Device Therapy for Prevention of Sudden Death

ICDs have demonstrated efficacy in the primary and secondary prevention of SCD through their treatment, not prevention, of VT/VF with defibrillation. Patients with sustained ventricular arrhythmias (>30 seconds) or cardiac arrest without a reversible cause have a class 1 recommendation for secondary-prevention ICD placement. ICD placement is recommended for the primary prevention of SCD in patients with ischemic or nonischemic cardiomyopathy, ejection fraction less than 35%, and New York Heart Association functional class II or III heart failure. Patients with heart failure and interventricular conduction defects (predominantly left bundle branch block) often benefit from cardiac resynchronization therapy or cardiac resynchronization therapy in combination with a defibrillator (see Heart Failure).

In the past, ICDs were implanted almost exclusively using a transvenous approach. New techniques allow for implantation of defibrillators in the lateral chest at the midaxillary line adjacent to the heart with tunneling of the lead under the skin next to the sternum. Subcutaneous defibrillators have several advantages, including reduced risk for device infection.

Infection is a major and chronic risk of implanted cardiac devices. Pacemaker and defibrillator infections, even of the pocket alone, must be managed aggressively to reduce morbidity and mortality. However, device infection presentation may be insidious and underwhelming, potentially limited to only pain or erythema over the pocket. Nevertheless, any patient suspected of having a cardiac device infection should be referred urgently for specialist evaluation. Empiric antibiotics alone (without blood cultures) may cloud or delay diagnosis, and diagnostic aspiration of the device pocket is never indicated because of the risk for introducing infection in an uninfected pocket. Effective treatment of cardiac device infection usually includes complete extraction of all hardware, debridement of the pocket, sustained antibiotic therapy, and re-implantation at a new location after infection has been eradicated.

KEY POINTS

- Implantable cardioverter-defibrillators are effective for primary and secondary prevention of sudden cardiac death.
- Infection of cardiac implanted devices may present insidiously but requires urgent and specialized evaluation, often necessitating complete hardware removal for a durable cure.

Valvular Heart Disease

General Principles

Valvular heart disease (VHD) involves cardiac dysfunction due to structural or functional valve abnormalities resulting from failure of the valves to either competently close (regurgitation) or effectively open (stenosis). VHD affects approximately 20 million persons in the United States. Although there are congenital forms, VHD is largely age dependent, with a prevalence of 3% to 6% in persons aged 65 years or older.

Many heart valve lesions progress slowly, causing patients to limit their activity unconsciously in response; therefore, a careful history and detailed physical examination are essential. Exertional dyspnea is the most common symptom. Depending on the lesion and severity, other symptoms include angina, syncope, palpitations, lower extremity edema, and increasing girth (ascites). Typical physical examination findings for valvular and other cardiac lesions are described in **Table 21**. Twelve-lead ECG, chest radiography, and transthoracic echocardiography (TTE) are the essential tests used to evaluate VHD.

To facilitate the timing of monitoring and intervention, VHD is classified into four stages (A through D), which consider risk factors, presence of symptoms, lesion severity, ventricular response to the volume or pressure overload caused by the lesion, effect on the pulmonary or systemic circulation, and heart rhythm changes (**Table 22**). Surveillance intervals for echocardiographic evaluation based on disease severity are listed in **Table 23** on page 64.

Medical therapy, although often effective for symptom palliation, has not been shown to prevent VHD progression or improve long-term survival in patients with VHD. Surgery, however, can be a life-saving intervention in select patients, and surgical risk calculation is a key component of the patient evaluation. Risk calculation involves assessment of the patient's age, morbidities, frailty, and impediments specific to the procedure under consideration (e.g., previous chest irradiation for a sternotomy approach). Risk calculators derived from national databases can assist in estimating risk for morbidity and mortality for surgical valve procedures. One such calculator, the Society of Thoracic Surgeons Adult Cardiac Surgery Risk Calculator, is available at http://riskcalc.sts.org/stswebriskcalc. Although risk calculators contain many data inputs, frailty and some other important patient and procedural characteristics are not factored into the calculations. Therefore, a comprehensive approach is required for determining patient surgical risk and candidacy. Frailty, which is variably defined as a geriatric syndrome of decline in several physiologic systems and processes, portends an increased risk for mortality in patients undergoing surgery and can be measured preoperatively (see MKSAP 19 General Internal Medicine 1).

For all patients in whom surgical or interventional therapy is being considered, a multidisciplinary approach with a heart team consisting of a cardiologist, a surgeon, and an interventional cardiologist is recommended. Evaluations in centers with specialized expertise in VHD (e.g., a Heart Valve Center of Excellence) is also advised for patients in whom intervention is being considered when there are no symptoms, multiple or complex morbidities are present, or surgical valve repair is favored over valve replacement.

TABLE 21. Valvular and Other Cardiac Lesions and Their Associated Examination Findings

Cardiac Condition	Characteristic Murmur	Location	Radiation	Associated Findings	Severity and Pitfalls
Aortic stenosis	Midsystolic; crescendo-decrescendo	RUSB	Right clavicle, carotid; apex	Enlarged, nondisplaced apical impulse; S_4; bicuspid valve without calcification will have systolic ejection click followed by murmur	Severe aortic stenosis findings may include decreased A_2; high-pitched, late-peaking murmur; diminished and delayed carotid upstroke; radiation of murmur to both clavicles and carotids Radiation of murmur down the descending thoracic aorta may mimic mitral regurgitation
Aortic regurgitation	Diastolic; decrescendo	LLSB (valvular) or RLSB (dilated aorta) (best heard sitting and leaning forward)	None	Enlarged, displaced apical impulse; S_3 or S_4; increased pulse pressure; bounding carotid and peripheral pulses	Acute severe regurgitation murmur may be masked by tachycardia and short duration of murmur Severity in chronic regurgitation is difficult to assess by auscultation
Mitral stenosis	Diastolic; low-pitched, decrescendo	Apex (best heard in left lateral decubitus position)	None	Loud S_1; tapping apex beat; opening snap after S_2 if leaflets mobile; irregular pulse if atrial fibrillation present	Interval between S_2 and opening snap is short in severe mitral stenosis Intensity of murmur correlates with transvalvular gradient P_2 may be loud if pulmonary hypertension present
Mitral regurgitation	Systolic; holo-, mid-, or late systolic	Apex	Axilla or back; occasionally anteriorly to precordium	Systolic click in mitral valve prolapse; S_3; apical impulse hyperdynamic and may be displaced if dilated left ventricle; in mitral valve prolapse, Valsalva maneuver moves onset of clicks and murmur closer to S_1; handgrip maneuver increases murmur intensity	Acute severe regurgitation may have soft or no holosystolic murmur, mitral inflow rumble, or S_3
Tricuspid regurgitation	Holosystolic	LLSB	LUSB	Merged and prominent *c* and *v* waves in jugular venous pulse; murmur increases during inspiration	Right ventricular impulse below sternum Pulsatile, enlarged liver with possible ascites Murmur may be high-pitched if associated with severe pulmonary hypertension
Tricuspid stenosis	Diastolic; low-pitched, decrescendo; increased intensity during inspiration	LLSB	None	Elevated central venous pressure with prominent *a* wave, signs of venous congestion (hepatomegaly, ascites, edema)	Low-pitched frequency may be difficult to auscultate, especially at higher heart rate
Pulmonary valve stenosis	Systolic; crescendo-decrescendo	LUSB	Left clavicle	Pulmonic ejection click after S_1 (diminishes with inspiration)	Increased intensity of murmur with late peaking
Pulmonary valve regurgitation	Diastolic; decrescendo	LLSB	None	Loud P_2 if pulmonary hypertension present	Murmur may be minimal or absent if severe due to minimal difference in pulmonary artery and right ventricular diastolic pressures

(Continued on the next page)

TABLE 21. Valvular and Other Cardiac Lesions and Their Associated Examination Findings *(Continued)*

Cardiac Condition	Characteristic Murmur	Location	Radiation	Associated Findings	Severity and Pitfalls
Benign (innocent) flow murmur	Midsystolic; grade 1/6 or 2/6 in intensity	RUSB	None	Normal intensity of A_2; normal splitting of S_2; no radiation	May be present in conditions with increased flow (e.g., pregnancy, fever, anemia, hyperthyroidism)
Hypertrophic obstructive cardiomyopathy	Systolic; crescendo-decrescendo	LLSB	None	Enlarged, hyperdynamic apical impulse; bifid carotid impulse with delay; increased intensity during Valsalva maneuver or with squatting to standing	Murmur may not be present in nonobstructive hypertrophic cardiomyopathy
Atrial septal defect	Systolic; crescendo-decrescendo	RUSB	None	Fixed split S_2; right ventricular heave; rarely, tricuspid inflow murmur	May be associated with pulmonary hypertension with increased intensity of P_2, pulmonary valve regurgitation
Ventricular septal defect	Holosystolic	LLSB	None	Palpable thrill; murmur increases with handgrip maneuver	Murmur intensity and duration decrease as pulmonary hypertension develops (Eisenmenger syndrome) Cyanosis if Eisenmenger syndrome develops

A_2 = aortic component of S_2; LLSB = left lower sternal border; LUSB = left upper sternal border; P_2 = pulmonic component of S_2; RLSB = right lower sternal border; RUSB = right upper sternal border.

TABLE 22. Stages of Progression of Valvular Heart Disease

Stage	Definition	Description
A	At risk	Patients with risk factors for development of VHD
B	Progressive	Patients with progressive VHD (mild to moderate severity and asymptomatic)
C	Asymptomatic severe	Asymptomatic patients who have the criteria for severe VHD: C1: Asymptomatic patients with severe VHD in whom the left or right ventricle remains compensated C2: Asymptomatic patients with severe VHD, with decompensation of the left or right ventricle
D	Symptomatic severe	Patients who have developed symptoms as a result of VHD

VHD = valvular heart disease.

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KEY POINTS

- Many heart valve lesions progress slowly, causing patients to limit their activity unconsciously in response; therefore, a careful history and detailed physical examination are essential.

(Continued)

KEY POINTS *(continued)*

- Medical therapy, although often effective for symptom palliation, has not been shown to prevent disease progression or improve long-term survival in patients with valvular heart disease.
- For all patients with valvular heart disease in whom surgical or interventional therapy is being considered, a multidisciplinary approach with a heart team consisting of a cardiologist, a surgeon, and an interventional cardiologist is recommended.

Aortic Stenosis

Clinical Presentation and Evaluation

Aortic stenosis may be congenital, as in persons with a bicuspid aortic valve, or acquired. The most common cause is degeneration of the valve that occurs with aging (**Figure 30**); severe lesions occur in approximately 3% of persons aged 65 years or older. Other acquired causes include rheumatic disease and chest irradiation. Although rheumatic disease of the mitral valve frequently occurs in isolation, rheumatic aortic valve disease almost never occurs without mitral valve involvement. Chest irradiation (e.g., mantle therapy for non-Hodgkin lymphoma) commonly results in a combination of stenosis and regurgitation.

Aortic stenosis causes chronic pressure overload of the left ventricle (LV), leading to concentric LV hypertrophy and myocardial interstitial fibrosis. Diastolic dysfunction follows, with eventual systolic heart failure and pulmonary congestion. The disease typically progresses with a decrease in the aortic

fraction ≤55%) thought to be due to aortic regurgitation or for patients with severe aortic regurgitation who are undergoing other cardiac surgery. Surgical treatment of aortic regurgitation is reasonable in asymptomatic patients with severe AR, normal left ventricular function, and significant LV dilatation (end-systolic dimension >50 mm or indexed end-systolic dimension >25 mm/m^2). In patients with isolated severe aortic regurgitation who have indications for SAVR and are candidates for surgery, TAVI should not be performed.

Medical therapy, preferably with dihydropyridine calcium channel blockers, ACE inhibitors, or angiotensin receptor blockers (ARBs), is indicated in patients with chronic aortic regurgitation and concomitant hypertension. Therapy with ACE inhibitors or ARBs and β-blockers is reasonable in severe aortic regurgitation with symptoms or LV dysfunction when surgery is not an option.

Significant acute aortic regurgitation due to aortic dissection is a surgical emergency, requiring aortic dissection repair and aortic valve replacement or repair. For other acute causes, the indications for surgery depend on the regurgitation severity, presence of symptoms, and hemodynamic stability of the patient.

KEY POINTS

- Characteristic clinical findings of chronic aortic regurgitation include bounding peripheral pulses, displacement of the left ventricular apex, and a diastolic decrescendo murmur heard along the right or left sternal border.
- In chronic aortic regurgitation, surgery with traditional open aortic valve replacement is advised for patients with symptoms or left ventricular dysfunction, or who have severe asymptomatic aortic regurgitation and are undergoing other cardiac surgery; in symptomatic patients who are not surgical candidates, medical therapy is appropriate.
- Medical therapy with dihydropyridine calcium channel blockers, ACE inhibitors, or angiotensin receptor blockers is recommended for patients with chronic aortic regurgitation and concomitant hypertension.
- Emergent surgery is indicated for patients with acute aortic regurgitation due to aortic dissection.

Bicuspid Aortic Valve Disease

Bicuspid aortic valve disease affects approximately 1% to 2% of the general population. Bicuspid morphology leads to abnormal shear forces and predisposes to early degeneration of the valve, resulting in stenosis in most patients (up to 75%) (see Figure 30) and pure regurgitation in a small minority of patients (2%-10%). Patients with a bicuspid aortic valve typically present with an asymptomatic finding of a systolic ejection murmur in adolescence or young adulthood and gradually progress to severe disease in the fifth or sixth decade of life. More than one third of those older than 70 years with severe aortic stenosis have an underlying bicuspid valve.

Bicuspid valvulopathy is often accompanied by aortic abnormalities, independent of the severity of aortic stenosis or regurgitation, and may be associated with aneurysm, dissection, or coarctation. Therefore, in patients with a bicuspid aortic valve, the ascending aorta and aortic arch should be examined for aortopathy with TTE. CMR angiography or CT angiography is indicated when echocardiographic assessment is suboptimal. Lifelong serial imaging is indicated if abnormalities are detected. The imaging modality and frequency depend on several factors, including the nature (stenosis, regurgitation, or aneurysm) and severity of the abnormality, age of the patient, family history, and candidacy for surgery. Importantly, bicuspid aortic valve is a heritable abnormality, and first-degree relatives of patients with a bicuspid aortic valve and aortopathy may be considered for screening with echocardiography.

Management of bicuspid aortic valve disease is determined by the predominant lesion type (stenosis or regurgitation) and its severity. In patients with a bicuspid valve undergoing surgery for severe aortic stenosis or regurgitation, surgical repair of the ascending aorta is reasonable when the aortic dimension is 4.5 cm or greater. In the absence of surgical indications for a stenotic or regurgitant aortic valve, surgical repair of the ascending aorta or aortic sinuses is recommended when the aortic dimension is greater than 5.5 cm and may be reasonable when the dimension is greater than 5.0 cm with an additional risk factor for dissection (e.g., family history, rate of progression ≥0.5 cm/year).

No medical therapies slow aortic dilatation in patients with aortopathy and a bicuspid aortic valve. Blood pressure should be controlled in patients with concomitant hypertension.

KEY POINTS

- Bicuspid morphology predisposes to early degeneration of the aortic valve, resulting in stenosis in most patients and pure regurgitation in few patients.
- Patients with a bicuspid aortic valve typically present with an incidental systolic ejection murmur in adolescence or young adulthood and gradually progress to severe disease in the fifth or sixth decade of life.
- Management of bicuspid aortic valve disease follows the recommendations for the predominant valve lesion type (aortic stenosis or regurgitation) and its severity.

Mitral Stenosis

Clinical Presentation and Evaluation

The leading cause of mitral stenosis is rheumatic heart disease, which is more common in women than in men (4:1 ratio). Although relatively rare in the United States, rheumatic heart disease is frequent in populations with limited access to

treatment for streptococcal pharyngitis. Rheumatic heart disease results in fusion of the mitral commissures and, in more advanced forms, calcification of the valve and abnormalities in the subvalvular apparatus (**Figure 33**). Other causes of mitral stenosis are parachute mitral valve, chest irradiation, and severe mitral annular calcification. Mitral annular calcification is more common in the elderly and is associated with inflammatory disorders, peripheral artery disease, and chronic kidney disease.

The natural history of mitral stenosis is characterized by slow progression over decades, with gradual left atrial (LA) enlargement and preservation of LV function. Symptoms may arise from low cardiac output (fatigue), pulmonary congestion (dyspnea), and pulmonary hypertension with right-sided heart failure (lower extremity edema). Pregnancy, with the resulting increased blood volume and cardiac output, may also precipitate symptoms. Symptoms are typically exertional because exercise shortens diastolic filling time and increases the transvalvular flow and diastolic mitral gradient, leading to worsening of LA hypertension. Patients also can present with systemic embolization, atrial fibrillation, or, in severe cases, hemoptysis. Heart failure is the cause of death in approximately 60% of patients with mitral stenosis, with thromboembolism causing most others.

Clinical findings when the valve is pliable include a tapping LV impulse, a loud S_1, an increased pulmonic component of S_2, a diastolic opening snap, and a diastolic rumble or low-pitched murmur at the apex (see Table 21). Signs of pulmonary or systemic congestion may be present depending on lesion severity and the patient's volume status.

TTE is highly accurate for assessing disease severity, pulmonary pressures, and RV function as well as for identifying concomitant valvular lesions (see Table 23). Additional imaging studies or cardiac catheterization is rarely required for diagnosis. Severe mitral stenosis is defined by a mitral valve area of 1.5 cm^2 or less, which usually corresponds to a mean mitral gradient of more than 5 to 10 mm Hg at a normal heart rate. In patients with a discrepancy between the clinical and echocardiographic findings, exercise echocardiography or exercise testing during cardiac catheterization should be

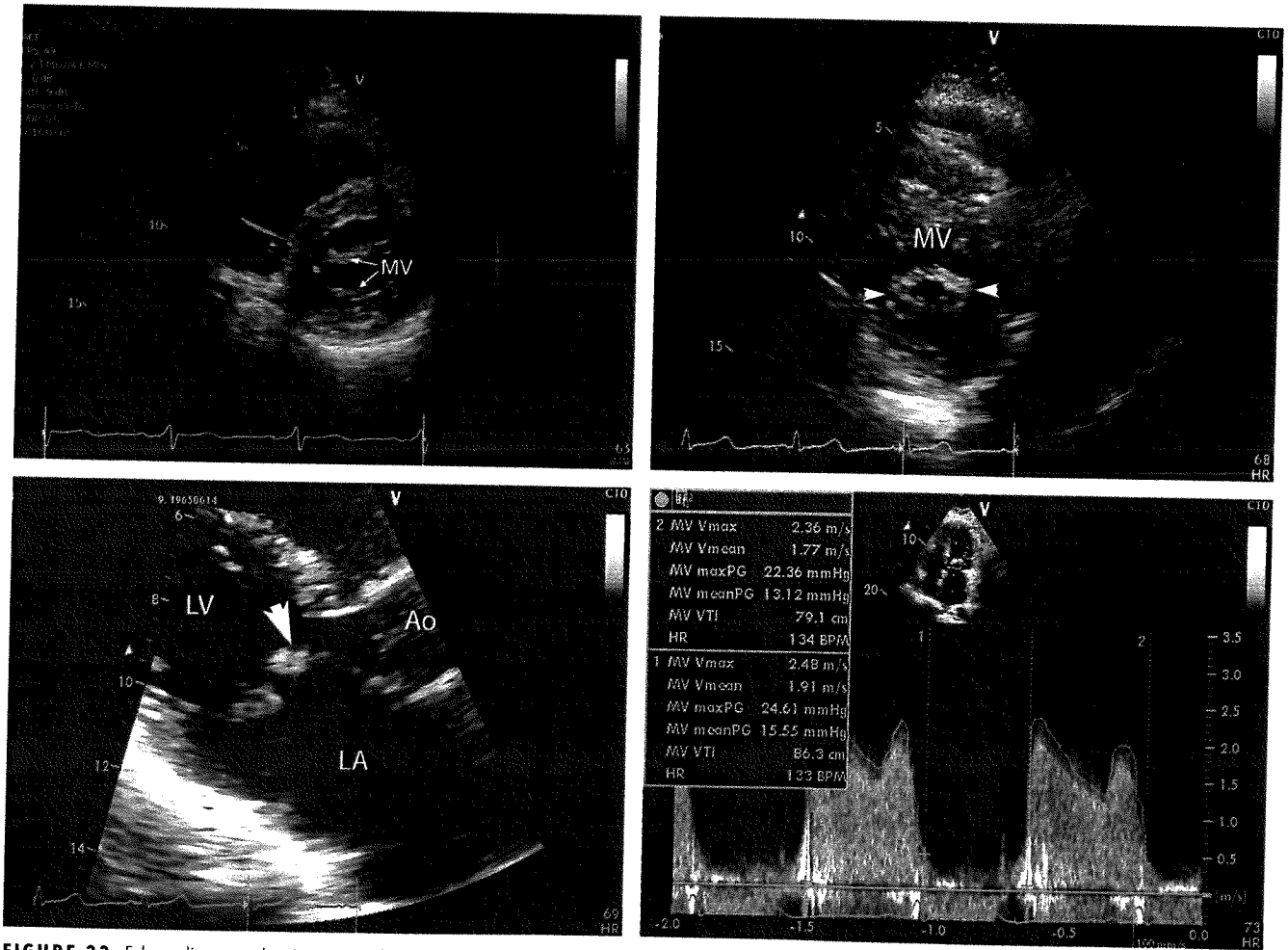

FIGURE 33. Echocardiograms showing a normal mitral valve (MV) (*top right panel*) and rheumatic mitral stenosis with commissural fusion (*arrowheads, top left panel*). In the bottom left panel, diastolic doming (*arrowhead*) is present with a "hockey stick" deformity from mitral stenosis. In the bottom right panel, a Doppler echocardiogram shows a mitral gradient of 13 mm Hg, consistent with severe stenosis. Ao = ascending aorta; LA = left atrium; LV = left ventricle.

pursued to assess the response of the mitral gradient and pulmonary pressures.

Management

Percutaneous balloon mitral commissurotomy (PBMC) is the procedure of choice for patients with significant rheumatic mitral stenosis when the valve is pliable and not severely calcified. PBMC is indicated for symptomatic patients with severe rheumatic mitral stenosis and favorable valve morphology and is reasonable in asymptomatic patients with severe rheumatic mitral stenosis and a pulmonary artery systolic pressure greater than 50 mm Hg. PBMC should not be performed in patients with LA thrombus or moderate or severe mitral regurgitation, both of which are optimally evaluated with TEE. In appropriately selected patients, the success rate with PBMC is 95%, and complications occur in fewer than 5% of patients. Mitral valve surgery is indicated in patients with severe mitral stenosis, New York Heart Association functional class III or IV symptoms, and a nonpliable valve and in asymptomatic patients with severe mitral stenosis undergoing concomitant cardiac surgery for another indication.

Nearly 50% of patients with mitral stenosis have atrial fibrillation, and without anticoagulation, these patients have a risk for thromboembolism of 20% to 25%. Patients with moderate to severe mitral stenosis and concomitant atrial fibrillation should receive a vitamin K antagonist such as warfarin, with a goal INR of 2.0 to 3.0. Anticoagulation is also indicated in patients with a history of LA thrombus or systemic embolization. Notably, clinical trials of direct-acting oral anticoagulants in atrial fibrillation excluded patients with moderate to severe mitral stenosis; therefore, the efficacy and safety of these agents have not been demonstrated in this population.

Because the mitral gradient is heavily dependent on transvalvular flow, medical therapy with negative chronotropic agents, diuretics, and long-acting nitrates can be effective for symptom palliation in patients who are not candidates for interventional or surgical therapy.

KEY POINTS

- In patients with mitral stenosis, transthoracic echocardiography is highly accurate for assessing disease severity, pulmonary pressures, and right ventricular function as well as identifying concomitant valvular lesions.
- Percutaneous balloon mitral commissurotomy is indicated for patients with symptomatic severe mitral stenosis and favorable valve morphology.
- Anticoagulation is recommended for patients with mitral stenosis and a history of atrial fibrillation, left atrial thrombus, or systemic embolization.

Mitral Regurgitation

Clinical Presentation and Evaluation

Mitral regurgitation may arise from dysfunction of any portion of the complex valve apparatus (leaflets, annulus, chordae, papillary muscles, or LV free walls) and may present acutely or chronically. Causes of acute mitral regurgitation include infective endocarditis, papillary muscle ischemia or rupture, trauma (e.g., injury from PBMC or blunt force), or degenerative disease with chordal rupture and flail leaflet. Chronic mitral regurgitation is classified as primary, involving any portion of the mitral annulus, or secondary, involving causes other than the annulus (e.g., ventricular dysfunction). Common causes of primary mitral regurgitation are mitral valve prolapse (also known as myxomatous or degenerative mitral valve disease), radiation therapy, rheumatic disease, and cleft mitral valve.

Mitral regurgitation results in volume overload with LV dilatation and LA hypertension, which may progress and cause pulmonary hypertension and RV failure. In acute mitral regurgitation, heart failure symptoms often occur abruptly because of insufficient time for adaptive chamber dilatation and, in some patients, can result in cardiogenic shock. The systolic murmur in acute mitral regurgitation may be brief because of the rapid equalization of LA and LV pressures, and echocardiography with color flow imaging can underestimate the severity of the regurgitation. Thus, when acute mitral regurgitation is suspected, comprehensive assessment to identify the potential causes should be pursued, and additional imaging with TEE should be considered.

Chronic primary mitral regurgitation is predominantly caused by mitral valve prolapse, which affects approximately 2% of the general population in the United States. Echocardiography in patients with chronic primary mitral regurgitation may show a range of abnormalities, including prolapse (**Figure 34**), gross degeneration of one or both leaflets (Barlow syndrome), or chordal rupture with flail leaflet. Barlow syndrome is more common in young adult patients. In patients who are relatively older, fibroelastic deficiency predominates and frequently results in chordal rupture. In patients with chronic secondary mitral regurgitation, ventricular dysfunction causes mitral regurgitation through papillary muscle displacement and tethering of the mitral leaflets, which impairs coaptation. The mitral valve apparatus is normal in patients with chronic secondary mitral regurgitation (**Figure 35**).

The physical examination in patients with chronic mitral regurgitation is notable for a blowing holosystolic murmur at the apex. In patients with mitral valve prolapse, one or more systolic clicks may precede the murmur, and variation in severity, preload, and afterload can lead to differences in murmur onset (holosystolic, midsystolic, or late systolic). In patients with LV dilatation, the apical impulse may be displaced laterally, and an S_3 may be audible, especially in patients with secondary mitral regurgitation due to LV dysfunction.

TTE readily and accurately assesses the severity of primary mitral regurgitation. Severe primary mitral regurgitation is defined by several parameters, the most common of which are an effective regurgitant orifice area of 0.4 cm^2 or larger, regurgitant volume of 60 mL or more, and vena contracta of

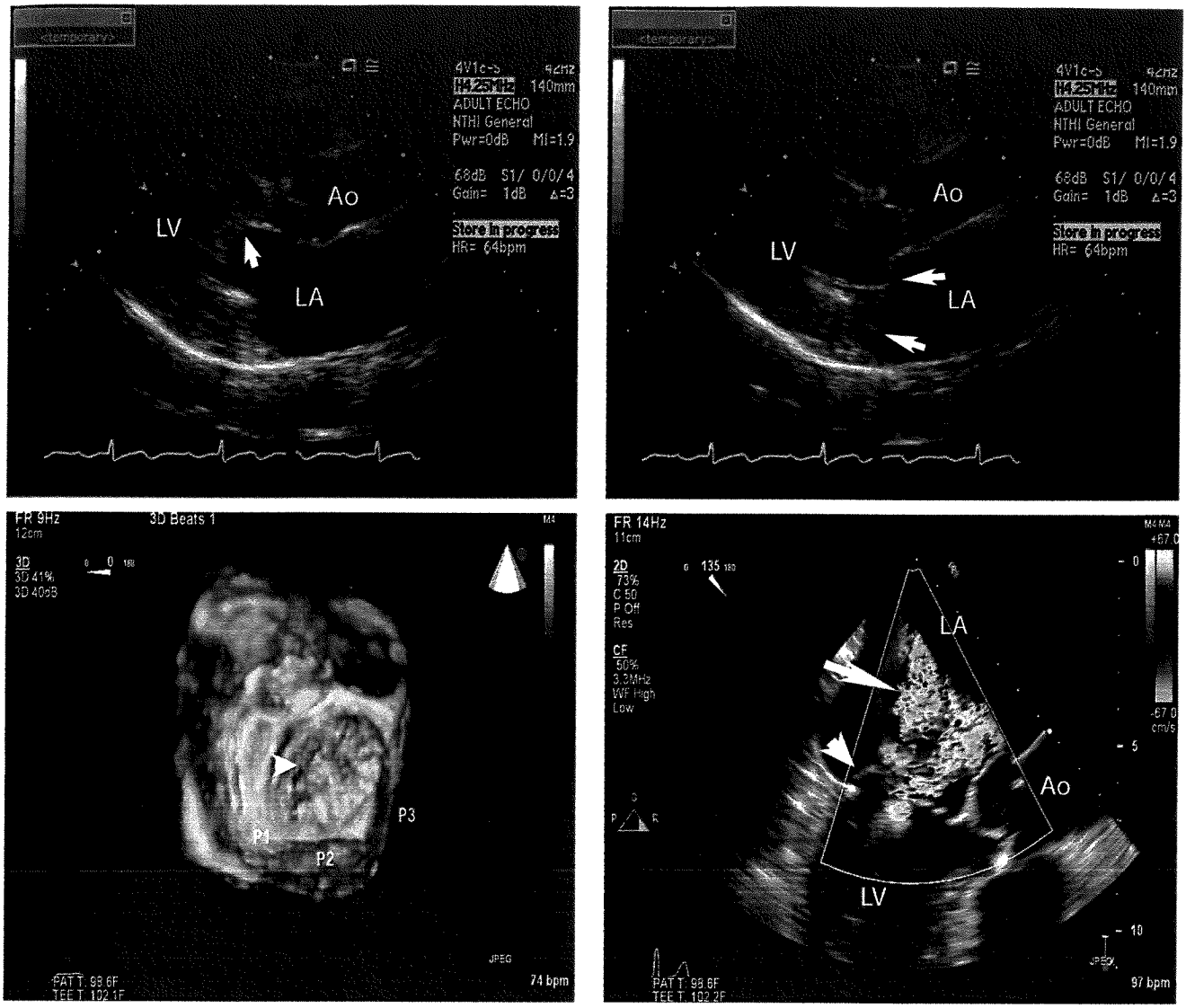

FIGURE 34. Mitral valve prolapse. Echocardiogram showing normal opening of the mitral valve (*arrow, top left panel*), which then prolapses into the left atrium during systole (*arrows, top right panel*). In a different patient with myxomatous degeneration, a flail portion of the posterior mitral valve is present (*bottom panels*); torn chordae are seen (*arrowheads*), leading to severe regurgitation seen on color flow imaging (*arrow, bottom right panel*). Ao = ascending aorta; LA = left atrium; LV = left ventricle.

0.7 cm or larger. When TTE provides insufficient or discordant information, TEE or CMR imaging is indicated.

Appropriate follow-up of asymptomatic patients with mitral regurgitation is outlined in Table 23.

Management

Medical therapy and surgical intervention can be life-saving in patients with acute severe mitral regurgitation. Vasodilator therapy with a titratable drug, such as nitroprusside, decreases aortic impedance and mitral regurgitation, thereby improving forward cardiac output. An intra-aortic balloon pump can be used to decrease afterload and augment systemic and coronary perfusion pressures. Prompt surgical correction should be considered for all patients with acute severe mitral regurgitation.

Patients with chronic severe primary mitral regurgitation generally do poorly without surgery, particularly when there are significant symptoms, flail leaflet, or LV dilatation. In one study of 458 patients with asymptomatic severe primary mitral regurgitation, the 5-year survival rate was only 58%. Surgical repair of the mitral valve is indicated for chronic severe primary mitral regurgitation in symptomatic patients (regardless of LV systolic function) and asymptomatic patients with LV dysfunction (ejection fraction ≤60% and/or LV end-systolic dimension ≥40 mm). Surgical repair is reasonable in asymptomatic patients with preserved LV function when the expected repair success rate is greater than 95% and the operative risk is less than 1%. Surgical repair is preferred over replacement in all patients, and patients should be referred to a surgical center with expertise to improve the chances of repair. Medical therapy with vasodilators is not beneficial in patients with primary mitral regurgitation in the absence of symptoms or LV dysfunction.

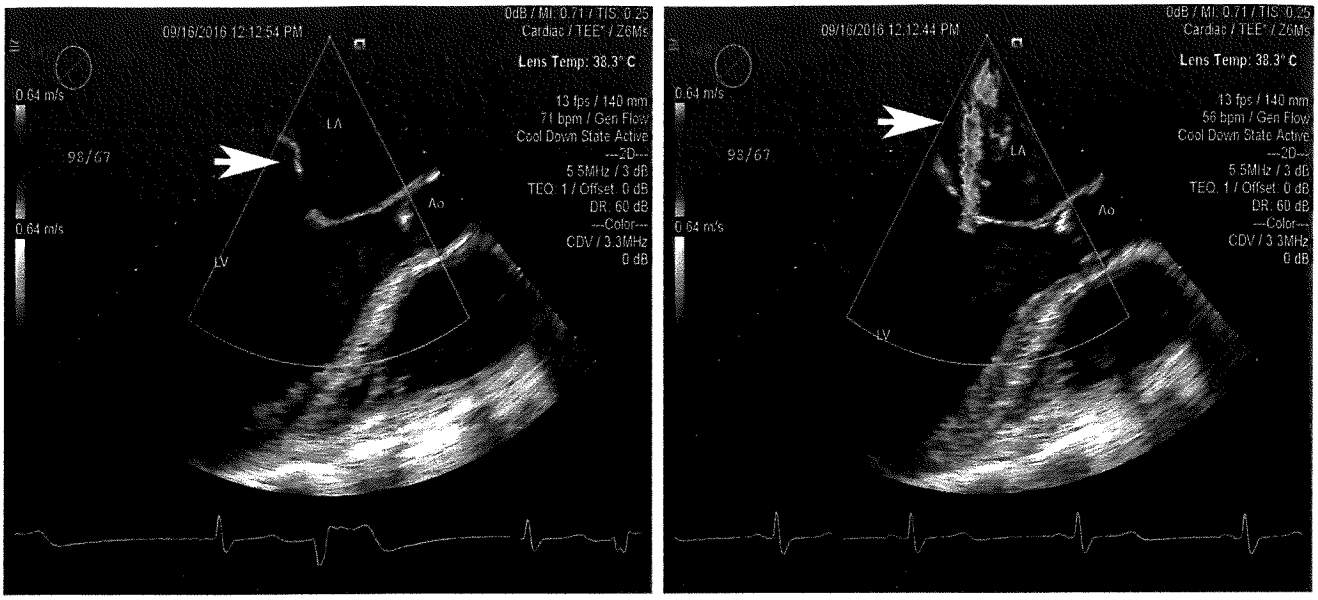

FIGURE 35. Echocardiograms demonstrating secondary mitral regurgitation in a patient with previous inferior myocardial infarction. Tethering of the posterior leaflet (*arrow, left panel*) is present due to the previous infarction and left ventricular remodeling. Mitral regurgitation (*arrow, right panel*) is evident on color flow imaging. Ao = ascending aorta; LA = left atrium; LV = left ventricle.

For patients who are not surgical candidates, transcatheter mitral valve repair with a clip device (transcatheter edge-to-edge repair [TEER]) improves coaptation of the mitral valve leaflets, leading to increased valve closure and a reduction in regurgitation (**Figure 36**). In selected patients with primary mitral regurgitation, success rates with TEER are approximately 90%, with a procedural mortality rate of approximately 2%.

In patients with chronic secondary mitral regurgitation, the primary goal of therapy is to address the underlying ventricular dysfunction with guideline-directed medical therapy and, if indicated, cardiac resynchronization therapy (see Heart Failure). Guideline-directed medical therapy for ventricular dysfunction includes an ACE inhibitor, ARB, or angiotensin receptor–neprilysin inhibitor; a β-blocker; a diuretic; and/or an aldosterone antagonist. Benefits of valve repair or replacement in patients with secondary mitral regurgitation are less certain, although studies have demonstrated favorable LV remodeling after surgery. Surgery for secondary severe mitral regurgitation is reasonable for those undergoing coronary artery bypass grafting, but mitral regurgitation may recur after repair because of primary LV dysfunction. TEER has been approved by the FDA for patients with chronic secondary mitral regurgitation and refractory symptoms despite optimal medical therapy for heart failure; however, the effect of TEER on mortality and heart failure hospitalization in this population has varied in studies.

KEY POINTS

- Patients with acute mitral regurgitation may present with acute heart failure; these patients may be difficult to diagnose clinically or with echocardiography.

(Continued)

KEY POINTS *(continued)*

- Surgery for chronic severe primary mitral regurgitation is indicated in the presence of symptoms, left ventricular dilatation, or decreasing ejection fraction.
- Surgical repair is preferred over replacement in patients with chronic primary mitral regurgitation; patients should be referred to a surgical center with expertise to improve the chances of repair.
- Transcatheter mitral valve repair with implantation of a clip device may be considered for patients with chronic severe primary mitral regurgitation who are at high surgical risk.
- Patients with chronic secondary mitral regurgitation should be treated with guideline-directed medical therapy for ventricular dysfunction; surgical intervention may be considered for those undergoing concomitant cardiac surgery.

Tricuspid Valve Disease

Tricuspid regurgitation, the most common form of tricuspid valve disease, is frequently functional (or secondary) and clinically asymptomatic. Causes of tricuspid regurgitation include cor pulmonale (or pulmonary hypertension) with RV failure, pacemaker or defibrillator lead placement, trauma, congenital abnormalities, and infective endocarditis. When symptomatic, patients may present with fatigue from low cardiac output as well as signs and symptoms of right-sided failure, such as elevated jugular venous pulse (a large *c-v* wave), a palpable RV lift, hepatic congestion with pulsatile liver, and peripheral edema. The murmur of tricuspid regurgitation is typically a