–1185.
7.• Anyanwu AC, Adams DH: **Etiologic classification of degenerative mitral valve disease: Barlow's disease and fibroelastic deficiency.** *Semin Thorac Cardiovasc Surg* 2007, **19:**90–96.

A review of the differentiating features between the major etiologies of degenerative mitral regurgitation.

8. Zoghbi WA, Enriquez-Sarano M, Foster E, et al.: **Recommendations for evaluation of the severity of native valvular regurgitation with two-dimensional and Doppler echocardiography.** *J Am Soc Echocardiogr* 2003, **16:**777–802.
9.•• Enriquez-Sarano M, Avierinos JF, Messika-Zeitoun D, et al.: **Quantitative determinants of the outcome of asymptomatic mitral regurgitation.** *N Engl J Med* 2005, **352:**875–883.

Quantitatively defined severe mitral regurgitation shown to reduce long-term survival in asymptomatic patients.

10. Pu M, Thomas JD, Vandervoort PM, et al.: **Comparison of quantitative and semiquantitative methods for assessing mitral regurgitation by transesophageal echocardiography.** *Am J Cardiol* 2001, **87:**66–70.
11.• Adams DH, Anyanwu A: **Pitfalls and limitations in measuring and interpreting the outcomes of mitral valve repair.** *J Thorac Cardiovasc Surg* 2006, **131:**523–529.

Discusses the flaws in the mitral valve repair literature and approaches to improving quality of outcome data.

12. Maslow AD, Regan MM, Haering JM, et al.: **Echocardiographic predictors of left ventricular outflow tract obstruction and systolic anterior motion of the mitral valve after mitral valve reconstruction for myxomatous valve disease.** *J Am Coll Cardiol* 1999, **34:**2096–2104.
13. Gillinov AM, Cosgrove DM III: **Modified sliding leaflet technique for repair of the mitral valve.** *Ann Thorac Surg* 1999, **68:**2356–2357.
14. Maslow AD, Singh A: **Mitral valve repair: to slide or not to slide-precardiopulmonary bypass echocardiogram examination.** *J Cardiothorac Vasc Anesth* 2006, **20:**842–846.
15• Bridgewater B, Hooper T, Munsch C, et al.: **Mitral repair best practice: proposed standards.** *Heart* 2006, **92:**939–944.

Consensus statement suggesting the need for regulation of mitral valve surgery and the role for specialist mitral valve surgeons.

16. Adams DH, Anyanwu AC: **The cardiologist's role in increasing the rate of mitral valve repair in degenerative disease.** *Curr Opin Cardiol* 2008, **23:**105–110.
17.• Bonow RO, Carabello BA, Kanu C, et al.: **ACC/AHA 2006 guidelines for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines (writing committee to revise the 1998 Guidelines for the Management of Patients With Valvular Heart Disease): developed in collaboration with the Society of Cardiovascular Anesthesiologists: endorsed by the Society for Cardiovascular Angiography and Interventions and the Society of Thoracic Surgeons.** *Circulation* 2006, **114:**e84–e231.

Practice guidelines for managing patients with valvular heart disease.

18• Flameng W, Meuris B, Herijgers P, Herregods MC: **Durability of mitral valve repair in Barlow disease versus fibroelastic deficiency.** *J Thorac Cardiovasc Surg* 2008, **135:**274–282.

Long-term study suggesting differences in outcome after degenerative mitral valve repair may be linked to etiology.