

Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 8e >

Chapter 54: Valvular Emergencies

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INTRODUCTION

In contrast to the dramatic symptoms associated with acute valvular dysfunction, most valvular heart disease encountered in the ED is chronic and incidentally noted on exam. Adaptive responses preserve cardiac function and can delay the diagnosis of chronic valvular disease for decades but contribute to eventual cardiac dysfunction. Compared with the general population, patients with clinically evident valvular heart disease have a 2.5-fold higher death rate and a 3-fold increased rate of stroke.¹

THE NEWLY DISCOVERED MURMUR

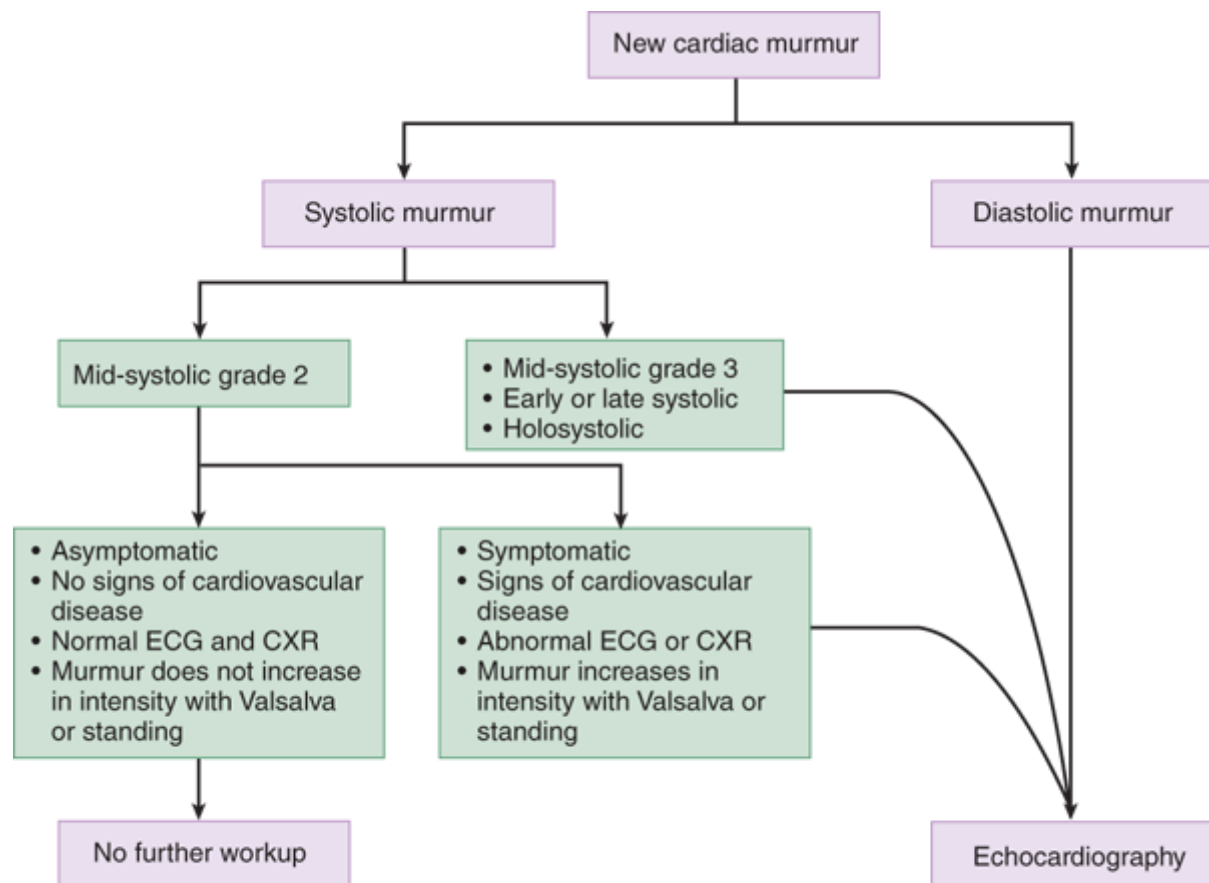
After discovering a new murmur, the first step in the ED is to determine the clinical significance. Benign or physiologic murmurs do not cause symptoms or findings compatible with cardiovascular disease; they are generally soft systolic ejection murmurs that begin after S_1 , end before S_2 , and are not associated with abnormal heart sounds. Systolic murmurs may be associated with anemia, sepsis, volume overload, or other conditions causing an increased cardiac output. Evaluation and treatment are focused on the underlying trigger rather than the murmur itself. Patients without chest pain, dyspnea, fever, or other signs attributable to valvular disease do not need emergent echocardiography but should be referred for eventual imaging.

Any diastolic murmur or new systolic murmur with symptoms at rest is pathologic and warrants emergent echocardiographic imaging. Patients with syncope from suspected aortic stenosis require admission for monitoring and echocardiography ([Figure 54-1](#)). [Table 54-1](#) presents a grading system for murmurs. Another consideration in the newly diagnosed murmur is the possibility of endocarditis, especially in suspected valvular insufficiency (see [chapter 155](#), "Endocarditis").

TABLE 54-1
A Grading System for Murmurs

Grade	Description
1	Faint, may not be heard in all positions
2	Quiet, but heard immediately with stethoscope placement onto the chest wall
3	Moderately loud
4	Loud
5	Heard with stethoscope partly off the chest wall
6	Heard when stethoscope is entirely off the chest wall

FIGURE 54-1.
Algorithm for evaluation of newly discovered systolic murmur. CXR = chest x-ray.



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MITRAL STENOSIS

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Mitral stenosis prevents normal diastolic filling of the left ventricle. Despite declining frequency, rheumatic heart disease remains the most common cause worldwide. Rheumatic carditis causes fusion of valvular commissures, matting of chordae tendineae, and eventual calcification and limited mobility of the valve. Valvular obstruction is slowly progressive, often with 20 to 40 years before onset of symptoms. Mitral valve obstruction causes left atrial pressure to rise, resulting in left atrial enlargement, pulmonary congestion, pulmonary hypertension, and frequently

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atrial fibrillation. In severe disease, pulmonary hypertension may lead to pulmonic and tricuspid valve incompetence, pulmonary edema, right-sided heart failure, and bronchial vein rupture.

Mitral annular calcification is a slowly progressive nonrheumatic cause of mitral stenosis. It is more common among women, elderly, and those with hypertension or with chronic renal failure.² Due to its slow progression, mitral annular calcification rarely causes severe symptoms.

CLINICAL FEATURES

Exertional dyspnea is the most frequent presenting complaint in patients with mitral stenosis. It is often precipitated by anemia or infection, which increases cardiac demand. Orthopnea and premature atrial contractions are also common. Symptoms associated with left- or right-sided heart failure may occur with more severe obstruction. Systemic emboli are a risk, especially when accompanied by atrial fibrillation. Due to earlier recognition and treatment, hemoptysis is a rare presenting symptom, although it can be massive.

Signs of mitral stenosis include a mid-diastolic rumbling murmur with crescendo toward S_2 . With the onset of atrial fibrillation, the presystolic accentuation of the murmur disappears. Typically, the S_1 is loud and is followed by a loud opening snap that is high-pitched and best heard to the right of the apex ([Table 54-2](#)). The apical impulse is small and tapping due to an underfilled left ventricle. Systemic blood pressure is typically normal or low. If pulmonary hypertension is present, signs may include a thin body habitus, peripheral cyanosis, and cool extremities due to compromised cardiac output. Auscultatory findings are less obvious.

TABLE 54-2

Comparison of Heart Murmurs, Sounds, and Signs

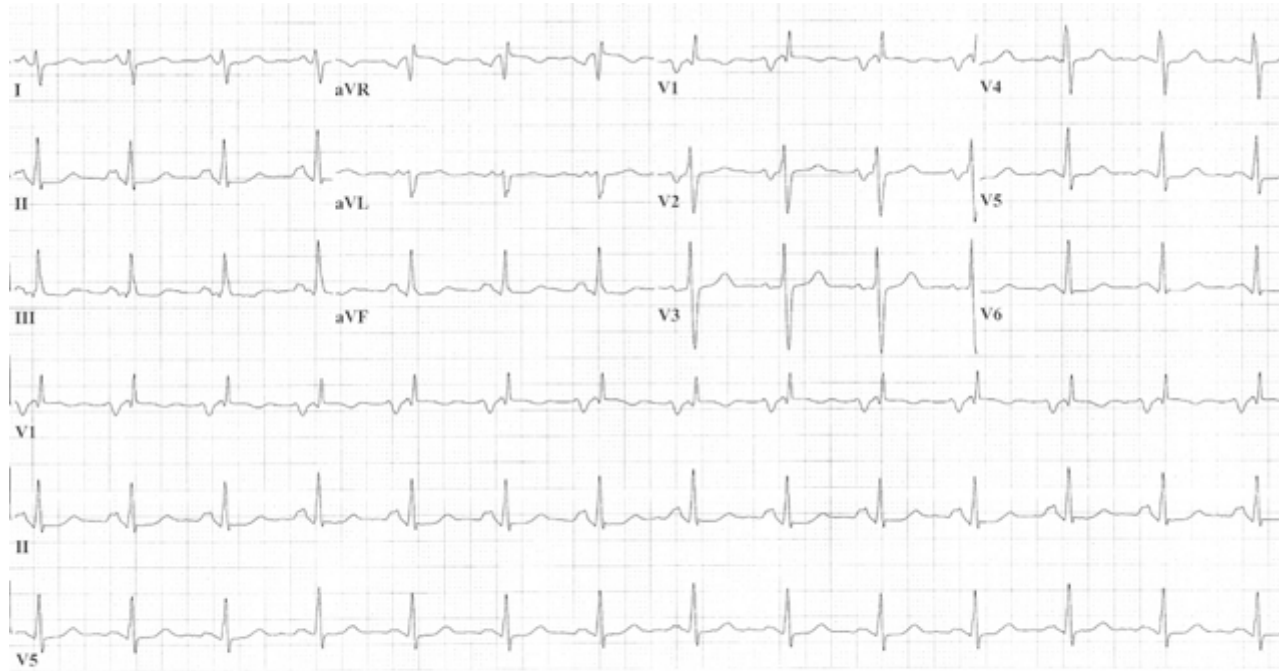
Valve Disorder	Murmur	Heart Sounds and Signs
Mitral stenosis	Mid-diastolic rumble, crescendos into S ₂	Loud snapping S ₁ , small apical impulse, tapping due to underfilled ventricle
Mitral regurgitation	Acute: harsh apical systolic murmur starts with S ₁ and may end before S ₂ Chronic: high-pitched apical holosystolic murmur radiating into S ₂	S ₃ and S ₄ may be heard
Mitral valve prolapse	Click may be followed by a late systolic murmur that crescendos into S ₂	Mid-systolic click; S ₂ may be diminished by the late systolic murmur
Aortic stenosis	Harsh systolic ejection murmur	Paradoxical splitting of S ₂ , S ₃ , and S ₄ may be present; pulse of small amplitude with a slow rise and sustained peak
Aortic regurgitation	High-pitched blowing diastolic murmur immediately after S ₂	S ₃ may be present; wide pulse pressure

DIAGNOSIS AND TREATMENT

The ECG may demonstrate notched or biphasic P waves and right axis deviation in patients with mitral stenosis ([Figure 54-2](#)). On chest radiograph, straightening of the left heart border, indicating left atrial enlargement, is a typical and early radiographic finding. Eventually, findings

FIGURE 54-2.

The ECG in mitral stenosis, demonstrating left atrial enlargement and right axis deviation. Note abnormal P waves in lead V₂.

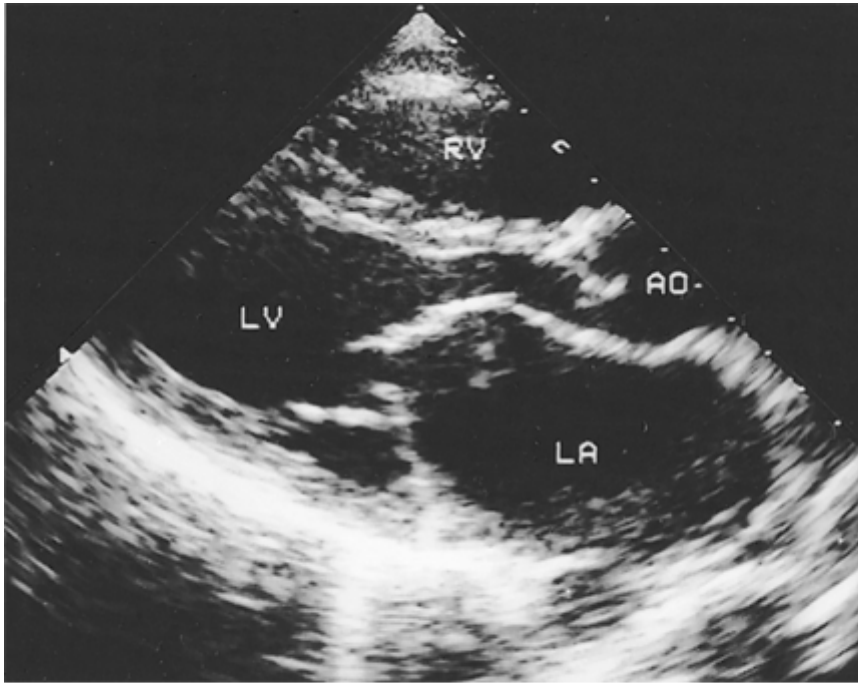


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The diagnosis is confirmed with echocardiography ([Figure 54-3](#)). In candidates for surgical repair, transesophageal echocardiography best assesses the degree of mitral regurgitation and the presence of a left atrial thrombus.³

FIGURE 54-3.

Parasternal long-axis view of mitral stenosis. The left atrium is enlarged, mitral opening is limited, and doming of the anterior mitral leaflet is present. AO = aorta; LA = left atrium; LV = left ventricle; RV = right ventricle. [Reproduced with permission from Fuster V, O'Rourke RA, Walsh RA, Poole-Wilson P (eds): *Hurst's the Heart*, 12th ed. © 2008, McGraw-Hill, New York.]



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Medical management focuses primarily on symptom control and anticoagulation. Patients in atrial fibrillation with rapid ventricular response or with dyspnea on exertion may benefit from heart rate control (see [chapter 18](#), "Cardiac Rhythm Disturbances"). Experts recommend anticoagulation if the left atrial diameter is greater than 55 mm or the patient has atrial fibrillation, a left atrial thrombus, or history of systemic emboli. In patients who present with hemoptysis from mitral stenosis–induced pulmonary hypertension, bleeding may be severe enough to require blood transfusion, emergent bronchoscopy, and consultation with a thoracic surgeon.

The most important ED action for patients with suspected but asymptomatic mitral stenosis is recognition and referral. The primary treatment for symptomatic disease is mechanical intervention, by balloon valvotomy, valve repair, or valve replacement, all optimally performed before the onset of severe pulmonary hypertension.

MITRAL REGURGITATION

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

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Mitral regurgitation occurs when a dysfunctional valve allows retrograde blood flow from the left ventricle into the left atrium during systole. Most patients with mitral regurgitation follow a chronic and slowly progressive course. The most common cause is fibroelastic deficiency syndrome, seen primarily in the elderly.⁴ **Mitral valve prolapse** is another cause of mitral regurgitation and is typically found in younger patients. Secondary mitral regurgitation occurs when a dilated left ventricle causes papillary muscle displacement and valve dysfunction.

In chronic mitral regurgitation, the left atrium dilates to accommodate increasing regurgitant flow to keep left atrial pressure near normal. Stroke volume is augmented, maintaining effective forward flow despite a large regurgitant volume. In contrast, acute mitral regurgitation results in new-onset pulmonary vascular congestion and peripheral edema. Cardiogenic shock may also exist from impaired forward blood flow. **Acute mitral regurgitation** is typically caused by papillary muscle or chordae tendineae rupture from myocardial infarction or valve leaflet perforation from infective endocarditis. Blunt thoracic trauma and spontaneous chordae tendineae rupture are other rare causes.

CLINICAL FEATURES

Symptoms of acute mitral regurgitation are severe dyspnea, tachycardia, and pulmonary edema. Cardiogenic shock or cardiac arrest can develop rapidly. Typical signs include an S_4 gallop and a harsh apical systolic murmur loudest in early or mid-systole, diminishing before S_2 (Table 54-2).

Even with severe symptoms, the murmur may not be audible over breath sounds in the noisy ED environment. A hyperactive apical impulse may be apparent. In the setting of ischemia, chest pain may be masked by overwhelming dyspnea.

Chronic mitral regurgitation can be tolerated for years without symptoms. Eventually exertional dyspnea develops, sometimes associated with palpitations or atrial fibrillation. Signs include a late systolic left parasternal lift and lateral displacement of the apical impulse. A high-pitched holosystolic murmur is best heard in the fifth intercostal space and mid-left thorax and radiates to the axilla. The first heart sound is soft and often obscured by the murmur. An S_3 is usually heard and is followed by a short diastolic rumble, indicating increased flow into the left ventricle. Given its slowly progressive course, signs of systemic thromboembolism may be the first suggestion of mitral regurgitation associated with atrial fibrillation.

DIAGNOSIS AND TREATMENT

Think of acute mitral regurgitation in any patient with new-onset and marked pulmonary edema, especially in patients with near-normal heart size on chest radiograph or in those who do not respond to conventional therapy. Obtain an ECG to look for signs of ischemia, frequently in the inferior or anterior walls. In chronic mitral regurgitation, the ECG may demonstrate left atrial enlargement and left ventricular hypertrophy, and t

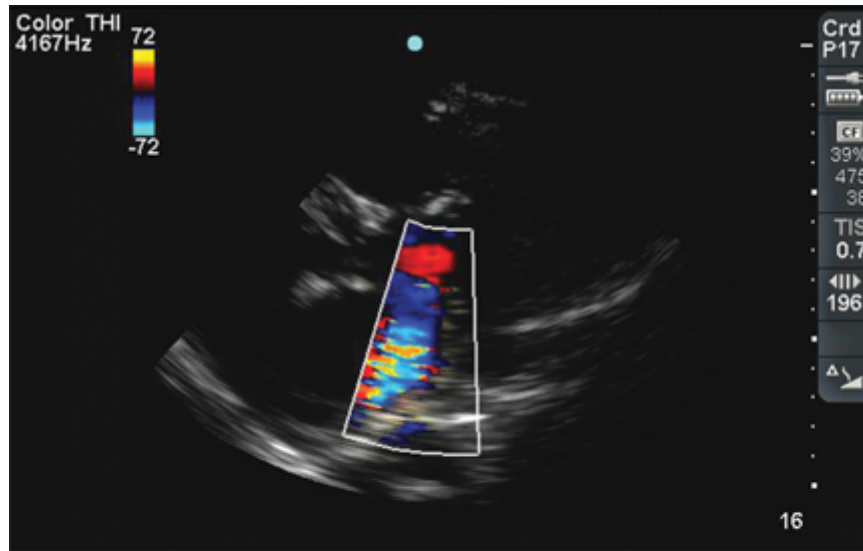
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tricular enlargement that is proportional to the severity of the regurgitant volume. Transthoracic

echocardiography ([Figure 54-4](#)) diagnoses mitral regurgitation, but may underestimate the severity of regurgitation. If transthoracic echocardiography is nondiagnostic, cardiac MRI or transesophageal echocardiography may be undertaken.³ Exercise stress echocardiography may also be useful.

FIGURE 54-4.

An echocardiogram demonstrating severe mitral regurgitation. Color flow Doppler shows regurgitant flow back into the left atrium.



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For acute mitral regurgitation due to papillary muscle rupture, emergency surgery is the treatment of choice. In the ED, start therapy with [oxygen](#) and positive-pressure ventilation for respiratory failure (see [chapter 53](#), "Acute Heart Failure"). Nitrates provide afterload reduction, which results in increased forward flow into the aorta and partially restores mitral valve competence as left ventricular size diminishes. Inotropic therapy with dobutamine may be necessary. Aortic balloon counterpulsation increases forward flow and mean arterial pressure while diminishing regurgitant volume and left ventricular filling pressure.

Treatment of severe mitral regurgitation from acute myocardial infarction includes emergent revascularization.⁵ Endocarditis as a cause of acute mitral valve insufficiency requires specific evaluation and treatment (see [chapter 155](#), "Endocarditis").

Medical therapy may improve regurgitant flow and allow a delay to surgery. The key is emergency cardiology and surgical consultation while
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Medically optimizing care.

In patients with chronic mitral regurgitation, treat acute symptoms. Control atrial fibrillation with rapid ventricular response with β -blockers or calcium channel blockers, and start anticoagulation to avoid embolization. Long-term management is best decided by the primary care provider or a cardiologist.

MITRAL VALVE PROLAPSE

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Mitral valve prolapse is a systolic billowing of one or both leaflets into the left atrium occurring with or without mitral regurgitation. It is characterized by myxomatous degeneration of the valve caused by heritable defects in connective tissue proteins. It is the most common valvular heart disease in industrialized countries, affecting approximately 2.4% of the population.⁶ Morbidity and mortality are greatly influenced by the presence of concomitant mitral regurgitation.⁷

CLINICAL FEATURES

Most patients with mitral valve prolapse are asymptomatic. Associated symptoms can include nonclassic chest pain, palpitations, fatigue, anxiety, and dyspnea unrelated to exertion. Signs such as scoliosis, pectus excavatum, and low body weight can also be associated. If exercise induces symptoms, morbidity increases.⁸

The classic auscultatory finding is a mid-systolic click ([Table 54-2](#)). Maneuvers that decrease preload, such as Valsalva or standing, will cause the click to occur earlier in diastole. Increasing preload by squatting or afterload by hand grips causes the systolic click to move later in systole. A late systolic murmur that crescendos into S_2 is present in some patients.

DIAGNOSIS AND TREATMENT

The diagnosis is unlikely to be made in the ED. The emergency evaluation focuses on late complications such as atrial fibrillation or heart failure. The ECG is usually normal, as is the chest radiograph unless scoliosis or pectus excavatum is seen. If mitral valve prolapse is suspected, refer the patient to a cardiologist for outpatient echocardiography to confirm the diagnosis and to identify any associated mitral regurgitation.⁹

ED treatment is rarely required. Patients with palpitations attributed to mitral valve prolapse may respond to oral β -blockers, but that treatment is best decided by the primary care physician. Antithrombotic therapy is not routinely recommended unless complicated by

transient ischemic attacks, stroke, or atrial fibrillation.^{10,11} Patients with mitral valve prolapse *and* concomitant mitral regurgitation require endocarditis prophylaxis (see [chapter 155](#), "Endocarditis").⁹

AORTIC STENOSIS

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Aortic stenosis is a structural abnormality of the aortic valve that prevents left ventricular outflow. In the United States, the most common cause of adult aortic stenosis is degenerative calcification (calcific aortic stenosis), associated with increasing age, hypertension, smoking, elevated cholesterol, and diabetes.¹² Rheumatic heart disease is a major cause of aortic valve disease worldwide. Bicuspid aortic valves and congenital heart disease are causes as well, especially in younger patients. The prevalence of aortic stenosis is about 3% of patients >74 years old.¹³

The typical course involves a long asymptomatic period, during which the left ventricle hypertrophies to preserve ejection fraction. Ventricular hypertrophy eventually impairs diastolic filling and increases myocardial [oxygen](#) demand. Slowly, aortic valve obstruction worsens, cardiac output diminishes, and systemic blood flow and coronary blood flow are impaired.

CLINICAL FEATURES

The classic triad of aortic stenosis is dyspnea, chest pain, and syncope. However, many patients with severe stenosis (aortic valve area <1.0 cm²) are asymptomatic. Often, a long asymptomatic period is followed by stepwise onset of symptoms starting with dyspnea followed by chest pain, then syncope, and finally signs of heart failure. Once symptoms start, mortality increases.¹⁴ Decreased exercise tolerance and exertional dyspnea or dizziness may be unnoticed or unreported prior to more ominous symptoms.

Classic physical examination findings are a late peaking systolic murmur at the right second intercostal space, radiating to the carotids, a single or paradoxically split S₂, an S₄ gallop, and a diminished carotid pulse with a delayed upstroke (pulsus parvus et tardus; [Table 54-2](#)). Brachioradial delay may also be a useful early finding. Simultaneously palpate the patient's right brachial artery and right radial artery. Any palpable delay between the brachial and radial pulses is abnormal. A narrowed pulse pressure, with or without hypotension, is another important clinical finding.

Aortic stenosis with atrial fibrillation can have dire consequences. Patients with AS typically have diastolic dysfunction and are dependent on [Loading \[Contrib\]/a11y/accessibility-menu.js](#) maintain cardiac output. Without effective atrial contraction, cardiac output drops dramatically, especially if

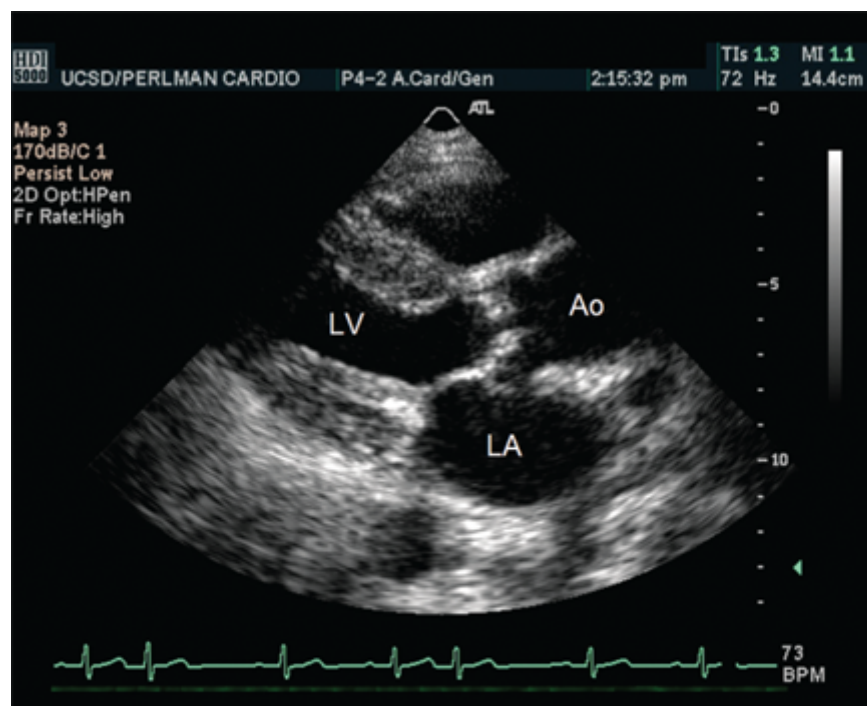
the patient is given [nitroglycerin](#) to treat chest pain or dyspnea.

DIAGNOSIS AND TREATMENT

ECG and chest radiograph findings lack sensitivity and specificity. The ECG usually demonstrates left ventricular hypertrophy, and a left or right bundle-branch block may be present in up to 10% of patients. The chest radiograph is normal early in the disease, but eventually signs of left ventricular hypertrophy and congestive heart failure develop. Transthoracic echocardiography confirms the diagnosis and determines severity ([Figure 54-5](#)). Low-dose dobutamine stress echocardiography may be useful to identify patients with severe aortic stenosis prior to the development of classic symptoms.³

FIGURE 54-5.

Parasternal long-axis plane demonstrating a thickened, stenotic aortic valve. Ao = aorta; LA = left atrium; LV = left ventricle. [Reproduced with permission from Fuster V, O'Rourke RA, Walsh RA, Poole-Wilson P (eds): *Hurst's the Heart*, 12th ed. © 2008, McGraw-Hill, New York.]



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Treat pulmonary edema with [oxygen](#) and positive-pressure ventilation as necessary. Negative inotropic drugs, such as β -blockers or calcium channel blockers, are often poorly tolerated. **Use nitrates, vasodilators, and diuretics with caution** because reducing preload or afterload may cause significant hypotension. New-onset atrial fibrillation may require cardioversion to maintain cardiac output.

Most patients with newly symptomatic aortic stenosis are admitted. Without surgery, 40% to 50% of patients with classic symptoms die within 1 year.^{15,16} Patients discharged from the ED should avoid vigorous physical activity and be seen promptly by a cardiologist. Endocarditis in isolated aortic stenosis is uncommon, and antibiotic prophylaxis is not recommended routinely (see [chapter 155](#), "Endocarditis").⁹

AORTIC REGURGITATION

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

Aortic regurgitation occurs when valve leaflets fail to close fully, causing blood to flow from the aorta into the left ventricle during diastole. The course is usually slowly progressive, with many patients remaining asymptomatic for decades. Over time, aortic regurgitation increases left ventricular wall stress, leading to hypertrophy. Increased stroke volume followed by a rapid pressure drop during diastole causes wide pulse pressures. Tachycardia shortens diastole, which decreases regurgitant volume and mutes symptoms early in the disease. In contrast, increased afterload during stress or isometric exercise exacerbates regurgitant flow and may precipitate symptoms. Over time, the combination of increasing left ventricular dilatation and hypertrophy compromises systolic function, and reduced cardiac output results in symptoms of heart failure.

Among patients in the Framingham Heart Study receiving echocardiography, aortic insufficiency was documented in 13% of men and 8.5% of women, mostly of trace or minor severity.¹⁷ Approximately half of cases are due to valvular leaflet problems from bicuspid aortic valves, infective endocarditis, or rheumatic disease. Nonvalvular causes include aortic dissection, Marfan's syndrome, or aortitis.¹⁸ Aortic regurgitation is also frequently associated with aortic stenosis, and at times, associated regurgitation may be severe.

CLINICAL FEATURES

Acute aortic regurgitation generally presents rapidly, with dyspnea and pulmonary edema being the most common presenting symptoms. To maintain cardiac output, tachycardia develops, but is often inadequate, resulting in cardiogenic shock or cardiac arrest. Sudden-onset ripping or tearing interscapular pain suggests **aortic dissection**. Fever or a history of IV drug abuse suggests endocarditis.

On physical exam, aortic regurgitation is associated with a high-pitched blowing diastolic murmur heard immediately after S_2 , in the second or third intercostal space at the left sternal border ([Table 54-2](#)). In acute disease, the murmur may be inaudible due to tachycardia, tachypnea, and rales. A systolic ejection murmur due to increased stroke volume and an S_3 due to ventricular dilatation may also be heard. In the left lateral decubitus position, a mid-diastolic rumble (Austin Flint murmur) may be appreciated using the bell of the stethoscope at the cardiac apex. Patients often have a widened pulse pressure. The classic "water hammer pulse" (Corrigan pulse) is a peripheral pulse with a quick rise in upstroke due to increased stroke volume followed by collapse from a rapid fall in diastolic pressure. Other classic findings include accentuated precordial apical thrust, pulsus bisferiens, Duroziez sign (a "to-and-fro" femoral murmur), de Musset sign (pulsatile head bobbing), and Quincke sign (capillary pulsations visible at the proximal nail bed while pressure is applied at the tip).

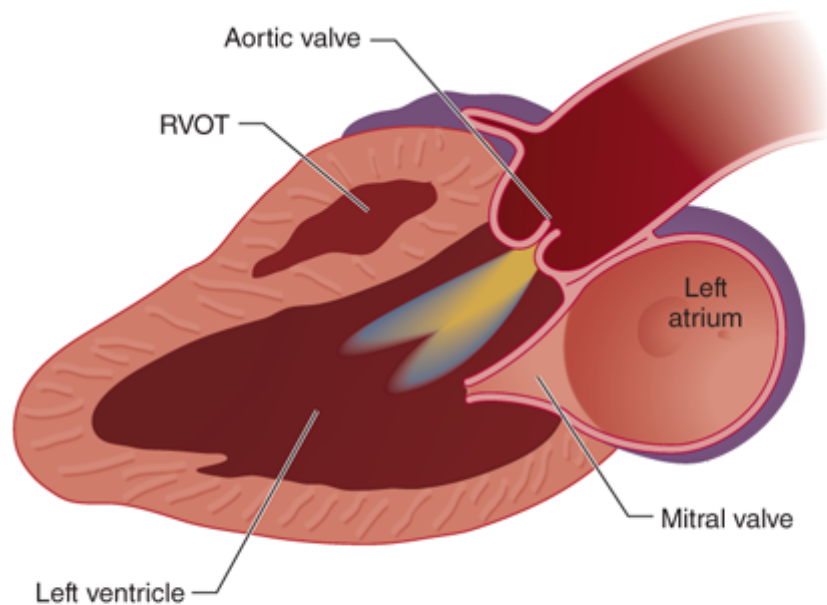
Patients with chronic aortic regurgitation typically present with exertional dyspnea or fatigue. Chest pain may occur from myocardial ischemia due to low diastolic pressures that decrease coronary blood flow. Palpitations may be caused by large stroke volume or premature ventricular contractions. Symptoms of left ventricular failure may occur late in the course of the disease, but greater than one quarter of patients with chronic aortic regurgitation who die or develop left heart dysfunction do so before symptoms occur.⁹

DIAGNOSIS AND TREATMENT

Echocardiography confirms the diagnosis and determines the cause and severity of regurgitation.³ Unstable patients require bedside transthoracic echocardiography ([Figure 54-6](#)). Chest x-ray may show acute pulmonary edema without cardiac enlargement. In aortic regurgitation due to aortic dissection, the chest x-ray may have additional findings associated with dissection such as a widened mediastinum (see [chapter 59](#), "Aortic Dissection and Related Aortic Syndromes"). If aortic dissection is suspected, CT with contrast is needed to assess the aorta, and transesophageal echocardiography is needed to assess valvular function. ECG findings in patients include sinus tachycardia and are generally nonspecific. Ischemic changes or ST elevation may be seen from aortic dissection involving the coronary arteries. In chronic aortic regurgitation, the chest x-ray reveals cardiomegaly, aortic dilatation, and, possibly, evidence of congestive heart failure. The most common ECG abnormality is left ventricular hypertrophy.

FIGURE 54-6.

An illustration of an echocardiogram demonstrating aortic regurgitation. Note regurgitant flow back into the left ventricle. RVOT = right ventricular outflow tract. [Courtesy of P. Lynch, MD, Yale Cardiology Department and Cancer Imaging Program, with permission.]



Left Parasternal Long Axis View

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Acute aortic regurgitation requires immediate surgical intervention. Treat pulmonary edema with [oxygen](#) and intubation for respiratory failure. [Nitroprusside](#), combined with inotropic agents such as dobutamine or [dopamine](#), can augment forward flow and reduce left ventricular end-diastolic pressure. Diuretics and nitrates are usually ineffective. **Although β -blockers are commonly used in aortic dissection, avoid these in acute aortic regurgitation** because they block the compensatory tachycardia that is critical in maintaining cardiac output. Intra-aortic balloon counterpulsation is also contraindicated because it worsens regurgitant flow. Despite intensive medical management, death from ventricular dysrhythmias, pulmonary edema, or cardiogenic shock is common. In patients with only mild acute aortic regurgitation due to endocarditis, antibiotics may be adequate treatment without acute surgical intervention (see [chapter 155](#)).

Chronic aortic regurgitation is usually treated with vasodilators such as angiotensin-converting enzyme inhibitors or dihydropyridine calcium channel blockers.³ If acute symptoms such as pulmonary edema or chest pain occur, admit the patient for stabilization and further management. Patients who become symptomatic, have a low ejection fraction, or have significant left ventricular dilatation are candidates for aortic valve replacement.

EPIDEMIOLOGY AND PATHOPHYSIOLOGY

The incidence of true right-sided valvular heart disease is not known because normal subjects frequently have a small amount of tricuspid and pulmonary valve regurgitation at baseline. Pathologic **tricuspid regurgitation** is usually due to elevated right heart pressure or volume overload, such as from pulmonary hypertension, chronic lung disease, pulmonary embolism, or atrial septal defects. **Tricuspid stenosis** is rare and is generally accompanied by regurgitation. The **pulmonic valve** is the least likely valve to be affected by acquired disease. Most pulmonic valvular disease is congenital (see [chapter 126](#), "Congenital and Acquired Pediatric Heart Disease"), although pulmonary hypertension, rheumatic heart disease, and carcinoid syndrome can rarely cause some degree of pulmonic valve disease. Acute onset of symptomatic tricuspid disease is most often due to endocarditis. Tricuspid valve endocarditis typically involves aggressive organisms, such as *Staphylococcus aureus*, which can cause rapid valve destruction.

CLINICAL FEATURES

Clinically significant right-sided valvular disease causes signs and symptoms of right heart failure such as jugular venous distention, peripheral edema, hepatomegaly, splenomegaly, and ascites. Exertional dyspnea is often the first symptom in patients with right-sided valvular disease associated with pulmonary hypertension. Patients with tricuspid valve regurgitation from endocarditis are often acutely ill with signs of sepsis. The murmur of tricuspid valve regurgitation is soft, blowing, and holosystolic. It is best heard along the lower left sternal border and increases with inspiration. A systolic waveform in the jugular vein, hepatic pulsations, and systolic eyeball propulsion may be seen in severe tricuspid incompetence. Tricuspid valve stenosis is associated with a rumbling crescendo-decrescendo diastolic murmur occurring just before S_1 . This murmur is best heard along the lower left sternal border, increases with inspiration, and is often preceded by an opening snap.

Pulmonic stenosis often presents with exertional dyspnea, syncope, chest pain, and the signs and symptoms of right heart failure. There is a harsh systolic murmur, best heard in the left second intercostal space, which increases with inspiration. A loud ejection click may precede the murmur, and an S_4 is often heard. Pulmonic regurgitation is associated with a high-pitched and blowing diastolic murmur (Graham Steell murmur), which increases in intensity during inspiration and is best heard over the left second and third intercostal spaces. An S_3 gallop is often present. Patients with severe right-sided valvular disease typically have a palpable right ventricle thrill or heave.

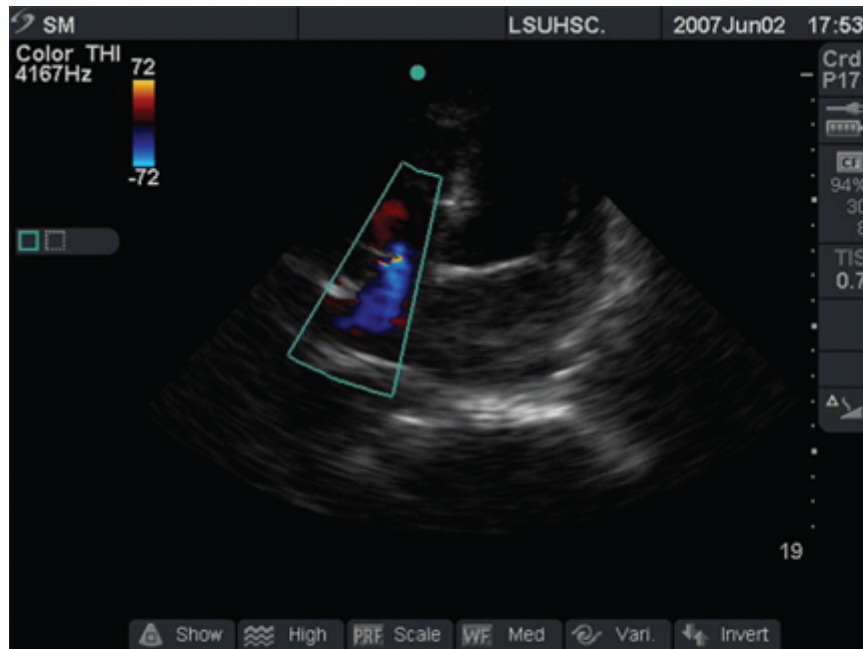
DIAGNOSIS AND TREATMENT

The diagnosis of right-sided valvular heart disease requires echocardiography ([Figure 54-7](#)), with transesophageal echocardiography being more sensitive. Chest radiography and ECG findings lack sensitivity or specificity. Chest radiography may show signs of right

atrial and ventricular enlargement. In pulmonic stenosis, there may be dilatation of the left pulmonary artery. ECG may demonstrate right atrial enlargement and signs of right ventricular hypertrophy.

FIGURE 54-7.

An echocardiogram demonstrating tricuspid regurgitation. Note regurgitant flow into the right atrium.



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Treatment of right-sided valvular heart disease is aimed at the underlying cause. Treat those with endocarditis with antibiotics. Patients with functional tricuspid or pulmonic regurgitation should receive treatment aimed at the underlying cause of the pulmonary hypertension or right-sided failure. Diuretics treat the effects of elevated venous pressure, such as lower extremity edema, ascites, and hepatic congestion, but use with caution to avoid volume depletion or electrolyte abnormalities.¹⁹ Patients with symptomatic pulmonic or tricuspid stenosis may be candidates for balloon valvotomy, and those with severe tricuspid regurgitation due to a structural valve abnormality may require valve replacement.

PROSTHETIC VALVE DISEASE

Prosthetic valves are divided into two basic groups: mechanical and bioprosthetic. Mechanical valves are more durable with lower failure rates but have a higher risk for thromboembolic complications. Life-long anticoagulation is necessary to reduce the thrombotic risk. Bioprosthetic valves, from porcine, bovine, or human sources, are less thrombogenic but are more likely to fail and require repeat surgery.

Systemic thromboembolism is a common complication of mechanical heart valves. Without anticoagulation, the risk of valve thrombosis or thromboembolism is about 8%, and falls to 1% to 2% per year with anticoagulation.^{20,21} Embolic risk is highest during the first 3 postoperative months, and emboli are more common from mitral rather than from aortic valves. Antiplatelet therapy is recommended for all patients with prosthetic valves. In all patients with mechanical valves, lifelong anticoagulation is recommended.¹⁰ The rate of bleeding complications is dependent on the type and intensity of anticoagulation. Major bleeding complications from [warfarin](#) occur in approximately 1.4% of prosthetic valve patients per year.²²

Prosthetic valves may malfunction in a number of ways, including thrombosis, dehiscence of sutures, gradual degeneration, or even sudden fracture. Symptoms are often slowly progressive, but in acute failures, severe symptoms and death may occur before corrective surgery can be accomplished.

Prosthetic valve endocarditis occurs in up to 6% of patients within 5 years of surgery.²³ Early cases (within the first year) are more commonly caused by *Staphylococcus epidermidis* and *S. aureus*.²⁴ Late cases of endocarditis are caused by similar organisms as those affecting native valves.²⁴ The most frequent organism is *Streptococcus viridans*, but *Serratia* and *Pseudomonas* are also implicated. Regardless of source, prosthetic valve endocarditis carries a high mortality rate.²⁵

CLINICAL FEATURES

Although valve replacement relieves valvular obstruction and regurgitation, cardiac remodeling persists, and many patients have persistent cardiac symptoms after valve replacement. Long-standing volume or pressure overload leads to ventricular dysfunction, and many patients continue to have dyspnea and symptoms of heart failure. Patients are also likely to have concomitant coronary artery disease, systemic hypertension, or atrial fibrillation.

Symptoms of prosthetic valve dysfunction depend on the type and location of the valve. Patients with prosthetic valves experience some symptoms specific to the presence of the artificial valve. Thromboembolism may cause systemic symptoms such as transient neurologic symptoms, amaurosis fugax, or self-limited ischemic episodes in the extremities or organs. Major embolic events include stroke, mesenteric

infarction, or sudden death. Prophylaxis against thrombotic complications of prosthetic valves with systemic anticoagulation may cause major bleeding, with hemorrhagic stroke being the most common lethal bleeding complication.

Acute onset of respiratory distress, pulmonary edema, and cardiogenic shock may be associated with mechanical valve failure, tearing of a bioprosthesis, or a large clot obstructing the valve or preventing closure. Failures often result in sudden death before corrective surgery can be done. A paravalvular leak also presents with congestive heart failure. The severity of symptoms is dependent on leak size and how rapidly the leak develops. Slowly progressive development of heart failure may occur with gradual accumulation of a prosthetic valve thrombus.

Patients with bioprostheses usually have a normal S_1 and S_2 , with no abnormal opening sounds. Mechanical valves normally have a loud, clicking, metallic sound associated with valve closure. Systolic murmurs of prosthetic aortic valves are common, but loud diastolic murmurs should be considered pathologic. A "quiet" mechanical valve is concerning. A loud holosystolic murmur indicates prosthetic mitral valve dysfunction. Aortic bioprostheses usually cause a short mid-systolic murmur, and mitral bioprostheses may cause a short diastolic rumble.

DIAGNOSIS OF PROSTHETIC VALVE DYSFUNCTION OR COMPLICATIONS

Think of potential prosthetic valve dysfunction in any patient with a valve replacement and new or progressive dyspnea, congestive heart failure, decreased exercise tolerance, or chest pain. Suspect thromboembolism, septic embolism, or intracranial hemorrhage in any patient with a prosthetic valve and new focal neurologic deficit. Finally, consider endocarditis in prosthetic valve patients with persistent fever or fever without a clear source.

Echocardiography is the diagnostic test of choice. Chest radiography lacks sensitivity and specificity but may show a change in valve position or signs of heart failure. Obtain a head CT in patients with focal neurologic deficits to evaluate for hemorrhage or embolic stroke. Patients on [warfarin](#) may require a complete blood count and coagulation studies. Obtain blood cultures for suspected endocarditis.

TREATMENT AND DISPOSITION

Emergency treatment for acute prosthetic valve dysfunction requires cardiology and cardiothoracic surgery consultation. Emergent surgery and thrombolytic therapy are potential therapies for acute valve thrombosis. Lesser degrees of obstruction should be treated by optimizing anticoagulation. Obtain consultation before discharging a patient with suspected prosthetic valve dysfunction.

REVERSAL OF ANTICOAGULATION WITH PROSTHETIC VALVES

Management of patients with prosthetic valves in the ED requires knowledge of the recommendations on anticoagulation and reversal of excessive anticoagulation. Mechanical mitral valves require an INR of 2.5 to 3.5, whereas bileaflet mechanical valves in the aortic position require an INR of 2.0 to 3.0.^{3,10} [Aspirin](#) is recommended for all patients with prosthetic valves—mechanical or bioprosthetic.¹⁰

For the emergency physician, the greatest dilemma regarding anticoagulation is not who should be on anticoagulation, but how to treat supratherapeutic anticoagulation with or without bleeding. An INR >5 poses a significant risk of excess bleeding, but rapid changes in anticoagulation pose an equally ominous risk of valve thrombosis and thromboembolism. Patients with an INR of 5 to 10 without bleeding may be treated by withholding [warfarin](#) or administering 1.0 to 2.5 milligrams of oral vitamin K. **Patients with severe bleeding complications are best treated with fresh frozen plasma or prothrombin complex concentrate.**³ **Avoid parenteral, high-dose vitamin K due to risk of overcorrection.**⁹

PREGNANT WOMEN WITH VALVULAR DISEASE

The changes in cardiovascular physiology during pregnancy may be poorly tolerated by patients with underlying valvular disease. Increases in cardiac output and blood volume accentuate the murmurs associated with mitral or aortic stenosis. Regurgitant aortic or mitral murmurs may be attenuated due to pregnancy-associated decreases in systemic vascular resistance. In general, asymptomatic mild lesions are well tolerated and pose low maternal and fetal risks. However, more pronounced disease that produces symptoms or is associated with pulmonary hypertension or left ventricular dysfunction is risky and requires high-risk obstetric follow-up. Neonatal complications associated with symptomatic maternal valvular lesions include prematurity, intrauterine growth retardation, respiratory distress syndrome, intraventricular hemorrhage, and death.²⁶

Patients with mild valvular heart disease can often be managed medically during pregnancy. Those with moderate or severe stenotic valvular heart disease may require balloon valvotomy or surgery. Patients with aortic or mitral regurgitation who have severe symptoms refractory to medical management may also require surgery. Endocarditis prophylaxis is unnecessary in patients with valvular heart disease when undergoing vaginal or cesarean delivery.⁹

Pregnant women with valvular heart disease may present in extremis, with dyspnea, pulmonary edema, angina, or syncope. These are generally treated in a similar fashion as discussed above. Pregnancy is associated with a hypercoagulable state due to changes in circulating hormone levels as well as venous stasis. Pregnant patients with valvular heart disease or prosthetic valves are at increased risk for thromboembolic events, and anticoagulation is recommended.³ [Warfarin](#) in pregnancy is associated with teratogenic effects, spontaneous abortion, prematurity, and stillbirth. Unfractionated and low-molecular-weight heparins are safe in pregnancy, but require close monitoring. [Heparin](#) therapy should be titrated to maintain an activated partial thromboplastin time ratio of at least twice the control. However, the effectiveness of [heparin](#) thromboembolic prophylaxis is

not well established in pregnant women despite being a common path.^{9,26} Low-dose [aspirin](#) (81 to 162 milligrams) once daily is recommended during the second and third trimesters for pregnant patients with mechanical or bioprosthetic valves.

PRACTICE GUIDELINES

Practice guidelines include the 2014 American College of Cardiology/American Heart Association Guidelines for the Management of Patients with Valvular Heart Disease³ and the Joint Task Force on the Management of Valvular Heart Disease of the European Society of Cardiology and the European Association for Cardio-Thoracic Surgery Guidelines on the Management of Valvular Heart Disease.¹⁹

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USEFUL WEB RESOURCES

American College of Cardiology. Access to practice guidelines, publications, and educational resources—<http://www.acc.org>

American Heart Association. Access to practice guidelines, heart disease statistics, and continuing medical education materials—
<http://www.americanheart.org>

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The Auscultation Assistant. Contains audio files of heart sounds and murmurs—<http://www.med.ucla.edu/wilkes/intro.html>

Yale Echocardiography Atlas. Provides still images and clips of echocardiograms—http://www.yale.edu/imaging/echo_atlas/contents

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