A popular saying holds that if one can hear a heart murmur in the middle of a loud and busy emergency department, then by definition the murmur is significant. Whether or not this is actually true, it does capture the frustration emergency physicians feel when trying to diagnose or manage valvular pathologic conditions with familiar yet limited tools, such as the stethoscope, chest radiograph, bedside ultrasound, and electrocardiogram (ECG). Sometimes the emergency physician makes a first diagnosis of a problematic heart valve by noting a previously unmentioned murmur on examination. At other times, patients present to the emergency department (ED) with known valvular disease. Valvular pathologic conditions may be at the very root of the patient’s chief complaint, for example, in aortic stenosis causing syncope or acute left-sided valve failure leading to hemodynamic collapse. More often, a valvular pathologic condition is a chronic, sideline issue that, nevertheless, needs to be taken into account when managing patient’s other medical needs.
This article focuses on the valve-related issues the emergency physician will face, from the trauma patient with a mechanical valve who may need his or her anticoagulation reversed to the febrile patient with a new murmur.

THE DISORDERED HEART VALVE

Epidemiology and Pathophysiology

In the developing world, rheumatic fever remains an important cause of valvular disease. But elsewhere, antibiotics for streptococcal infections have greatly reduced rheumatic heart disease prevalence, making valvular disease in the medically privileged nations generally either an inherited or congenital issue (ie, related to Marfan-associated dissection or bicuspid aortic valve), or, more often, a result of age-associated wear and tear.

Prosthetic valves

As the population ages, we are more and more likely to encounter patients with valvular disease and the sequela related to repair and treatment of this disease. Valvular disease now accounts for up to 20% of all cardiac surgeries in the United States. There are approximately 40,000 replacement valve operations performed each year in the United States, with more than 80 different types of artificial valves, from mechanical to bioprosthetic.

Aortic stenosis

Left-sided valvular lesions are the most commonly encountered in clinical practice, in part because right-sided valvular lesions have a prolonged latent asymptomatic period. In turn, aortic stenosis is the most common of all valvular diseases in the developed world. It is usually caused by calcification of a congenital bicuspid valve, age-related calcification of a normal aortic trileaflet valve, or rheumatic heart disease. Patients follow a well-defined disease course and rarely become symptomatic until older than 60 years. Once symptomatic with anginal symptoms, syncope, or congestive heart failure, patients have a precipitous decline in their clinical course and have an average survival of 2 to 5 years.

Left-sided regurgitation

Acute left-sided regurgitation can be life threatening and patients often present in extremis. Acute aortic regurgitation is most often caused by infective endocarditis, aortic dissection, or blunt chest trauma. Acute mitral regurgitation is caused by flail leaflet or chordae tendinae rupture caused by infective endocarditis, blunt chest trauma, or myxomatous disease. It can also be caused by a papillary muscle rupture caused by ischemic disease. Regardless of aortic or mitral valve failure, the acute regurgitant volume leads to increased filling pressures and causes massive volume overload. Patients present with the sudden onset of pulmonary edema, hypotension, and cardiogenic shock.

Infective endocarditis

A far less dramatic presentation, although also dangerous, can be seen with infective bacterial endocarditis, which may declare itself with little more than low-grade persistent fevers. The incidence and mortality of infective endocarditis has not decreased over the past 30 years. Although once a streptococcal disease affecting young adults with rheumatic heart disease, it is now predominantly a staphylococcal disease affecting a population with new risk factors. After staphylococci, viridans group streptococci and coagulase negative staphylococci are the most prevalent. Implanted prosthetic valves in an aging population are especially prone to become infected,
as are stenotic or incompetent native valves. Left-sided valves are more frequently infected. Right-sided endocarditis, particularly of the tricuspid valve, is particularly associated with intravenous (IV) drug abuse.\textsuperscript{8,9}

\textbf{Clinical Presentation and Work-up}

\textbf{Aortic stenosis}

Patients with symptomatic aortic stenosis typically present with anginal symptoms, syncope, or congestive heart failure. It is imperative that emergency physicians carefully auscultate for a crescendo-decrescendo systolic ejection murmur in these patients, especially if given a history of aortic stenosis or having a loud murmur. Not being able to hear a harsh systolic murmur in these patients is a cause for alarm. It often signifies critical disease progression because the stenosed valve impedes forward blood flow causing the systolic ejection murmur to become more and more quiet. An ECG may show signs of left ventricular hypertrophy from the chronic disease process. A chest radiograph may show cardiomegaly and increased pulmonary congestion.\textsuperscript{5} A poststenotic aortic dilatation is a characteristic finding.\textsuperscript{10}

\textbf{Left-sided regurgitation}

Patients with an acute left-sided valvular regurgitation, whether from an incompetent aortic or mitral valve, present with an abrupt onset of symptoms, including dyspnea, pulmonary edema, hypotension, and cardiogenic shock. It is a challenging and time-sensitive diagnosis to make and requires a high degree of suspicion. Patients with an acute regurgitation are often tachycardic, tachypneic, and hypotensive with coarse rales and signs of heart failure on examination. Auscultation may yield a soft, early diastolic murmur. Patients with chronic, decompensated symptoms may have signs of left atrial enlargement or left ventricular hypertrophy on ECG or chest radiograph. Those with acute regurgitation will often have a normal ECG. A chest radiograph will show signs of mild pulmonary congestion in patients with chronic conditions and severe pulmonary edema in those with acute disease. All patients with suspected acute regurgitation should have an emergent echocardiogram.\textsuperscript{11}

\textbf{Infective endocarditis}

Patients with acute infective endocarditis often present toxic in appearance with high fevers, chills, and rigors. Subacute infective endocarditis follows a more indolent course, and patients often have poorly localized symptoms with low-grade fevers, fatigue, and malaise. Infective endocarditis will rarely be definitively diagnosed in the ED because of the criteria needed for diagnosis. It must, however, always be suspected in patients with a fever, especially those with a new murmur or valvular risk factors. Fever is the most common vital sign abnormality. Acute phase reactants, such as C-reactive protein and erythrocyte sedimentation rate, are often elevated.\textsuperscript{9}

The modified Duke criteria (Table 1) are the standard criteria used to guide the diagnosis of endocarditis.\textsuperscript{12} The two major criteria are positive blood cultures and evidence of endocardial involvement on echocardiogram. There are also five minor criteria including fever, risk factors, and the classic signs of splinter hemorrhages, Janeway lesions and Roth spots—related to septic emboli to the fingernails, palms, and retina. The classic signs appear in a minority of patients, and their absence does not exclude the diagnosis of infective endocarditis. A firm diagnosis of endocarditis can be made by having either both major criteria, one major criteria and three minor criteria, or all five minor criteria. That sort of certainty, however, can be pursued on the in-patient services. In the Emergency Department, the goal is to entertain the diagnosis and begin the workup. Three sets of blood cultures should be drawn, with at least 1 hour apart between the first and third culture. At least one set should be
drawn from a separate site, given the possibility that patients may only intermittently shed bacteria to become bacteremic. Two sets of blood cultures have a sensitivity of approximately 95%, whereas 3 sets of blood cultures have a sensitivity of approximately 99%. Minor criteria include the classic signs of splinter hemorrhages, Janeway lesions, and Roth spots. These criteria are related to septic emboli to the fingernails, palms, and retina. They appear in a minority of patients, and the absence of these signs does not exclude the diagnosis of infective endocarditis.

### Treatment and Management

#### Aortic stenosis

As previously noted, patients who are symptomatic with aortic stenosis, whether with angina, syncope, or congestive heart failure, have an average survival rate of 2 to 5 years from the onset of symptoms. Many cardiologists and cardiothoracic surgeons will consider valve replacement at the time of symptom onset. These patients should be admitted to the hospital for a formal echocardiogram to better evaluate the stenotic lesion and for the consideration of valve replacement.

In the ED, medical resuscitation of unstable patients with critical aortic stenosis involves aggressive fluid resuscitation because these patients are heavily preload dependent. Diuretics and nitrates to treat congestive heart failure should, thus, be used with caution in these patients to avoid a precipitous drop in systemic vascular resistance. There has been some recent evidence to suggest nitrates may be beneficial to promote contractility by alleviating left ventricular filling pressures and augmenting coronary blood flow. It should be noted that these patients were closely monitored with invasive devices in an intensive-care-unit (ICU) setting and nitrates should be used with caution in the ED. When needed, inotropic medications, such as dopamine and dobutamine, are still more preferable than nitrates.

Surgical valve replacement is the definitive treatment. An intra-aortic balloon pump has been shown to be a useful adjunct in improving the hemodynamic stability of these patients while awaiting surgery. Minimally invasive valve-replacement techniques are becoming increasingly common and are a promising treatment modality in the management of these patients who are often poor surgical candidates.

#### Left-sided regurgitation

The management of acute left-sided regurgitation starts, as with all patients in the ED, with the ABCs: airway, breathing, and circulation. The airway often needs to be managed early in these patients, given the propensity to develop pulmonary edema from cardiogenic shock. Aggressive fluid resuscitation and inotropes to augment forward flow are the mainstays of medical therapy. Intra-aortic balloon pumps are

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Table 1

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<tr>
<th>Modified Duke criteria for the diagnosis of infective endocarditis</th>
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<tr>
<td><strong>Major Criteria</strong></td>
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<tr>
<td>Blood cultures positive for infective endocarditis</td>
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<tr>
<td>Echocardiogram positive for infective endocarditis</td>
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<tr>
<td><strong>Minor Criteria</strong></td>
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<tr>
<td>Predisposition (predisposing heart condition or intravenous drug use)</td>
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<tr>
<td>Fever greater than 38°C</td>
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<td>Vascular signs (ie, septic emboli, Janeway lesions)</td>
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<td>Immunologic signs (ie, Osler nodes, Roth spots)</td>
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<td>Microbiological evidence (ie, cultures, serology)</td>
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contraindicated for acute aortic regurgitation because they may augment the regurgitant volume through the aortic valve. They can be a useful adjunct for acute mitral regurgitation. Surgical valve replacement is the definitive treatment.11

**Infective endocarditis**

Emergency management of patients with suspected infective endocarditis includes drawing blood cultures, stabilizing patients hemodynamically, and starting the appropriate antibiotics. If patients are sent into the ED to rule out endocarditis with positive blood cultures from their outpatient provider, there are specific recommended antibiotic regimens. For undifferentiated patients with a suspicion of infective endocarditis, treatment depends on whether this infected valve is a native valve or prosthetic valve, and if a prosthetic valve, whether it is considered early (<12 months after surgery) or late.

Treatment of native valve endocarditis and late prosthetic valve endocarditis should cover infections caused by staphylococci, streptococci, HACEK (Haemophilus, Actinobacillus, Cardiobacterium, Eikenella, Kingella) species, and Bartonella. Proposed empiric antibiotic regimens include ampicillin-sulbactam or ampicillin-clavulanic acid with gentamicin; or vancomycin with gentamicin and ciprofloxacin. Treatment of early prosthetic valve endocarditis should include coverage of methicillin-resistant Staphylococcus aureus. The proposed empiric antibiotic regimen should include vancomycin with gentamicin and rifampin.9

**ESSENTIAL QUESTIONS**

**How Should the Emergency Physician Approach the New, Asymptomatic Heart Murmur?**

Some murmurs are incidental findings that, nevertheless, need an outpatient follow-up. The American College of Cardiologists (ACC) and the American Heart Association (AHA) recommend that no follow-up is needed if the murmur is midsystolic, grade II or softer, and thought to be innocent or functional by an experienced listener. But an asymptomatic murmur that is diastolic, continuous, holosystolic, late systolic, associated with ejection clicks, or that radiates to the neck or back needs to be evaluated by echocardiography.16 There are no specific recommendations on the timing of this evaluation.

**What are the Most Important Murmurs?**

The answer to this question depends on the clinical context. In older patients who are febrile without an obvious source, any new murmur would be concerning because it could possibly indicate endocarditis. In a febrile IV drug user, a right-sided murmur is particularly telling, again as possibly indicating endocarditis, particularly of the tricuspid valve.

However, in reality there are probably only 3 major murmurs for which an ED physician requires expertise (Table 2). Two of them are the systolic murmurs of aortic stenosis and mitral regurgitation. Both of these murmurs occur with systole (ie,
immediately after or instead of the S1 heart sound). Aortic stenosis is generally heard loudest at the right upper sternal border and radiates to the carotids. Mitral regurgitation is generally heard over the apex of the heart and radiates to the axilla. A third important valvular problem, aortic regurgitation, presents with a blowing diastolic murmur (ie, a soft murmur immediately after or instead of the S2 heart sound). In patients with ripping or tearing back pain, such a soft-blowing murmur after S2 could herald a life-threatening aortic dissection or aneurysm.

**How can the Emergency Physician Determine What Kind of Prosthetic Valve Patients have?**

It is useful to identify the type of prosthetic valve that patients have implanted, especially to guide auscultation for abnormal heart sounds. When the ED physician does not hear the expected sounds on auscultation, he or she must be acutely concerned for valve malfunction. Some rules of thumb include the following: Prosthetic aortic valves should produce a closing click at the end of systole (during S2). Prosthetic mitral valves should produce a late diastolic click (closing as S1 begins). Tissue valve closing sounds mimic native valves, and you may hear a normal S1 and S2 with no murmur.

Patients are instructed to carry cards in their wallets identifying the serial number and the type of valve that they have implanted along with the surgeon and the hospital at which the operation took place. If this information is not available, chest radiography is of immense value in identifying the type of mechanical valve.

The lateral radiograph is the most useful. Bisecting the heart in half and also from the apex to the base divides the lateral chest radiograph into 4 regions: The aortic valve is in the anterosuperior region. The pulmonic valve is in the posterosuperior region. The tricuspid valve is in the anteroinferior region. The mitral valve is in the posteroinferior region. Anteroposterior radiography can be similarly used, but it is less reliable because the valves are closer in proximity to one another. Dividing the heart into 4 regions again: The aortic valve is in the left upper quadrant. The pulmonic valve is in the right upper quadrant. The mitral valve is in the left lower quadrant. The mitral valve is in the right lower quadrant. Chest radiography is also useful in identifying the direction the valve is pointing. For example, the direction of a valve along the aortic outflow tract may suggest that it is an aortic valve.

**When is Prophylactic Antibiotic Therapy Mandated in Patients with Prosthetic Valves?**

The percentage of valves that become infected is 1% to 6%, an incidence rate that has prompted the practice of giving antibiotics prophylactically to patients with artificial heart valves, congenital heart disease, or a history of endocarditis. The AHA and the ACC recently updated their 2008 guidelines regarding such prophylaxis. The AHA/ACC guidelines now recommend a single dose of antibiotic (typically, amoxicillin 2 g by mouth) given 30 to 60 minutes before dental procedures that manipulate the gingival tissue or the periapical region or perforate the oral mucosa. In patients allergic to penicillins, the guidelines recommend a cephalosporin or macrolide (cephalexin 2 g or azithromycin 500 mg, for example). Patients who cannot take oral antibiotics can be given ampicillin, cefazolin, clindamycin, or ceftriaxone doses intravenously.

Routine prophylactic antibiotics are no longer indicated for wound management or for gastrointestinal, genitourinary, or respiratory tract procedures. So, it is safe to perform a bronchoscopy or a transesophageal echo, place a Foley catheter, incise and drain an abscess, or suture up a minor laceration without prior antibiotics. The
AHA/ACC guideline committee does note that some clinicians may wish to continue antibiotic prophylaxis for patients with bicuspid aortic valve, coarctation of the aorta, severe mitral valve prolapse, or hypertrophic obstructive cardiomyopathy.\textsuperscript{18}

**What are the Early Complications Associated with a Recently Implanted Artificial Heart Valve?**

Prosthetic heart valve complications can be divided into early (within 3 months of surgery) and late complications (>3 months from surgery). The most dangerous complication in the early postoperative period is pericardial effusion leading to cardiac tamponade. Cardiac tamponade occurs when fluid accumulates in the pericardium causing compression of the right ventricle during diastole. This condition leads to the bowing of the ventricular septum into the left ventricle causing decreased stroke volume and signs of obstructive shock. Patients classically present with Beck triad of hypotension, jugular venous distension, and muffled heart sounds. A measured pulsus paradoxus of greater than 10 mmHg is suggestive of tamponade physiology.\textsuperscript{19,20}

Bedside ultrasonography by the emergency practitioner can expeditiously diagnose cardiac tamponade before formal echocardiography. Classic echocardiographic signs of tamponade include visualizing right ventricular diastolic collapse and also seeing cardiac alternans, the swinging of the heart within the pericardial effusion. One can also see the pathophysiology behind pulsus paradoxus as previously described and visualize right ventricular dilation with inspiration and compression with expiration. It is also useful to visualize the inferior vena cava (IVC) using M mode under sonography. The increased right atrial filling pressures cause persistent IVC dilation with minimal (if any) respiratory variation.\textsuperscript{21}

The emergency treatment is pericardiocentesis. Postoperative patients, however, are likely to have clotted blood in the pericardial sac that may be difficult to aspirate with a needle. If needle pericardiocentesis is unsuccessful, the patients’ freshly closed midline sternotomy incision can be opened with a scalpel and a gloved hand can be inserted into the pericardium to remove the clot.\textsuperscript{22}

Atrial fibrillation is another complication that often occurs after the operation. It should be managed with antiarrhythmics in conjunction with the patients’ cardiothoracic surgeon.\textsuperscript{20}

A postoperative inflammation of the pericardium can also be seen and is termed postpericardiotomy syndrome. It usually presents 1 to 6 weeks after an operation and patients typically present with fever, pleuritic chest pain, pain worse in the supine position, or shortness of breath. Pleural effusions or cardiomegaly (secondary to associated pericardial effusion) may be seen on chest radiograph. ECG may show diffuse ST elevation and PR depression as this is an inflammatory condition similar to pericarditis. Laboratory values may be remarkable for leukocytosis, elevated sedimentation rate, and elevated C-reactive protein. Treatment is with antiinflammatory medications or corticosteroids.\textsuperscript{20}

**What are the Late Complications Associated with a Long-Standing Artificial Heart Valve?**

Late complications associated with an implanted artificial heart valve relate largely with anticoagulation. Patients that are inadequately anticoagulated may present with heart valve thrombosis. Patients with an acute thrombosis of a heart valve present with signs and symptoms of obstructive shock. The diagnosis is made by echocardiography. The treatment of unstable patients is thrombolysis followed by anticoagulation. Patients unresponsive to thrombolysis must be taken emergently to the operating room.\textsuperscript{23}
Mechanical valves can fracture and prosthetic valves can be worn down. Patients with a leaflet fracture or ruptured valve present in extremis with acute onset shortness of breath, pulmonary edema, and shock. In the ED, these patients need to be emergently intubated and receive fluids, pressors, and inotropes while awaiting definitive surgical management. They should also get an emergent echocardiogram, either in the ED or the ICU.24

*When can Anticoagulation be Reversed in Patients with an Artificial Heart Valve?*

By creating turbulent blood flow and shearing forces, artificial heart valves create a predisposition to form clots (which, as in atrial fibrillation, can then be showered off through the arterial system as emboli). Patients with mechanical valves, thus, need to be on lifelong anticoagulation, with relatively high target international normalized ratios (INRs). The usual range is from 2.5 to 3.5. Only the ball-and-cage model of aortic valve has a lower INR target of 2.0 to 3.0. Bioprosthetic valves, such as porcine valve transplants, are less thrombogenic and, thus, generally only require anticoagulation for the first 3 months.25

Given the high risk for thrombosis, and the correspondingly aggressive anticoagulation, it is not surprising that a major concern with patients with artificial heart valves revolves around the risk for clotting or bleeding events.

Emergent reversal of anticoagulation, in the face of life-threatening gastrointestinal bleeding, trauma, or hemorrhagic stroke, for example, can be accomplished with fresh frozen plasma (FFP). The use of FFP is preferable to the use of vitamin K for fear of creating a hypercoagulable state with high-dose vitamin K and to enable more precise control over lowering the INR to a target level.16

There is a paucity of data on the safety of reversing anticoagulation. Anecdotally, there have been cases of thromboembolism of a valve from a subtherapeutic INR in just 1 to 2 days. For example, one of the authors managed a patient with a subarachnoid hemorrhage and a St. Jude valve whose anticoagulation was reversed for just 24 hours. Within 2 hours of reinitiating anticoagulation with a heparin bridge, the patient developed left-sided facial and arm paralysis, initially raising concern that her hemorrhage was expanding. Her heparin was turned off while emergent head imaging was arranged; this revealed not an expansion of the bleeding, but a new ischemic stroke, likely from the artificial valve.

One retrospective study looked at 28 patients with mechanical valves that needed anticoagulation in the setting of a life-threatening bleed. The patients received vitamin K or FFP, and anticoagulation was withheld for 11 to 19 days. The study noted that none of the patients had thromboembolic complications from their valves, although there were 4 in-hospital deaths from bleeding complications. The conclusion was that the thromboembolic risk is low in patients with prosthetic valves hospitalized for life-threatening bleed when anticoagulation is held.26

*How Safely can Anticoagulation be Held in Patients with an Artificial Heart Valve?*

There may be circumstances when an emergency practitioner is involved in coordinating an elective procedure for a patient and may be asked to withhold anticoagulation. According to the ACC/AHA guidelines, patients with a modern, bileaflet mechanical aortic valve and no other risk factors are at a relatively low risk for valve-related thrombosis. In fact, when these patients are headed for an elective procedure of some sort, the recommended approach is to simply hold warfarin therapy for 2 to 3 days, enough so that INR decreases less than 1.5, and then restart warfarin a day after the procedure, with no heparin bridging.16
The risk of a valve-related thrombosis is much higher for artificial mitral or tricuspid valves. Patients are also considered high risk for thrombosis if they have any mechanical valve associated with additional risk factors, including atrial fibrillation, left ventricular dysfunction, a previous thromboembolism, or a known hypercoagulable condition. For these high-risk patients, warfarin is also held before elective surgery or procedures. But therapeutic heparin dosing is added in when the INR decreases to less than 2 (typically 48 hours before surgery), with heparin stopped only 4 to 6 hours before the procedure and reinitiated as soon afterwards as bleeding stability allows.16

REFERENCES