Valvular Heart Disease: Changing Concepts in Disease Management

Acute Valvular Regurgitation

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Acute severe valvular regurgitation is a surgical emergency, but accurate and timely diagnosis can be difficult. Although cardiovascular collapse is a common presentation, examination findings to suggest acute regurgitation may be subtle, and the clinical presentation may be nonspecific. Consequently, the presentation of acute valvular regurgitation may be mistaken for other acute conditions, such as sepsis, pneumonia, or nonvalvular heart failure. Although acute regurgitation may affect any valve, acute regurgitation of the left-sided valves is more common and has greater clinical impact than acute regurgitation of right-sided valves.

Data to guide appropriate management of patients with acute regurgitation are sparse; there are no randomized trials, and much of the literature describes either small series or the experiences of specific centers. Despite these limitations, the available data are sufficient to allow identification of general principles as well as development of applicable guidelines from both the American College of Cardiology/American Heart Association and European Society of Cardiology. The guidelines recommend valve surgery for symptomatic patients with aortic or mitral regurgitation, including those with acute regurgitation. The data and guidelines emphasize overarching clinical principles, including the need for a high clinical suspicion of acute regurgitation, timely use of echocardiography, and, in the majority of patients, rapid progression to surgery.

Causes

Causes of acute regurgitation overlap with causes of chronic regurgitation and vary depending on the valve affected (Table 1). Endocarditis may affect either the aortic or mitral valve, whereas other causes are unique to the specific valve involved. The majority of causes of acute regurgitation present as an acute or subacute event. However, acute regurgitation can occur in patients with chronic regurgitation, when regurgitant severity is exacerbated by factors such as coronary ischemia, chordal rupture, or leaflet perforation from endocarditis. Acute regurgitation of either the aortic or mitral valve may result from procedural complications of percutaneous valve procedures. In addition, acute prosthetic valve regurgitation is seen more frequently as more patients undergo valve surgery. Acute prosthetic valve regurgitation is usually due to a tear of a bioprosthetic leaflet or thrombosis of a mechanical valve, although perivalvular regurgitation can occur, particularly in prosthetic valve endocarditis.

Acute aortic regurgitation is most commonly due to endocarditis, but there are a variety of less common causes as well. Aortic dissection, whether due to Marfan syndrome, bicuspid aortic valve, or atherosclerotic disease, may present with aortic regurgitation. Blunt trauma may result in leaflet rupture. Another less common cause is rupture of a fenestration in the aortic leaflet.

Acute mitral regurgitation may result from either “organic” or “functional” causes. Organic causes are those that result in permanent structural disruption of the valve, such as leaflet perforation from endocarditis, chordal rupture in myxomatous valve disease, or papillary muscle rupture due to myocardial infarction. Functional mitral regurgitation results from abnormalities of the left ventricle, such as cardiomyopathies in which the papillary muscles are laterally displaced, or acute ischemia, in which an akinetic wall segment and papillary muscle impair mitral valve closure. The distinction between organic and functional causes is an important one because treatment of organic causes requires surgical repair, whereas functional causes may improve with treatment of the underlying myocardial ischemia, infarction, or cardiomyopathy.

Functional mitral regurgitation is more often chronic than acute. However, processes that result in rapid decline of ventricular function may cause acute functional mitral regurgitation as part of the presentation of acute heart failure. Examples include myocarditis or rapidly developing cardiomyopathies such as Takotsubo cardiomyopathy (left apical ballooning) and peripartum cardiomyopathy. Emphasizing the variability in pathological process, a study demonstrated that mitral regurgitation in Takotsubo cardiomyopathy can result from outflow tract obstruction and systolic anterior mitral leaflet motion due to apical ballooning with preserved basal ventricular function. Rheumatic carditis can cause acute mitral regurgitation through a combination of leaflet inflammation and myocardial dysfunction, with some data suggesting that the degree of valve dysfunction drives outcomes. Although uncommon in industrialized nations, acute rheumatic carditis remains a significant issue in developing countries.
Pathophysiology

Most clinicians are familiar with the pathophysiology and hemodynamic impact of chronic regurgitation, but the stark differences between acute and chronic regurgitation are important to understand to make an accurate diagnosis of acute regurgitation. Chronic regurgitation of either the aortic or mitral valve affords time for the ventricle to dilate to accommodate the regurgitant volume. This adaptation maintains forward stroke volume and cardiac output despite the regurgitant volume. Correspondingly, left ventricular end-diastolic pressure remains normal unless there is coexistent pathology that impairs diastolic function.

The lack of time for adaptation to additional blood volume leads to the cascade of events typical of acute regurgitation. Acute aortic and mitral regurgitation share some common hemodynamic sequelae, despite the differences in pathogenesis and valve location within the circulation. In both circumstances, the left ventricle is not able to adequately compensate for the regurgitant volume, and excessive backward blood flow impairs forward stroke volume. Compensatory tachycardia may preserve cardiac output initially, but eventually hypotension, organ failure, and other evidence of cardiogenic shock will develop. Pulmonary capillary wedge pressure increases abruptly and pulmonary edema develops, although by different mechanisms depending on the valve involved. Notably, acute exacerbation of chronic regurgitation may result in similar hemodynamic changes.

Aortic Valve Regurgitation

Acute aortic regurgitation differs from chronic aortic regurgitation in several important ways. In acute aortic regurgitation, normal ventricular size results in a marked increase in end-diastolic pressure relative to the regurgitant volume. Impaired forward stroke volume yields a decreased systolic pressure and a narrow pulse pressure. Although there is some degree of compensation by a Frank-Starling mechanism, the ventricle is functioning on a steep pressure-volume curve because of the lack of chamber dilation. This contrasts with chronic regurgitation, in which end-diastolic pressures are relatively low, and the additional stroke volume manifests as an increased systolic pressure (Table 2). Therefore, reliance on pulse pressure as an indicator of regurgitation may significantly underestimate the severity of acute aortic regurgitation. In chronic aortic regurgitation, increased systolic pressure results in increased afterload. This is not seen in acute regurgitation given the low stroke volume. However, if acute regurgitation has resulted in shock, sympathetic activation and the renin-angiotensin cascade may result in increased systemic vascular resistance.

Abnormal mitral valve function due to acute hemodynamic changes may further impair stroke volume. Marked elevation of left ventricular diastolic pressure can cause early closure of the mitral valve, and tachycardia will limit mitral inflow, resulting in decreased ventricular filling. The presence of preexisting chronic aortic regurgitation and ventricular enlargement may blunt the hemodynamic impact of acutely worsened regurgitation. Conversely, preexisting disease processes that impair diastolic function, such as hypertension or aortic stenosis, may result in markedly more dramatic clinical presentation of acute aortic regurgitation.

Coronary ischemia may develop as a consequence of aortic regurgitation. Decreased diastolic coronary flow decreases myocardial perfusion, whereas elevated end-diastolic pressures and tachycardia increase myocardial oxygen demand. This supply-demand mismatch is obviously further exacerbated if obstructive coronary lesions are present or if aortic dissection impairs coronary flow.

**Table 1. Causes of Acute Regurgitation**

<table>
<thead>
<tr>
<th>Aortic Regurgitation</th>
<th>Mitral Regurgitation</th>
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</thead>
<tbody>
<tr>
<td>Endocarditis</td>
<td>Chordal rupture</td>
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<tr>
<td>Aortic dissection (type A)</td>
<td>Endocarditis</td>
</tr>
<tr>
<td>Ruptured fenestration</td>
<td>Papillary muscle rupture</td>
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<tr>
<td>Blunt chest trauma</td>
<td>Papillary displacement due to ischemia*</td>
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<tr>
<td>Prosthetic valve dysfunction</td>
<td>Acute rheumatic fever with carditis</td>
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<tr>
<td></td>
<td>Acute cardiomyopathy*</td>
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<td></td>
<td>Prosthetic valve dysfunction</td>
</tr>
</tbody>
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**Table 2. Comparison of Findings in Acute and Chronic Severe Regurgitation**

<table>
<thead>
<tr>
<th></th>
<th>Acute</th>
<th>Chronic</th>
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<tbody>
<tr>
<td><strong>Hemodynamics</strong></td>
<td></td>
<td></td>
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<tr>
<td>Cardiac output</td>
<td>↓</td>
<td>N</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>N ↓</td>
<td>↓</td>
</tr>
<tr>
<td>Systolic pressure</td>
<td>↓</td>
<td>↑</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure</td>
<td>↑ ↑</td>
<td>N</td>
</tr>
<tr>
<td>Left ventricular size</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Examination</strong></td>
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<tr>
<td>Diastolic murmur</td>
<td>Soft, early</td>
<td>Holodiastolic, decrescendo</td>
</tr>
<tr>
<td>S1</td>
<td>Soft</td>
<td>Normal</td>
</tr>
<tr>
<td>S2</td>
<td>Loud P2</td>
<td>Normal</td>
</tr>
<tr>
<td>S3</td>
<td>Present</td>
<td>Absent</td>
</tr>
<tr>
<td><strong>Mitril regurgitation</strong></td>
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<tr>
<td>Cardiac output</td>
<td>↓</td>
<td>N</td>
</tr>
<tr>
<td>Ejection fraction</td>
<td>N ↓</td>
<td>↑</td>
</tr>
<tr>
<td>Left ventricular end-diastolic pressure</td>
<td>↑ ↑</td>
<td>N</td>
</tr>
<tr>
<td>Left atrial compliance</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td>Left ventricular size</td>
<td>N</td>
<td>↑</td>
</tr>
<tr>
<td><strong>Examination</strong></td>
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</tr>
<tr>
<td>Murmur</td>
<td>Soft, decrescendo</td>
<td>Holosystolic</td>
</tr>
<tr>
<td>S3</td>
<td>May be present</td>
<td>Absent</td>
</tr>
<tr>
<td>V waves of CVP</td>
<td>May be present</td>
<td>Absent</td>
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</tbody>
</table>

* N indicates normal; ↑, increased from normal; ↓, decreased from normal; CVP, central venous pressure.
regurgitation increases volume into a normally compliant left atrium, resulting in a marked increase in left atrial pressure (Table 2). A significant V wave may be evident in either condition, although it is more pronounced in acute regurgitation. Because of the increased left atrial pressure from acute mitral regurgitation, pulmonary edema is a common consequence. Preexisting conditions may affect tolerance of acutely increased left atrial and left ventricular volume. Patients with a history of chronic mitral regurgitation and preserved ventricular function may tolerate the marked increase in volume better, whereas patients with impaired ventricular function may quickly decompensate with acute worsening of mitral regurgitation. Those patients with pulmonary edema associated with ST-segment elevation myocardial infarction often have coexistent mitral regurgitation, with often underestimated severity and a poor prognosis. As with acute aortic regurgitation, there is some degree of initial compensation afforded by increased preload, but the inability of the ventricle and atrium to accommodate the increased volume results in marked increase in left ventricular end-diastolic and left atrial pressures.

**Clinical Presentation**

The majority of patients with acute aortic or mitral regurgitation will present with dyspnea, hemodynamic instability, and symptoms of shock, including weakness, dizziness, and altered mental status. Symptoms at presentation may also reflect the underlying pathogenesis of acute regurgitation, such as severe chest pain from aortic dissection or fever from endocarditis. A subset of patients with acute mitral regurgitation may present solely with new-onset dyspnea, without evidence of impending cardiovascular collapse, and may therefore be misdiagnosed with a noncardiogenic pulmonary process or heart failure from another cause (Figure 1).

On examination, tachycardia, hypotension, peripheral vasodilatation, and other evidence of cardiac shock are common. As listed in Table 2, examination findings typically seen in chronic regurgitation may be absent or subtle. For the aforementioned reasons, the findings of chronic regurgitation related to ventricular enlargement, such as apical displacement, are typically absent, and murmurs are frequently soft. The presence of tachycardia and tachypnea further impairs the detection of faint murmurs.

In acute aortic regurgitation, the rapid equilibration of left ventricular and aortic diastolic pressures results in a faint early diastolic murmur, in contrast to the louder decrescendo diastolic murmur of chronic significant aortic regurgitation. Early closure of the mitral valve due to elevated left ventricular end-diastolic pressures yields a soft S1, lack of aortic leaflet coaptation during valve closure results in a soft A2, and, if pulmonary hypertension is present, there may be a loud P2, findings not typical of chronic regurgitation. The eponymous peripheral signs associated with chronic aortic regurgitation typically reflect increased pulsatility from increased stroke volume and wide pulse pressure. Because of the diminished stroke volume and decreased pulse pressure of acute aortic regurgitation, these signs are not typically present. With acute severe mitral regurgitation, rapid equilibration of ventricular and atrial pressures during systole results in a faint systolic murmur rather than the holosystolic murmur typically heard with chronic mitral regurgitation. Reliance on examination findings alone to diagnose acute regurgitation or estimate severity is fraught with potential error, and therefore additional diagnostic testing is needed.

**Diagnostic Testing**

Electrocardiography typically demonstrates sinus tachycardia with nonspecific ST- and T-wave abnormalities. Evidence of ischemic ST changes may be seen if regurgitation is mediated by ischemia or if the hemodynamic circumstances exacerbate coronary insufficiency. Chest x-ray will typically demonstrate a normal-sized left heart and pulmonary edema. Those patients with preexisting left ventricular dilation may have cardiomegaly, whereas those with aortic dissection may have a widened mediastinum. Rarely, acute mitral regurgitation may direct regurgitant flow preferentially to a single pulmonary vein, with edema seen most prominently in that lung segment. This finding is easily confused with pneumonia, particularly if the patient has endocarditis or is not profoundly ill.

The diagnosis of acute regurgitation is made by echocardiography. The presence of severe aortic or mitral regurgitation and normal left ventricular size should immediately raise the possibility of acute regurgitation. Further suspicion should be raised if ventricular function appears normal or
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Hyperdynamic because ejection fraction is typically not significantly decreased in acute regurgitation.\(^{17}\) Echocardiographic findings of acute severe aortic and mitral regurgitation are shown in Table 3. Quantitative measures of regurgitant severity that are useful in chronic regurgitation are less useful in acute regurgitation. Measures of effective regurgitant orifice area and regurgitant volume can be inaccurate in acute regurgitation, particularly in the face of tachycardia. Hemodynamic data have demonstrated the variability in effective regurgitant orifice area and regurgitant volume in acute regurgitation depending on afterload and loading conditions.\(^{18,19}\) Thus, rarely will quantitative measures contribute significantly to management decisions in acute regurgitation.

In addition to assessment of severity and ventricular function, transthoracic echocardiography should also demonstrate the mechanism of regurgitation, such as dissection or ruptured mitral chordae. The acuity of regurgitation may be difficult to assess by echocardiography alone in patients with a history of chronic regurgitation because ventricular size is enlarged, and Doppler findings may be present because of chronic regurgitation. In these cases, comparison with prior studies and clinical examination of the patient will aid in determination of acuity.

Color Doppler on transthoracic echocardiography may underestimate regurgitation severity, particularly if the jet is eccentric. Transesophageal echocardiography may be indispensable in identifying the severity and mechanism of regurgitation if a transthoracic study is inconclusive, particularly with prosthetic valve dysfunction. Additionally, transesophageal echocardiography is important in planning operative repair options, including identification of leaflet or annulus involvement, and in establishing annular size to guide valve replacement options. Particularly if one plans to use an aortic homograft or to evaluate the feasibility of a Ross repair, transesophageal echocardiographic data on annular size are important. However, if the transesophageal echocardiography results will not materially change the decision to pursue surgery, transesophageal echocardiography can be done in the operating room (Figures 1 and 2).

Cardiac catheterization is generally not indicated in the preoperative assessment of patients with acute regurgitation. The exception is patients with acute coronary syndromes complicated by acute mitral regurgitation, for whom revascularization alone may improve regurgitation or for whom both revascularization and mitral valve surgery are needed. For those patients without ischemia as a potential underlying mechanism of regurgitation, such as those with mitral regurgitation due to chordal rupture or aortic regurgitation, the time to obtain a cardiac catheterization and the contrast load may be poorly tolerated. For those with aortic dissection, cardiac catheterization is rarely needed and may worsen dissection. Additional imaging to evaluate possible underlying causes of acute regurgitation, such as computed tomography or magnetic resonance imaging, may be needed, particularly if aortic dissection is a concern not addressed fully by echocardiography.\(^{20}\)

Rapid diagnosis is of the utmost importance because the mortality of acute severe regurgitation is high if untreated. Therefore, any diagnostic modality needed to make an accurate timely diagnosis should be used, but once the diagnosis is made, definitive treatment should not be delayed for diagnostic studies that will not significantly alter the course of care. Acute coronary syndromes and ischemia benefit diagnostically from cardiac biomarkers, and brain natriuretic peptide results can help to distinguish cardiac from noncardiac causes of dyspnea and shock. This is particularly true in those patients presenting with unilateral pulmonary edema.\(^{16}\)

### Treatment

#### Medical Therapy

The treatment of acute aortic regurgitation is surgery to repair or replace the valve. Medical therapy may be used to stabilize the patient en route to surgery; however, surgery should not be delayed in favor of efforts at medical management.\(^{16}\) While the surgical team is being readied, vasodilators such as nitroprusside may be used to improve forward flow, and inotropes such as dobutamine may improve cardiac output. Medical therapy is not a substitute for surgery, however.\(^{21,22}\) Intra-aortic balloon pump use is contraindicated in acute aortic regurgitation because balloon inflation during diastole is detrimental to left ventricular hemodynamics.
Patients with organic causes of mitral regurgitation are similar to those with acute aortic regurgitation. Although supportive therapy may provide some stability en route to the operating room, the fundamental abnormality is structural and requires surgical intervention. Thus, the most important distinction in assessing acute mitral regurgitation is determining the underlying cause. If organic, with disruption of the normal valve structure, surgery is the mainstay of therapy. If functional, medical therapy may be sufficient to avoid operative intervention; however, if medical therapy fails, surgical options need to be reexplored. Additionally, revascularization is a necessity in those patients undergoing surgery for acute mitral regurgitation if significant obstructive coronary disease is present. Unlike in acute aortic regurgitation, the intra-aortic balloon pump may be beneficial in acute mitral regurgitation.

Medical therapy for patients with acute functional mitral regurgitation is directed at the underlying pathophysiology. In patients with ischemia or infarction, restoration of blood flow to the affected territory may be sufficient to improve regurgitation. Data suggest that severe mitral regurgitation in patients with cardiogenic shock is not uncommon, approaching 7% of patients in the Should We Emergently Revascularize Occluded Coronaries in Cardiogenic Shock (SHOCK) registry, and that the presence of acute mitral regurgitation in these patients is a very poor prognostic sign, with an observed mortality of 55%, improving to only 39% in patients selected for emergency surgery. The SHOCK trial demonstrated that early revascularization improves outcomes at 6 months in patients with cardiogenic shock and acute myocardial infarction, and further analysis demonstrated the prognostic importance of mitral regurgitation in these patients. Short- and long-term survival was inversely related to the degree of mitral regurgitation, arguing for more aggressive treatment of those patients with significant mitral regurgitation in the setting of acute infarction, with improved mortality in those patients undergoing early revascularization. Thus, if surgical revascularization is needed, the presence of severe mitral regurgitation should encourage rather than discourage surgical intervention.

In those with acute regurgitation in the setting of acutely developing or worsening cardiomyopathies, aggressive heart failure therapy may be sufficient to improve the degree of mitral regurgitation. In addition to pharmacotherapy, mechanical support may be of use in patients with functional mitral regurgitation. The intra-aortic balloon pump can be an effective support mechanism in patients with acute functional mitral regurgitation of any cause but is particularly useful for those patients with underlying myocardial ischemia or cardiomyopathy. The role of left ventricular assist devices is not well studied in acute mitral regurgitation but can be used in the setting of acutely decompensated heart failure not responsive to medical therapy.

For patients with valve thrombosis resulting in acute regurgitation, thrombolytic therapy may be an alternative to surgery. However, both the American College of Cardiology/American Heart Association and European Society of Cardiology guidelines recommend surgery as first-line treatment in patients with significant symptomatic prosthetic valve dysfunction, with thrombolytics reserved for proven valve thrombosis when surgery is high risk or unavailable or if there is a small thrombus burden and few symptoms.

### Surgical Treatment: Acute Aortic Regurgitation

#### Anesthetic Considerations

Anesthetizing a critically ill patient with acute aortic regurgitation can be a significant challenge. Induction is particularly risky, and the surgical team should be present in the room at the time of induction, ready to initiate early cardiovascular support if necessary.

The pathophysiology of acute aortic regurgitation presents unique challenges to the anesthesiologist. Low diastolic pressure, tachycardia, and increased wall stress may impair coronary flow, and if induction further reduces blood pressure, coronary ischemia may exacerbate patient instability. Consequently, the anesthesiologist must attempt to avoid tachycardia and hypotension during intubation and induction of anesthesia because there may not be latitude to accelerate care to compensate for worsened hypotension and coronary ischemia. Hemodynamic monitoring is also crucial, and in many cases monitoring lines will have been placed before arrival in the operating room. The rational use of inotropic drugs, vasoconstrictors, sedatives, and anesthetic agents is essential to ensure optimal outcomes.

#### Surgical Considerations

In the aortic position, surgical options are defined by the pathophysiology of disease, anatomy, and anticipated long-term outcomes. Myocardial protection is a significant concern in this setting because the incompetent aortic valve and possible associated aortic root pathology make antegrade cardioplegia ineffective or possibly dangerous. Reliable placement of a retrograde coronary sinus cannula for delivery of cardioplegia is essential. Adjunct handheld cardioplegia can be delivered subsequently via coronary ostia after the aortic valve and root have been properly assessed. Additionally, use of a left ventricular vent, placed via the right superior pulmonary vein, may aid in ventricular decompression and induction of a diastolic arrest. Other protective adjuncts such as systemic hypothermia on cardiopulmonary bypass or topical cooling have been used frequently but have unclear overall value.

#### Aortic Valve Endocarditis

Intraoperative transesophageal echocardiography and direct assessment of annular involvement are important in planning the surgical procedure. Careful assessment of the annulus is essential to ensure that (1) it is intact and (2) there are no undrained abscesses. In cases of aortic annulus involvement, the use of a homograft is generally indicated (Figure 3). Although aortic homografts are also at risk for prosthetic valve endocarditis, the additional tissue included with the homograft, including the left ventricular outflow tract and the anterior leaflet of the mitral valve, is sometimes essential for successful aortic root reconstruction. In cases of endocarditis limited to the valve leaflets, leaflet excision and aortic valve replacement are indicated, without additional annular procedures. There is no evidence that a mechanical versus a bioprosthetic valve has a differential rate of infection after replacement for endocarditis.
In some centers, particularly in children, the use of a pulmonary autograft (Ross procedure) may be used. This is controversial, however. As with elective aortic valve surgery, the risk and benefits of the Ross repair are debatable. Theoretically, there is a benefit of providing a durable aortic valve replacement that may grow with a child and not require future intervention. However, the exchange of “single-valve disease” for “2-valve disease” and data suggesting that the neoaortic valve requires replacement in 10% to 20% of patients within 10 years of operation may argue that the long-term outcomes offset the potential benefit. This is a particularly important decision in the case of an unstable patient who may not tolerate the additional bypass time necessary to successfully perform a Ross repair. Consequently, the use of a Ross repair in the acute setting is limited to a very specific patient population in specific centers with extensive surgical success in Ross repairs. Finally, depending on the clinical status of the patient and the overall status of the aortic root, some patients benefit from a simple valve replacement with anticipated root reconstruction after the patient has had an opportunity to recover from multisystem organ failure.

Aortic Dissection
The pathophysiology of acute aortic regurgitation in the setting of a type A aortic dissection varies. The strategy for addressing the acute aortic regurgitation in that setting is dependent on its pathogenesis. In the setting of a normal trileaflet aortic valve, aortic valve resuspension and interposition graft replacement of the ascending aorta can frequently address valvular regurgitation by raising the commissural heights to normal and reattaching the sinuses of Valsalva to the aortic wall. When an aortic dissection is present in the setting of an abnormal aortic valve, such as a bicuspid aortic valve or an ascending aortic aneurysm, our practice is to perform a modified Bentall procedure using a valved conduit and direct coronary reimplantation. One center reported their experience with aortic root replacement using mechanical conduits and aortic allografts, noting no difference in 1- and 5-year survival based on conduit choice, including those patients with aortic dissection. Both aortic root remodeling procedures (Yacoub procedure) and aortic root reimplantation procedures (David procedures) have been used successfully in the setting of aortic dissection as well but require more experienced surgical expertise for consistently excellent results. The possibility of coronary ischemia due to coronary artery involvement from dissection or technical problems with the coronary artery reimplantation needs to be considered in repair options to achieve optimal outcomes.

Fenestration/Trauma
Disrupted leaflets due to trauma or a ruptured fenestration can be repaired in certain instances. A leaflet perforation can be patched with sutures and an appropriately sized piece of autologous or bovine pericardium. However, a low threshold for valve replacement should be the norm. Blunt chest trauma resulting in aortic valve regurgitation is most often a result of loss of commissural suspension, leaflet perforation, or dissection. As with other causes of acute aortic regurgitation, surgical therapy is tailored to the intraoperative anatomy.

Surgical Treatment: Acute Mitral Regurgitation

Anesthetic Considerations
Although patients with acute mitral regurgitation often are not as unstable as patients with acute aortic regurgitation, these patients nonetheless remain hemodynamically tenuous and can demonstrate acute cardiopulmonary collapse at any time. As with acute aortic regurgitation, all members of the surgical team should be present during induction in case urgent initiation of cardiopulmonary bypass is needed. If the cause of acute mitral valvular regurgitation is profound ongoing myocardial ischemia or infarction with either significant systolic or diastolic dysfunction, additional anesthetic considerations must be made to optimize myocardial oxygen supply and demand, preload and afterload, and coronary perfusion pressure. If not done before arrival in the operating room, placement of an intra-aortic balloon pump to improve coronary perfusion may be considered before induction of anesthesia.

Surgical Considerations
The pathogenesis of acute mitral regurgitation determines its management. In a series of acute mitral regurgitation, the causes were acute myocardial infarction (45%), degenerative valvular disease (26%), and infective endocarditis (28%); each of these causes is managed by different strategies, and each has different outcomes. Unlike aortic regurgitation, for which repair is rarely possible, mitral regurgitation may be treated with either repair or replacement.

Surgical exposure of the mitral valve is critical, and both surgical incision approaches may need to be considered: a median sternotomy or an anterolateral or full lateral right thoracotomy incision. Minimally invasive approaches to acute mitral valvular regurgitation should be considered by only the most experienced surgeons. Expeditious use of...
cardiopulmonary bypass, optimal myocardial protection, excellent valve exposure, and short pump runs will lead to the best results and are less likely achieved through a minimally invasive approach. Flexibility in the approach to the mitral valve can also be important, and a variety of techniques should be considered, particularly if there is complex endocarditis and extensive annular reconstruction is needed. Potential approaches may include the most standard lateral Sonnegaard’s groove incision in the left atrium, biatrial incisions, anterior approaches beneath the aortic root, or transection of the superior vena cava with a more anterolateral approach to the left atrium, which is particularly useful if endocarditis is more complex and the annular reconstructions are more extensive.

**Ischemic Mitral Regurgitation**

Repair or replacement of the mitral valve in the setting of acute regurgitation due to myocardial ischemia mandates attention to revascularization. Careful review of preoperative coronary angiography, if available, and preparation for aortocoronary bypass grafting are essential. Because these patients tend to be hemodynamically tenuous, time taken for left internal mammary preparation may be ill advised, and bypass grafting can be completed with venous conduits. Myocardial protection in the setting of acute myocardial infarction is challenging. Both antegrade and retrograde cardioplegia are usable in this setting. Delivery of antegrade cardioplegia via newly placed venous conduits can augment myocardial protection. Given the ischemic nature of the myocardium, metabolically enhanced, slow warm induction may have beneficial effects. Furthermore, controlled reperfusion at the conclusion of the operation may also allow for more brisk recovery of myocardial function. In general, intra-aortic balloon counterpulsation is maintained for at least 24 hours after the conclusion of the operation.

Papillary muscle rupture is a rare complication of myocardial infarction, occurring in 1% to 3% of myocardial infarctions, with a mortality of 80% with medical therapy alone. \(^{31,32}\) Historically, operative mortality was as high as 67%, and patients were frequently denied surgery. With the addition of bypass grafting to mitral valve replacement or repair, operative mortality now is <10%. \(^{33}\)

**Mitral Valve Endocarditis**

When endocarditis is the underlying cause of acute regurgitation, the principle of excising all sites of active infection drives the therapeutic approach. Once the infection is excised, then the decision to repair (leaflet), replace (chord or entire valve), or reconstruct (annulus) will be critical. The most difficult reconstructions involve the fibrous trigone of the heart, where the annular support of both the aortic and mitral valves is involved, the tissues are edematous and friable, and the 3-dimensional visualization for the reconstruction can be difficult, even for the experienced surgeon. Availability of aortic homografts may be optimal in cases of infective endocarditis, in which both the mitral and aortic annuli are involved with active infection or abscess.

Reoperative mitral valve surgery, such as in the case of prosthetic valve endocarditis, poses unique challenges to the surgeon. Manipulation of the mitral annulus, coupled with ongoing infection and previous scar, can result in atrioventricular disruption with even the most gentle of maneuvers. This catastrophic event is highly lethal and must be considered and anticipated if possible.

**Mitral Valve Repair**

Valve repair is always preferable to valve replacement, when possible. Valve repair is more likely to be an option in acute mitral regurgitation than in acute aortic regurgitation. Once again, therapeutic options and surgical priorities must be judged on the basis of pathogenesis, acute pathophysiology, underlying pathology, and comorbidities. Keys to success are accurate assessment of preoperative data, good surgical judgment, and expeditious operations. When the technical results are unacceptable with the initial procedure, use of a second pump run and repeated repair or replacement are necessary. Thus, repair should be undertaken only in those situations in which procedural success is likely because an unstable patient may not tolerate additional bypass time. In those situations with confounding myocardial ischemia or infarction, additional attention to detail must come into play in planning and executing the operation, including optimal myocardial protection, appropriate coronary revascularization, rational use of inotropic drugs, and selected use of mechanical circulatory assistance. The extent and priorities of the operation will be affected by many issues, including the presence of papillary muscle rupture; chordal rupture; annular dilation; hibernating, stunned, or infarcted myocardium; and the severity of coronary artery disease.

All techniques for mitral valve repair can come into play: annuloplasty with rings, annular reconstruction with pericardium, leaflet resection, leaflet reconstruction with pericardium, chordal replacement, vegetation excision, and edge to edge repair.
Valve repair is more common in situations in which acute annular dilation is the cause, such as ischemia, or in which the pathology only involves the posterior leaflet, such as myxomatous valve disease or endocarditis (Figure 4), with or without annular dilation. The ability to repair the mitral valve dramatically lessens when there is annular involvement with infection or anterior leaflet involvement with degenerative disease.

**Mitral Valve Replacement**

Mitral valve replacement is needed when repair is not possible or there is likely to be significant residual mitral regurgitation after attempted repair. Patients with acute mitral regurgitation are unlikely to tolerate reexploration and replacement after an unsuccessful repair, particularly if the attempted repair was complex and time consuming. Therefore, mitral valve replacement may be the preferable choice if valve anatomy is unfavorable or when the only option for repair is complex. This is especially true with tissue destruction due to endocarditis, in which case, more commonly than not, annular involvement precludes simple repair with predictable excellent initial results. The important surgical goal in valve replacement is to avoid a periprosthetic leak because of inadequate visualization or poor tissue quality and to preserve both posterior and anterior chords whenever possible. There is no specific benefit of mechanical versus bioprosthetic prosthesis in degenerative, ischemic, or infectious situations, although mechanical prostheses are usually preferred for durability, low profile, ease of insertion, and the possible need for anticoagulation for other acute or chronic reasons, such as arrhythmia.

**Clinical Outcomes**

Broadly applicable clinical outcomes in acute valvular regurgitation are difficult to ascertain. The cause of acute valvular regurgitation is variable for both aortic and mitral valves, the time to intervention is quite variable, the complexity of intraoperative repair is unique, and comorbid conditions are common; outcomes are dependent on multiple factors. Variability in outcomes is also due to incidence and prevalence of the disease entity, patient selection, era of reporting, and surgeon experience. Despite the difficulties, certain disease entities have some available data. A recent report of the outcomes of endocarditis in a multinational patient cohort
emphasized the high mortality of the disease, despite improved medical and surgical therapy. Although mortality has decreased over time, 30-day mortality remains 15% to 20%. In a series with 48% of patients undergoing surgery, mortality was increased in those with pulmonary edema but decreased in those who underwent surgery. The SHOCK trial emphasizes the need for early revascularization to improve long-term outcomes of patients with acute mitral regurgitation in acute myocardial infarction. Mortality remains high in patients with aortic dissection, with 31.4% mortality for unstable patients undergoing surgery for type A dissections. The presence of shock increases the risk of poor outcomes, and therefore, when possible, operative intervention before the onset of shock is one means of improving results. The cause of acute regurgitation also drives outcomes, as demonstrated by a recent report of a series of patients undergoing surgery for acute severe mitral regurgitation that demonstrated an overall 30-day mortality of 22.5%. The cause of acute regurgitation also drives outcomes, and therefore, when possible, operative intervention before the onset of shock is one means of improving results.

**Conclusion**

Acute valvular regurgitation is a surgical emergency that requires appropriate diagnosis and rapid intervention for optimal outcomes (Figure 5). Because the examination findings of acute regurgitation are different and often more subtle than those of chronic regurgitation, the diagnosis is often missed when a patient presents with dyspnea and shock. A high index of suspicion and echocardiography are important in rapid diagnosis, and surgical treatment should proceed as quickly as possible. Surgical mortality remains high; however, medical therapy is not sufficiently effective to obviate the need for surgery.

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**Disclosures**

None.

**References**


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