because it provides no advantages over epinephrine. Likewise, atropine should not be used for the treatment of asystole or pulseless electrical activity arrest. Any reversible causes (such as tamponade) should be identified and treated.

Patients with VT/VF should be shocked, followed by immediate resumption of CPR and reassessment of the rhythm in 2 minutes. Epinephrine should be administered after the second shock and every 3 to 5 minutes thereafter. Amiodarone should be given as a bolus if VT/VF continues despite three shocks and epinephrine administration. A second dose of amiodarone can be given if VT/VF persists.

Device Therapy for Prevention of Sudden Death

ICDs have demonstrated efficacy in the primary and secondary prevention of SCD. Patients with sustained ventricular arrhythmias (>30 seconds) or cardiac arrest without a reversible cause have a class I recommendation for secondary prevention ICD placement. Patients with heart failure who meet specific criteria should undergo ICD placement for primary prevention (see 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.

Patients with ICDs need to avoid strenuous upper extremity exercises, including weight lifting, because of concern for lead stress and subsequent fracture. Inappropriate detection of VT/VF and shocks can result from electromagnetic interference; therefore, patients need to avoid devices that pose risks, such as arc welding equipment and high-voltage machinery. Patients with ICDs who are undergoing invasive procedures or surgery should be evaluated by their electrophysiologist for device reprogramming recommendations.

In the past, ICDs were implanted almost exclusively by 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.

KEY POINT

- Implantable cardioverter-defibrillators are effective for primary and secondary prevention of sudden cardiac death.

Device Infection

Device infections have many different forms, ranging from pocket infections to endocarditis. Most infections are due to *Staphylococcus epidermidis* and *Staphylococcus aureus*. Symptoms of cardiac device infection include fever, chills, malaise, lassitude, and failure to thrive, particularly in the elderly.

Physical examination of the pocket may reveal erythema, swelling, drainage, or wound dehiscence. In patients suspected of having device infection, several blood cultures, an erythrocyte sedimentation rate, and a C-reactive protein level should be obtained. A transesophageal echocardiogram should be obtained to evaluate for intracardiac or lead vegetations. Aspirating the device pocket is never indicated because this can damage the leads or introduce infection in a sterile or uninfected pocket. PET-CT can also identify infection of the device pocket or leads if other testing is inconclusive.

Treatment of cardiac device infection includes complete extraction of all hardware, debridement of the pocket, sustained antibiotic therapy, and reimplantation at a new location after infection has been eradicated.

Valvular Heart Disease

General Principles

Valvular heart disease (VHD) is characterized by underlying functional or anatomic abnormalities in the cardiac valves that result in regurgitation or stenosis. VHD is common, occurring in approximately 20 million persons in the United States. Although there are congenital forms, VHD is largely age dependent, and 3% to 6% of those aged 65 years and older are affected.

A thorough history and physical examination are essential in evaluating for VHD. Many heart valve lesions are slowly progressive, and patients may unconsciously limit their activity in response to a worsening of underlying VHD. The most common symptom is exertional dyspnea. Other symptoms include angina, syncope, palpitations, lower extremity edema, and ascites, depending on the lesion and severity. Typical physical examination findings for valvular and other cardiac lesions are described in **Table 21**. Twelve-lead electrocardiography, chest radiography, and transthoracic echocardiography (TTE) with two-dimensional imaging may be performed to evaluate for VHD.

For clinical monitoring and timing of intervention, VHD is classified into four stages (A to D), which consider the presence of symptoms, severity of the lesion, 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 58.

Surgery can be a life-saving intervention in select patients, and surgical risk calculation is a key component of the patient evaluation. Surgical risk is determined through an assessment of the patient's age, morbidities, frailty, and impediments specific to the procedure being considered (for example, prior chest radiation therapy for a sternotomy approach). Risk calculators derived from national databases can assist in estimating risk for morbidity and mortality for various surgical valve procedures. One such calculator, the Society of Thoracic Surgery Adult Cardiac Surgery Risk Calculator, is available at riskcalc.sts.org. Although risk calculators contain many data input fields, it is important to note that frailty and some other important patient and procedural characteristics are not part of these online assessment tools.

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 down the descending thoracic aorta may mimic mitral regurgitation
Aortic regurgitation	Diastolic; decrescendo	LLSB (valvular) or RLSB (dilated aorta) (heard best 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 (heard best 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
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, decreases with amyl nitrite	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.

Reproduced with permission from Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, et al; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63:2438-88. [PMID: 24603192] doi:10.1016/j.jacc.2014.02.537. Copyright 2014, Elsevier.

Therefore, a comprehensive, holistic approach is required for determining patient surgical risk and candidacy. Frailty, which is variably defined as a geriatric syndrome characterized by declines in several physiologic systems and processes, portends an increased risk for mortality in patients undergoing surgery and can be measured preoperatively (see MKSAP 18 General Internal Medicine). H

For all patients in whom surgical or interventional therapy is being considered, a multidisciplinary approach with a heart team consisting of cardiologists, surgeons, and interventional cardiologists is recommended. Evaluations in centers with specialized expertise in VHD (for example, a Heart Valve Center of Excellence) is also advised for patients in whom intervention is being considered when (1) there are no symptoms; (2) multiple or complex morbidities are present; or (3) surgical valve repair is favored over valve replacement.

Medical therapy, although often effective for symptom palliation, has not been shown to prevent progression of VHD or improve long-term survival in patients with VHD.

KEY POINTS

- Many heart valve lesions progress slowly, leading patients to unconsciously limit their activity in response; therefore, a careful history and detailed physical examination are essential.
- 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 cardiologists, surgeons, and interventional cardiologists is recommended.
- Medical therapy, although often effective for symptom palliation, has not been shown to prevent progression of valvular heart disease or improve long-term survival in patients with valvular heart disease.

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)	Clinical evaluation yearly; echo every 3-5 y
	Mild (V_{max} 2.0-2.9 m/s or mean gradient <20 mm Hg)	Clinical evaluation yearly; echo every 1-2 y
	Moderate (V_{max} 3.0-3.9 m/s or mean gradient 20-39 mm Hg)	Clinical evaluation yearly; echo every 6-12 mo
	Severe (V_{max} ≥4 m/s or mean gradient ≥40 mm Hg, AVA ≤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)	
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 ≥50%; LVESD ≤50 mm	Clinical evaluation every 6-12 mo; echo every 6-12 mo, more frequently for dilating LV
	EF <50%; 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 >30 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 Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP 3rd, Guyton RA, et al; American College of Cardiology/American Heart Association Task Force on Practice Guidelines. 2014 AHA/ACC guideline for the management of patients with valvular heart disease: executive summary: a report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines. J Am Coll Cardiol. 2014;63:2438-88. [PMID: 24603192] doi:10.1016/j.jacc.2014.02.537

Aortic Stenosis

Clinical Presentation and Evaluation

Aortic stenosis may be congenital, such as in persons with a bicuspid aortic valve, or acquired. The most common cause is degeneration of the valve that occurs with aging; severe lesions occur in approximately 3% of persons aged 65 years and older (**Figure 20**). Other causes include rheumatic disease and chest radiation. Although rheumatic disease of the mitral valve frequently occurs in isolation, rheumatic aortic valve disease almost never occurs without mitral valve involvement. Chest radiation (for example, mantle therapy for non-Hodgkin lymphoma) commonly causes a mixture of both valvular stenosis and regurgitation.

Aortic stenosis results in 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. Exertional dyspnea, syncope, and angina are the most common presenting symptoms; however, symptoms may not

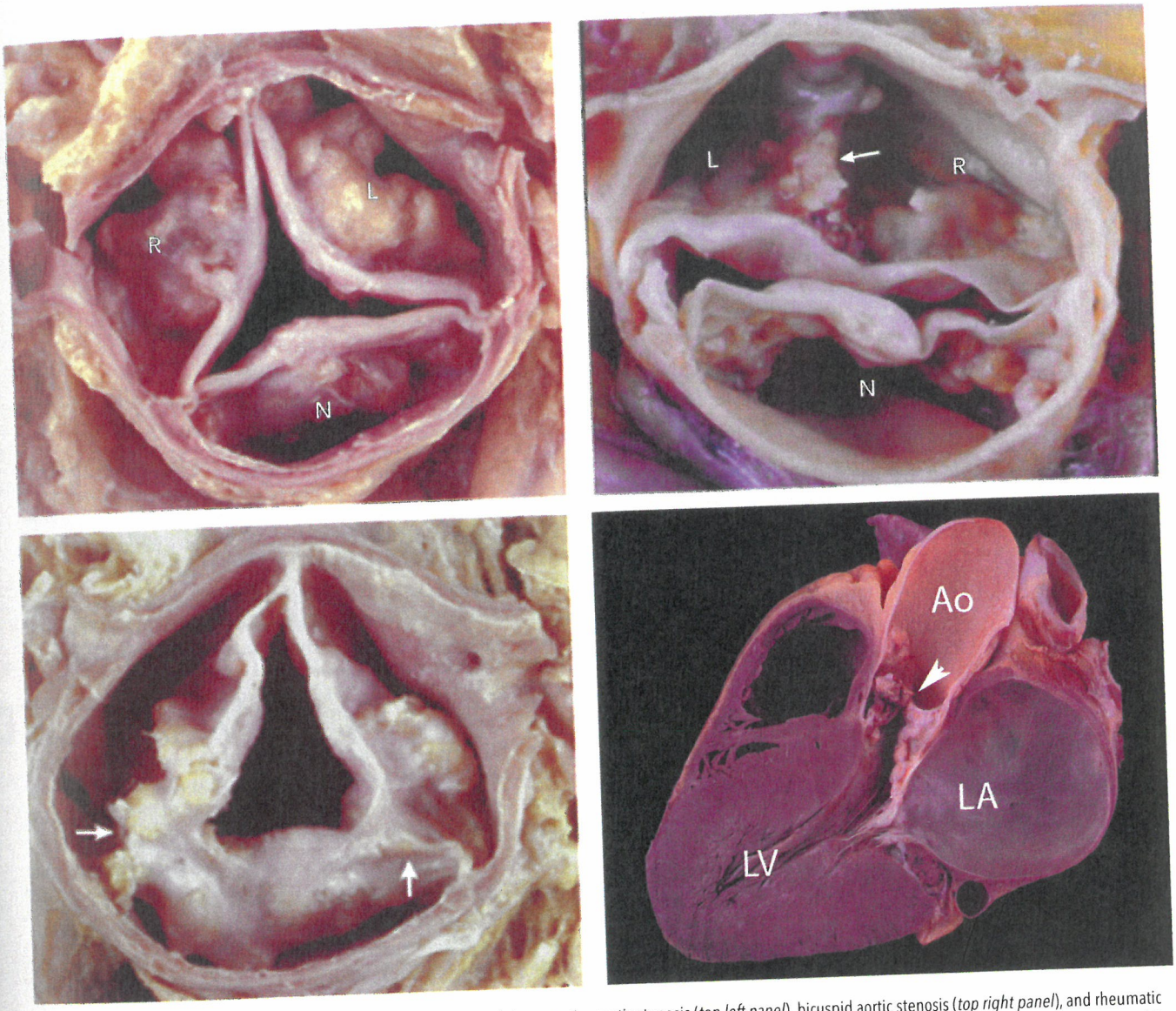

FIGURE 20. Aortic stenosis. Gross specimens showing pathology of degenerative aortic stenosis (*top left panel*), bicuspid aortic stenosis (*top right panel*), and rheumatic aortic disease (*bottom left panel*). The raphe between the left (L) and right (R) aortic cups is fused in this case of bicuspid 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.

appear until stenosis is severe. The disease typically progresses with a decrease in the aortic 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. Among asymptomatic patients with severe aortic stenosis, 75% will die or develop symptoms within 5 years. Once symptoms occur in patients with severe aortic stenosis, 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).

In patients with severe aortic stenosis, the characteristic physical findings 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). Physical findings that suggest severe aortic stenosis should be promptly evaluated (see Table 21).

The primary imaging modality for the evaluation of aortic stenosis is TTE (**Figure 21**). Echocardiography can determine the cause and severity of aortic stenosis (such as the gradient and valve area) as well as LV function and wall thickness. In some patients, echocardiography may underestimate the severity of aortic stenosis. Further evaluation with cardiac catheterization, during which the cardiac output and the gradient across the aortic valve can be measured, is required when there are discrepancies between the findings on physical

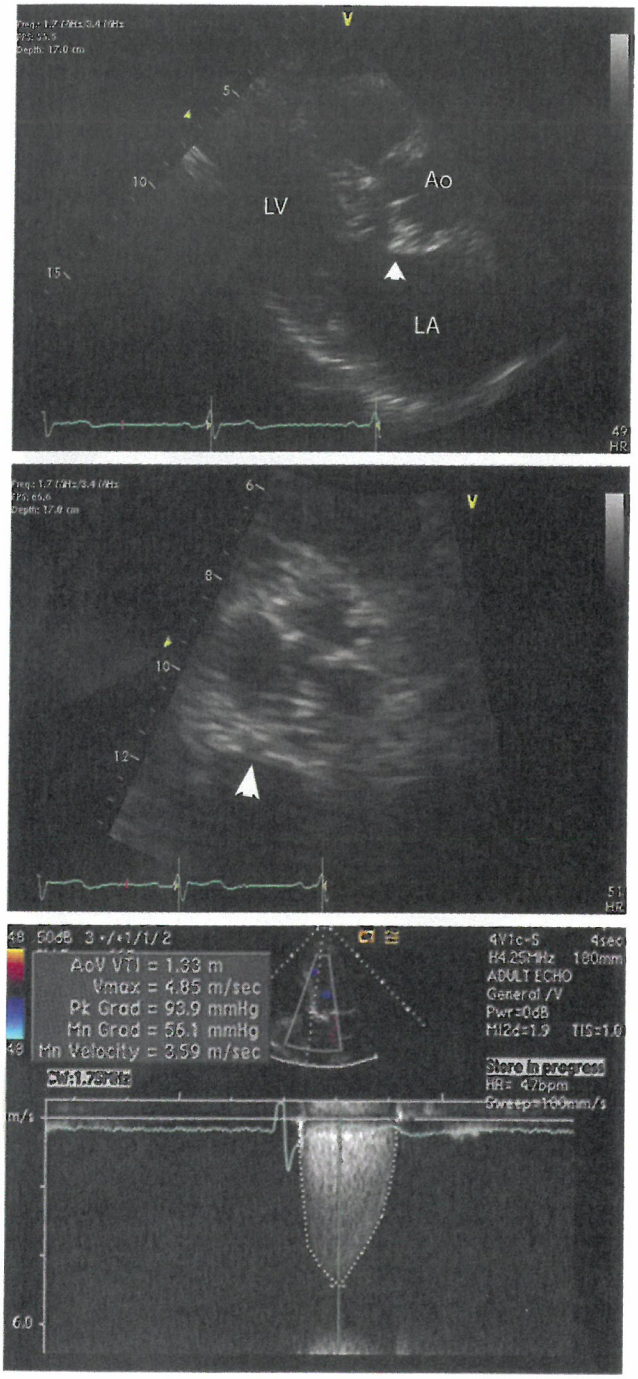

FIGURE 21. Echocardiographic findings in aortic stenosis. Calcific aortic stenosis (*arrowhead*) is present in parasternal long-axis (*top panel*) and short-axis (*middle panel*) views. Left ventricular (LV) hypertrophy is also present. Doppler echocardiogram shows a mean aortic gradient of 56 mm Hg, consistent with severe aortic stenosis (*bottom panel*). Ao = ascending aorta; LA = left atrium.

examination and the echocardiographic results in symptomatic patients being considered for surgery.

Severe aortic stenosis is typically defined by a small valve area (≤1.0 cm^2) and either high peak velocity (>4 m/s) 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: patients with severe LV dysfunction and low cardiac output and patients with preserved LV function and paradoxical low-flow, low-gradient aortic stenosis. In the former group, dobutamine echocardiography or an invasive hemodynamic study is needed to distinguish true aortic stenosis from pseudostenosis. With pseudostenosis, dobutamine increases cardiac output and the opening forces on the aortic valve, causing the valve area to increase out of the severe range. With true 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. In patients with paradoxical low-flow, low-gradient aortic stenosis, low stroke volume (<35 mL/m^2) results from a combination of small LV size and high aortic impedance to flow (hypertension). Determination of lesion severity in paradoxical aortic stenosis requires consideration of the hemodynamics, valve morphology (such as degree of degeneration), presence of LV hypertrophy, and clinical presentation of the patient. In patients with either low-flow, low-gradient severe aortic stenosis 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 (1) the presence of symptoms (such as dyspnea, angina, presyncope, or syncope), (2) LV systolic dysfunction (ejection fraction <50%) in an asymptomatic patient, or (3) a concomitant cardiac surgical procedure for another indication (such as simultaneous coronary artery bypass grafting or ascending aorta surgery). Aortic valve replacement may be considered in asymptomatic patients with abnormal results on supervised exercise testing, such as those with poor exercise tolerance, abnormal electrocardiographic 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 replacement [TAVR]) (**Figure 22**). SAVR and TAVR 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 assessment by a multidisciplinary heart team. TAVR is currently indicated for symptomatic patients with trileaflet aortic stenosis who are at intermediate or high surgical risk and who do not have concomitant severe aortic regurgitation. Randomized trials comparing TAVR with SAVR in low-risk patients are ongoing.

Although the pathophysiology of aortic stenosis is known to be inflammatory, randomized trials of medical therapy, specifically statins, have not found this therapy to be effective in slowing disease progression. For patients with coexistent

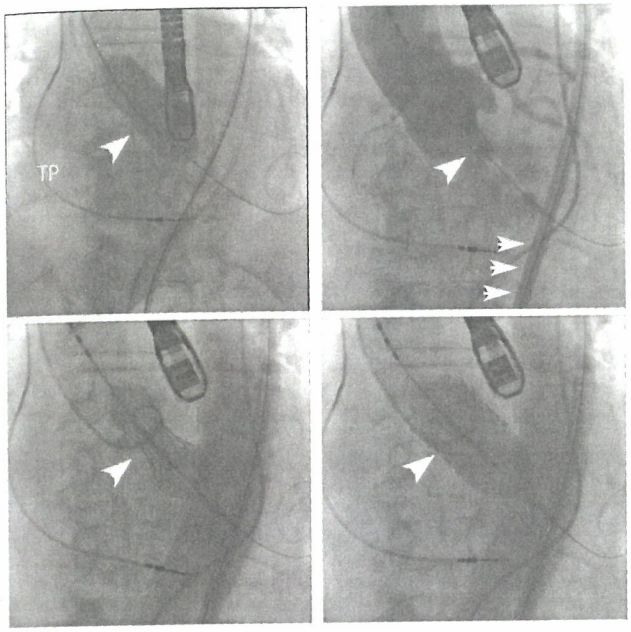

FIGURE 22. Transcatheter aortic valve replacement. *Top left panel:* Balloon aortic valvuloplasty (*arrowhead*) is first performed. *Top right panel:* Using a transfemoral approach (*arrows*), a transcatheter aortic valve (*arrowhead*) is positioned at the aortic annulus using aortography. *Bottom left panel:* The prosthesis (*arrowhead*) is then slowly inflated using rapid pacing from a temporary pacemaker (TP), which creates ventricular standstill. *Bottom right panel:* The prosthesis is fully deployed.

hypertension or heart failure, guideline-directed medical therapy is recommended. Vasodilators should be used with caution in patients with aortic stenosis and heart failure symptoms. In select cases, balloon valvuloplasty may be used to bridge patients to therapy with TAVR or SAVR.

KEY POINTS

- The most common cause of severe aortic stenosis is degeneration of the aortic valve.
- The characteristic physical 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 surgical candidates.
- Aortic valve replacement is a life-prolonging intervention in patients with severe aortic stenosis; the patient's surgical risk and the presence of symptoms determine whether aortic valve replacement is performed using open surgery or a transcatheter approach.
- Transcatheter aortic valve replacement is indicated for symptomatic patients with trileaflet aortic stenosis who are at intermediate or high surgical risk and who do not have concomitant severe aortic regurgitation.

Aortic Regurgitation

Clinical Presentation and Evaluation

Aortic regurgitation may be caused by aortic root pathology or intrinsic valve disease and can manifest acutely or chronically. Causes of chronic aortic regurgitation include ascending aortic dilatation and valve abnormalities due to bicuspid disease, calcific degeneration, rheumatic involvement, or chest radiation. Causes of acute aortic regurgitation are endocarditis, blunt chest trauma, iatrogenic causes (such as complications of balloon aortic valvuloplasty), and 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 can eventually lead to symptoms, including shortness of breath, fatigue, or angina. Physical 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 can also result in an early-peaking systolic ejection murmur. In patients with acute regurgitation, the abrupt onset of volume overload may not be well tolerated, and these patients can present with acute heart failure or even cardiogenic shock. Additionally, patients with acute regurgitation may not have bounding pulses because stroke volume has not markedly increased, and murmurs may be softer or shorter in duration, owing to the rapid equalization of pressures between the aorta and LV.

TTE is recommended for the evaluation of aortic regurgitation and LV function. When endocarditis is suspected and transthoracic imaging is suboptimal, transesophageal echocardiography (TEE) is advised. As an alternative, cardiac magnetic resonance (CMR) imaging and invasive angiography also can be used to determine the severity of regurgitation. Criteria for severe aortic regurgitation include a jet width that occupies 65% of the LV outflow tract or more, vena contracta 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 is also typically dilated in chronic aortic regurgitation. For patients suspected of having an aortic root abnormality, an evaluation with CMR imaging, CT, or TEE is recommended.

Management

Acute aortic regurgitation due to aortic dissection is a surgical emergency. For other acute causes, the indications for surgery depend on severity, presence of symptoms, and the hemodynamic stability of the patient. In cases of chronic aortic regurgitation, surgery with traditional open aortic valve replacement is advised for patients with symptoms (typically, dyspnea or angina), those with LV dysfunction (ejection fraction <50%), or patients undergoing other cardiac surgery. Surgical treatment of aortic regurgitation is reasonable in cases of significant LV

CONT. dilatation (end-systolic diameter >50 mm or indexed end-systolic dimension >25 mm/m^2). Aortic valve repair without valve replacement may be performed in centers of expertise. Follow-up of asymptomatic patients is based on severity of regurgitation and other factors (see Table 23).

Medical therapy, preferably with dihydropyridine calcium channel blockers (nifedipine, isradipine, felodipine, nicardipine, nisoldipine, lacidipine, and amlodipine), ACE inhibitors, or angiotensin receptor blockers, is recommended in patients with chronic aortic regurgitation in the setting of hypertension. In the absence of hypertension, medical therapy is appropriate for symptomatic patients who are not surgical candidates.

KEY POINTS

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

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 20) and pure regurgitation in a small minority of patients (2%-10%). 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. More than one third of those older than 70 years with severe aortic stenosis have an underlying bicuspid valve.

A bicuspid aortic valve is often accompanied by abnormalities in the aortic arch, independent of the severity of aortic stenosis or regurgitation, and may be associated with aneurysms, dissection, or coarctation. Therefore, in patients with a bicuspid aortic valve, the aortic arch should be examined for aortopathy with CMR imaging, echocardiography, or cardiac CT; serial imaging is indicated if abnormalities are detected. The imaging modality and frequency depend on several factors, including the location and severity of the abnormalities, age of the patient, family history, and candidacy for surgery (see Diseases of the Aorta). Importantly, bicuspid aortic valve disease is heritable, and first-degree relatives should be screened for its presence with echocardiography.

Management of bicuspid aortic valve disease depends on the predominant lesion type (aortic stenosis or regurgitation) and its severity. In patients with a bicuspid valve who are undergoing surgery for severe aortic stenosis or regurgitation, surgical repair of the ascending aorta is advised when the aortic diameter is greater than 4.5 cm. In the absence of surgical indications for a stenotic or regurgitant aortic valve, surgical repair of the ascending aorta or aortic sinuses is advised when the aortic diameter is greater than 5.5 cm or when the diameter is greater than 5.0 cm with additional risk factors for dissection (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 severity of the valvular disease.

Mitral Stenosis

Clinical Presentation and Evaluation

The leading cause of mitral stenosis is rheumatic heart disease, which has a higher predilection for women than men (female-to-male ratio of 4:1). Although relatively uncommon 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 23**). Other causes of mitral stenosis are parachute mitral valve, chest radiation, 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 a slow progression over decades, with gradual enlargement of the left atrium (LA) and preservation of LV function. Symptoms