Valvular heart disease is the fifth most common cardiovascular disorder; the first four are hypertension, coronary artery disease, congestive heart failure, and stroke. Valvular heart disease may develop acutely, but more commonly it is a chronic process, evolving over many years. Severe valvular heart disease eventually leads to heart failure and dysrhythmias unless the course of the disease is interrupted. Valvular heart disease affects persons of all ages who have valvular dysfunction due to congenital or acquired causes. Most valve repairs for congenital valvular disorders are performed immediately after birth or during childhood. Common causes of acquired valvular heart disease include degenerative heart disease, rheumatic heart disease, and infective endocarditis. Less common causes include trauma, lupus erythematosus, tumors, syphilis, cancer, and arthritic disease.

Significant advances have been made in the past several decades in the early assessment and management of valvular heart disease. Innovations in diagnostic tools, pharmacological developments, and improved invasive and surgical techniques have contributed to these advances. Cardiac catheterization, color flow Doppler imaging, transesophageal echocardiography, and cardiac magnetic resonance imaging have revolutionized the diagnostic approach. Pharmacological agents such as diuretics, nitrates, digitalis, anticoagulants, calcium channel blockers, phosphodiesterase inhibitors, antidyssrhythmics, and antibiotics have improved the medical management of valvular heart disease. Balloon valvuloplasty provides patients with mitral stenosis and aortic stenosis an invasive yet nonsurgical option for treatment of valve disease. Balloon valvuloplasty has been used successfully to treat mitral stenosis in patients without heavily calcified valves and has been used as a temporizing procedure in patients for whom surgery is a risk.

Great strides have been made in the surgical treatment of heart valve disease. In the 1950s, surgery was used to repair damaged heart valves. In the early 1960s, the first valve replacement surgery with a prosthetic valve was performed. Valve replacement became the preferred surgical technique, because prosthetic valves were easy to insert and reliable. Complications associated with valve replacement include wear of the prosthetic valve, thromboembolism, hemorrhage due to use of anticoagulants, and prosthetic valve endocarditis. According to Frankel and Brest, after valve replacement surgery, the disease process that involved the native valve is replaced by another disease process, one that involves the prosthetic valve. Therefore, surgeons turned with renewed interest to repair and reconstruction of cardiac valves. Valve repair eliminates or at least minimizes many of the problems associated with valve replacement.

The focus of this article is repair of cardiac valves for adults with acquired valvular disorders. The prevalence of cardiac valve disease and valve dysfunction is reviewed, and the most common techniques for repair of the mitral and aortic valves are described. Outcomes after valve repair are discussed, and specific priorities in patients' care are emphasized.
Prevalence of Cardiac Valve Disease

Although less information is available on the epidemiology of valvular heart disease than on that of atherosclerotic disease, both diseases are common and are associated with appreciable morbidity and mortality. A steady decrease has occurred in the incidence of rheumatic heart disease, the most common single cause of valvular heart disease. However, rheumatic fever continues to be prevalent, especially in developing countries, in the tropical areas of the world, and in isolated pockets of developed countries. In the late 19th century, reports of acute rheumatic fever worldwide were numerous. In the United States, hospitals in Philadelphia, New York, and Boston reported thousands of cases of rheumatic fever; however, in the early 1900s, incidence reports in the United States were sparse and were not duplicated to note trends. The best records are available from Denmark, where the annual incidence of the disease, which was 200 per 100000 in 1862, had decreased to 50 per 100000 by 1940 and to 11 per 100000 by 1962. Likewise, a steady decrease has occurred in the incidence of rheumatic heart disease in the United States. In 1950, approximately 15 000 persons in the United States died of rheumatic fever or rheumatic heart disease. From 1987 to 1997 the death rate for rheumatic heart disease decreased by 33.2%. Although the prevalence of rheumatic heart disease is decreasing, it is estimated that currently 1.8 million persons in the United States have the disease and that approximately 35 000 patients are hospitalized annually because of rheumatic heart disease.

The Mitral and Aortic Valves

The Mitral Valve

The mitral valve lies between the left atrium and the left ventricle. The valve consists of 2 leaflets: a large anterior (aortic) leaflet and a small posterior (mural) leaflet. The leaflets join at 2 commissures (the lateral and medial) and are supported by a subvalvular mechanism. The subvalvular mechanism consists of the papillary muscles and the chordae tendineae (Figure 1). The mitral valve leaflets arise directly from the mitral annulus, with the anterior leaflet attaching to one third of the annulus and the posterior leaflet attaching to approximately two thirds of the annulus. The chordae tendineae originate from the fibrous tips of the papillary muscles and connect into the free edges and the undersurfaces of the mitral leaflets. Primary chordae are attached to the...
leaflet edges, and secondary chordae are attached to the undersurface of the leaflet. Additional tertiary chordae arise from the posterior left ventricular wall and attach to the undersurface of the posterior leaflet. The chordae divide and subdivide into more than 100 chordae that support the 2 mitral valve leaflets. The chordae tendineae and the papillary muscles prevent the prolapse of the leaflets into the left atrium during systole and contribute to the competency of the mitral valve.

**The Aortic Valve**

The aortic valve consists of 3 cusps or leaflets: the left cusp, the right cusp, and the noncoronary cusp (Figure 2). The aortic valve lies between the left ventricle and the ascending aorta. The aortic valve does not have a subvalvular mechanism. The aortic cusps open as blood is propelled into the aorta and close when the pressure in the aorta is greater than the pressure in the left ventricle.

**Value Dysfunction**

Valvular disorders most often affect the valves on the left side of the heart, the mitral and aortic valves. The mitral and aortic valves are constantly exposed to high pressures and mechanical stress that can lead to cardiac valve damage. When a valve is open, blood flows from one chamber through the valve to the next. A pressure gradient does not exist between the 2 chambers or between the structures (chamber or vessel) above and below the valve. However, as valvular dysfunction develops and progresses, pressure gradients between the 2 structures develop.

Valvular dysfunction is a gradual process; the valve or valves affected become stenotic or insufficient. A stenotic valve does not open entirely, resulting in a decreased amount of forward blood flow. An insufficient or regurgitant valve does not close completely, so some blood flows backward instead of forward.

The 3 most common types of acquired valve disorders are degenerative disease, rheumatic disease, and infective endocarditis. Degenerative disease occurs as a gradual destruction of the valve due to constant wear and tear. The degeneration occurs slowly and is characterized by increased thickening, stiffening, and calcification of the leaflets. Valve calcification develops as a person ages. Calcification of the valve occurs as collagen fibers become thick and disoriented, the amount of mucopolysaccharides decreases, and progressive accumulation of fatty tissue occurs.

Valve calcification develops as a person ages. Calcification of the valve occurs as collagen fibers become thick and disoriented, the amount of mucopolysaccharides decreases, and progressive accumulation of fatty tissue occurs. The development of calcium infiltration reduces mobility of the valve leaflets, increases tension on the chordae, elevates the leaflets, and facilitates chordal elongation or rupture. Rheumatic disease begins with fusion of the valve leaflets at the commissures. Then the area of fusion enlarges, leading to fibrosis and constriction of the chordae tendineae and thickening and calcification of the leaflets. Infectious endocarditis directly damages valve tissue.

**Cardiac Valve Surgery**

Valvular heart disease is usually a chronic, progressive disease. Cardiac compensatory mechanisms often maintain a state of equilibrium for years before valvular function deteriorates to the point at which signs and symptoms are evident and more definitive therapy is needed. The natural history of valvular heart disease has been dramatically altered during the past 30 years by medical and surgical management.

Surgical methods for improving the function of diseased cardiac valves include valve reconstruction and valve replacement with mechan-
ical prostheses, biological prostheses, or homograft valves. Although valve reconstruction can be performed to improve the function of insufficient or stenotic valves, most surgeries are performed because of valve insufficiency.

Replacement of the mitral valve is indicated for patients with severe valve calcification, marked subvalvular stenosis, or mitral stenosis accompanied by marked mitral regurgitation. Replacement of the native aortic valve with a prosthetic valve is used for patients with severely damaged valves. Valve replacement also is used when valve repair is unsuccessful.

The type of valve surgery is usually established at the time of the operation. Preoperative prediction of the operation that can be performed is based on the location and type of valvular disease and the skill and experience of the surgeon.

Timing of Cardiac Valve Surgery

Patients with cardiac valve disease are monitored closely by a cardiologist to determine the best time for valve surgery. Data obtained with transesophageal echocardiography are used to decide the best time for cardiac surgery and the type of repair needed. The data aid in determining the location and severity of leaflet prolapse; leaflet mobility and restriction; the point of leaflet coaptation; presence and severity of annular calcification; chordal fusion; and site, direction, and size of regurgitant blood flow. Cardiac valve surgery is commonly performed as soon as signs and symptoms of valvular dysfunction begin to occur. Ideally, surgery is performed before the development of left ventricular dysfunction or atrial enlargement that may lead to atrial dysrhythmias.

Minimally Invasive Cardiac Valve Surgery

Traditionally, cardiac valve surgery involved a median sternotomy incision, cardiopulmonary bypass, and cannulation of the right atrium and the aorta. Recently, valve surgery has been performed by using minimally invasive approaches.

Minimally invasive surgery is performed by using instruments designed to provide intracardiac retraction and to allow the surgeon to work through small thoracotomy incisions or ports. Percutaneous cardiopulmonary bypass, an intra-aortic balloon catheter for aortic occlusion, and instillation of a cardioplegic solution are used. Video-assisted techniques may be used. The early results of minimally invasive cardiac repair surgeries have been promising.

Valve Repair and Reconstructive Techniques

The term valvuloplasty is used to describe both valve repair and valve reconstruction. Although valve repair and reconstruction can be used to treat aortic valve disease, they are more commonly used to treat mitral valve dysfunctions. The mitral valve is more complex than the aortic valve both anatomically and functionally.

Mitral Valve Repair

Open Mitral Commissurotomy

Open mitral commissurotomy is used when the mitral valve is stenotic because of fused commissures. The fused commissures are incised from the annulus to the center of the mitral valve. The goals of open mitral commissurotomy are to improve leaflet mobility and to increase the size of the valve orifice. The procedure is most effective when the valve leaflets are thin and pliable. The length of the commissurotomy must be precise. If the incision extends too far toward the annulus, an annuloplasty may become necessary.

Open mitral commissurotomy is used early in the disease process. Indications for use of open mitral commissurotomy rather than mitral valve replacement include a loud opening snap on auscultation, good leaflet mobility on ventriculograms and echocardiograms, and no evidence of valvular calcification on radiographs. Surgery is recommended for patients when cardiac catheterization reveals that the mitral valve area is less than 1.5 cm².

Reshaping the Mitral Valve Annulus

Surgery for mitral insufficiency is recommended on the basis of the
patient’s signs and symptoms and the results of cardiac catheterization and echocardiography. An annuloplasty involves reconstruction of an insufficient mitral valve and is used to repair a deformed or dilated mitral valve annulus. The valve annulus is composed of a fibromuscular ring.

Two surgical techniques may be used if the mitral valve annulus is enlarged. Figure 4 depicts the first technique, in which the valve annulus is sutured to reduce the size of the enlarged annulus. The sutures are placed at both commissures, incorporating only the posterior annulus. In the second technique, a prosthetic annuloplasty ring is inserted (Figure 5). The annuloplasty ring is sewn to the mitral valve annulus to reshape the annulus.

Annuloplasty rings can be preshaped (rigid or semirigid) or flexible. The size and shape of the mitral valve annulus change during diastole and systole because of the contraction and relaxation of muscle bundles posteriorly surrounding the mural part of the mitral annulus. Historically, annuloplasty rings were rigid. Rigid rings were intended to remodel annular deformity associated with chronic atrial and ventricular enlargement, stabilize mitral valve repair by reducing tension on reconstructed valvular parts, enhance leaflet coaptation by reducing the mitral surface area, and prevent further annular dilatation. Today, more flexible annuloplasty rings are available. Borghetti et al found that a flexible annuloplasty ring (glutaraldehyde-treated autologous pericardium) improved valvular function. A more flexible annuloplasty ring resembles the patient’s inherent mitral valve annulus because the valve annulus can enlarge during diastole and become smaller during systole.

In decalcification of the mitral valve annulus, calcium is removed, the annulus is reconstructed, and further valve repair is done as indicated. Extensive valve calcification may necessitate mitral valve replacement.

Chordae Tendineae Repair or Reconstruction

Mitral insufficiency can also be related to dysfunction of the subvalvular mechanism. Elongated chordae or detached chordae contribute to prolapse of the anterior or posterior valve leaflets, resulting in mitral insufficiency. As described previously, the chordae tendineae support the valve leaflets and prevent the leaflets from prolapsing into the left atrium during systole. If the chordae are elongated or some of them are detached, they cannot provide the necessary valve support, resulting in prolapse of the leaflets.
into the left atrium during systole. As the mitral valve leaflets prolapse, mitral insufficiency occurs. Usually the posterior valve leaflet is affected.38

Shortening elongated chordae tendineae preserves accurate function of valve leaflets. The chordae tendineae are shortened and attached to the mitral valve leaflet or to the papillary muscle. Figure 6 depicts a technique used to shorten elongated chordae tendineae in which the chordae tendineae are folded onto themselves and then sutured to the valve leaflet. Elongated chordae tendineae can also be folded and tucked within the papillary muscle or folded and sutured to the exterior of a papillary muscle (Figure 7).

Ruptured chordae of the anterior leaflet may be replaced by transposing chordae from the posterior leaflet.39 Ruptured chordae can also be replaced by new chordae made of natural or artificial materials, such as polytetrafluoroethylene suture material.38-40 This procedure is referred to as chordal replacement (Figure 8).

Reconstruction of chordae tendineae is used to elongate fibrotic chordae. Shortened or fused chordae tendineae are excised. Shortened chordae are longitudinally cut in an effort to lengthen the chordae and improve support of the valve leaflets. If several chordae tendineae fuse, a fenestration is performed by excising a triangular segment of fibrous tissue (Figure 9).

**Valve Leaflet Repair**

Myxomatous and degenerative changes in the leaflet tissues with subsequent elongation or even rupture of the chordae can result in mitral insufficiency.41 Changes occur in the posterior or anterior leaflets. Repair of a valve leaflet may involve resecting a segment of the prolapsed leaflet (Figure 10). A prosthetic annuloplasty ring is usually required to provide additional support to the valve annulus after valve leaflet resection.

Valve leaflet reconstruction may be needed to repair damaged leaflets. Extensive infection of the mitral valve necessitates valvular replacement. However, localized infection and limited destruction of leaflets or chordae are treated with valve repair.39 The infected leaflets may be repaired with patches of glutaraldehyde-treated autologous or bovine pericardium.39-40 New chordae tendineae (neochords) are created from polytetrafluoroethylene sutures.38-40 Hvass et al41 reported successful transfer of a posterior tricuspid leaflet (with chordae and papillary muscle attached) to repair a torn posterior mitral valve leaflet.

**Papillary Muscle Repair**

Papillary muscles may become torn or detached during periods of myocardial ischemia. The damaged papillary muscles can be surgically reattached.

**Assessment of Mitral Valve Surgery**

The effectiveness of mitral valve repair and reconstruction is evaluated at the end of surgery. Traditionally, isotonic sodium chloride solution was injected into the left ventricle to assess valve competence. More commonly today, transesophageal echocardiography is used in the operating room at the end of the surgery to evaluate the effectiveness of the valve repair. If problems are noted, additional repair or reconstruction is necessary or the valve may need to be replaced.

**Aortic Valve Repair**

Repair of the aortic valve is not as developed as is repair of the mitral valve. Repair of a stenotic or insufficient aortic valve is more difficult than repair of the mitral valve.
because the closing mechanism is more precise.42

Open aortic commissurotomy may be used on stenotic or fused aortic valves. The commissures are incised in an attempt to open the stenotic valve.

Calcification of the valve cusps may contribute to aortic stenosis. Calcium deposits are carefully removed from the aortic valve. Before aortic valve prostheses became available, manual debridement of severely stenotic aortic valves was performed at some centers.43

Once aortic valve prostheses were available, fewer aortic valve repairs were done because of the high prevalence of recalcification and restenosis after valve debridement.43 The recent success of mitral valve repair and the application of the ultrasonic surgical aspirator have prompted renewed interest in aortic valve repair.44-48

Repair for treatment of aortic regurgitation may be more promising than repair for treatment of aortic stenosis. Aortic regurgitation is due to cusp retraction (by fibrosis or calcification), prolapse, and perforation or is due to dilatation of the aortic root.39 Cusp retraction is treated by extending or replacing the cusp with glutaraldehyde-treated bovine or autologous pericardium.39 A triangular section of a prolapsed aortic valve cusp is removed in an effort to eliminate aortic regurgitation. Pericardial patches are used to repair perforations in the aortic valve cusps. Valve resuspension may decrease or eliminate the amount of regurgitation caused by dilatation of the aortic valve annulus. Before valve resuspension, pathological aortic tissue is resected to restore the geometry of the aortic root.60 Fibrotic valve leaflets contribute to aortic insufficiency. Carpentier42 developed a technique to shave the edges of thickened valve leaflets to improve or eliminate aortic insufficiency.

As with mitral valve repair, accurate repair of the aortic valve is essential. The effectiveness of the repair is assessed at the end of surgery by using transesophageal echocardiography.

**Surgical Outcomes**

**Mitral Valve Repair Versus Mitral Valve Replacement**

A mitral commissurotomy is performed to improve valve function in a stenotic mitral valve with little calcification. Surgical commissurotomy improves outcomes for patients with New York Heart Association class III and class IV heart failure.24 Cohn et al50 reported that 95% of patients with New York Heart Association class III heart failure had survived 10 years after commissurotomies. The mean operative mortality rates for commissurotomy and valve replacement are 1% to 3% and 5% to 10%, respectively.

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**Figure 7** Repair of an elongated chordae tendineae by suturing within a papillary muscle (A) and suturing to the side of a papillary muscle (B).

Reprinted from Khonsari,49-50 with permission.
Mitral valve reconstruction offers another surgical option for patients experiencing acute mitral insufficiency due to ischemia.

Rankin et al\textsuperscript{51} reported that hospital mortality rates were significantly lower for patients undergoing valve repair (26\%) than for patients undergoing mitral valve replacement (53\%).

Outcomes related to cardiac valve repair surgery were investigated in multiple studies\textsuperscript{20,37,40,52-71} Most of the studies involved patients who had mitral valve repair (Tables 1 and 2). Mitral valve repair has been used to treat patients with mitral insufficiency, mitral stenosis, and mixed mitral valve disease (both mitral stenosis and mitral insufficiency). Successful outcomes have been reported for patients who had mitral valve repair for treatment of mitral valve dysfunction caused by degenerative valve disease, rheumatic valve disease, and other causes, including endocarditis. A variety of types of valve repair surgeries have been used, including annuloplasty with and without prosthetic rings, leaflet resection, leaflet patching, chordal shortening, chordal transfer, chordal replacement, debridement, commissurotomy, and papillary muscle reattachment.

Mitral valve repair and mitral valve replacement have been compared (Table 1). Overall, the operative mortality is lower after mitral valve repair than after mitral valve replacement.\textsuperscript{52,57,67,70} For patients who had mitral valve repair, survival rates at 4.5 to 15 years were either the same or better than the rates for patients who had mitral valve replacement.\textsuperscript{52,57,67,70} Freedom from reoperation rates were also similar for both groups of patients: 87\% to 100\% for valve repair and 80\% to 92\% for valve replacement.\textsuperscript{52,57,67,70}

Reported operative mortality rates for patients who had mitral
Valve repair were 0% to 5.4% (Tables 1 and 2). Patients who had mitral valve repair and no additional cardiac surgical procedures had operative mortality rates of 3.3% or less. Successful long-term survival rates (>10 years) have been reported for patients who had mitral valve repair. At 10 to 15 years after mitral valve repair surgery, freedom from reoperation rates were from 67% to 98%. Mitral valve repair surgery can be performed with few long-term complications, including thromboembolism and endocarditis.

Aortic Valve Repair Versus Aortic Valve Replacement

Aortic valve repair has been used successfully to relieve stenosis and avoid early aortic valve insufficiency. King et al performed mechanical decalcification of the aortic valve on 92 patients. At 5, 10, and 15 years after surgery, the freedom from reoperation rates were 77%, 48%, and 38%, respectively. Although ultrasonic decalcification may adequately relieve stenosis, the unacceptable prevalence of late regurgitation and the prevalence of restenosis have limited the use of this technique.

Izumoto et al repaired the aortic valves of 63 patients who had aortic regurgitation. The 5-year survival rate was 95.1%, and the freedom from reoperation rate was 78%. Izumoto and colleagues concluded that surgery for aortic valve repair is not yet an adequate alternative because of the prevalent need for reoperation. This conclusion was confirmed by Gillinov et al, who found that patients operated on for combined aortic and mitral valve repair had a 10-year survival rate of 62% and a freedom from reoperation rate of 65%.

Additional data need to be collected after aortic valve repairs. At this point, the early and long-term effects of aortic valve repairs are unknown.

Management of Patients After Valve Repair Surgery

Compared with valve replacement, valve repair is usually recommended earlier in the disease process. The advantage of this earlier treatment is that the patient is usually younger, the valve dysfunction is less advanced, and left ventricular function may be preserved. If surgery is performed purely for mitral valve repair or reconstruction, an uncomplicated postoperative surgical recovery is expected. However, often cardiac valve repair surgery is performed concomitantly with other cardiac surgical procedures, including tricuspid or aortic valve surgery and coronary artery bypass graft surgery. Postoperative management varies according to the type of cardiac surgery performed. Achievement of normothermia, oxygenation, fluid and electrolyte balance, hemodynamic stability, and comfort and early detection of potential complications are essential.

Care of patients after cardiac valve repair is similar to care of patients after cardiac valve replacement. In the immediate postoperative period, assessment of the patient’s hemodynamic status is important. The major goals of management are to optimize preload, enhance contractility, and reduce afterload. Patients with valvular disease often have increased cardiac filling pressures. Although postoperatively the dysfunctional cardiac valve is repaired, the heart requires time to adjust to the improved hemodynamic function. Preoperative pulmonary artery pressures are a useful guide for postoperative management. Patients usually do better in the postoperative period if
Fluid replacement is adjusted on the basis of presurgical right atrial and pulmonary artery wedge pressures. Vasoactive agents may be required in the immediate postoperative period to optimize cardiac output. Inotropic agents may be necessary to improve cardiac contractility, and vasodilators may be necessary to decrease systemic vascular resistance and aid in afterload reduction.

Continuous monitoring for potential cardiac dysrhythmias is essential in the postoperative period. Conduction disturbances commonly occur after valve surgery, because the mitral and aortic valves lie close to the conduction pathways. Transient or permanent heart block may occur because of edema, ischemia, or damage to the conduction pathways. Conduction disturbances may necessitate temporary epicardial pacing and at times even permanent pacemaker therapy.

Atrial dysrhythmias are a common complication after cardiac surgery and occur with increased frequency after cardiac valve surgery. Prophylactic beta-blockade is usually started preoperatively and continued postoperatively to prevent atrial dysrhythmias. Anticoagulation therapy is started after cardiac valve replacement. Although long-term anticoagulation is usually not necessary after cardiac valve repair, short-term anticoagulation therapy (3 months) is commonly started after cardiac valve repair.

### Table 1: Studies comparing mitral valve repair and mitral valve replacement

<table>
<thead>
<tr>
<th>Study</th>
<th>Valve dysfunction</th>
<th>Type of valve surgery</th>
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<tbody>
<tr>
<td>Duran et al.</td>
<td>Majority of patients had mixed mitral valve disease (72%); the remainder had mitral insufficiency (17%) and mitral stenosis (11%). The cause of valve dysfunction for most patients (&gt;90%) was rheumatic heart disease.</td>
<td>Patients had mitral valve repair (with a flexible prosthetic ring and additional valvular, commissural, and subvalvular repairs as needed) (n = 212) or mitral valve replacement (with a Hancock porcine bioprosthesis) (n = 307). Additional cardiac surgical procedures were performed for 57% of the patients (eg, aortic valve surgery).</td>
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<tr>
<td>Perier et al.</td>
<td>Majority of patients had mixed mitral valve disease (48.5%); remainder had mitral insufficiency (39%) and mitral stenosis (12.5%). The cause of the valve dysfunction was rheumatic heart disease (66.5%), degenerative disease (28%), and other causes (5.5%).</td>
<td>Patients had mitral valve repair (with a prosthetic ring) (n = 100), mitral valve replacement with a porcine valve (n = 100), Starr-Edwards tilting disc valves (n = 100), and Björk-Shiley valves (n = 100).</td>
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<td>Grossi et al.</td>
<td>The majority of patients had mitral valve insufficiency (76%); the remainder had mixed mitral valve disease (24%). Valvular dysfunction was caused by degenerative disease (40%), rheumatic heart disease (33%), and other causes (27%).</td>
<td>Patients had either mitral valve reconstruction with ring annuloplasty (n = 725) or a mitral valve replacement with a St Jude Medical valve (n = 514). Additional cardiac surgical procedures were performed for 52% of the patients (eg, coronary artery bypass graft surgery).</td>
</tr>
<tr>
<td>Mohty et al.</td>
<td>All of the patients had mitral insufficiency.</td>
<td>Patients had either mitral valve repair (n = 679) or mitral valve replacement (n = 238).</td>
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<tr>
<td>Sternik et al.</td>
<td>All of the patients had mitral insufficiency due to infective endocarditis</td>
<td>Patients (n = 44) had mitral valve surgery. 64% had mitral valve replacement, and 36% had mitral valve repairs, including leaflet prosthetic patches, leaflet resection, and artificial chordae.</td>
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Association guidelines for the management of patients with valvular heart disease recommend a 3- to 5-day overlap of treatment with heparin and warfarin and discontinuation of the heparin when the patient has an international normalized ratio (INR) of 2.0 to 3.0. Long-term aspirin therapy may be recommended for patients after valve repair. Long-term anticoagulation therapy is necessary for patients who have a history of thrombus formation, atrial fibrillation, left atrial dilatation, or a mechanical valve.

Teaching patients about anticoagulation therapy is essential. Patients need frequent assessment and monitoring of INRs. Monthly determination of the INR is indicated; the ratio should be maintained at 2.0 to 3.5 (or within the desired range as prescribed by the cardiologist). Patients should report the occurrence of bruising, bleeding, epistaxis, and hemoptysis.

Dieticians should review nutritional precautions with patients and the patients’ families before patients are discharged from the hospital. Patients are advised what foods have vitamin K and are told to avoid eating large amounts of yellow and dark green vegetables and fatty foods, which increase the absorption of vitamin K. Because vitamin K promotes clotting of the blood, eating many foods with increased amounts of the vitamin can reduce the effectiveness of warfarin.

Patients should avoid alcohol and medications (eg, aspirin) that may interact with warfarin, because both potentiate the anticoagulation effect of warfarin.

Bacterial endocarditis is a serious and potentially life-threatening complication of cardiac valve surgery. Lifelong antibiotic prophylaxis is recommended for patients with acquired valvular dysfunction.

### Mortality
- Hospital mortality was 1.9% for patients with mitral valve repair and 11.4% for patients with mitral valve replacement.
- Hospital mortality was 2% for patients with mitral valve repair and 12%, 13%, and 12%, respectively, for patients with mitral valve replacement.
- Operative mortality was 7.2% for patients who received a St Jude valve and 5.4% for patients who had valve annuloplasty (no significant difference). Isolated mitral valve operation had a mortality rate of 2.5% for valve replacement and 2.2% for mitral valve repair.
- Mortality rate for mitral valve repair was less than the mortality rate for mitral valve replacement.
- Hospital mortality rate was 21% for patients who had mitral valve replacement and 0% for patients who had valve repair.

### Survival
- Survival at 4.5 years was 96.4% for patients with mitral valve repair and 81% for patients with mitral valve replacement.
- Survival rates at 7 years were 82% (valve repair), 56% (porcine bioprosthesis), 61% (Starr-Edwards), and 60% (Bjork-Shiley).
- Survival rate was 89.3% (valve replacement) and 84.1% (valve repair) at 8 years. For patients with isolated, nonrheumatic mitral valve disease, 8-year survival rate was better in the mitral valve reconstruction group (95.4%) than in the St Jude group (91.6%) (P = .03).
- Survival rates at 15 years were better for mitral valve repair than for mitral valve replacement for repair of posterior leaflets (41% vs 31%) and for repair of anterior leaflets (42% vs 31%). Survival rates were 79% at 5 years for patients who had mitral valve replacement and 100% at 3 years for patients who had mitral valve repair.

### Reoperation
- Freedom from reoperation at 7 years was 87% (valve repair), 80% (porcine bioprosthesis), 92% (Starr-Edwards), and 82% (Bjork-Shiley).
- Freedom from cardiac death and reoperation at 8 years was 83.4% for the St Jude group and 73.1% for the valve reconstruction group.
- Freedom from reoperation at 15 years was similar for patients having repair and replacement.
- Freedom from reoperation was 82% at 5 years for patients who had mitral valve replacement and 100% at 3 years for patients who had mitral valve repair.
<table>
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<tr>
<td>Cosgrove et al, 56 1986</td>
<td>Patients had mitral insufficiency caused by degenerative disease (80%), rheumatic heart disease (11%), and other causes (9%).</td>
<td>Patients (n = 117) had mitral valve repair (ring annuloplasty, resection of posterior leaflet, chordal shortening, commissurotomy, annuloplasty without ring, leaflet patching, chordal transfer, debridement, and/or suture repair). Forty-eight percent had mitral valve repair only; the remainder had mitral valve repair and additional cardiac surgery (eg, coronary artery bypass surgery).</td>
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<td>Lessana et al, 55 1990</td>
<td>Patients had mitral valve insufficiency (54%) or mixed mitral valve disease (46%) caused by rheumatic heart disease (65.5%), degenerative valve disease (30.5%), and other causes (4%).</td>
<td>Valve repair procedures included prosthetic annuloplasty ring, commissurotomy resection of chordae, resection of nodules, leaflet resection, chordal shortening, and/or transfer of chordae. Additional cardiac surgical procedures were performed for 49% of the patients (eg, aortic valve surgery). A subset of this sample (17%) included children.</td>
</tr>
<tr>
<td>DeLoche et al, 54 1990</td>
<td>Patients had mitral valve insufficiency due to degenerative disease (58%), rheumatic heart disease (38%), or other causes (4%).</td>
<td>Patients had mitral valve repair, including annuloplasty, leaflet resection, chordal shortening, leaflet mobilization, commissurotomy, and papillary muscle reimplantation. Additional cardiac surgical procedures were performed for 17% of the patients (eg, tricuspid valve reconstruction).</td>
</tr>
<tr>
<td>Alvarez et al, 53 1996</td>
<td>All of the patients had mitral valve insufficiency caused by degenerative disease.</td>
<td>All patients (N = 155) had mitral valve reconstruction surgery. Only 3% of patients had a ring annuloplasty performed. Additional cardiac surgical procedures were performed for 19% of the patients (eg, coronary artery bypass surgery).</td>
</tr>
<tr>
<td>Carpentier et al, 52 1996</td>
<td>All of the patients had mitral insufficiency caused by extensive calcification of the mitral valve annulus.</td>
<td>All patients (N = 67) had mitral valve repair.</td>
</tr>
<tr>
<td>David et al, 50 1998</td>
<td>All of the patients had mitral insufficiency due to degenerative disease.</td>
<td>All patients (N = 324) had mitral valve repair: 165 had chordal replacement with expanded polytetrafluoroethylene (ePTFE) sutures to replace chordae tendineae, and 159 had the valve repaired without the use of ePTFE (leaflet resection, chordal shortening, and/or chordal transfer). The majority of patients also had prosthetic ring annuloplasty. Additional cardiac surgical procedures were performed for 26% of the patients (eg, aortic valve replacement).</td>
</tr>
<tr>
<td>Borghetti et al, 49 2000</td>
<td>All of the patients had mitral valve insufficiency. The cause of the valve dysfunction was degenerative disease in the majority of patients (91%).</td>
<td>All patients (N = 44) had the mitral valve repaired with quadrangular resection of the posterior leaflet; 23 patients had an autologous pericardial ring inserted, and 21 had a Carpentier-Edwards rigid ring inserted.</td>
</tr>
<tr>
<td>Kobayashi et al, 48 2000</td>
<td>All of the patients had mitral valve insufficiency. Mitral insufficiency was due to prolapse of both valve leaflets (39%), prolapse of the anterior leaflet (47%), and prolapse of the posterior leaflet (14%). The cause of the valve dysfunction was degenerative disease in the majority of patients (88%); the remainder had an infective cause (12%).</td>
<td>All patients (N = 74) had mitral valve repair with chordal replacement with ePTFE; 50% also had repair of the mitral valve leaflets, and 41% had the Maze procedure.</td>
</tr>
</tbody>
</table>
**Mortality**

| Operative mortality was 2% for patients who had mitral valve repair only and 7% for patients who had mitral valve repair combined with additional cardiac surgical procedures. |
| Operative mortality rate was 4%. |
| Operative mortality was 5.3%. |
| Operative mortality rate was 3.9%. |
| Hospital mortality rate was 3.3%. |
| Operative mortality was 1%. |
| Hospital mortality was 0%. |
| Hospital mortality was 1.4%. |

**Survival**

| Survival at 2 years was 91%. |
| Survival rate was 73% at 15 years. At 15 years, 94% of patients were free of thromboembolic events, and 97% were free of endocarditis. |
| Survival rate was 46% at 15 years. At 15 years, freedom from infective endocarditis was 96%, and freedom from thromboembolism was 90.4%. Overall functional status of patients was good: 98% of patients were New York Heart Association class I or class II. |
| Survival rate was 93% at 7 years. |
| Survival rate was 75% at 10 years. At 10 years, the freedom from stroke was 94%, freedom from transient ischemic attacks was 92%, and freedom from endocarditis was 99%. |
| All patients were alive at 4-year follow-up. No complications occurred. Patients with a flexible pericardial ring had improved left ventricular function. |
| Event-free survival rates (as assessed by the freedom from cardiac death, thromboembolism, reoperation, and anticoagulation-related hemorrhage) were 91.3% at 5 years and 71.6% at 10 years. |

**Reoperation**

| Freedom from reoperation at 2 years was 96%. |
| Freedom from reoperation was 67% at 13 years in the group with rheumatic heart disease and 98% at 10 years in the group with degenerative heart disease. |
| At 15 years 87% of patients were free from reoperation. |
| Freedom from reoperation was 84.9% at 15 years. |
| At 7 years freedom from reoperation was 87%. |
| At 10 years the freedom from reoperation was 96%. |
| No reoperations were needed at 4-year follow-up. |
| Actuarial reoperation-free rates were 94.3% at 5 years and 81.7% at 5 years. |

Continued
Kuwaki et al., 2000

All patients had mitral valve disease. All patients had mitral valve insufficiency or combined mitral insufficiency and mitral stenosis. The causes of the mitral valve disease were degenerative (62%), rheumatic (17%), endocarditis (13%), and ischemic (8%).

Nagy et al., 2000

All patients had mitral valve disease.

Phillips et al., 2000

All patients had mitral valve disease. All patients had mitral valve insufficiency due to anterior leaflet prolapse.

Alfieri et al., 2001

All patients had mitral valve insufficiency. The causes were degenerative disease (80.8%), rheumatic heart disease (9.6%), endocarditis (6.1%), and ischemia (2.3%). Both valve leaflets were prolapsed in 57%; the anterior leaflet was prolapsed in 25%, the posterior leaflet was prolapsed in 12%, and the free edge of the valve was eroded in 5%.

Braunberger et al., 2001

All patients had mitral valve disease. All patients had mitral valve insufficiency due to degenerative disease (90%) or bacterial endocarditis (10%). Mitral valve insufficiency was due to prolapse of the posterior valve leaflet in 61%, both leaflets in 20%, and the anterior leaflet in 18%.

Cozzi et al., 2001

All patients had mitral valve insufficiency.

Dreyfus et al., 2001

All patients had mitral valve insufficiency with anterior leaflet prolapse. For most patients, mitral insufficiency was due to the degenerative process.

Tomita et al., 2002

All patients had moderate-severe mitral insufficiency with prolapse of both leaflets with dysfunction of both anterior and posterior mitral valve leaflets.

Fasol and Mahdjoobian, 2002

Patients had mitral valve insufficiency due to degenerative disease.

Piciche et al., 2002

All patients had mitral valve insufficiency. The causes of the mitral valve disease were degenerative (79%) and rheumatic (21%).

<table>
<thead>
<tr>
<th>Study</th>
<th>Valve dysfunction</th>
<th>Type of valve surgery</th>
</tr>
</thead>
<tbody>
<tr>
<td>Kuwaki et al., 2000</td>
<td>All patients had mitral valve insufficiency or combined mitral insufficiency and mitral stenosis. The causes of the mitral valve disease were degenerative (62%), rheumatic (17%), endocarditis (13%), and ischemic (8%).</td>
<td>All patients (N = 86) had mitral valve repair, including annuloplasty, leaflet resection, chordal implantation, and commissurotomy.</td>
</tr>
<tr>
<td>Nagy et al., 2000</td>
<td>All patients had mitral valve disease.</td>
<td>All of the patients (N = 130) had mitral valve repair (mitral plication annuloplasty with a semicircular buttressed suture around the posterior leaflet); 83 had combined procedures including coronary artery bypass graft (40 patients) and aortic valve replacement (43 patients), and 65 had a rerepair of the mitral valve.</td>
</tr>
<tr>
<td>Phillips et al., 2000</td>
<td>All patients had mitral valve insufficiency due to anterior leaflet prolapse.</td>
<td>All of the patients (N = 121) had mitral valve annuloplasty; 46 (38%) had chordal shortening and 75 (62%) had chordal replacement with ePTFE sutures.</td>
</tr>
<tr>
<td>Alfieri et al., 2001</td>
<td>All patients had mitral valve insufficiency. The causes were degenerative disease (80.8%), rheumatic heart disease (9.6%), endocarditis (6.1%), and ischemia (2.3%). Both valve leaflets were prolapsed in 57%; the anterior leaflet was prolapsed in 25%, the posterior leaflet was prolapsed in 12%, and the free edge of the valve was eroded in 5%.</td>
<td>All patients had mitral valve repair.</td>
</tr>
<tr>
<td>Braunberger et al., 2001</td>
<td>All patients had mitral valve disease. All patients had mitral valve insufficiency due to degenerative disease (90%) or bacterial endocarditis (10%). Mitral valve insufficiency was due to prolapse of the posterior valve leaflet in 61%, both leaflets in 20%, and the anterior leaflet in 18%.</td>
<td>All patients (N = 162) had a Carpentier annuloplasty ring inserted. 78% of patients also had valve resection and 30% of patients had chordae shortened or transposed.</td>
</tr>
<tr>
<td>Cozzi et al., 2001</td>
<td>All patients had mitral valve insufficiency.</td>
<td>All patients (N = 121) had mitral valve repair (quadrangular resection of the posterior mitral leaflet, triangular resection of the anterior mitral leaflet, chordal transposition, neochord sutures, and/or annuloplasty rings).</td>
</tr>
<tr>
<td>Dreyfus et al., 2001</td>
<td>All patients had mitral valve insufficiency with anterior leaflet prolapse. For most patients, mitral insufficiency was due to the degenerative process.</td>
<td>All patients (N = 132) had mitral valve repair; 70% had repositioning of the papillary muscle and 30% had chordal shortening.</td>
</tr>
<tr>
<td>Tomita et al., 2002</td>
<td>All patients had moderate-severe mitral insufficiency with prolapse of both leaflets with dysfunction of both anterior and posterior mitral valve leaflets.</td>
<td>All patients (N = 17) had repair of the mitral valve with artificial chordae (CV-4 ePTFE sutures). All patients had suture annuloplasties to correct annular dilatation.</td>
</tr>
<tr>
<td>Fasol and Mahdjoobian, 2002</td>
<td>Patients had mitral valve insufficiency due to degenerative disease.</td>
<td>All patients (N = 37) had mitral valve repair involving resection of the posterior leaflet, triangular resection of the anterior leaflet, and a ring annuloplasty.</td>
</tr>
<tr>
<td>Piciche et al., 2002</td>
<td>All patients had mitral valve insufficiency. The causes of the mitral valve disease were degenerative (79%) and rheumatic (21%).</td>
<td>All patients (N = 66) had mitral valve repair involving resection of the anterior or posterior leaflet, placement of artificial chordae, commissurotomy, annulus decalcification, and a ring annuloplasty. 45% had additional procedures (including repair or replacement of the aortic valve and coronary artery bypass graft surgery).</td>
</tr>
<tr>
<td>Mortality</td>
<td>Survival</td>
<td>Reoperation</td>
</tr>
<tr>
<td>-----------------------------------</td>
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<td>----------------------------------------------</td>
</tr>
<tr>
<td>Hospital mortality was 2.3%</td>
<td>Actuarial survival at 10 years was 83.2%. At 10 years, freedom from thromboembolism was 90.9%, and freedom from endocarditis was 98.5%.</td>
<td>Freedom from reoperation at 10 years was 86.8%.</td>
</tr>
<tr>
<td>Hospital mortality was 3%</td>
<td>Survival rate was 95.2% at 2 years.</td>
<td>At 2 years, 6.6% of patients required mitral valve replacement.</td>
</tr>
<tr>
<td>Hospital mortality was 0.8%</td>
<td>Survival rate was 94.4% at 5 years.</td>
<td>Freedom from reoperation at 5 years was 90%.</td>
</tr>
<tr>
<td>Hospital mortality was 0.7%</td>
<td>Survival rate was 95% at 4 years.</td>
<td>Risk of reoperation was 1.4% in patients who had chordal replacement and 14.8% in patients who had chordal shortening.</td>
</tr>
<tr>
<td>Hospital mortality was 1.9%</td>
<td>The Kaplan-Meier 20-year survival rate was 48%, which is similar to the survival rate of a normal population with the same age structure.</td>
<td>Freedom from reoperation at 20 years was 95%.</td>
</tr>
<tr>
<td>Hospital mortality was 2.3%</td>
<td>Survival rates at 8 years were better for patients who had repositioning of the papillary muscle (96.3%) than for patients who had chordal shortening (88.1%).</td>
<td>Freedom from reoperation at 4 years was 94%.</td>
</tr>
<tr>
<td>Hospital mortality was 0%</td>
<td>All patients were alive at 4-year follow-up.</td>
<td>No data reported.</td>
</tr>
<tr>
<td>Hospital mortality was 0%</td>
<td>Survival rate at 2 years was 97%.</td>
<td>Freedom from reoperation at 2 years was 100%.</td>
</tr>
<tr>
<td>Hospital mortality was 0%</td>
<td>Survival rate at 3 years was 94%.</td>
<td>Freedom from reoperation at 3 years was 93%.</td>
</tr>
</tbody>
</table>
Patients who have valve repair still must take antibiotics prophylactically before dental work, gastrointestinal procedures, genitourinary procedures, surgery, and other invasive procedures to minimize the risk of cardiac valve infection.

Any patient who has had an episode of rheumatic fever is at high risk for recurrent episodes of acute rheumatic fever. Patients with a history of rheumatic fever should take antibiotics throughout life as prophylaxis against the recurrence of rheumatic carditis.25

After discharge, patients are followed up by their cardiac surgeon within the first few weeks of surgery. The interval for routine follow-up depends on each patient’s needs. Patients receiving anticoagulation therapy require frequent monitoring of their INR. After anticoagulation therapy is discontinued, patients usually have follow-up with a cardiologist at least yearly.

Future

As the number of cardiac valve repairs increases, most likely more patients with coronary artery disease and valve dysfunction will have coronary artery bypass graft surgery and valve repair. In addition, an increased number of patients will have one dysfunctional valve repaired and another dysfunctional valve replaced.

An increasing number of patients will also have rerepair of an originally repaired cardiac valve. The rerepair may involve procedures needed to make adjustments to earlier repairs, or the original repair may be intact but the valve may need repair of newly developed valvular dysfunction. Although the prevalence of rerepaired cardiac valves is low, successful rerepairs have been reported.80,81,83-85

Minimally invasive approaches are currently being used for valve repair.86,87,106 New approaches to minimally invasive valve repair will be used for patients who have valve repair in the future. It is anticipated that 3-dimensional surgical visualization systems will further enhance visualization of cardiac valves.40

Technology will continue to change cardiac surgery. Currently computer-enhanced mitral valve surgery is being performed.86,88 The computer-enhanced telemanipulation system offers the potential for endoscopic valve repair with hands-free surgery. Repair of the mitral valve is performed via remote control; the surgeon sits at a surgical console directing computer-enhanced instruments used to perform the surgery. The surgeon’s motions are sensed by motion sensors, computed, and transferred online to the tip of the end-effectors (within the thoracotomy incision). From the console where the surgeon sits, he or she can communicate with the operating room staff via wireless microphones. This technology has been used successfully in repair of valve leaflets and chordae and placement of annuloplasty valve rings. Surgeons claim that totally endoscopic mitral valve repair surgery can be successfully performed with this technology.82

The future will bring advances in tissue transfer. Transfer of autologous tissue from the posterior leaflet of the tricuspid valve has been used to repair torn mitral valve leaflets.80,81 Tissue-engineered valve leaflets are being investigated and may be used in the future.

No nursing research has been done in patients having cardiac valve repair, so many research possibilities exist. Studies of preoperative preparation, postoperative management, and recovery at home are needed.

Conclusion

The increase in the number of valve repairs will continue. If a valve can be repaired, an attempt to fix the dysfunctional valve is beneficial. The native valve is preserved, and the risks of chronic anticoagulation and prosthetic valve failure are avoided.86 Although, the repair may not last a lifetime, it may delay surgery for valve replacement and thus delay the risks associated with prosthetic valve replacement. Outcome data for mitral valve repair are promising. Additional outcome data for aortic valve repair are needed.

According to Grunkemeier and colleagues,84 valve repair, when practical, should be considered preferable to replacement, in both mitral and aortic positions, although aortic valve repair has not yet stood the test of time.

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Advances in Cardiac Surgery: Valve Repair
Debra Lynn-McHale Wiegand

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