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33

Acquired Valvular Heart Disease

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DATABASE FOR NURSING MANAGEMENT

Definition, Classification, and Epidemiology

Valvular heart disease continues to be a common source of cardiac dysfunction and mortality. Competent cardiac valves maintain a unidirectional flow of blood through the heart as well as to the pulmonary and systemic circulations. Diseased cardiac valves that restrict the forward flow of blood because they are unable to open fully are referred to as *stenotic*. Stenotic valves elevate afterload and cause hypertrophy of the atria or ventricles pumping against the increased pressure. Cardiac valves that close incompetently and permit the backward flow of blood are referred to as *regurgitant, incompetent*, or *insufficient*. Regurgitant valves cause an elevated volume load and dilation of the cardiac chambers receiving the blood reflux. Valvular dysfunction may be primarily stenotic or regurgitant, or it is a “mixed” lesion, a valve that neither opens nor closes adequately. Valvular heart disease is usually described by the duration of the dysfunction (acute vs. chronic), the valves involved, and the nature of the valvular dysfunction (stenosis, insufficiency, or a combination of stenosis and insufficiency). The degree of cardiac dysfunction is defined by the New York Heart Association's (NYHA) Functional and Therapeutic Classification. Acquired valvular heart disease most commonly affects, and is most symptomatic with, the aortic and mitral valves. This chapter focuses on the mitral and aortic valves, with a brief discussion of tricuspid valve disease. Because the cause of pulmonic disease is primarily congenital, it is not presented (see Chapter 35).

Causes of Acquired Valvular Heart Disease

Rheumatic Heart Disease

Rheumatic fever is the most commonly acquired cause of valvular heart disease in childhood (Khan, 1996). Tissues involved in rheumatic fever include the lining and valves of the heart, skin, and connective tissue (Fig. 33-1). Rheumatic fever results as a complication of group A streptococcal upper respiratory tract infections, occurring in approximately 3% of those with streptococcal pharyngitis 2 to 3 weeks after acute rheumatic fever. Rheumatic fever is an acute systemic, inflammatory disease that occurs as a response to streptococcal infections (Bhola & Gill, 2001). Group A streptococcal throat infection is responsible for initial and recurrent attacks of rheumatic fever. Lymphatic

channels from the tonsils are thought to transmit group A streptococci to the heart.

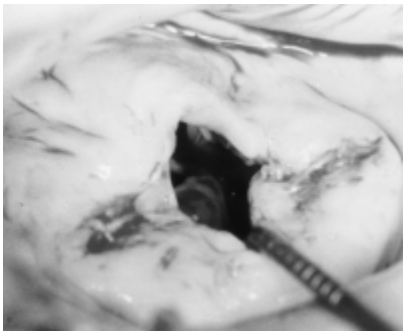


Figure 33-1. Rheumatic mitral valve with leaflet thickening and commissural fusion. (From Alpert, J. S., Sabick, J., & Cosgrove, D. M. [1998]. Mitral valve disease. In E. J. Topol, R. M. Califf, J. M. Isner et al. [Eds.], *Textbook of cardiovascular medicine* [p. 511]. Philadelphia: Lippincott-Raven.)

Although acute rheumatic fever is still common in other countries, it has declined in frequency in the United States since mid century, even though there is a persistently high frequency of streptococcal pharyngitis (Burge & DeHoratius, 1993). Reasons for the decline in rheumatic fever include the use of antibiotics to treat and prevent streptococcal infections, as well as improved social conditions such as decreased crowding, better housing and sanitation, and access to health care. Rheumatic fever persists in underdeveloped countries in which socioeconomic conditions enable the spread of streptococcal bacteria and limit access to adequate health care.

Acute rheumatic fever involves diffuse exudative and proliferative inflammatory reactions in the heart, joints, and skin. Major diagnostic criteria include carditis, polyarthritis, chorea, subcutaneous nodules, and erythema marginatum (pink, circinate skin rash). Manifestations with minor diagnostic importance include arthralgias, fever, acute-phase reactants in the blood (C-reactive protein), elevated erythrocyte sedimentation rate, and a prolonged PR interval on the electrocardiogram (Kaplan, 1998).

Carditis is the most important clinical manifestation of acute rheumatic fever, causing inflammation of the endocardium, myocardium, and pericardium. Myocarditis is characterized by interstitial inflammation that may affect cardiac conduction. Pericardial inflammation may result in a fibrinous exudate and small to moderate amounts of serous fluid in the pericardial sac (Abraham et al., 1991). Endocarditis causes extensive inflammatory changes, resulting in scarring of the heart valves and acute heart failure. Warty lesions of eosinophilic material build-up at the bases and edges of the valves. As the lesions progress, granulation tissue and subsequent vascularization develop, and fibrosis occurs. The annulus, cusps, and chordae tendineae are scarred and, as a

result, they thicken and shorten. Acute heart failure develops because of interstitial myocarditis. Fibrinoid degeneration develops, followed by the appearance of Aschoff nodules, the characteristic pathologic lesion of acute rheumatic fever. As Aschoff nodules heal, fibrous scars remain. In severe cases, death from acute heart failure may result. Carditis frequently does not cause any symptoms and is detected only when the patient

seeks help because of arthritis or chorea.

Auscultatory signs of aortic and mitral insufficiency are frequently apparent. In more than 90% of patients with carditis, the mitral valve is affected. When the mitral valve is affected, there may be a high-pitched, blowing, pansystolic murmur. A Carey-Coombs murmur, a low-pitched, mid-diastolic murmur of short duration, may be noted at the apex. The Carey-Coombs murmur may be attributed to swelling and stiffening of mitral valve leaflets, increased flow across the valve, and alteration in left ventricular compliance. The regurgitation of the aortic valve results in diastolic murmurs, whereas involvement of the tricuspid valve is rarely appreciated during the acute phase (Abraham et al., 1991).

Rheumatic fever can be prevented by aggressive treatment of the initial episode of streptococcal pharyngitis: penicillin G, 500 mg as the first dose and then 250 mg four times daily for a duration of 10 days. If a patient is penicillin allergic, then clarithromycin 500 mg twice daily for 7 to 14 days or clindamycin 150 mg every 8 hours can be substituted (Khan, 1996).

Infective Endocarditis

Infective endocarditis is an endovascular infection that supports continuous bacteremia from the source of the infection, usually a vegetation on a heart valve (Towns & Reller, 2003). Although incidence of infective endocarditis is low, between 1.5 and 6 cases per 100 cases per year, morbidity and mortality are high (Sexton & Spelman, 2003). Rheumatic heart disease, as well as other cardiac lesions such as calcific aortic stenosis, hypertrophic cardiomyopathy, and congenital heart disease, and the presence of prosthetic heart valves predispose to endocarditis. Intravenous drug abusers are at risk for infective endocarditis caused by recurrent bacteremias related to injection from contaminated needles and localized infections at injection sites. Patients with long-term intravenous lines or dialysis catheters are also at increased risk. Acute endocarditis can also occur in normal heart valves from infection somewhere else in the body. In patients with community-acquired, native valve endocarditis, *Staphylococcus aureus* is the predominant cause of acute disease (Karchmer, 1998). Pathogens that are most commonly responsible for subacute endocarditis include streptococci, enterococci, coagulase-negative staphylococci, and the HACEK group of organisms (*Hemophilus* species, *Actinobacillus actinomycetemcomitans, Cardiobacterium hominis, Eikenella* species, and *Kingella kingae*). The *S. aureus* infecting intravenous drug users is frequently methicillin-resistant (Karchmer, 1998). Clinical presentations of endocarditis range from fever and malaise to symptoms related to systemic emboli (Table 33-1).

Table 33-1 CLINICAL MANIFESTATIONS OF INFECTIVE ENDOCARDITIS

Symptoms	Physical Examination Findings

Fever	Fever
Chills and sweats	Changing or new heart murmur
Malaise	Evidence of systemic emboli
Weigth loss	Splenomegaly
Anorexia Stroke symptoms Myalgias	Janeway lesions (small hemorrhages on palms or soles of feet)
Arthralgias Confusion	Splinter hemorrhages (hemorrhagic streaks at fingernail trips)
Congestive heart failure	Osler' nodules (small, tender nodules on finger or toe pads)

The pathologic process of endocarditis requires that several conditions exist to permit infection to grow in the heart and to promote an environment that supports growth on the endocardial surface. For endocarditis to occur, there must be: (1) endocardial surface injury; (2) thrombus formation at the site of injury; (3) bacteria in the circulation; and (4) bacterial adherence to the injured endocardial surface (Chan et al., 1993). The complications of infective endocarditis include congestive heart failure, paravalvular abscess formation, and embolic events to the brain or other organs, sepsis, pericarditis, renal failure, and metastatic abscesses (Sexton & Spelman, 2003). The reduction in mortality for infective endocarditis over the past 30 years from 25% to 30% down to 10% to 20% may be largely related to aggressive surgical intervention in cases complicated by congestive heart failure, invasive abscesses, and prosthetic valve infections (Olaison & Pettersson, 2003).

Blood cultures are an essential diagnostic tool in infective endocarditis. Three separate sets of blood cultures drawn from different venipuncture sites, obtained over 24 hours, usually identify the organism. Patients with infective endocarditis that

remain culture-negative may have fastidious organisms or may have received intravenous antibiotics before blood samples were drawn. In acute endocarditis, antibiotic therapy

should be started after blood cultures have been obtained using strict aseptic technique and optimal skin prep (Towns & Reller, 2003). The clinical approach in acute endocarditis includes appropriate antibiotics and monitoring for complications (Display 33-1). The usual course is 6 full weeks of intravenous antibiotics. Patients who do not respond well to standard antibiotic therapy may be referred for surgical valve replacement (Display 33-2).

DISPLAY 33-1

CLINICAL APPROACH TO ENDOCARDITIS

Establish diagnosis

- Blood cultures
- Physical examination findings
- Echocardiography
- Establish source that seeded endocarditis

Start appropriate antibiotics based on blood cultures

Monitor telemetry for conduction defects

Treat valvular regurgitation with afterload reduction agents

Repeat blood cultures 3 days after antibiotics started to ensure response

Insert long-term intravenous access for antibiotics

Monitor drug levels when appropriate

Monitor for systemic emboli

Echocardiography is frequently used to verify the presence of vegetations on the valves (Fig. 33-2). Transthoracic echocardiography (TTE) is less sensitive than transesophageal (TEE) echocardiography in identifying vegetations (45% to 75% vs. 90% to 94%) (Karchmer, 1998). Transesophageal echocardiography is also useful to identify paravalvular leaks and annular abscesses seen in prosthetic valve endocarditis. Although TEE is more sensitive, some clinicians recommend that TTE be obtained first and to perform TEE only if the TTE images are inadequate or suspicion of infective endocarditis remains high and the initial TTE was negative (Sachdev et al., 2003).

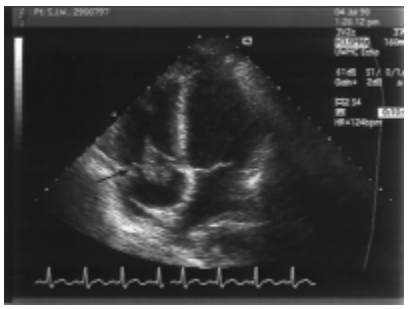


Figure 33-2. Two-dimensional echocardiogram view of vegetation on tricuspid valve in 27-year-old woman with endocarditis (*arrow*).

DISPLAY 33-2

INDICATIONS FOR CARDIAC SURGERY IN INFECTIVE ENDOCARDITIS

Heart failure with hemodynamic instability

Persistent bacteremia and fever despite optimal antibiotic therapy

Paravalvular abscess or fistula

Recurrence of endocarditis after full course of antibiotics

Systemic emboli

Heart failure due to prosthetic valvular dysfunction

Valve dehiscence (in prosthetic valvular endocarditis)

New conduction system defects

Fungal endocarditis

Prevention of endocarditis in high-risk populations, such as those with rheumatic heart disease or structural valve disease, is essential. Patients at risk (Display 33-3) who are undergoing procedures that may cause a transient bacteremia, such as dental or genitourinary procedures, should be treated prophylactically using recommended guidelines (for complete guidelines, refer to *Prevention of Bacterial Endocarditis: Recommendations by the American Heart Association*) (Dajani et al., 1997). The treatment for infective endocarditis is prolonged high-dose antibiotic therapy and valve replacement for those who have evidence of severe valve dysfunction (Chan, 1993).

Miscellaneous Causes of Valvular Disease

In addition to rheumatic fever and endocarditis, there are other causes of acquired valvular heart disease. Degenerative changes of the tissue, such as myxomatous degeneration, calcification, and those associated with Marfan syndrome, can cause valvular dysfunction. Trauma or infection may affect the supportive or subvalvular apparatus. Dilation of the ventricles caused by chronically elevated preloading may dilate an atrioventricular valve opening to the point that the leaflets no longer

approximate and the valve becomes incompetent. Coronary heart disease (CHD) and myocardial infarction (MI) can affect the papillary muscles of the right and left ventricles, causing either dysfunction caused by ischemia or frank flail of atrioventricular valve leaflets caused by papillary muscle rupture. Systemic diseases such as lupus erythematosus and scleroderma may also cause valvular dysfunction.

DISPLAY 33-3

RISK FACTORS FOR INFECTIVE ENDOCARDITIS

Recent dental procedure or periodontal disease

History of congentital heart disease

History of valvular heart disease

Long-term in-dwelling intravenous line

Genitourinary infections or instrumentation

Prosthetic valve (mechanical or biologic)

History of intravenous drug abuse

Hemodialysis

Diagnostic Testing for Valvular Heart Disease

The diagnosis of valvular heart disease is based on patient history, physical assessment, and diagnostic testing. Some tests, such as the ECG and the chest radiograph, may be relatively insensitive in diagnosing valvular heart disease, even though they are part of standard screening tests in patients with heart dysfunction. Diagnostic tests that are more specific and quantitative for valvular dysfunction include echocardiography mode, two-dimensional, Doppler ultrasonography, or transesophageal), right and left heart catheterization, nuclear imaging, and exercise testing (Buccino et al., 1993). Diagnostic findings for specific valvular lesions are noted in the sections discussing each abnormality.

Mitral Stenosis

Cause

The predominant cause of mitral stenosis is rheumatic fever. The mitral valve is the valve most often damaged by rheumatic carditis (Dalen & Fenster, 2000). Rheumatic fever causes thickening and decreased mobility of the mitral valve leaflets associated with fusion of the commissures. Uncommon, nonrheumatic causes of mitral stenosis include malignant carcinoid syndrome, severe mitral annular or leaflet calcification, congenital absence of one of the papillary muscles resulting in a parachute deformity of the mitral valve, neoplasm, endocardial vegetations, and degenerative calcification of an implanted tissue prosthetic heart valve (Fann et al., 1997).

Pathology

The rheumatic process causes the mitral valve to become fibrinous, resulting in leaflet thickening, commissural or chordal fusion, and calcification. As a result, the mitral valve apparatus becomes funnel-shaped with a narrowed orifice. Fusion of the mitral valve commissures results in narrowing of the principal orifice, whereas interchordal fusion obliterates the secondary orifices.

Pathophysiology

The normal mitral valve area is 4 to 6 cm^2. Once the cross-sectional area of the mitral valve is reduced to 2 cm^2 or less, a pressure gradient between the left atrium and left ventricle occurs. The reduced orifice impedes left atrial emptying. Increased left atrial

pressure and dilation occurs along with left atrial hypertrophy in an attempt to maintain normal diastolic flow into the left ventricle. Increased left atrial pressure is transmitted to the pulmonary circuit, resulting in pulmonary hypertension and pulmonary congestion. As the left atrium distends and pressure rises, atrial conduction fibers are stretched, stimulating onset of atrial fibrillation (Chan et al., 1993). Patients have left-sided congestive heart failure (CHF) without left ventricular dysfunction. Mitral stenosis has a sparing effect on the left ventricle. Symptoms of mitral stenosis are usually related to obstruction of the mitral valve rather than ventricular dysfunction. As pulmonary pressure increases, right-sided heart failure may occur.

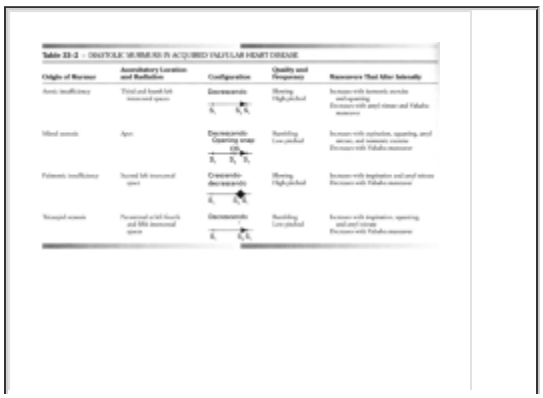
Clinical Manifestations

Women have mitral stenosis four-times more frequently than men do (Braunwald, 1998). Women who had previously been asymptomatic with mitral stenosis may become symptomatic and even experience severe hemodynamic decompensation during pregnancy (Teerlink & Foster, 1998). Most patients remain asymptomatic for several years and may not have symptoms until the fourth or fifth decades of life (Carabello, 1998).

Mild dyspnea on exertion occurs as the most common symptom of mild mitral stenosis (valve area of 1.6 to 2.0 cm^2). As mitral stenosis becomes more severe (valve area of 1 to 1.5 cm^2), dyspnea, fatigue, paroxysmal nocturnal dyspnea, and atrial fibrillation may occur. When mitral stenosis becomes severe (valve area of 1 cm^2 or less), symptoms include fatigue and dyspnea with mild exertion or rest. Patients often have a cough or hoarseness and may have hemoptysis (Khan, 1996). With advanced mitral stenosis, pulmonary hypertension and symptoms of right-sided heart failure occur (i.e., edema, hepatomegaly, ascites, elevated jugular venous pressure). Increased left atrial pressure, atrial fibrillation, and stagnation of left atrial blood flow can result in formation of mural thrombi, with resultant embolic events, including cerebral vascular accidents.

Physical Assessment

In severe mitral stenosis, on auscultation, there are four typical findings, including: (1) an accentuated S_1; (2) an opening diastolic snap; (3) a mid-diastolic rumble noted best at the apex (in sinus rhythm, followed by presystolic accentuation); and (4) an increased pulmonic S_2 intensity associated with pulmonary hypertension (Table 33-2). It usually takes 2 or more years after the rheumatic episode for development of the typical murmur of mitral stenosis (Khan, 1996).



Patients with mitral stenosis may exhibit malar blush (pink discoloration of the cheeks). Patients with severe mitral stenosis may have weak pulses secondary to reduced cardiac output. The apical pulse is tapping in quality and is nondisplaced. A lower left parasternal lift or heave caused by right ventricular hypertrophy may be present. Cardiac rhythm is often irregular, indicating atrial fibrillation.

Diagnostic Tests

Echocardiography is used in the evaluation of mitral stenosis to: (1) quantify the valve area and gradient; (2) quantify the degree of mitral insufficiency; (3) define the degree of left atrial

enlargement; (4) assess mitral annular calcification; (5) assess pulmonary artery pressures and degree of pulmonary hypertension; and (6) evaluate right- and left-sided ventricular function. Transesophageal echocardiography provides better detail of the mitral valve and provides better visualization of atrial thrombus (Brady, 2003).

Cardiac catheterization is used less in diagnosis of mitral stenosis as echocardiography techniques improve. Cardiac catheterization does allow for accurate assessment of valve area and can also identify associated mitral regurgitation. For patients with known or suspected CHD, coronary angiography can delineate coronary anatomy. Right heart catheterization can evaluate right heart and pulmonary artery pressures.

Electrocardiography is nonspecific and does not indicate the severity of mitral stenosis. If the patient remains in sinus rhythm and left atrial enlargement has occurred, characteristic P mitrale (broad, bifid P waves in leads II and V_1) may be identified. Right axis deviation and right ventricular hypertrophy may be noted in severe mitral stenosis. Atrial fibrillation is common in patients with long-standing mitral stenosis and is usually coarse in appearance.

Chest radiography correlates with the degree of mitral stenosis. As mitral stenosis becomes more severe, the chest radiograph demonstrates straightening of the left heart border caused by left atrial enlargement, elevation of the left mainstem bronchus caused by distention of the left atrium, and distribution of blood flow from the lower to upper lobes. Although heart size remains normal, central pulmonary arteries become prominent. Kerley B lines and interstitial edema are often present.

Medical Management

Medical therapy for mitral stenosis is aimed at preventing the complications of systemic embolization and bacterial endocarditis, as well as atrial fibrillation if it occurs (Dalen & Fenster, 2000). Patients who have asymptomatic mitral stenosis require only antibiotic prophylaxis. Patients with mild pulmonary congestion can be managed with diuretics alone. Beta-blockers can be used to reduce heart rate and improve diastolic filling time. When patients have atrial fibrillation, digoxin, beta-blockers, or calcium channel blockers can be

used for ventricular response rate control. Patients with atrial fibrillation require anticoagulation to prevent thrombus formation in the atrium. Once the patient has symptoms of NYHA functional class III or IV despite adequate medical management, mechanical correction of mitral stenosis by balloon valvuloplasty or surgery should be performed.

Interventional and Surgical Management

Percutaneous Mitral Catheter Balloon Valvuloplasty

Percutaneous mitral catheter balloon valvuloplasty is an alternative, less invasive procedure than surgical treatment for mitral stenosis. Balloon valvuloplasty is performed in the cardiac catheterization laboratory by a cardiologist experienced with invasive techniques. A small balloon valvuloplasty catheter is introduced percutaneously at the femoral vein and passed into the right atrium. The catheter is then directed transseptally and positioned across the mitral valve.

Inflation of either one large balloon (23 to 25 mm) or two smaller balloons (12 to 18 mm) stretches the valve leaflets (Fig. 33-3). Separation of the commissures and fracture of nodule calcium are the apparent mechanisms that improve valve movement and function (Braunwald, 1992). The best results from this technique to date have been in patients with rheumatic mitral stenosis with commissural fusion. An echocardiographic scoring system rates leaflet thickening, leaflet mobility,

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calcification, and subvalvular deformity, with a maximum score of 4 in each division. Patients with a total echo score of ≤8 respond most favorably (Palacios et al., 2002). Balloon valvuloplasty has been associated with complications including systemic embolization (1% to 3%), severe mitral regurgitation (3% to 5%), and death (0% to 1%) (Mayes et al., 1999). An atrial septal defect may also occur in as many as 10% of patients undergoing balloon valvuloplasty as a result of the transseptal approach, but this closes or decreases in most patients (Mazur et al., 1999). Results have been promising, with the average gradient reduction being approximately 18 to 6 mm Hg and, on the average, an increase in calculated valve area of 50% to 100%. Mitral balloon valvuloplasty is reserved for patients who continue to be symptomatic despite adequate medical therapy.

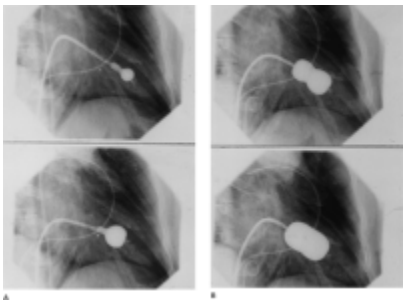

Figure 33-3. Mitral valvuloplasty: Inoue's technique. (*A*) Inflation of distal portion of balloon, which is then pulled back and anchored at the mitral valve. (*B*) Inflation of proximal and middle portions of balloon. At full inflation, the narrowed “waist” of the balloon has disappeared. (From Vahanian, A. S. [1998]. Valvuloplasty. In E. J. Topol, R. M. Califf, J. M. Isner et al. [Eds.], *Textbook of cardiovascular medicine* [p. 2157]. Philadelphia: Lippincott-Raven.)

Balloon valvuloplasty has become increasingly useful in women who experienced hemodynamic decompensation during pregnancy (Teerlink & Foster, 1998).

Surgical Treatment

Surgical replacement of the mitral valve is required when there is severe mitral regurgitation coexisting with mitral stenosis or if the mitral stenosis is not amenable to percutaneous balloon valvuloplasty. Although some valves with mitral stenosis may be repaired by open commissurotomy and reconstruction, heavily calcified rheumatic mitral valves often are beyond the point of repair. The usual prosthetic valve of choice in mitral stenosis is a mechanical prosthesis, because patients already require life-long anticoagulation because of atrial fibrillation. For young women who wish to become pregnant, a bioprosthesis may be recommended.

Tricuspid Valve Disease

Tricuspid regurgitation is primarily “functional” rather than structural and occurs secondary to dilation of the right ventricle and the annulus of the tricuspid valve. Functional tricuspid regurgitation frequently accompanies mitral stenosis and pulmonary hypertension because of the increased pressure and volume load on the right ventricle. Symptoms include signs of right-sided heart failure, large V waves in their right atrial or central venous pressure trace, and pulsatile neck veins. Other causes of tricuspid regurgitation include trauma, infective endocarditis, right atrial tumor, and tricuspid valve prolapse. The murmur of tricuspid regurgitation is a holosystolic murmur heard along the left sternal border and may extend over

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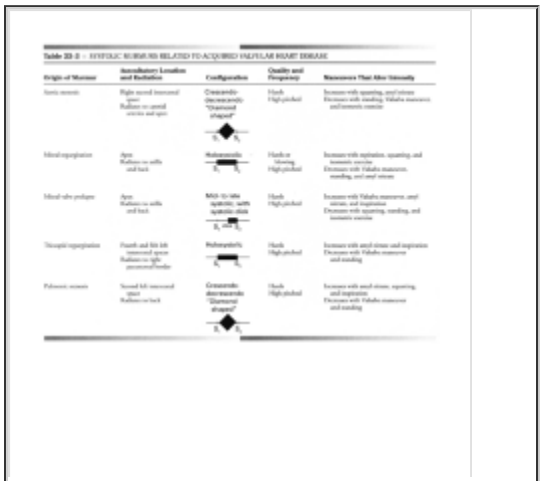
the precordium, sounding like the murmurs of mitral regurgitation and ventricular septal defect (Table 33-3). Patients with mild tricuspid regurgitation normally do not require treatment. Medical treatment is aimed at reducing pulmonary artery pressures and right heart afterload. If tricuspid regurgitation is severe and symptomatic, surgical intervention with annuloplasty repair may be performed. Tricuspid valve replacement is performed only if repair is not feasible or fails (Otto, 1999).



Acquired tricuspid stenosis is uncommon, recognized in approximately 5% of patients with rheumatic heart disease, and usually does not occur without involvement of the mitral valve (Ewy, 2000). Tricuspid stenosis has a pathologic process similar to that of mitral stenosis. The murmur of tricuspid stenosis is comparable to the murmur of mitral stenosis, including an opening snap followed by a diastolic rumble. The murmur of tricuspid stenosis is a diastolic decrescendo murmur along the left sternal border (see Table 33-2). Common symptoms of tricuspid stenosis include fatigue, minimal orthopnea, paroxysmal nocturnal dyspnea, hepatomegaly, and anasarca. Although there is limited experience with balloon valvuloplasty, when tricuspid stenosis is severe, surgical repair with direct commissurotomy and annuloplasty is usually performed at the same time as mitral valve intervention (Otto, 1999).

Prosthetic Valves

Prosthetic cardiac valves have been used since the mid 1960s to treat acquired valvular heart disease. Because no “perfect” prosthetic valve exists, the patient with valvular heart disease is managed medically as long as it is safely feasible. Timing of valve replacement depends on the patient's functional status, ventricular dysfunction, and the natural course of the lesion.

Before a decision is made to use a particular valve, factors in valve design, specifically durability, thrombogenic potential, and hemodynamic properties, are weighed against annulus size and certain clinical conditions such as the desirability of long-term anticoagulation. Table 33-4 summarizes the characteristics considered in selection of prosthetic valves. Because of their proven durability, mechanical valves are most often chosen for patients younger than age 65 to 70 years, unless contraindicated (e.g., previous bleeding

problems, desire to become pregnant, or poor compliance with medication and follow-up). Prosthetic heart valves differ in design, echocardiography image, and radiologic appearance (Fig. 33-4).

Table 33-4 SELECTION OF TYPE OF PROSTHETIC VALVE BASED ON PATIENT CHARACTERISTICS

Biologic Valve	Mechanical Valve
History of bleeding	Age <65 years

Inability to take warfarin	Already on anticoagulation
Desire to become pregnant	History of embolic cerebral vascular accident
History of thrombosis with mechanical valve	History of atrial fibrillation
Age >65 years	

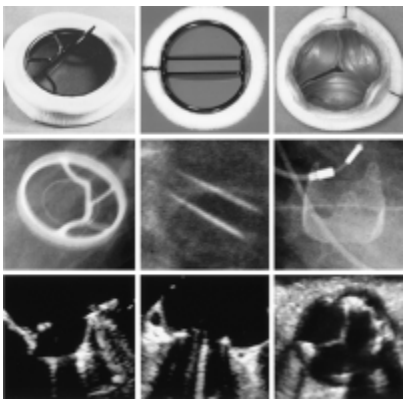


Figure 33-4. radiographic (*middle row*), and echocardiographic (*bottom row*) appearance of prosthetic valves. From left to right: Bjork-Shiley single tilting disk, St. Jude's Medical bileaflet mechanical valve, and Carpentier-Edwards xenograft (radiographs courtesy of Dr. Carolyn van Dyke). (Adapted from Garcia, M. L. [1998]. Prosthetic valve disease. In E. J. Topol, R. M. Califf, J. M. Isner et al. [Eds.], *Textbook of cardiovascular medicine* [p. 580]. Philadelphia: Lippincott-Raven.)

Mechanical Valves

Although the age of patients undergoing valve replacement in the United States continues to increase, mechanical valves dominate the market with a 60% to 40% market share advantage over biological valves (Wernly & Crawford, 1998). Mechanical (nonbiologic) valves have excellent durability but are usually thrombogenic. Bileaflet and tilting-disk valves are the mechanical valves in common use today. Caged-ball valves are used less frequently in the United States but may be used in other areas of the world. In patients with aneurysm or dissection of the ascending aorta, composite grafts of conduit and mechanical valves may be used.

Bileaflet valves, such as St. Jude, ATS (advancing the standard), and the CarboMedic are low-profile valves that have centrally mounted leaflets attached to the seating ring with butterfly hinges. These hinges allow the leaflets to open to 85 degrees, making these valves the least obstructive of the mechanical valves. Made of pyrolytic carbon, these valves produce nearly central flow with little turbulence (Edmunds et al., 1991). The two leaflets swing open in systole, resulting in three separate flow areas (Grunkemeier et al.,

1994). With adequate anticoagulation, thromboembolic risk is low with bileaflet valves.

The *tilting-disk valve* is a low-profile valve consisting of a disk that sits in a seating ring; the flat or convexoconcave disk tilts in response to pressure changes. The Medtronic Hall valve is a tilting-disk valve commonly used today. Tilting-disk valves open to an angle of 60 to 75 degrees in relation to the seating ring. When open, tilting-disk valves produce a minor and major orifice for blood to pass through. Tilting disks have more central flow, but usually more turbulence, than caged-ball valves.

Tilting disks close with an audible click. The technology for production of tilting-disk valves has evolved so that a single piece of metal is used to avoid welded struts. In the past, welded struts fractured and caused fatal results, as did the older Bjork-Shiley convexoconcave valve, which is no longer, manufactured or implanted (Edmunds et al., 1991).

Caged-ball valves have been used since the 1960s and have an excellent durability record. Changes in pressure cause the ball to move forward and back within its caged structure. Flow is directed laterally through the valve rather than centrally. Because of its high profile, the caged-ball valve prosthesis can become obstructive, especially when used in patients with small aortic roots or small left ventricles. The Starr-Edwards and the Sutter (formerly SmeloffCutter) are two of the most common caged-ball valves used. Caged-ball prostheses have been largely abandoned in favor of lower-profile bileaflet valves.

Tissue Valves

Tissue (biologic) valves are characterized by having low rates of thrombotic episodes associated with their use. Porcine or bovine tissue is strengthened and made nonviable by treatment with glutaraldehyde. Homografts are tissue valves from cadavers. They are preserved cryogenically, but are difficult to procure, and their longevity has not been well proven. The main advantages of tissue valves are the associated low rates of thromboembolism and the subsequent decrease in patient morbidity when anticoagulant therapy is not required. Nonthrombogenicity is particularly important for those patients in whom long-term anticoagulation should be avoided, such as children, young adult women, patients older than age 70 years, or people with a history of bleeding.

The Hancock porcine valve, the Medtronic Mosaic porcine bioprosthesis (treated with alpha oleic acid to retard calcification),

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and the Carpentier-Edwards porcine valve are xenografts using porcine aortic valves preserved with glutaraldehyde under pressure, mounted on a stent (Jamieson, 2003). The Carpentier-Edwards pericardial bioprosthesis is made of leaflets fashioned from bovine pericardium fixed without pressure in glutaraldehyde.

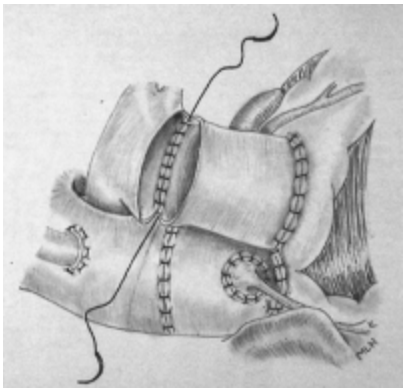
Stentless bioprosthetic porcine xenograft valves such as the St. Jude Medical-Toronto, the Medtronic Freestyle Stentless, and the Edwards Prima Plus porcine bioprosthesis have been developed to improve the durability and enhance the hemodynamic performance of porcine aortic valves. Stentless aortic biological valves were developed secondary to the recognition that conventional bioprosthesis have limitations of long-term durability and residual obstruction that may impede left ventricular mass regression (Goldman & Mallidi,

2003). Because of the structural similarity to aortic allografts, stentless bioprostheses adapt to the aortic root and reproduce the anatomy of the native aortic valve (Luciani et al., 1998). Use of the stentless aortic bioprosthesis has resulted in enhanced survival and hemodynamic superiority (David, 1998). It is expected that reducing mechanical stress on valve leaflets, and the associated degeneration of the bioprosthesis, may be slowed. Thus, stentless xenografts may prove more durable than commonly used stented valves (Luciani et al., 1998).

Homografts or *allografts* from human cadavers are virtually free of any associated thrombosis. They are especially useful in patients with small aortic roots or in patients with active endocarditis. Earlier homografts were preserved with glutaraldehyde and demonstrated early failure. Homografts are now stored “fresh” after harvesting in an antibiotic solution and are then cryopreserved, increasing their longevity to at least 10 years. Valve failure is uncommon and usually the result of progressive valve incompetence (Doty et al., 1998). Even though the homografts are human tissue, there does not appear to be any problem with antigenicity (Whittlesay & Geha, 1991). Aortic allografts have demonstrated excellent freedom from thromboembolism, endocarditis, and progressive valve incompetence (Doty et al., 1998). Because of lack of availability, use of homografts has been limited.

In the *Ross procedure* (also known as pulmonary autograft), the aortic valve is replaced with a pulmonary autograft, and the native pulmonary valve is replaced with a pulmonic allograft. Although this procedure introduced by Donald Ross in 1967 (Elkins, 2003) was originally developed for pediatric application, it has been expanded to adult surgery as well. In patients undergoing the Ross procedure, the native pulmonary valve is excised and then implanted in the aortic position (autograft); a pulmonary homograft (allograft) is implanted into the pulmonic position (Fig. 33-5). The pulmonary autograft has been shown to be resistant to degeneration and calcification (Ross, 1987). Potential clinical and hemodynamic advantages of the pulmonary autograft over the aortic homograft include potential for growth when used in the pediatric population, increased cellular viability, enhanced durability, and possibly internal innervation of the cusps (Santini, 1997). The 30-day mortality for the Ross procedure as reported by the International Ross Registry is 3.3% (140 of 4,197 patients) (International Ross Registry, 2003). The actuarial freedom from pulmonary autograft valve replacement is 90% ± 3% at 13 years (Elkins, 2003). Although the Ross procedure is gaining acceptance, especially in young adults who wish to avoid anticoagulation, there is concern that it offers no better result than the aortic homograft, which is a simpler procedure, with less morbidity. Pulmonary autografts require significantly longer operating time but do not seem to affect early and midterm outcomes compared with aortic homografts (Santini, 1997).

Figure 33-5. Illustration of Ross procedure. Suture line of pulmonary homograft is shown. (From Elkins, R. C. [1998]. Valve repair and valve replacement in children, including the Ross procedure. In L. R. Kaiser, I. L. Kron, & T. L. Spray [Eds.], *Mastery of cardiothoracic surgery* [p. 947]. Philadelphia: Lippincott-Raven.)



Minimally Invasive Valve Surgery

Minimally invasive valve surgery is now used for both aortic valve replacement and mitral valve repair and replacement. Minimally invasive surgical approaches are possible because a wide assortment of technological advances, such as endoscopic and surgical equipment, have been developed. Although patients undergoing minimally invasive valve surgery still require cardiopulmonary bypass, classic median sternotomy may be avoided, thus reducing pain, improving cosmetic results, and expediting recovery. These patients have a lower requirement for erythrocytes, express greater satisfaction, and have lower hospital charges (approximately 20% less than in patients with standard mitral valve and aortic valve approaches) (Cohn et al., 1997). As minimally invasive valve surgery continues to evolve, it will likely become a mainstay in the treatment of valvular heart surgery.

Aortic valve replacement can be performed through an upper “T” mini-sternotomy without intraoperative difficulties. Postoperative pain is reduced and recovery is expedited, with patients discharged to home as early as postoperative day 3 (Izzat et al., 1998). Two minimally invasive techniques for mitral valve surgery have been used: a right parasternal approach (Fig. 33-6) developed at the Cleveland Clinic and a mini-thoracotomy (Chitwood et al., 1997). Compared with

patients with median sternotomy, patients undergoing mitral valve replacement through the right parasternal approach had a shortened length of stay and reduced direct hospital costs (Cosgrove et al., 1998). The mini-thoracotomy approach with video assistance can be used safely in patients undergoing mitral valve repair or replacement; compared with standard techniques, the mini-thoracotomy resulted in less morbidity, earlier discharge, and lower cost (Chitwood et al., 1997). The majority of clinical series demonstrates that minimally invasive port-access approach to mitral valve surgery has low morbidity and mortality, with echocardiographic outcomes equivalent to conventional mitral valve surgery (Sharony et al., 2003). More recently, minimally invasive mitral valve surgery has evolved to include computed-assisted robotic techniques in current clinical trials. The daVinci Surgical System allows the surgeon to operate from a console through an end-affector using micro wrist instruments, which are mounted on robotic arms, inserted through the chest wall (Chitwood, 2003).

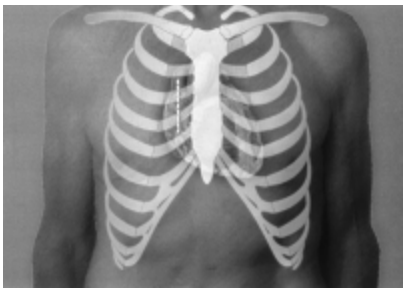


Figure 33-6. Example of one approach to minimally invasive mitral valve surgery. An 8- to 10-cm incision is made from the lower border of the second costal cartilage to the upper border of the fifth costal cartilage. (From Alpert, J. S., Sabick, J., & Cosgrove, D. M. [1998]. Mitral valve disease. In E. J. Topol, R. M. Califf, J. M., Isner et al. [Eds.], *Textbook of cardiovascular medicine* [p. 526]. Philadelphia: Lippincott-Raven.)

Complications of Prosthetic Valves

Thromboembolism remains the most common complication of patients with prosthetic valves. Anticoagulant therapy with warfarin is begun in all patients 48 hours after surgery and is continued for 6 to 12 weeks. All patients with mechanical valves require life-long anticoagulation because of the risk of thrombosis and embolization. The highest thromboembolic risk for mechanical and biologic valves occurs in the first few days to months after implantation, before the valve is fully endothelialized. Even with anticoagulation, the risk of thromboembolism is 1% to 2% per year for patients with mechanical valves (McAnulty & Rahimtoola, 1998). The American Heart Association and the American College of Cardiology recommend INR of 2.0 to 3.0 for mechanical aortic valves and INR of 2.5 to 3.5 for mechanical mitral valves (Goldsmith et al., 2002). Of mechanical valves, the caged-ball valves have the highest rate of thromboembolism, and the St. Jude valves have the lowest (Garcia, 1998). Tissue valves other than homografts also usually require anticoagulation for 6 to 12 weeks after surgery, after which patients have their therapy converted to aspirin. The overall risk of thromboembolism with biologic valves is 0.6% to 0.7% per year (McAnulty & Rahimtoola, 1998). Homografts or the Ross procedure require no anticoagulation.

Prosthetic valvular thrombosis is a serious complication and can result in severe hemodynamic compromise. In patients with prosthetic valves who are not anticoagulated into a therapeutic range, thrombosis of the prosthetic valve can occur. Valve thrombosis can occur with either mechanical or bioprosthetic heart valves but occurs most often in prosthetic valves in the mitral position (Garcia, 1998). Thrombus or pannus formation on the valve may occlude the orifice or entrap the pivoting mechanisms, causing acute stenosis or regurgitation. Symptoms of valve thrombosis include embolic events and CHF. Valve thrombosis can be diagnosed with transesophageal echocardiography (McAnulty & Rahimtoola, 1998). Emergent valve replacement usually is indicated for large thrombi, but if the patient is not a surgical candidate, thrombolytic agents may be used (Garcia, 1998).

The rate of *bacterial endocarditis* is approximately 3% the first year after valve replacement and 0.5% each year thereafter (McAnulty & Rahimtoola, 1998). Although symptoms of

prosthetic valve endocarditis are similar to those of native valve endocarditis, the infection may be difficult to control with antibiotics alone because of prosthetic material involved.

Early prosthetic valve endocarditis (within the first 60 days) carries a high mortality rate of 20% to 70% (Garcia, 1998). Early prosthetic valve endocarditis occurs in less than 1% of valve replacement patients and frequently requires the patient to undergo additional operations (Crawford & Durack, 2003). The most common organism in early prosthetic valve endocarditis is *Staphylococcus epidermidis*. Eighty percent of these staphylococci are methicillin-resistant, suggesting that these infections may be nosocomial in nature (Garcia, 1998). Fever, heart failure, new murmur, and embolic events are common manifestations.

Late prosthetic valve endocarditis (more than 60 days after surgery) occurs most commonly in patients with bioprosthetic valves in the aortic position. Urinary infection, dental procedures, and urologic procedures are the most common sources identified (Garcia, 1998). The incidence is less than 1% per year and is generally caused by the same bacterial species that cause subacute bacterial endocarditis (Crawford & Durack, 2003). For patients who do not respond to antibiotic therapy or who have local invasion of the annulus, embolic events, fungal infection, heart failure, or prosthetic valve dysfunction, repeat valvular replacement is indicated (McAnulty & Rahimtoola, 1998).

Prosthesis malfunction is uncommon for the first 10 years after mechanical valve implantation. The best-known problems with mechanical failures were those affecting the Bjork-Shiley convexoconcave tilting-disk valves first manufactured in 1978, with the peak incidence of valve failure in the 1981 to 1982 models. Subsequent modifications improved the valve area but also increased stress forces. As a result, by 1994, 564 strut fractures were reported in these Bjork-Shiley valves; two

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thirds of those strut fractures were fatal (Garcia, 1998). Although these valves have been withdrawn from the market, approximately 40,000 had been implanted worldwide. Because acute valve strut fracture can be fatal, patients with these valves should be evaluated for partial strut fracture using high-resolution cineradiography. Prophylactic replacement of these valves is not advocated (Wernly & Crawford, 1998).

Valve degeneration is the primary complication of patients with tissue prostheses. Degeneration of biologic prostheses can occur as lipid or calcium deposits cause valve cusps to stiffen and stenose. The incidence of structural failure requiring replacement is approximately 30% at 10 years after valve replacement (Garcia, 1998). Failure of tissue valves often occurs slowly over months to years and presents as progressive heart failure. Prosthetic valve degeneration and failure are most easily diagnosed with echocardiography.

Paravalvular leaks between the prosthetic ring and the annulus occur because of tearing of the suture line, spontaneously or after infection. Presence of a new murmur and signs of heart failure alert the clinician to paravalvular leaks. The patient's clinical course should be followed up; when the leak becomes significant, surgical repair or replacement is indicated. Hemolysis may also accompany paravalvular leaks.

Hemolytic anemia is a consequence of shortened red cell survival time in all patients with prosthetic valves. Movement of the valve ball or disk causes varying degrees of destruction

of the red blood cells. Hemolysis may also occur with paravalvular leak. Commonly, hemolysis is mild and the patient can compensate by increasing red blood cell production. Rarely, hemolytic anemia occurs. Chronic intravascular hemolysis results in loss of iron in the urine; iron deficiency anemia may result after several years.

Mitral Insufficiency

Cause

Mitral insufficiency (also termed *regurgitation*) may be either chronic or acute (Table 33-5). Acute mitral regurgitation is caused by chordal rupture, MI, trauma, myxomatous valvular degeneration, mitral valve prolapse, or endocarditis (Carabello, 2000). Chronic mitral regurgitation may be the result of a number of abnormalities including, but not limited to, rheumatic heart disease, injury after radiation, cardiomyopathies, infiltrative disease, ischemic damage to the subvalvular apparatus, infective endocarditis, myxomatous degeneration, hypertrophic cardiomyopathy, diet-drug–induced lesions, or marked left ventricular dilation (Enriquez-Sarano et al, 2000).

Table 33-5 ETIOLOGIES OF ACQUIRED MITRAL REGURGIATION

Chronic Mitral Regurgitation	Acute Mitral Regurgitation
Rheumatic heart disease	Myocardial infarction causing: Papillary muscle rupture or dysfunction
Ischemia to subvalvular apparatus	
Infective endocarditis	Rupture of chordae
Myxomatous degeneration	Infective endocarditis
Hypertrophic cardiomyopathy	Trauma
	Myxomatous degeneration with chordal rupture
Left ventricular dilation	

Systemic lupus erythematosus	
Marfan's syndrome	
Calcification of annulus	
Ankylosing spondylitis	
Scleroderma	
Ehlers-Danlos syndrome	
Prosthetic paravalvular leak	
Deterioration of prosthetic mittal valve	

Pathology

Primary mitral regurgitation occurs when the mitral valve annulus, leaflets, chordae, or papillary muscles are affected by ischemia, collagen disease, infection, calcification, trauma, or degenerative changes, causing incompetent coaptation of the mitral leaflets. Secondary mitral regurgitation occurs with ventricular dilation when ventricular geometry is changed, causing malalignment of the papillary muscles. Although it is sometimes difficult to distinguish between primary and secondary regurgitation, primary regurgitation is often more severe than insufficiency secondary to annular dilation (Braunwald, 1992).

Pathophysiology

Mitral regurgitation occurs as the result of inadequate closure of the mitral valve, allowing regurgitant flow back into the left atrium during each left ventricular systole. Its severity depends on the volume of regurgitant flow. Regurgitant flow into the left atrium reduces forward flow, stroke volume, and cardiac output (Khan, 1996). Regurgitant flow also increases left atrial pressure, causing left atrial dilation and pulmonary congestion. During diastole, the regurgitant volume returns to the left ventricle and increases its volume load.

In chronic mitral regurgitation, persistent volume overload results in progressive ventricular dilation and mild hypertrophy. Although ventricular dilation and hypertrophy are initially

compensatory, over time, chronic volume overload may result in decreased systolic function of the left ventricle and lead to heart failure (Khan, 1996). In acute mitral regurgitation, neither the left atrium nor the ventricle has had sufficient time to adjust to the increased volume load. Left atrial pressure rises quickly, resulting in pulmonary congestion and edema.

Clinical Manifestations

Patients with acute versus chronic mitral regurgitation vary in clinical presentation and physical examination findings. In acute mitral regurgitation, symptoms progress rapidly. Symptoms are typically those of left ventricular failure. The patient is usually tachycardic to compensate for the reduced forward stroke volume. Patients are dyspneic secondary to pulmonary congestion and edema; they are often orthopneic and have paroxysmal nocturnal dyspnea and poor exercise tolerance. Patients may also have signs of biventricular failure because

right-sided failure may occur secondary to pulmonary hypertension. Patients in acute mitral regurgitation often present to the emergency room with reports of sudden inability to breathe. New-onset atrial fibrillation can occur. Patients with ischemic mitral insufficiency or papillary muscle rupture may also report chest pain.

During the compensatory phase of chronic mitral regurgitation, patients may be relatively asymptomatic for years. Initial signs of mitral regurgitation include exertional dyspnea, orthopnea, paroxysmal nocturnal dyspnea, cough, palpitations, new atrial fibrillation, and lower extremity edema. Symptoms may occur so gradually that patients may present subacutely to the clinic with symptoms as vague as fatigue and inability to sleep.

Physical Assessment

On examination, the most easily noted characteristic of either chronic or acute mitral regurgitation is the holosystolic murmur, which is heard best at the apex and radiates to the axilla (see Table 33-3). The murmur of mitral regurgitation may vary somewhat depending on the underlying cause. Patients may have an S_3 gallop in moderate to severe regurgitation caused by high diastolic flow into the ventricle. An S_4 gallop is uncommon in chronic mitral regurgitation. However, in acute mitral regurgitation, an S_4 gallop is common because the left atrium and ventricle are noncompliant. The patient with rheumatic heart disease may also have a diastolic murmur related to coexisting mitral stenosis.

Because of left ventricular dilation, patients with chronic mitral regurgitation have an easily palpated, left laterally displaced point of maximal impulse. Patients with a markedly enlarged left atrium may have a left parasternal lift because of anterior displacement of the apex. Patients with acute or decompensated chronic mitral regurgitation may be anxious and diaphoretic because of left ventricular failure. Blood pressure may be normal to low and pulse pressure may be narrowed secondary to decreased stroke volume. Jugular venous pressure can be normal or elevated in the patient with right-sided heart failure. Breath sounds can range from basilar crackles to dullness secondary to pleural effusion. In addition, hepatosplenomegaly, hepatojugular reflux, peripheral edema, and ascites may be

present in the patient with right-sided heart failure.

Diagnostic Tests

Transthoracic echocardiography can identify the structural cause of the mitral regurgitation as well as gauge left atrial size, left ventricular dimensions and performance, pulmonary artery pressures, and right heart function. Color flow Doppler allows for assessment of severity of regurgitation. *Transesophageal echocardiography* is better than transthoracic echocardiography for defining mitral valve anatomy and discriminating prosthetic valves and paravalvular leaks.

Cardiac catheterization is used to identify coexisting coronary artery disease and to grade the severity of mitral regurgitation. Left ventriculography can assess left ventricular function and distinguish any wall motion abnormalities. Right heart catheterization quantifies pulmonary artery pressures and allows for evaluation of the large V waves in the pulmonary artery wedge tracing.

Electrocardiography in chronic mitral regurgitation may demonstrate left ventricular hypertrophy and left atrial enlargement or P mitrale (characterized by M-shaped P waves). Atrial fibrillation may occur with acute and chronic mitral regurgitation. Patients with ischemic papillary muscle dysfunction may demonstrate ischemic changes, and patients with papillary muscle rupture can show acute inferior, posterior, or anterior MI.

Chest radiography in chronic mitral regurgitation shows left ventricular hypertrophy and left atrial enlargement. Calcification of the mitral valve annulus and apparatus may also be seen. In acute or decompensated chronic mitral regurgitation, pulmonary vascular redistribution and pulmonary edema can be observed. If the heart is of normal size, the degree of mitral regurgitation is so mild or so acute that eccentric left ventricular hypertrophy has not had time to develop.

Medical Management

Medical therapy for mitral regurgitation is geared toward afterload reduction to promote forward flow and minimize regurgitation back into the left atrium and pulmonary vasculature. In patients with acute or decompensated chronic mitral regurgitation, intravenous vasodilators such as nitroprusside can reduce filling pressures and ventricular cavity size and promote forward flow with afterload reduction. Intravenous diuretics are used to reduce volume overload. In acutely ill patients refractory to medications, intra-aortic balloon counterpulsation can be used further to reduce afterload while maintaining coronary perfusion with diastolic augmentation.

In patients with chronic mitral regurgitation or those in acute heart failure who are being weaned from intravenous inotropes and vasodilators, other afterload-reducing agents, such as angiotensin-converting enzyme (ACE) inhibitors, nitrates, or hydralazine, may be used. Diuretics can treat chronic and acute volume overload. Some practitioners continue to advocate the use of digoxin, especially for patients in atrial fibrillation. In the patient with chronic but compensated mitral valve regurgitation, mitral surgery can be safely deferred or avoided. The patient should be carefully monitored, however, and referred for mitral valve repair or replacement before significant left ventricular dysfunction or pulmonary

hypertension occurs.

Surgical Management

Surgical Intervention

Two surgical approaches are used to treat mitral regurgitation. Mitral valve repair uses reconstructive techniques as well as a rigid prosthetic ring to repair the mitral valve apparatus, thus sparing the valve and avoiding the consequences of valve replacement (Fig. 33-7). Mitral valve replacement involves implantation of a prosthetic valve with attempted preservation of at least part of the mitral valve apparatus (Reardon & David, 1998), which contributes to left ventricular function (Fig. 33-8).

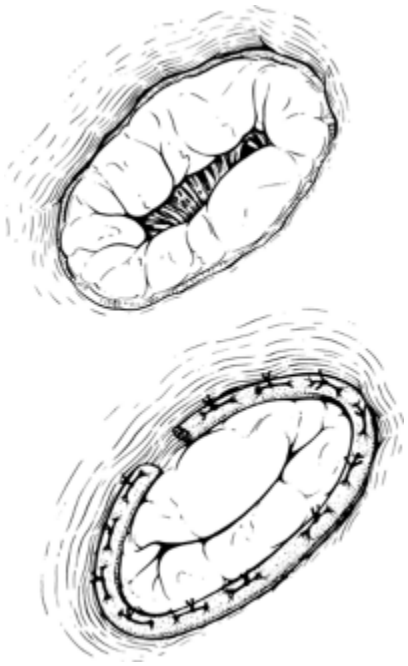

Figure 33-7. (*Top*) Regurgitant mitral valve (note large primary orifice). (*Bottom*) Completed mitral valve annuloplasty with ring sutured in place.

Figure 33-8. Valve replacement with chordal preservation. (From Chitwood, W. R. [1998]. Mitral valve repair: Ischemic. In L. R. Kaiser, I. L. Kron, & T. L. Spray [Eds.], *Mastery of cardiothoracic surgery* [p. 321]. Philadelphia: Lippincott-Raven.)

In patients with chronic mitral regurgitation, mitral replacement should occur before the patient has had irreversible left ventricular dysfunction. Mitral valve replacement or repair can preserve left ventricular function and ejection fraction. Patients with NYHA class II symptoms should be considered for surgery. Factors contributing to increased operative risk

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include reduced left ventricular ejection fraction, increased left ventricular end-systolic volume, older age, concomitant coronary artery disease, previous cardiac surgery, and pulmonary hypertension (Fann et al., 1997).

Mitral Valve Repair

In selected patients, mitral valve repair may be undertaken for patients with mitral insufficiency as an alternative to replacement. Surgical techniques involve reconstructing the leaflets and annulus in such a way as to narrow the orifice. These procedures consist of direct suture of the valve cusps, repair of the elongated or ruptured chordae tendineae (chordoplasty), or repair of the valve annulus (annuloplasty). With an annuloplasty, the incompetent valve is remodeled using a ring prosthesis that is attached to the leaflets and the annulus. Mitral valve repair has demonstrated excellent short-term and long-term results with low perioperative mortality rate (not >2% in most reported series). Carpentier and associates report 94% and 92% freedom from re-operations at 10 and 20 years, respectively (Hampton & Verrier, 2003).

Mitral Valve Replacement

In patients with acute mitral regurgitation secondary to MI, coronary angiography should be performed to define coronary anatomy for concomitant coronary bypass surgery at the time of mitral valve repair or replacement. In patients with acute mitral regurgitation secondary to MI, the mortality rate can be as high as 50% secondary to acute left ventricular failure (Fann et al., 1997).

Mitral Valve Prolapse

Cause

Mitral valve prolapse refers to a number of conditions in which one or both of the mitral valve leaflets becomes superior to the plane of the annulus during systole (Braunwald, 1998). The posterior leaflet is most often affected. Mitral valve prolapse (MVP) is also known as Barlow syndrome or click-murmur syndrome. It is the most common cause of significant isolated mitral regurgitation (Karon, 1997) and has been reported to be one of the most common heart disorders, with an overall prevalence of 2.4% (Playford & Weyman, 2001). Although MVP occurs most commonly in women, with a peak incidence in the fourth decade of life, severe mitral regurgitation associated with mitral valve prolapse is more common in men (Fann et al., 1997). The most common cause of MVP is myxomatous degeneration. Marfan syndrome, Ehlers-Danlos syndrome, rheumatic heart disease, and ischemic papillary muscle dysfunction also cause mitral valve prolapse. In addition, MVP has a hereditary component transmitted as an autosomal dominant trait (Braunwald, 1992).

Pathology

Patients with MVP have redundant myxomatous tissue with excess deposits of proteoglycans in the middle or spongiosa layer of the valve.

Histologically, collagen fragmentation and disorganization as well as elastic fiber are present. Acid mucopolysaccharide material accumulates in the valve leaflets. The mitral valve

leaflets, annulus, and chordae tendineae may also demonstrate disrupted collagen structure and extensive myxomatous change. Myxomatous changes may also occur in the tricuspid, aortic, and pulmonic valves (Alpert et al., 1998).

Pathophysiology

Enlargement of the valve leaflets related to myxomatous degeneration causes systolic prolapse of one or both leaflets into the left atrium. Patients with MVP may have mitral regurgitation ranging in severity from none to severe. Persistent billowing of the valve causes stress to the underlying chordae and papillary muscles. Progressive mitral valvular degeneration can result in increasingly severe mitral regurgitation. If chordal rupture occurs, severe mitral regurgitation develops.

Supraventricular tachycardias (i.e., premature atrial contractions and paroxysmal supraventricular tachycardias) and ventricular arrhythmias may occur in patients with MVP. Although some patients with MVP have had sudden cardiac death, it is unclear what role MVP has in the cause. Some investigators believe that patients with MVP, history of syncope, complex ventricular arrhythmias, significant mitral regurgitation, and prolonged QT interval are at increased risk for sudden death (Kligfield & Devereux, 1995). Patients with MVP may also have autonomic nervous system dysfunction; specifically, mid-brain control of adrenergic and vagal responses may be abnormal. Heightened sympathetic nervous system tone may lead to a decease in left ventricular preload, resulting in MVP (Alpert et al., 1998).

Clinical Manifestations

Most patients with MVP are asymptomatic. Patients may have sharp, localized chest pain that is usually brief in duration. Although the cause of this chest pain is unclear if the patient does not have CHD, some authorities have suggested that the chest pain is cardiac in origin and is related to abnormal traction and tension on the papillary muscles (O'Rourke, 1998). Patients may have equivocal symptoms of anxiety, fatigue, palpitations, and orthostatic hypotension. As mitral regurgitation progresses, patients may note increasing dyspnea, fatigue, decreased exercise tolerance, orthopnea, and paroxysmal nocturnal dyspnea. Ruptured chordae with leaflet flail and acute mitral regurgitation result in symptoms of severe left ventricular failure.

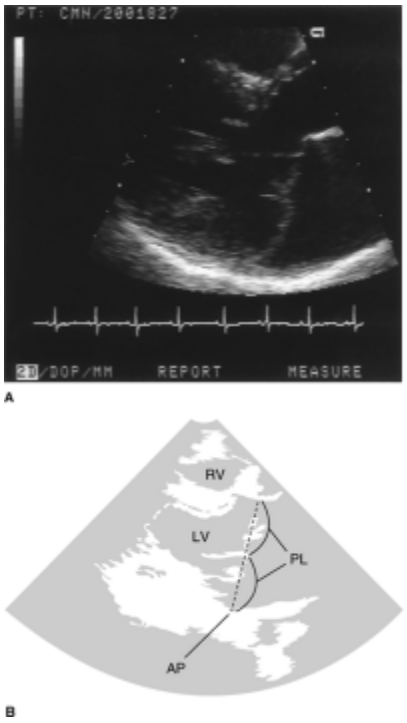
Physical Assessment

The classic auscultatory finding of mitral valve prolapse is mid-systolic click with mid to late systolic murmur (see Table 33-3). The click of MVP occurs when the elongated mitral valve apparatus reaches the end of its tether in mid systole (Carabello, 1998). The murmur occurs secondary to regurgitant flow when the mitral valve leaflets fail to approximate. Patients with mitral valve prolapse may have the murmur or click, or both. Findings may also vary over time. When the degree of mitral regurgitation is mild to moderate or less, heart rate and blood pressure may be normal. Additional physical findings may include thin body habitus, pectus excavatum, straight-back syndrome, and scoliosis.

Diagnostic Tests

Echocardiography plays a key role in the diagnosis of MVP. Abnormal systolic motion of one or both of the mitral valve leaflets superior to the annular plane can be seen (Fig. 33-9). Doppler echocardiography gives additional evidence of valve regurgitation. *Transesophageal echocardiography* provides a more detailed look at the mitral valve and chordal structures (Brady, 2003).

Figure 33-9. (*A*) Long-axis echocardiographic view of mitral valve with bileaflet prolapse above the annular plane into the left atrium. (*B*) Illustration corresponding to echocardiogram. RV, right ventricle; LV, left ventricle; AP, annular plane; PL, prolapsing leaflet.



Cardiac catheterization can be used to rule out CHD as the origin of chest pain. Left ventriculography can demonstrate

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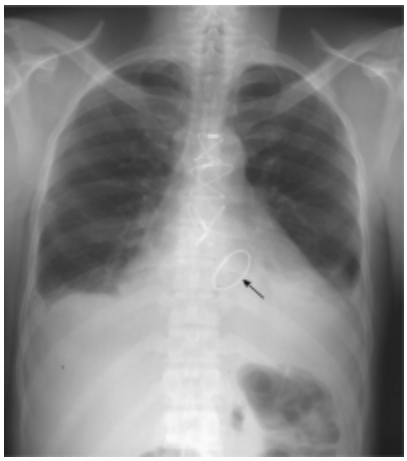
abnormal motion of the mitral valve and help determine the degree of regurgitation.

Electrocardiography is nondiagnostic. The ECG may be normal or have nonspecific ST-T-wave changes in the inferior leads (II, III, and aVF) and occasionally in the anterolateral leads (V_4 through V_6). The ST-T-wave changes may become more notable with exercise. Premature atrial and ventricular complexes may also be identified. *Exercise testing* may be used to help rule out the cause of the chest pain.

Chest radiography is often normal and is usually nondiagnostic for MVP. Patients with acute mitral regurgitation secondary to chordal rupture have pulmonary congestion but not cardiomegaly. Patients with chronic severe mitral regurgitation have an enlarged cardiac silhouette secondary to left atrial and left ventricular enlargement in addition to pulmonary congestion (Fig. 33-10).

Figure 33-10. Chest radiograph of 51-year-old man with history of mitral valve prolapse and repair (note annuloplasty ring marked with *arrow*). Patient's valve repair has failed and his mitral regurgitation is now severe. Patient is now in severe heart failure with notable bilateral pleural effusions and cardiomegaly.



Medical and Surgical Management

Medical Treatment

Asymptomatic patients with MVP require no therapy other than antibiotic prophylaxis. Opinions remain divided on whether patients with isolated click without murmur require antibiotic prophylaxis. Patients with the murmur of mitral regurgitation or echocardiographic evidence of mitral regurgitation are recommended to have antibiotic prophylaxis. Beta-blockers or calcium channel blockers may be used to help alleviate palpitations or chest pain syndrome.

Surgical Treatment

Patients with MVP and severe mitral regurgitation or flail leaflets should be evaluated for surgery. They can often undergo repair rather than replacement. For discussion of surgical options, refer to the section on surgical intervention for mitral regurgitation.

Prognosis

MVP is usually a benign condition. Most patients remain asymptomatic for their entire lives. However, in a small subset of patients, sudden cardiac death may occur secondary to arrhythmias. Patients with palpitations, syncope, or dizziness should be further evaluated and considered for treatment of arrhythmias (Shah, 1991).

Aortic Stenosis

Cause

Aortic stenosis is characterized by obstruction of the left ventricular outflow tract. Most commonly, left ventricular outflow obstruction is valvular, but it may be either supravalvular or subvalvular. The age at which aortic stenosis becomes symptomatic is determined by the underlying cause. Aortic stenosis occurring from ages 1 to 30 years usually represents

congenital aortic stenosis. Aortic stenosis presenting at the ages of 40 to 60 years is primarily rheumatic in origin or secondary to calcific aortic stenosis in a congenitally bicusp aortic valve. Past the age of 60 to 70 years, calcific degenerative stenosis is the most prevalent cause. Of the causes of aortic stenosis, senile/degenerative calcific aortic stenosis is most common.

Pathology

In senile/degenerative calcific aortic stenosis, cumulative wear and tear leads to calcification on an otherwise normal aortic valve. Calcific deposits prevent the cusps from opening normally in systole, resulting in stenosis. Risk factors for development of calcific aortic stenosis include male gender, elevated lipoprotein(a), height, hypertension, smoking, elevated low-density lipoprotein cholesterol, raised serum calcium, raised serum creatinine, and diabetes (Rajamannan et al., 2003). In patients with congenitally bicuspid aortic valves, abnormal flow through the valve leads to calcium deposition and restriction of cusp opening. In rheumatic aortic stenosis, inflammation and fibrosis of the valve result in fusion of the commissures as well as calcified masses in the aortic cusp (Chan, 1993).

Pathophysiology

Aortic stenosis typically progresses over a period of years. As the valve cusps become less mobile, the valve orifice decreases in size, resulting in an increasingly higher left ventricular systolic pressure necessary to eject blood across the stenosed valve. This increased left ventricular afterload results first in compensatory concentric left ventricular hypertrophy. Although initially adaptive in aortic stenosis, left ventricular hypertrophy leads to decreased ventricular compliance and diastolic dysfunction. As aortic stenosis becomes severe, left ventricular systolic function may also decline, resulting

in CHF. Late in the course, any coexisting mitral regurgitation increases because of an increased pressure gradient, which drives blood from the left ventricle into the left atrium (Braunwald, 1992).

Angina may result even in the absence of CHD because of an imbalance in myocardial oxygen supply and demand. Myocardial oxygen demand is increased secondary to increased left ventricular wall stress and muscle mass. Myocardial oxygen delivery is reduced as a result of decreased coronary perfusion pressure.

Syncope or near syncope can result secondary to reduced cerebral perfusion pressure, inappropriate left ventricular baroreceptor response, or arrhythmia. Orthostatic blood pressure changes may occur during exertion when arterial pressure drops because of systemic vasodilation in the setting of a fixed cardiac output. Increased left ventricular pressure may result in inappropriate baroreceptor response. Rapid atrial arrhythmias or ventricular arrhythmias may also cause “graying out” spells or frank syncopal episodes (Braunwald, 1992).

Clinical Manifestations

Patients with mild to moderate aortic stenosis are usually asymptomatic. As severe aortic

stenosis develops, the most common initial symptom is dyspnea on exertion, followed by angina and near syncope or syncope. CHF also may occur as a result of ventricular dysfunction or increasing mitral regurgitation. Less commonly, sudden death, probably caused by ventricular fibrillation, may be the presenting clinical feature.

Physical Assessment

Aortic stenosis is most readily detected by auscultation of its classic mid-systolic (systolic ejection) murmur (see Table 33-3). As aortic stenosis progresses, the murmur peaks progressively later in systole and decreases in intensity as cardiac output falls. The murmur may decrease or disappear over the sternum and reappear at the apex, causing the incorrect impression of mitral regurgitation (Gallavardin phenomenon) (Carabello, 1998). An S_4 gallop is usually present. The point of maximal intensity is sustained but may not be displaced. Blood pressure is normal to hypertensive until late in the disease progress. Jugular venous pressure is normal in most patients except those with severe aortic stenosis associated with heart failure. Reduction in stroke volume and cardiac output may cause diminished carotid upstrokes and late systolic peak (tardus) in severe or critical aortic stenosis.

Diagnostic Tests

Echocardiography is the principal modality used to diagnose and quantify aortic stenosis. Two-dimensional echocardiography defines valve leaflet thickening and cusp movement restriction as well as gauging left ventricular hypertrophy and evaluating ventricular function (Carabello, 1998). Aortic valve pressure gradient can be measured, aortic valve area calculated, and pulmonary artery pressures estimated. Echocardiography is the most important diagnostic imaging technique used to diagnose and follow aortic stenosis (Brady, 2003).

Cardiac catheterization is performed in patients with aortic stenosis primarily to rule out concomitant CHD. Left ventriculography can quantify the left ventricular ejection fraction. The transvalvular gradient can be established by direct pressure measurement. Right heart catheterization can better quantify pulmonary artery pressures and cardiac output.

Electrocardiography often shows a pattern of left ventricular hypertrophy, although its absence does not exclude the presence of critical aortic stenosis. In addition to QRS amplitude changes typically associated with left ventricular hypertrophy, the patient with aortic stenosis may demonstrate ST-T-wave changes typical of left ventricular strain (Braunwald, 1992).

Exercise testing in patients with mild to moderate aortic stenosis with equivocal symptoms may be cautiously accomplished in the hands of a cardiologist and can provide relevant information regarding exercise tolerance. In patients with known severe aortic stenosis and classic symptoms such as syncope, dyspnea, and chest pain, exercise testing carries increased risk of ventricular tachyarrhythmias and ventricular fibrillation and should not be preformed.

Gated blood pool radionuclide scans provide information regarding ventricular function similar to echocardiography and left ventriculography. Gated pool scans may be useful in

patients in whom left ventriculography cannot be performed (i.e., patients with elevated creatinine), or in those in whom the left ventricle cannot be clearly imaged with echocardiography.

Chest radiography may be negative even in advanced disease. Heart size may be normal or only minimally enlarged. The left ventricular border and apex may be rounded, demonstrating a boot-shaped silhouette. Identifiable calcification of the aortic valve and aorta may be present. As disease progresses, left atrial enlargement, pulmonary hypertension, and CHF may become evident. Poststenotic dilation of the proximal ascending aorta may be noted along the right heart border in the posteroanterior chest radiograph (Fig. 33-11).

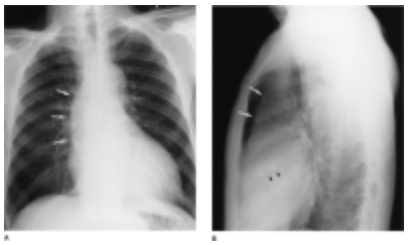


Figure 33-11. (*A*) Posteroanterior chest radiograph showing rounded border of left ventricle (*arrows*). (*B*) Lateral chest radiograph showing calcified aortic valve (*arrowheads*) and filling of the retrosternal airspace with dilated ascending aorta (*arrows*). (From Boxt, L. M. [1998]. Plain film examination of the chest. In E. J. Topol, R. M. Califf, J. M. Isner et al. [Eds.], *Textbook of cardiovascular medicine* [p. 511]. Philadelphia: Lippincott-Raven.)

Medical Management

Aside from antibiotic prophylaxis to prevent endocarditis, there is no effective medical management of aortic stenosis (Carabello, 1998). Because the course of aortic stenosis varies in its progression, patients should be carefully followed-up by their health care providers with serial physical examinations and periodic echocardiography. Patients with mild aortic stenosis undergo echocardiography every 2 years. Patients with asymptomatic severe aortic stenosis are followed-up with serial echocardiograms every 6 to 12 months. Patients are instructed regarding symptoms of aortic stenosis, including dyspnea, decreased exercise tolerance, shortness of breath, chest pain, near syncope, and syncope (Braunwald, 1992). Beta-blockers, diuretics, nitrates, and ACE inhibitors must be used with caution, because they may precipitate syncope or even cardiovascular collapse in the patient with severe aortic stenosis (Carabello, 1998).

Interventional and Surgical Management

Percutaneous Aortic Catheter Balloon Valvuloplasty

Percutaneous aortic catheter balloon valvuloplasty is accomplished by passing a guide wire across the stenotic aortic valve into

the apex of the left ventricle. A balloon-tipped catheter is advanced retrograde across the stenotic valve. The balloon is inflated, fracturing calcified nodules and separating the fused commissures. The aortic valve ring is also stretched to increase the size of the aortic valve orifice. Although results vary, aortic balloon valvuloplasty has been shown, on average, to increase aortic valve area from 0.5 to 0.8 cm^2 and to decrease the gradient from 60 to 30 mm Hg. In addition, left ventricular ejection fraction tends to rise in those patients with depressed left ventricular function (Braunwald, 1992).

Restenosis is a major problem in balloon aortic valvuloplasty in adults, occurring in approximately half of the patients within 6 months. Approximately one third of patients have recurrence of symptoms within 6 months. Because of the high restenosis rate, aortic balloon valvuloplasty in adults is reserved for those candidates unsuitable for surgery (i.e., the elderly with heart failure or pregnant women). Aortic balloon valvuloplasty may be used as a bridge or palliative procedure in these populations, and surgical replacement may be considered for a later time (Braunwald, 1992).

Aortic Valve Replacement

Aortic valve replacement is the only effective treatment for advanced aortic stenosis (Chan, 1993). The natural history of aortic stenosis is used as a guide to determine the timing of aortic valve replacement surgery. Patients with asymptomatic aortic stenosis have nearly the same survival rate as the age-matched general population. Once the patient experiences symptoms of angina, syncope, or heart failure, there is an abrupt decline in survival rate. In patients presenting with CHF, only 50% survive 2 years. For patients who present with syncope, the 3-year survival rate is only 50% without aortic valve replacement. The average life expectancy of patients with apnea is only 5 years without aortic valve replacement (Ross, 1968). Aortic valve replacement is recommended in all patients with severe, symptomatic aortic stenosis. Although the average perioperative mortality rate in most centers ranges from 2% to 8% (Braunwald, 1992), perioperative mortality rates range from less than 5% in young, healthy patients to as high as 30% in the frail elderly (Nishimura, 1997). Factors that increase the mortality risk at the time of aortic valve replacement include class III or IV heart failure, emergency surgery, aortic insufficiency, and cardiomegaly (Shine & Howland-Gradman, 1996). Even in patients with severe left ventricular dysfunction, improvement of symptoms and left ventricular ejection fraction occurs in most (Connolly et al, 1997). Selection of aortic valve prostheses was discussed earlier in this chapter.

Aortic Insufficiency

Cause

Aortic regurgitation may be caused by either intrinsic abnormalities of the aortic valve leaflets or disease of the aortic root. In rheumatic fever and endocarditis, the aortic leaflets are

directly affected. In congenitally bicuspid valves, the larger cusp may become redundant, resulting in diastolic prolapse and progressive aortic regurgitation. The aortic valve may

also become incompetent because of aortic root dilation. As the aortic root dilates, the aortic annulus becomes so large that the valve cusps no longer approximate, resulting in regurgitation.

Aortic root dilation is seen in patients with Marfan syndrome, rheumatic arthritis, ankylosing spondylitis, annuloaortic ectasia (associated with hypertension and aging), aortic dissection, syphilitic aortitis, and collagen vascular disease.

Pathology

Rheumatic fever leads to fibrinous infiltrates on the valve cusps, causing them to contract and become malaligned and incompetent. Patients with rheumatic disease may have a “mixed lesion” that includes both aortic regurgitation and aortic stenosis. In acute or subacute infective endocarditis of the aortic valve, tissue destruction of the leaflets causes cusp perforation or prolapse. Vegetations adherent to the aortic valve may also interfere with valve closure, causing incompetence. In patients with aortic root dilation or ascending aortic dissection, the aortic annulus becomes greatly enlarged, the aortic leaflets separate, and aortic incompetence follows.

Pathophysiology

Volume overload occurs secondary to regurgitant volume reentering the left ventricle from the aorta through the incompetent aortic valve. Retrograde flow occurs during diastole when left ventricular pressure is low and aortic pressure is high. The left ventricle is forced to pump the normal volume received from the left atrium as well as the regurgitant volume from the aorta.

Similar to mitral regurgitation, the hemodynamic presentation and the heart's ability to compensate differ depending on whether the aortic insufficiency is acute or chronic. In chronic aortic regurgitation, the left ventricle is subjected to pressure and volume overload. As a result, the left ventricle develops mild concentric hypertrophy to accommodate the pressure load and eccentric hypertrophy to compensate for the increased volume load. Patients with chronic aortic regurgitation may remain asymptomatic for years, until progressive left ventricular dilation and dysfunction result in CHF. In patients with acute aortic regurgitation, the left ventricle has not had time to compensate with either concentric or eccentric hypertrophy and cannot accommodate the large volume caused by acute aortic regurgitation. As a result, left ventricular and left atrial pressures rise sharply, causing acute CHF and pulmonary edema. Patients with acute aortic regurgitation usually require surgical intervention.

Clinical Manifestations

Patients with chronic aortic regurgitation are often asymptomatic for many years. Common symptoms of aortic regurgitation include fatigue and exertional dyspnea. Patients may report palpitations, dizziness, and the sensation of a forceful heartbeat, especially when lying on their left side. Angina may also be noted, but it occurs less frequently in aortic regurgitation than in aortic stenosis. As heart failure ensures, patients experience orthopnea, paroxysmal nocturnal dyspnea, and cough related to left-sided heart failure.

With acute aortic regurgitation, symptoms of left-sided heart failure develop rapidly.

Physical Assessment

The typical murmur of aortic regurgitation is a high-pitched, early diastolic decrescendo murmur with a blowing quality (see Table 33-2). Patients may also have a physiologic murmur of mitral stenosis caused by the regurgitant aortic jet, which partially prevents mitral valve closure (Austin Flint murmur). As the severity of aortic regurgitation increases, the murmur becomes louder and longer. In chronic aortic regurgitation, the point of maximal impulse is displaced laterally. Systolic hypertension and decreased diastolic pressure create a widened pulse pressure. Patients with chronic aortic regurgitation may have a host of other physical findings that may not be present in acute aortic regurgitation (Table 33-6).

Table 33-6 SPECIFIC PHYSICAL EXAMINATION FINDINGS IN AORTIC REGURGITATION

Sign	Phsical Description
Quincke's sign	Pulsatile flushing/blanching of nail bed with application of gentle pressure
de Musset's sign	Bobbing of head with each pulse
Corrigan's pulse (waterhammer)	Sharp systolic upstroke and diastolic collapse of pulse
Müller's sign	Bobbing of uvula with each pulse
Traube's sign	“Pistol shot” sound auscultated over the femoral arteries
Duroziez's sign	Biphasic femoral bruit auscultated with mild pressure
Hill's sign	Blood pressure higher in arms than legs

Diagnostic Tests

Echocardiography is helpful in identifying the cause of aortic regurgitation. Echocardiography can indicate left ventricular volume overload by the increased internal diameter of the ventricular chamber during systole and diastole. Doppler echocardiography is the best noninvasive means to detect aortic regurgitation. Transesophageal echocardiography is especially useful in imaging the ascending and descending aorta in patients with suspected aortic dissection.

Cardiac catheterization should be performed to visualize and quantify the extent of regurgitation before surgery. However, physical findings and noninvasive tests are sufficient to establish the diagnosis of aortic insufficiency. In patients with known or suspected CHD, coronary angiography should be performed. In patients with aortic root dilation, aortic root angiography may be performed concurrently with coronary angiography.

Radionuclide imaging can be used to estimate ejection fraction and determine myocardial perfusion defects in patients with concomitant CHD.

Exercise testing may be used to establish exercise tolerance and to evaluate asymptomatic patients.

Electrocardiography may be normal in patients with acute aortic regurgitation or in patients with mild to moderate

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chronic regurgitation. Patients with moderate to severe chronic regurgitation may have left-axis deviation and a pattern of left ventricular strain (Q waves in I, aVL, and V_3 to V_6, with small R wave in V_1). Intraventricular conduction defects may occur with left ventricular dysfunction or annular abscess.

Chest radiography may show only CHF in the patient with acute aortic regurgitation because compensatory left ventricular dilation has not yet occurred. In chronic aortic regurgitation, the chest radiograph demonstrates marked cardiomegaly with inferior and leftward displacement of the apex (Braunwald, 1992). Dilation of the ascending aorta and a widened mediastinum may be noted in patients with aortic dissection. In patients with a dilated aortic root or dissection, computed tomography or magnetic resonance imaging may be necessary to better-delineate the ascending aorta, transverse arch, and proximal descending aorta.

Medical Management

Patients who have asymptomatic aortic regurgitation should receive appropriate antibiotic prophylaxis as well as afterload reduction with vasodilators. In patients with asymptomatic, chronic, or severe aortic regurgitation and normal left ventricular function, nifedipine reduced left ventricular size and mass and further improved left ventricular function (Scognamiglio et al., 1994), thus reducing and delaying the need for aortic valve replacement. Nifedipine is superior to hydralazine, which does not decrease left ventricular mass or reduce left ventricular dimension (Khan, 1996). Diltiazem and verapamil are contraindicated in aortic regurgitation because they have a more potent negative inotropic effect and may produce bradycardia, which may worsen heart failure. It is probably

reasonable to substitute other dihydropyridine calcium channel blockers if nifedipine is poorly tolerated (Carbello, 1998). ACE inhibitors may be used to reduce afterload, although they are not as well studied in this population of patients.

Patients with moderate to severe aortic regurgitation should not participate in vigorous exercise or competitive sports. Patients with chronic severe aortic regurgitation should be followed-up with physical examination and echocardiography every 6 to 12 months (Rahimtoola, 1998). Sodium nitroprusside reduces preload and afterload and can be used to stabilize patients with acute aortic regurgitation before surgery. Intra-aortic balloon counterpulsation cannot be used because inflation of the balloon during diastole would increase the regurgitant volume into the left ventricle, which acutely worsens left ventricular dilation and heart failure.

Surgical Management

Acute aortic regurgitation requires urgent aortic valve replacement. Without adequate time for compensatory mechanisms to develop, aortic regurgitation triggers rapid onset of CHF, tachycardia, and diminished cardiac output. It is desirable to treat patients with acute aortic regurgitation secondary to infective endocarditis with a minimum of 48 hours of appropriate intravenous antibiotics before implanting a prosthetic valve. In patients with active endocarditis who are hemodynamically unstable, use of cadaveric human aortic homografts may minimize the risk of prosthetic valve endocarditis. Patients who have aortic regurgitation caused by ascending aortic dissection or dilation require replacement of the ascending aorta as well.

In chronic aortic regurgitation, the aortic valve must be replaced before irreversible left ventricular dysfunction. In asymptomatic patients, it is usually recommended that the aortic valve be replaced when left ventricular function begins to deteriorate. Surgery is recommended when the echocardiographic left ventricular ejection fraction is.55 or less, the end-diastolic diameter approaches 75 mm, or the end-systolic diameter reaches 50 mm. When symptoms of heart failure develop, aortic valve surgery should be performed regardless of echocardiography findings, because new-onset heart failure indicates that the heart has met the limits of compensation (Carbello, 1998).

REFERENCES

Alpert, J. S., Sabick, J., & Cosgrove, D. M. (1998). Mitral valve disease. In E. J. Topol, R. M. Califf, J. M. Isner et al. (Eds.), *Textbook of cardiovascular medicine* (pp. 503–532). Philadelphia: Lippincott-Raven.

Abraham, M. T., & Cherin, G. (1991). Rheumatic fever. In K. Chatterjee, M. D. Cheitlin, J. Karliner et al. (Eds.), *Cardiology: An illustrated text/reference* (pp. 10.98–10.106). Philadelphia: JB Lippincott.

Bhola, R., & Gill, E. (2001). Rheumatic heart disease and mitral stenosis. In O. V.

Adair, *Cardiology secrets* (2nd ed., pp. 226–235).

Brady, T. J., Grist, T. M., Westra, S. J., et al. (2003). Valvular. In T. J. Brady, T. M. Grist, S. J. Westra, et al, *Pocket radiologist: Cardiac top 100 diagnoses* (pp. 53–75). Salt Lake, UT: Amirsys.

Braunwald, E. (1992). Valvular heart disease. In E. Braunwald (Ed.), *Heart disease: A textbook of cardiovascular medicine* (4th ed., pp. 1007–1077). Philadelphia: WB Saunders.

Braunwald, E. (1998). Valvular heart disease. In A. S. Fauci, E. Braunwald, & K. J. Isselbacher (Eds.), *Harrison's principles of internal medicine* (14th ed., pp. 1311–1324). New York: McGraw-Hill.

Buccino, D., Tu, A. S., & Come, P. C. (1993). Diagnostic imaging and catheterization techniques. In L. S. Lilly (Ed.), *Pathophysiology of heart disease* (pp. 130–146). Philadelphia: Lea & Febiger.

Burge, D. J., & DeHoratius, R. J. (1993). Acute rheumatic fever. In A. N. Brest (Ed.), *Valvular heart disease: Comprehensive evaluation and treatment* (2nd ed., pp. 3–7). Philadelphia: F. A. Davis.

Carabello, B. A. (1998). Recognition and management of patients with valvular heart disease. In L. Goldman, & E. Braunwald (Eds.), *Primary cardiology* (pp. 370–389). Philadelphia: WB Saunders.

Carabello, B. A. (2000). Acute mitral regurgitation. In J. S. Alpert, J. E. Dalen, & S. H. Rahimtoola (Eds.), *Valvular heart disease* (3rd ed., pp. 143–155). Philadelphia: Lippincott Williams & Wilkins.

Chan. E., Duh, E., Stidham, B. et al. (1993). Valvular heart disease. In L. S. Lilly (Ed.), *Pathophysiology of heart disease* (pp. 130–146). Philadelphia: Lea & Febiger.

Chitwood, W.R., Wixon, C. L., & Elbeery, J. R. (1997). Video-assisted minimally invasive mitral valve surgery. *Journal of Thoracic & Cardiovascular Surgery*, 114, 773–781.

Chitwood, W. R. (2003). Robot-assisted mitral valve surgery. In K. L. Franco, & E. D. Verrier (Eds.), *Advanced therapy in cardiac surgery* (pp. 220–229). Hamilton, Ontario:

BC Decker.

Cohn, L. H., Adams, D. H., Couper, G. S., et al. (1997). Minimally invasive cardiac valve surgery improves patient satisfaction while reducing costs of cardiac valve replacement and repair. *Annals of Surgery*, 226, 421–426.

Connolly, H. M., Oh, J. K., Orszulak, T. A., et al. (1997). Aortic valve replacement for aortic stenosis with severe left ventricular dysfunction: Prognostic Indicators. *Circulation*, 95, 2395–2399.

Cosgrove, D. M., Sabik, J. F., & Navia, J. L. (1998). Minimally invasive valve operations. *Annals of Thoracic Surgery*, 65, 1538–1539.

Crawford, M. H. & Durack, D. T. (2003). Clinical presentation of infective endocarditis. *Cardiology Clinics*, 21, 159–166.

Dajani, A. S., Taubert, K. A., Wilson, W., et al (1997). Prevention of bacterial endocarditis: Recommendations by the American Heart Association. *Circulation*, 96, 358–366.

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Dalen, J. E., & Fenster, P. E. (2000). Mitral stenosis. In J. S. Alpert, J. E. Dalen, & S. H. Rahimtoola (Eds.), *Valvular heart disease* (3rd ed., pp. 75–112). Philadelphia: Lippincott Williams & Wilkins.

David, T. E., Puschmann, R., Ivanov, J., et al. (1998). Aortic valve replacement with the stentless and stented porcine valves: A case-match study. *Journal of Thoracic & Cardiovascular Surgery*, 116, 236–240.

Doty, J. R., Salazar, J. D., Liddicoat, J. R., et al. (1998). Aortic valve replacement with cryopreserved aortic allograft: Ten year experience. *Journal of Thoracic Cardiovascular Surgery*, 115, 371–379.

Edmunds, L. H., Addonizio, V. P., & Tepe, N. A. (1991). Valvular heart disease: Prosthetic valve replacement. In K. Chatterjee, M. D. Cheitlin, & J. Karliner et al. (Eds.), *Cardiology: An illustrated text/rReference* (pp. 940–941). Philadelphia: JB Lippincott.

Elkins, R. C. (2003). Pulmonary autograft. In K. L. Franco & E. D. Verrier (Eds.),

Advanced therapy in cardiac surgery (pp. 156–167). Hamilton, Ontario: BC Decker.

Enriquez-Sarano, M., Schaff, H. V., Tajik, A. J. & Frye, R. L. (2000). Chronic mitral regurgitation. In J. S. Alpert, J. E. Dalen, & S. H. Rahimtoola (Eds.), *Valvular heart disease* (3rd ed., pp. 113–141). Philadelphia: Lippincott Williams & Wilkins.

Ewy, G. A. (2000). Tricuspid valve disease. In J. S. Alpert, J. E. Dalen, & S. H. Rahimtoola (Eds.), *Valvular heart disease* (3rd ed., pp. 377–392). Philadelphia: Lippincott Williams & Wilkins.

Fann, J. I., Ingels, N. B., & Miller, D. C. (1997). Pathophysiology of mitral valve disease and operative indications. In L. H. Edmunds (Ed.), *Cardiac surgery in the adult* (pp. 959–990). New York: McGraw-Hill.

Garcia, M. J. (1998). Prosthetic valve disease. In E. J. Topol, R. M. Califf, J. M. Isner et al. (Eds.), *Textbook of cardiovascular medicine* (pp. 579–605). Philadelphia: Lippincott-Raven.

Goldman, B. S., & Mallidi, H. (2003). Update on Stentless valves. In K. L. Franco & E. D. Verrier (Eds.), *Advanced therapy in cardiac surgery* (pp. 196–206). Hamilton, Ontario: BC Decker.

Goldsmith, I., Turpie, A. C. G., & Lip, G. Y. (2002). ABC of Antithrombotic therapy: valvular heart disease and prosthetic heart valves. *BMJ*, 325, 1228–1231.

Grunkemeier, G. L., Starr, A., & Rahimtoola, S. H. (1994). Replacement heart valves. In R. A. O'Rourke (Ed.), *Hurst's the heart: Update I* (pp. 98–123). New York: McGraw-Hill.

Hamptom, C. R., & Verrier, E. D. (2003). Mitral valve repair. In K. L. Franco & E. D. Verrier (Eds.), *Advanced therapy in cardiac surgery* (pp. 207–219). Hamilton, Ontario: BC Decker.

International Ross Registry. (2003). Follow-up data. [Online]. Available: http://www.rossregistry.com (Accessed 8/28/03).

Izzat, M. B., Yim, A. P., El-Zufari, et al. (1998). Upper T mini-sternotomy for aortic valve operations. *Chest*, 114, 291–294.

Jamieson, W. R. E. (2003). Update on new tissue valves. In K. L. Franco & E. D. Verrier (Eds.), *Advanced therapy in cardiac surgery* (pp. 177–195). Hamilton, Ontario: BC Decker.

Kaplan, E. L. (1998). Acute rheumatic fever. In R. W. Alexander, R. C. Sclant, & V. Fuster (Eds.), *Hurst's the heart* (9th ed., pp. 1759–1787). New York: McGraw-Hill.

Karchmer, A. W. (1998). Approach to the patient with infective endocarditis. In L. Goldman & E. Braunwald (Eds), *Primary cardiology* (pp. 201–218). Philadelphia: WB Saunders.

Karon, B. L. (1997). Valvular regurgitation. In J. G. Murphy (Ed.), *Mayo Clinic cardiology review* (pp. 533–554). Armonk, NY: Futura.

Khan, M. G. (1996). Valvular heart disease and rheumatic fever. In M. G. Khan (Ed), *Heart Disease Diagnosis and Therapy: A Practical Approach* (pp. 415–460). Baltimore: Williams & Wilkins.

Kligfield, P., & Devereux, R. B. (1995). Arrhythmia in mitral valve prolapse. In P. R. Podrid & P. R. Kowey (Eds.), *Cardiac arrhythmia: Mechanisms, diagnosis, and management* (pp. 1253–1260). Baltimore: Williams & Wilkins.

Luciani, G. B., Bertolini, P., Vecchi, B. et al. (1998). Midterm results after aortic valve replacement with freehand stentless xenografts: A comparison of three prostheses. *Journal of Thoracic and Cardiovascular Surgery*, 115, 1287–1296.

Mayes, C. E., Cigarroa, J. E., Lange, R. A., et al. (1999). Percutaneous mitral balloon valvuloplasty. *Clinics in Cardiology*, 22, 501–503.

Mazur, W., Parilak, L. D., Kaluza, G., et al. (1999). Balloon valvuloplasty for mitral stenosis. *Current Opinion in Cardiology*, 14, 95–103.

McAnulty, J. H., & Rahimtoola, S. H. (1998). Antithrombotic therapy and valvular heart disease. In R. W. Alexander, R. C. Sclant, & V. Fuster (Eds.), *Hurst's the heart* (9th ed., pp. 1759–1787). New York: McGraw-Hill.

Nishimura, R. A. (1997). Valvular stenosis. In J. G. Murphy (Ed.), *Mayo Clinic cardiology review* (pp. 521–532). Armonk, NY: Futura.

Olaison, L., & Pettersson, G. (2003) Current best practices and guidelines: Indications for surgical intervention in infective endocarditis. *Cardiology Clinics*, 21, 235–251.

O'Rourke, R. A. (1998). Mitral valve prolapse syndrome. In R. W. Alexander, R. C. Sclant, & V. Fuster (Eds.), *Hurst's the heart* (9th ed., pp. 1821–1831). New York: McGraw-Hill.

Otto, C. M. (1998). Aortic stenosis: Clinical evaluation and optimal timing of surgery. *Cardiology Clinics*, 16(3), 353–373.

Otto, C. M. (1999). Right-sided valve disease. In C. M. Otto, *Valvular heart disease* (pp. 362–379). Philadelphia: W.B. Saunders.

Palacios, I. F., Sanchez, P. L. Harrell, L. C., et al. (2002). Which patients benefit from percutaneous mitral balloon valvuloplasty? *Circulation*, 105, 1465–1471.

Playford, D., & Weyman, A. E. (2001). Mitral valve prolapse: time for a fresh look. *Reviews in Cardiovascular Medicine*, 2(2), 73–81.

Rahimtoola, S. H. (1998). Aortic valve disease. In R. W. Alexander, R. C. Sclant, & V. Fuster (Eds.), *Hurst's the heart* (9th ed., pp. 1759–1787). New York: McGraw-Hill.

Rajamannan, N. M., Gersh, B., & Bonow, R. O. (2003). Calcific aortic stenosis: from bench to bedside- emerging clinical and cellular concepts. *Heart*, 89, 801–805.

Reardon, M. J., & David, T. E. (1998). Mitral valve replacement with preservation of the subvalvular apparatus. *Current Opinion in Cardiology*, 14, 104–110.

Ross, J. Jr., & Braunwald, E. (1968). Aortic stenosis. *Circulation* (Suppl. V), 38, 61–67.

Ross, D. N., Jackson, M., & Davies, J. (1987). Pulmonary autograft aortic valve replacement. *Circulation*, 75, 895–901.

Sachdev, M., Peterson, G. E., & Jollis, J. G. (2003). Imaging techniques for diagnosis of infective endocarditis. *Cardiology Clinics*, 21, 185–195.

Santini, F., Dyke, C., Edwards, S., et al. (1997). Pulmonary autograft versus homograft

replacement of the aortic valve: A prospective randomized trial. *Journal of Thoracic and Cardiovascular Surgery*, 113, 894–899.

Scognamiglio, R., Rahimtoola, S. H., Fasoli, G., et al. (1994). Nifedipine in asymptomatic patients with severe aortic regurgitation and normal left ventricular function. *New England Journal of Medicine*, 331, 689–694.

Sexton, D. J., & Spelman, D. (2003). Current best practices and guidelines: Assessment and management of complications in infective endocarditis. *Cardiology Clinics*, 21, 273–282.

Shah, P. M. (1991). Mitral valve prolapse. In K. Chatterjee, M. D. Cheitlin, J. Karliner et al. (Eds.), *Cardiology: An illustrated text/reference* (pp. 9.30–9.39). Philadelphia: JB Lippincott.

Sharony, R., Grossi, E. A. Ribakove, G. H., et al. (2003). Minimally invasive cardiac valve surgery. In K. L. Franco & E. D. Verrier (Eds.), *Advanced therapy in cardiac surgery* (pp.147–155). Hamilton, Ontario: BC Decker.

Shine, L., & Howland-Gradman, J. (1996). Aortic stenosis in the elderly: Valvuloplasty vs surgery. *American Journal of Nursing* (Suppl.), 7–11.

Teerlink, J. R., & Foster, E. (1998). Valvular heart disease in pregnancy. *Cardiology Clinics*, 16 (3), 573–594.

Towns, M. L., & Reller, L. B. (2003). Diagnostic methods: current best practices and guidelines for isolation of bacteria and fungi in infective endocarditis. *Cardiology Clinics*, 21, 197–205.

Wernly, J. A., & Crawford, M. H. (1998). Choosing a prosthetic heart valve. *Cardiology Clinics*, 16 (3), 491–504.

Whittlesay, D., & Geha, A. S. (1991). Selection and complications of cardiac valvular prosthesis. In A. E. Baue, A. S. Geha, G. L. Hammond et al. (Eds.), *Glenn's thoracic and cardiovascular surgery* (5th ed., pp. 1719–1728). Norwalk, CT: Appleton & Lange.