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

TABLE 23. Serial Evaluation of Asymptomatic Patients with Left-Sided Valvular Conditions

Factors Considered	Lesion Severity	Frequency of Evaluation
Aortic Stenosis		
Stenosis severity; rate of progression; LV systolic function; ascending aorta dilation if associated with bicuspid aortic valve	At risk (V_{max} <2 m/s)	
	Mild (V_{max} 2.0-2.9 m/s or mean gradient <20 mm Hg)	Clinical evaluation yearly; echo every 3-5 y
	Moderate (V_{max} 3.0-3.9 m/s or mean gradient 20-39 mm Hg)	Clinical evaluation yearly; echo every 1-2 y
	Severe (V_{max} ≥4 m/s or mean gradient ≥40 mm Hg, AVA typically ≤1.0 cm^2)	Clinical evaluation yearly; echo every 6-12 mo
	Very severe (V_{max} ≥5 m/s or mean gradient ≥60 mm Hg)	Clinical evaluation yearly; echo every 6-12 mo
Aortic Regurgitation		
Regurgitation severity; rate of progression; LV ejection fraction; LV chamber size; ascending aorta dilation if bicuspid aortic valve	Mild (VC <0.3 cm, ERO <0.10 cm^2, RV <30 mL/beat, RF <30%); normal EF	Clinical evaluation yearly; echo every 3-5 y
	Moderate (VC 0.3-0.6 cm, ERO 0.10-0.29 cm^2, RV 30-59 mL/beat, RF 30%-49%)	Clinical evaluation yearly; echo every 1-2 y
	Severe (VC >0.6 cm, ERO ≥0.3 cm^2, RV ≥60 mL/beat, RF ≥50%)	
	EF ≥55%; LVESD ≤50 mm	Clinical evaluation every 6-12 mo; echo every 6-12 mo, more frequently for dilating LV
	EF <55%; LVESD >50 mm	Clinical evaluation every 6-12 mo; echo every 6-12 mo, more frequently for dilating LV
Mitral Stenosis		
Stenosis severity	Mild and moderate (MVA >1.5 cm^2, diastolic pressure half-time <150 ms)	Clinical evaluation yearly; echo every 3-5 y
	Severe (MVA ≤1.5 cm^2, diastolic pressure half-time ≥150 ms or ≥220 ms with very severe stenosis, PASP >50 mm Hg)	Clinical evaluation yearly; echo every 1-2 y for MVA 1.0-1.5 cm^2, every year for MVA <1.0 cm^2
Mitral Regurgitation		
Regurgitation severity; rate of progression; EF; LV chamber size	At risk (VC <0.3 cm)	Clinical evaluation yearly; echo only if symptomatic
	Mild and moderate (VC <0.7 cm, ERO <0.40 cm^2, RV <60 mL/beat, RF <50%)	Clinical evaluation yearly; echo every 3-5 y for mild severity, every 1-2 y for moderate severity
	Severe (VC ≥0.7 cm, ERO ≥0.4 cm^2, RV ≥60 mL/beat, RF ≥50%)	Clinical evaluation every 6-12 mo; echo every 6-12 mo, more frequently for dilating LV

AVA = aortic valve area; echo = echocardiography; EF = ejection fraction; ERO = effective regurgitant orifice; LV = left ventricular; LVESD = left ventricular end-systolic dimension; MVA = mitral valve area; PASP = pulmonary artery systolic pressure; RF = regurgitant fraction; RV = regurgitant volume; VC = vena contracta width; V_{max} = maximum aortic jet velocity.

Recommendations based on Otto CM, Nishimura RA, Bonow RO, et al; Writing Committee Members. 2020 ACC/AHA guideline for the management of patients with valvular heart disease: a report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines. J Am Coll Cardiol. 2021;77:e25-e197. [PMID: 33342586] doi:10.1016/j.jacc.2020.11.018

valve area of approximately 0.12 cm^2 per year, but the rate depends on patient age, underlying severity of the stenosis, and comorbid conditions, such as kidney failure and hypertension. Exertional dyspnea, syncope, and angina are the most common symptoms; however, symptoms may not appear until stenosis is severe. Among asymptomatic patients with severe aortic stenosis, 75% will die or develop symptoms within 5 years. Once symptoms occur, life expectancy is generally only 1 to 2 years. Thus, serial evaluation every 6 to 12 months is recommended for patients with severe disease (see Table 23).

The characteristic clinical findings of severe aortic stenosis include a late-peaking systolic murmur, a diminished or absent aortic component of the S_2, and a delay in the carotid upstroke (pulsus tardus) that may be accompanied by a decreased pulse amplitude due to low cardiac output (pulsus parvus). Clinical findings suggestive of severe aortic stenosis should be promptly evaluated (see Table 21).

The primary imaging modality for evaluation of aortic stenosis is TTE (**Figure 31**). Echocardiography can determine the cause of stenosis, stenosis severity (with gradient and valve

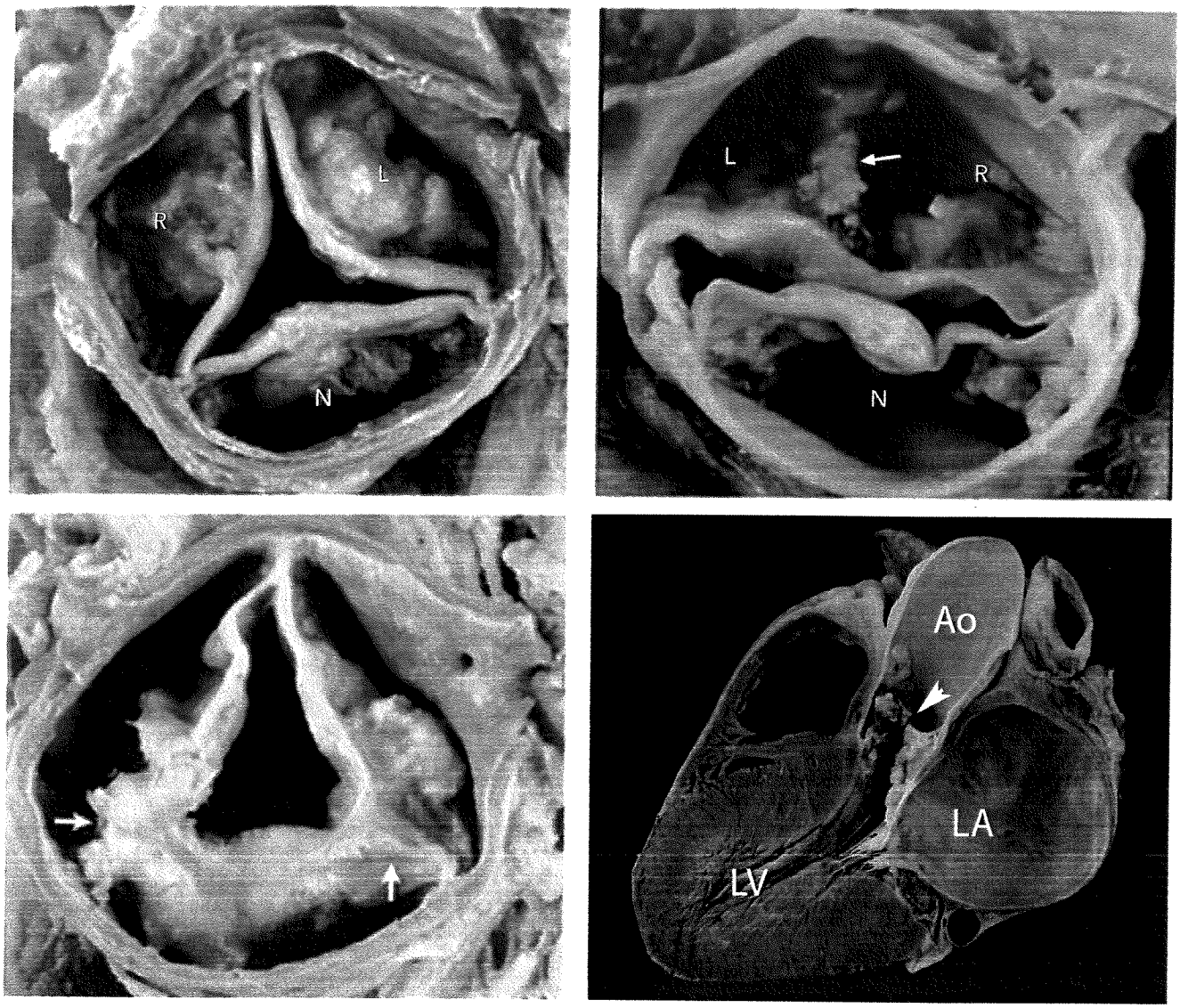

FIGURE 30. Aortic stenosis. Gross specimens showing pathology of degenerative aortic stenosis (*top left panel*), bicuspid aortic stenosis (*top right panel*), and rheumatic aortic disease (*bottom left panel*). The raphe between the left (L) and right (R) aortic cups is fused in this case of bicuspid aortic stenosis (*arrow in top right panel*). Fusion of the commissures is a distinctive feature of rheumatic disease (*arrows in bottom left panel*). Gross specimen showing severe left ventricular (LV) hypertrophy as a result of pressure overload from severe aortic stenosis (*arrowhead in bottom right panel*). Ao = ascending aorta; LA = left atrium; N = noncoronary cusp.

Images courtesy of Dr. William Edwards, Mayo Clinic.

area assessment), LV function and wall thickness, right ventricular (RV) function, pulmonary artery pressure, and the presence or absence of other valve pathology. In some patients, echocardiography may underestimate the severity of aortic stenosis. Further evaluation with cardiac catheterization, during which cardiac output and the aortic pressure gradient can be measured, is required when there are discrepancies between the clinical and echocardiographic findings in symptomatic patients being considered for intervention. Exercise stress testing is useful in asymptomatic patients with severe aortic stenosis to confirm asymptomatic status, but it should be performed under cardiologist supervision. Exercise stress testing is contraindicated in symptomatic severe aortic stenosis.

Severe aortic stenosis is typically defined by a small valve area (≤1.0 cm^2), high peak velocity (>4 m/s), and/or high mean gradient (>40 mm Hg). There are two patient subsets in which severe aortic stenosis may be present with a small valve area and either low velocity or low gradient—(1) patients with severe LV dysfunction and low cardiac output (low-flow, low-gradient aortic stenosis) and (2) patients with preserved LV function and paradoxical low-flow, low-gradient aortic stenosis. In the former group, dobutamine echocardiography or dobutamine cardiac catheterization is needed to distinguish true aortic stenosis from pseudostenosis. In pseudostenosis, dobutamine increases cardiac output and the opening forces on the aortic valve, causing the valve area to increase out of the

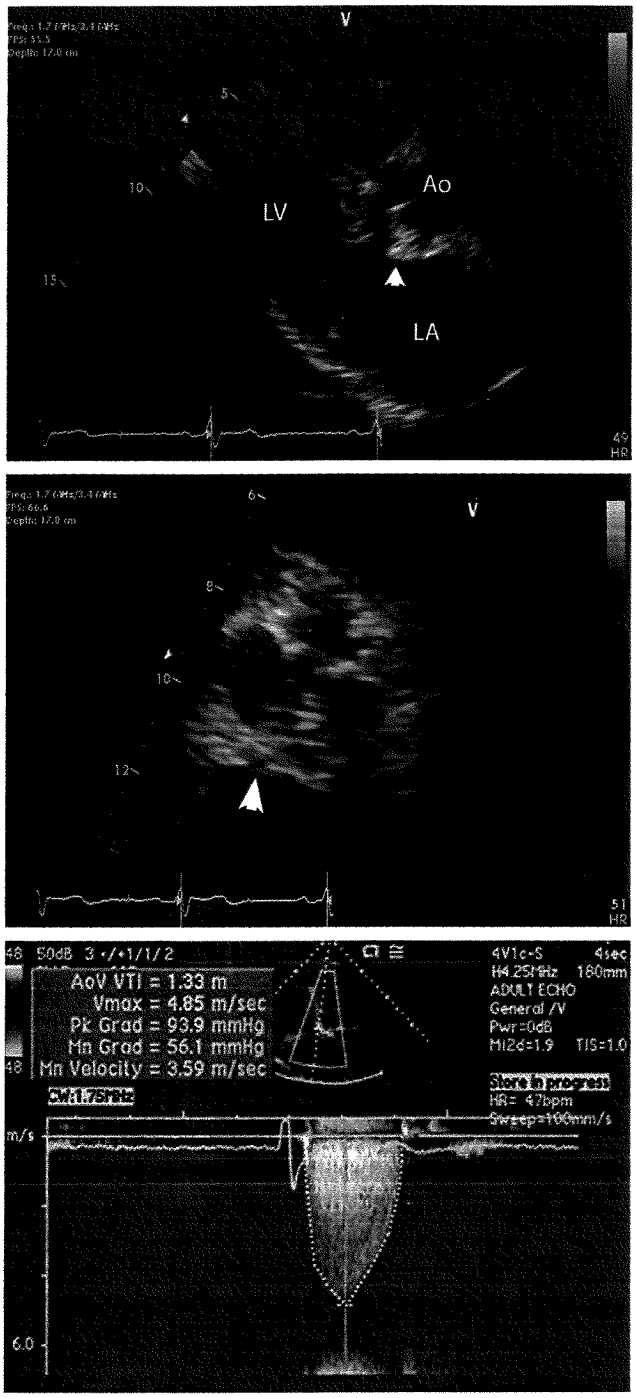

FIGURE 31. Echocardiographic findings in aortic stenosis. Calcific aortic stenosis (*arrowhead*) is present in parasternal long-axis (*top panel*) and short-axis (*middle panel*) views. Left ventricular (LV) hypertrophy is also present. Doppler echocardiogram shows a mean aortic gradient of 56 mm Hg, consistent with severe aortic stenosis (*bottom panel*). Ao = ascending aorta; LA = left atrium.

severe range. With low-flow, low-gradient aortic stenosis, the calculated valve area remains in the severe range with dobutamine administration, and the aortic valve gradient and velocity increase with increased stroke volume. Patients with paradoxical low-flow, low-gradient aortic stenosis have low stroke volume (<35 mL/m^2) resulting from a combination of small LV size and high aortic impedance to flow (e.g., hypertension) or other causes of low cardiac output (e.g., atrial fibrillation, pulmonary hypertension). Determination of lesion severity in paradoxical aortic stenosis requires consideration of the hemodynamics, valve morphology (e.g., degree of calcification), presence of LV hypertrophy, and clinical presentation of the patient. In patients with suspected low-flow, low-gradient severe aortic stenosis with normal or reduced left ventricular ejection fraction, measurement of aortic valve calcium score by CT is reasonable to further define severity. In patients with either low-flow, low-gradient severe aortic stenosis with reduced LV function or paradoxical low-flow, low-gradient severe aortic stenosis, observational studies have shown improved survival with aortic valve replacement compared with medical therapy.

Management

Aortic valve replacement is a life-prolonging procedure in patients with severe aortic stenosis. The indications for aortic valve replacement in severe aortic stenosis are the presence of symptoms (e.g., dyspnea, angina, presyncope, syncope, or heart failure), LV systolic dysfunction (ejection fraction <50%) in an asymptomatic patient, or a concomitant cardiac surgical procedure for other indications (e.g., coronary artery bypass grafting or ascending aorta surgery). Aortic valve replacement is reasonable in asymptomatic patients with very severe aortic stenosis and low surgical risk and asymptomatic patients with abnormal results on supervised exercise testing, such as poor exercise tolerance, abnormal ECG changes, or hypotension during testing.

Aortic valve replacement can be performed with open cardiac surgery (surgical aortic valve replacement [SAVR]) or via transcatheter approach (transcatheter aortic valve implantation [TAVI]) (**Figure 32**). SAVR and TAVI have similar procedural and long-term survival rates, with expected operative mortality rates of 1% to 3%. The choice between surgical and transcatheter interventions is based on the presence of symptoms and the patient's surgical risk, as determined through comprehensive multidisciplinary assessment. TAVI is recommended over SAVR for symptomatic patients with severe aortic stenosis who are older than 80 years or for younger patients with a life expectancy less than 10 years. TAVI is also recommended over SAVR for symptomatic patients of any age with severe aortic stenosis and a high or prohibitive surgical risk if predicted postprocedure survival is more than 12 months with an acceptable quality of life. For symptomatic patients who are aged 65 to 80 years, either SAVR or TAVI is appropriate following shared decision making. Neither SAVR nor TAVI is indicated in patients with limited expectation of survival due to comorbid conditions.

Although the pathophysiology of aortic stenosis is known to be inflammatory, randomized trials have shown medical therapy, specifically statins, to be ineffective in slowing disease progression. For patients with coexistent hypertension or heart failure, guideline-directed medical therapy is recommended. Vasodilators should be used with caution in patients with

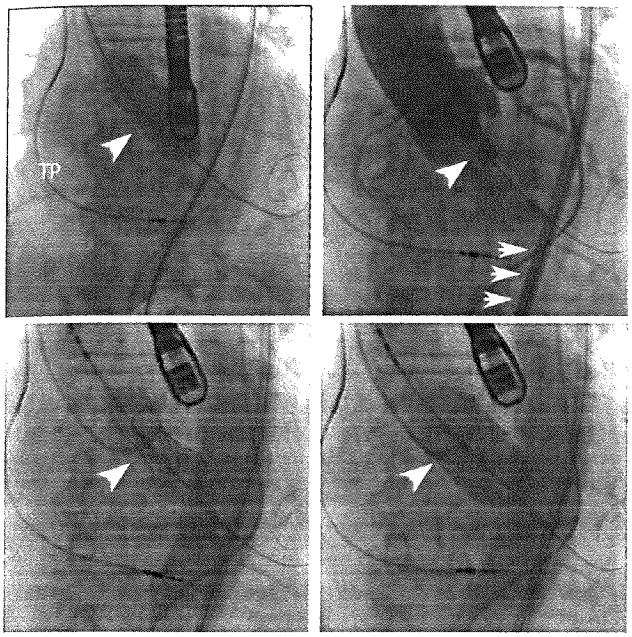

FIGURE 32. Transcatheter aortic valve implantation. *Top left panel:* Balloon aortic valvuloplasty (*arrowhead*) is first performed. *Top right panel:* Using a transfemoral approach (*arrows*), a transcatheter aortic valve (*arrowhead*) is positioned at the aortic annulus using aortography. *Bottom left panel:* The prosthesis (*arrowhead*) is then slowly inflated during rapid pacing from a temporary pacemaker (TP), which creates ventricular standstill. *Bottom right panel:* The prosthesis is fully deployed.

aortic stenosis and heart failure symptoms. In select cases, balloon valvuloplasty may be used to bridge unstable patients to therapy with TAVI or SAVR.

KEY POINTS

- The characteristic clinical findings of severe aortic stenosis include a late-peaking systolic murmur, a diminished or absent aortic component of the S_2, and a weak and delayed carotid upstroke.
- Echocardiography is accurate for defining the severity of aortic stenosis in most patients; when there is a discrepancy between the clinical and echocardiographic findings, cardiac catheterization should be considered in patients who are candidates for intervention.
- Patients with low-flow, low-gradient aortic stenosis have a small valve area but low velocity and/or low gradient due to low stroke volume; dobutamine echocardiography or cardiac catheterization can be used to distinguish pseudo-severe aortic stenosis from true severe aortic stenosis.
- Aortic valve replacement prolongs life in patients with symptomatic severe aortic stenosis; the patient's surgical risk determines whether valve replacement is performed using open surgery or a transcatheter approach.
- Transcatheter aortic valve implantation is indicated for symptomatic patients with trileaflet aortic stenosis at any level of surgical risk who do not have concomitant severe aortic regurgitation.

Aortic Regurgitation

Clinical Presentation and Evaluation

Aortic regurgitation, manifesting acutely or chronically, arises from aortic root pathology or intrinsic valve disease. Causes of chronic aortic regurgitation include ascending aortic dilatation and valve abnormalities due to bicuspid disease, calcific degeneration, rheumatic involvement, or chest irradiation. Acute aortic regurgitation may be caused by endocarditis, blunt chest trauma, iatrogenic damage (e.g., complications of balloon aortic valvuloplasty), or aortic dissection.

In chronic aortic regurgitation, volume overload causes progressive LV dilatation and eccentric hypertrophy. Chronic aortic regurgitation may be tolerated for many years but may eventually lead to symptoms, including shortness of breath, fatigue, or angina. Clinical findings result from the large stroke volume and LV dilatation and include bounding peripheral pulses, displacement of the LV apex, and a diastolic decrescendo murmur heard either along the right sternal border (suggesting root pathology) or left sternal border (suggesting valve pathology) (see Table 21). The large forward stroke volume also can cause an early-peaking systolic ejection murmur.

In acute regurgitation, the abrupt onset of volume overload may cause acute heart failure or even cardiogenic shock. Bounding pulses may not be present because stroke volume has not markedly increased, and murmurs may be softer or shorter in duration because of the rapid equalization of pressures between the aorta and LV.

TTE is indicated for evaluation of aortic regurgitation and LV function. In patients with moderate or severe aortic regurgitation and suboptimal TTE images or a discrepancy between clinical and TTE findings, transesophageal echocardiography (TEE), cardiac magnetic resonance (CMR) imaging, or cardiac catheterization is indicated. When endocarditis is suspected, TTE is the initial imaging test in most patients. TEE following TTE is recommended for patients with suboptimal TTE results or with high initial risk. CT angiography is indicated in patients with acute AR and aortic dissection because it is highly accurate and usually rapidly available.

Criteria for severe aortic regurgitation include a jet width that occupies 65% or more of the LV outflow tract, vena contracta (the width of the regurgitant jet at its most narrow portion) greater than 0.6 cm, holodiastolic flow in the descending aorta, regurgitation volume of 60 mL or more, and effective regurgitant orifice area of 0.3 cm^2 or greater. The LV typically is dilated in chronic aortic regurgitation. In patients suspected of having an aortic root abnormality, evaluation with CMR imaging, CT, or TEE is recommended. Surveillance is based on severity of regurgitation and other factors (see Table 23).

Management

In chronic aortic regurgitation, surgery with traditional open aortic valve replacement is advised for patients with symptoms (typically, dyspnea or angina) or LV dysfunction (ejection

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

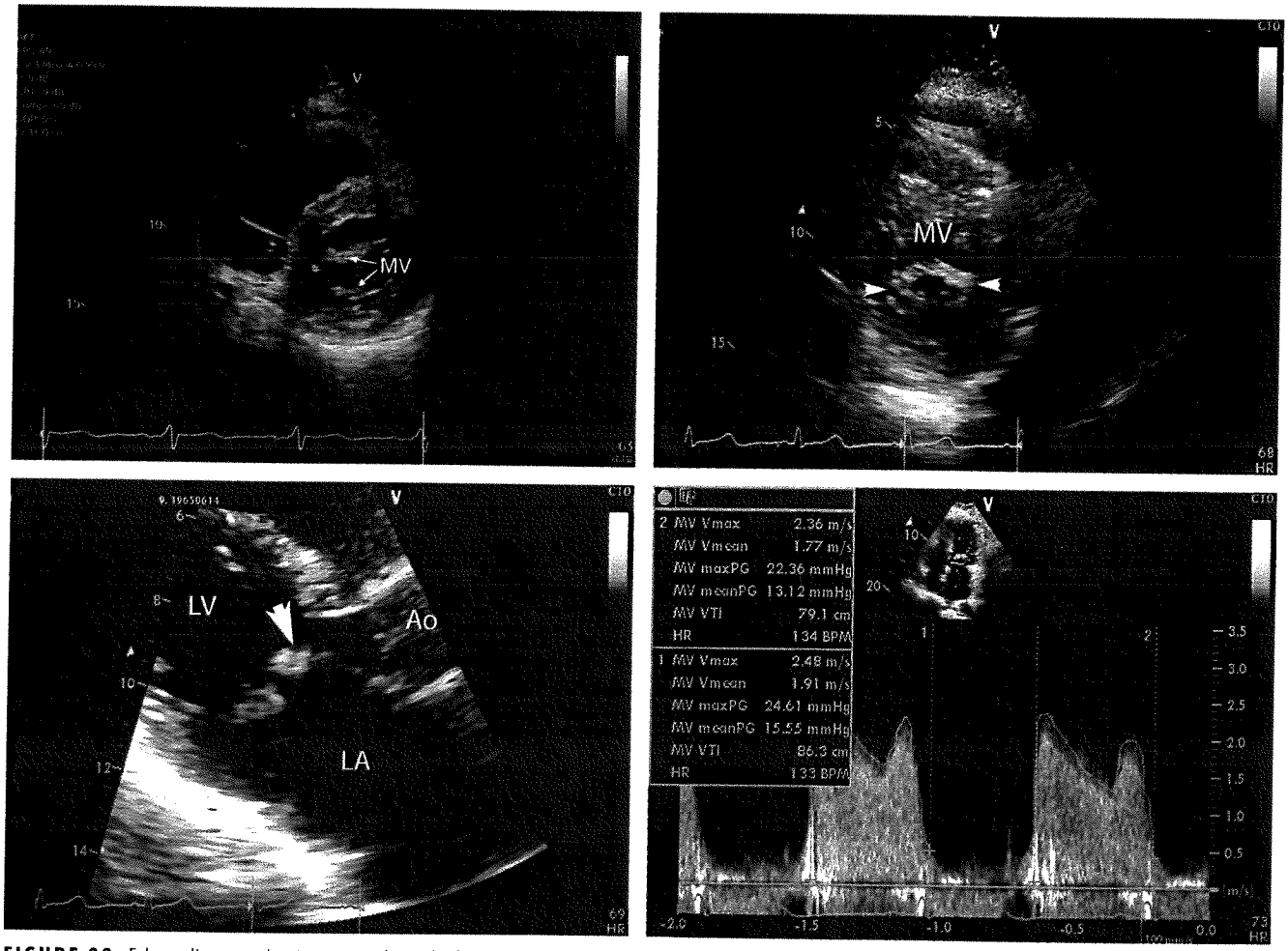

FIGURE 33. Echocardiograms showing a normal mitral valve (MV) (*top right panel*) and rheumatic mitral stenosis with commissural fusion (*arrowheads, top left panel*). In the bottom left panel, diastolic doming (*arrowhead*) is present with a "hockey stick" deformity from mitral stenosis. In the bottom 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.