Mitral Valve Disease

By John Ofenloch, MD, Andrew Sherman, MD, and Joshua Rovin, MD

Mitral valve disease is one of the most prevalent acquired valvular heart disorders. Typically, mitral valve disease is classified as mitral regurgitation or mitral stenosis. Mitral regurgitation has many causes including mitral valve prolapse, chordal rupture, leaflet fibrosis with restriction, and dilated cardiomyopathy with heart failure; while mitral stenosis is generally a sequela of rheumatic fever (group A streptococcal tonsillo-pharyngitis) in childhood. Treatment for mitral valve disease includes optimal medical therapy, surgical valve repair or replacement, and more recently less invasive catheter-based repair and replacement techniques. Unfortunately, mitral valve disease left untreated inevitably leads to heart failure and death. Treatment guidelines are evolving to encourage early referral for surgical therapy in severe mitral regurgitation.

Mitral Stenosis

Patients with mitral stenosis typically present more than 20 years after an initial episode of rheumatic fever. A definite clinical history of rheumatic fever can be obtained in 50-60 percent of patients, and women are affected more often than men by a 2:1 or 3:1 ratio.

The slow effects of rheumatic fever result in progressive thickening, scarring and calcification of the mitral valve leaflets and chordae. Fusion of the commissures and chordae decreases the mobility of the leaflets and restricts the size of the mitral orifice. The narrowing of the valve results in an increased pressure gradient across the valve in diastole leading to increased left atrial and pulmonary venous pressures. Left ventricular output and stroke volume are diminished in mitral stenosis, particularly in tachycardic patients.

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Elevated pulmonary pressures cause pulmonary congestion and edema typically manifested by exertional dyspnea, fatigue and atrial arrhythmias. Physical findings include an opening snap and mid-diastolic rumble. Medical treatment is aimed at alleviating pulmonary congestion with diuretics, controlling atrial fibrillation and anticoagulation to prevent embolic events. In general, surgical treatment is required, as medical therapy does not alter the mechanics of the valvular stenosis. Surgical options include mitral valve balloon commissurotomy, surgical mitral commissurotomy and mitral valve replacement. Mitral valve repair is generally not possible in these patients and commissurotomy remains only a palliative procedure or a bridge to further, more definitive, intervention. Mitral valve replacement is the best option to correct mitral stenosis.

Mitral Regurgitation

Patients with mitral regurgitation are typically classified as having structural (degenerative) or functional regurgitation. Treatment strategies and options are different for each category of mitral valve disease. The mitral valve apparatus is comprised of two leaflets, multiple chordae tendineae, and two papillary muscles that all interact dynamically during systole and diastole. While mitral valve prolapse is quite prevalent, only 5-10 percent of these patients develop significant mitral regurgitation. Importantly, 2 percent of the population overall suffers from severe mitral regurgitation.

Structural mitral regurgitation results from multiple valvular pathologies but most cases fall under the category of degenerative mitral valve disease. Within this category are two distinct etiologies. One, commonly referred to as Barlow's disease, involves myxomatous changes to the valve that results in excess tissue, thickened and tall leaflets with chordal elongation.

Fibroelastic deficiency is the second subtype that, in contradistinction to Barlow's disease, is due to connective tissue deficiency, rather than excess, and results in thin, inadequate leaflet tissue.

Categorizing degenerative mitral valve disease into one of these two entities is important because key aspects of surgical repair or replacement vary depending on this distinction. Fewer cases of mitral regurgitation are the result of rheumatic fever, infectious endocarditis, mitral annular calcification, congenital anomalies or collagen-vascular disorders.

Functional mitral regurgitation describes a process secondary to coronary artery disease or dilated cardiomyopathy. It isn't a primary pathologic problem with the valve leaflets, but rather a problem with the left ventricle. Ischemia due to coronary artery disease or myocardial infarction may result in regional wall motion abnormalities, which affect the interaction of the ventricle, papillary muscles, chordal structure and valve leaflets. Acute myocardial infarction may result in papillary muscle rupture, an acute structural abnormality that often progresses rapidly to acute heart failure. Generally, enlargement or dilation of the left ventricle distorts the valve apparatus inhibiting leaflet coaptation and leading to regurgitation.

Severe regurgitation reduces the impedance to left ventricular outflow with the volume of blood ejected into the left atrium increasing the left atrial pressure and reducing forward cardiac output. Chronic severe mitral regurgitation may not cause symptoms if the regurgitant load is well tolerated by compensatory atrial and ventricular changes. Ultimately, patients become symptomatic, developing shortness of breath, dyspnea on exertion, fatigue, orthopnea, paroxysmal nocturnal dyspnea, edema, palpitations and atrial fibrillation. The characteristic murmur of mitral regurgitation is holosystolic and best heard at the cardiac apex.
Treatment Options

Asymptomatic patients with mild or moderate mitral regurgitation do not require any specific medical or surgical treatment provided they remain normotensive with normal left ventricular function. Surgical treatment in patients with severe structural mitral regurgitation is indicated at the onset of any symptoms of heart failure. Stress echocardiograms or a six-minute walk test may be helpful to determine the presence of symptoms in less active patients. Severe mitral regurgitation is best quantified with the use of echocardiography. The following indices confirm the severity including:

1. EROA (effective regurgitant orifice area) > 0.40 cm²
2. Mitral regurgitant volume > 60 cc
3. Regurgitant fraction > 50%
4. PISA > 0.7 cm

Surgical treatment of asymptomatic patients with severe mitral regurgitation has undergone a transition to addressing patients sooner in their process rather than waiting for symptoms to develop. In fact, the ACC/AHA Valvular Guidelines have recommended surgical intervention in asymptomatic patients in the presence of any of the following criteria:

1. Left ventricular dysfunction (ejection fraction <= 60%)
2. Left ventricular dilation (LVESD >= 40 mm)
3. Atrial fibrillation
4. Elevated resting pulmonary artery pressures

There are also recent studies suggesting that even asymptomatic patients with severe structural mitral regurgitation without any of the above criteria should undergo surgery, provided that the center ensures a high likelihood of valve repair with an extremely low rate of complications. The supportive evidence demonstrates that repairing regurgitant valves early can decrease the likelihood of future heart failure and mortality. Over the past three years in the BayCare system, over 75 isolated mitral valve repairs have been performed with only a single mortality.

The reason that surgical and cardiology societies have taken a more aggressive stance toward early intervention on patients with severe mitral regurgitation is that surgical correction should be undertaken prior to a potentially irreversible decline in left ventricular function. The degree of deterioration in LV contractility is an important determinant in the patient’s prognosis.

In patients with ischemic or dilated cardiomyopathy with EF < 30 percent, mitral regurgitation carries a poor prognosis. In these patients, guideline directed medical therapy for heart failure is appropriate, including angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers, beta blockers and/or aldosterone antagonists. Biventricular pacing has been shown to decrease mitral regurgitation in cardiomyopathy as well. Medical therapy has no role to specifically treat structural mitral regurgitation but is the mainstay for functional mitral regurgitation.

Open surgical repair or replacement: These procedures are recommended for asymptomatic patients as well as selected asymptomatic patients as described above. Mitral valve repair is generally preferable to mitral valve replacement and provides enhanced survival in most patients, provided a durable repair can be performed. Depending on the etiology of the structural valve problem, a variety of repair techniques have been developed including resection of redundant tissue, placing neo-chords where appropriate, and providing support with a variety of prosthetic annuloplasty rings.

Several surgical approaches are available for mitral valve surgery depending on the scope of operation required. Conventional sternotomy allows excellent exposure and access to the mitral valve as well as other heart valves and coronary arteries. Mortality is typically less than one percent with hospital stays of four to five days. Return to normal activity generally takes four to six weeks.

Minimally invasive approach: Limited access incision in the right chest tailored for operations requiring mitral valve surgery only. Mortality is typically less than 1 percent with hospital stays of three to four days. Return to normal activities can occur in two to four weeks.

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Transcatheter approaches to mitral valve repair or replacement: These procedures are currently reserved for patients at higher risk for open surgical procedures. There are more than 20 new devices in development for less invasive treatment of mitral valve disease. MitraClip catheter-based edge-to-edge mitral valve repair is the only FDA-approved transcatheter device for the treatment of mitral regurgitation. This procedure is performed percutaneously via the femoral vein and doesn’t require a sternal, chest or groin incision. Thorough evaluation of mitral valve anatomy is required with a transesophageal echocardiogram (TEE) to determine the feasibility of transcatheter techniques. Within the BayCare system, the procedure is performed by the Heart Team and typically takes less than one hour. MitraClip mortality is less than 1 percent and the hospital stay is usually one night. Return to normal activities is almost immediate.

Extensive innovation in the field of transcatheter mitral valve techniques has occurred in the past several years and there are many developments under clinical trials. The team-based approach to designing the appropriate therapy for patients with mitral disease will be essential to maximize patient outcomes and preserve ventricular function, especially in patients with multi-valvular disease.

Comprehensive mitral valve surgery programs within BayCare are offered at the Bostick Heart Center at Winter Haven Hospital, St. Joseph’s Hospital Heart Institute (Tampa), and the Morgan Heart Hospital at Morton Plant (Clearwater). Each of these institutions provides excellent complex mitral valve surgery in addition to treatment for atrial fibrillation (Maze procedure).

MitraClip therapy is presently offered at St. Joseph’s Hospital in Tampa and Morton Plant Hospital in Clearwater. Additionally, the Structural Heart and Advanced Valve Care Center at Morton Plant Hospital is one of 10 hospitals in the United States that is participating in the feasibility trial for the Abbott Tendyne Transcatheter Mitral Valve (Replacement). Many exciting options are being developed by industry research and development programs.

Physicians and heart teams at all BayCare hospitals collaborate to ensure that patients have access to all available options for treatment of mitral valve disease. Screening of patients for the Tendyne mitral valve replacement study is currently underway at Morton Plant Hospital.