Incompetence of the aortic valve results in volume overload of the left ventricle. The distinction between acute and chronic forms of aortic regurgitation is important in terms of etiologies, associated diseases, prognosis, and treatment.

**ETIOLOGY AND PATHOGENESIS**

Common causes of acute aortic regurgitation include ascending aortic dissection with distortion of the normal valve architecture, infective endocarditis with destruction of a valve leaflet, traumatic disruption, and spontaneous rupture or prolapse of a valve cusp secondary to degenerative diseases of the valve. Acute aortic regurgitation also may occur with sudden dehiscence of the sewing ring of a prosthetic valve and after operative or balloon valvuloplasty.

The many causes of chronic aortic regurgitation are fundamentally related to two structural defects: those involving the valve leaflets and cusp or those involving the aortic root. Causes of valve leaflet disease include rheumatic heart disease, congenital abnormalities of the aortic valve (especially bicuspid valves), calcific degenerative valve disease, myxomatous degeneration, or infective endocarditis. Rheumatic disease is characterized by shortening and scarring of the cusps and is frequently accompanied by mitral valve involvement (Fig. 28-1). Congenitally bicuspid valves occur in up to 2% of the population, with a marked male predominance. This abnormality often presents as aortic stenosis or a mixed stenosis–regurgitation lesion, but 10% have pure regurgitation. Infective endocarditis may cause aortic regurgitation by several mechanisms, including perforation of a single leaflet or a flail leaflet or by weakening of the cusp and valve annulus as a result of an expanding aortic root abscess.

Aortic root disease is responsible for approximately half of all clinically significant regurgitation cases. Common aortic root problems causing aortic regurgitation include Marfan’s syndrome with annuloaortic ectasia and ascending aortic dissection that may distort valve structure and undermine support of the leaflets. In systemic hypertension, aortic regurgitation may occur because the presence of long-standing hypertension can result in dilation of the ascending aorta with distortion of the valve and chronic damage to the valve leaflets. Less common causes of aortic regurgitation include syphilitic aortitis, ankylosing spondylitis, osteogenesis imperfecta, systemic lupus erythematosus, rheumatoid arthritis, psoriatic arthritis, Behçet’s syndrome, ulcerative colitis, discrete subaortic stenosis, and ventricular septal defect with prolapse of an aortic cusp (Fig. 28-2).

**Natural History**

The natural history of chronic aortic regurgitation is incompletely known. Data from the presurgical era indicate that patients with chronic, severe aortic insufficiency who have angina or heart failure have a prognosis similar to those with severe aortic stenosis, with mortality rates of at least 10 to 20% per year. Asymptomatic patients with normal left ventricular (LV) function develop symptoms or LV dysfunction at a rate of about 4% annually, but the occurrence of sudden death is rare (<0.2% per year). It is important to note, however, that 25% of patients die or progress to LV dysfunction before manifesting symptoms, emphasizing the importance of serial quantitative assessments of LV function. The subset of asymptomatic patients who have LV dysfunction predictably has higher event rates, with more than 25% developing symptoms annually.

**CLINICAL PRESENTATION**

The presentation of aortic insufficiency varies with the onset (acute or chronic) and the degree of compensatory changes to volume overload (Table 28-1). In acute aortic regurgitation, the
Rheumatic Heart Disease
Aortic Insufficiency

Aortic insufficiency: Valve viewed from above; thickened, short cusps with triangular deficiency

Shortened cusps of aortic valve with exposure of sinuses and dilatation of aorta: “Jet lesion” on septal wall of L. ventricle

Concentric hypertrophy with some dilatation of L. ventricle resulting from aortic insufficiency, causing chordae tendineae to elongate and run in a relatively horizontal direction, thus impeding closure of mitral valve and leading to secondary mitral insufficiency
AORTIC INSUFFICIENCY

Figure 28-2

Syphilitic Heart Disease

Incompetent aortic valve with taut, separated cusps viewed from above

Dilated and markedly sclerotic thoracic aorta with widened aortic ring and narrowing of coronary ostia; hypertrophy of L. ventricle with regurgitant lesion on ventricular septum

Calcification of ascending aorta and dilatation of thoracic aorta

Stellate scar in media of aorta
presentation is often dramatic. LV hypertrophy has not developed, so ventricular compliance is normal and remains so despite the sudden regurgitation. The acute volume overload is poorly tolerated because the left ventricle is abruptly and dramatically distended, resulting in impaired systolic function (based on the diastolic pressure–volume curve). The left ventricle is unable to acutely compensate for the large regurgitant volume. Forward cardiac output is reduced and LV end-diastolic pressure is greatly increased. Tachycardia occurs in a futile attempt to increase cardiac output. The regurgitation results in premature mitral valve closure with occasional diastolic mitral regurgitation. As a result, the patient with acute regurgitation usually appears severely ill, manifesting tachycardia, hypotension, peripheral vasoconstriction, and pulmonary congestion and edema, but lacks many physical signs of chronic regurgitation. Fatigue, apathy, agitation, or a decline in mental function may develop as a manifestation of the abrupt decrease in forward cardiac output.

Chronic aortic regurgitation may be asymptomatic for years. When symptoms develop, they are usually indolent, reflecting the slow, progressive nature of the disease. Frequent complaints include exertional dyspnea, orthopnea, and paroxysmal nocturnal dyspnea, all reflecting congestive heart failure (CHF), and palpitations. Angina pectoris may be produced by concurrent coronary artery disease or low diastolic perfusion pressure and a reduction in coronary blood flow during diastole because of the aortic regurgitation–induced LV hypertrophy. As aortic regurgitation develops, the left ventricle slowly enlarges primarily with eccentric hypertrophy, although concentric hypertrophy also occurs from increased afterload (Fig. 28-3). LV end-diastolic volume increases in response to the regurgitation with an increase in chamber compliance; accordingly the increased end-diastolic volume is not associated with major increases in end-diastolic pressure. Increased stroke volume maintains normal forward cardiac output, usually without significant increases in heart rate. Greatly augmented stroke volume leads to many of the classic findings of chronic aortic regurgitation (Table 28-2 and Fig. 28-3); the patient sometimes experiences an unpleasant

**Table 28-1**

**Hemodynamic Features of the Stages of Severe Aortic Regurgitation**

<table>
<thead>
<tr>
<th></th>
<th>Acute Severe Regurgitation</th>
<th>Chronic, Severe Regurgitation (Compensated)</th>
<th>Chronic, Severe Regurgitation (Late Decompensation)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LV compliance</td>
<td>Not increased</td>
<td>Increased</td>
<td>No longer increased</td>
</tr>
<tr>
<td>LVEDP</td>
<td>↑↑↑</td>
<td>Normal</td>
<td>↑↑↑</td>
</tr>
<tr>
<td>LV dimensions</td>
<td>Normal</td>
<td>↑↑</td>
<td>↑↑</td>
</tr>
<tr>
<td>Aortic SBP</td>
<td>Normal or low</td>
<td>↑</td>
<td>Normal or low</td>
</tr>
<tr>
<td>Aortic DBP</td>
<td>Normal</td>
<td>↓↓</td>
<td>Normal</td>
</tr>
<tr>
<td>Pulse pressure</td>
<td>Normal / ↑</td>
<td>↑↑↑</td>
<td>Normal</td>
</tr>
<tr>
<td>LVEF</td>
<td>Normal</td>
<td>Normal / ↑</td>
<td>↓</td>
</tr>
<tr>
<td>Total stroke volume</td>
<td>↑</td>
<td>↑↑↑</td>
<td>↑</td>
</tr>
<tr>
<td>Heart rate</td>
<td>↑↑↑</td>
<td>Normal</td>
<td>↑↑</td>
</tr>
<tr>
<td>Regurgitant volume</td>
<td>Large</td>
<td>Very large</td>
<td>Large</td>
</tr>
<tr>
<td>Effective cardiac output</td>
<td>↓↓</td>
<td>Normal</td>
<td>↓</td>
</tr>
<tr>
<td>Arterial pulse volume</td>
<td>Normal / ↑</td>
<td>↑↑↑</td>
<td>Normal</td>
</tr>
</tbody>
</table>

↑ indicates slight increase; ↑↑↑, moderate increase; ↑↑↑↑, severe increase; ↓, slight decrease; ↓↓, moderate decrease; ↓↓↓, severe decrease; LV, left ventricular; LVEDP, left ventricular end-diastolic pressure; LVEF, left ventricular ejection fraction.

Arrows are not used in the first row because the changes in LV compliance are complex. In acute severe regurgitation, compliance is not really normal, but not increased. In last column, compliance is not really normal, but is reduced compared with the state described in the middle column.
Manifestations of Aortic Insufficiency

Cerebral insufficiency: Dizziness, pulsating headache

Pallor

Cervical and suprasternal pulsation

Dyspnea

Pulmonary congestion

Edema

R. heart failure

Bounding pulse (Corrigan pulse)

Blood pressure: Increased pulse pressure

"Pistol-shot" sound

"Cogwheel" respiration

Aorta dilated; exaggerated pulsation

Diastolic regurgitation through aortic valve

Coronary insufficiency (precordial pain)

L. ventricle dilated

Failure

Apex beat shifts to left and down;

Diastolic regurgitation through aortic valve

O. ventricular pressure: Increased pulse pressure

Soft, blowing, diastolic decrescendo murmur

L. ventricular enlargement

L. ventricular hypertrophy and dilatation: Increased voltage of QRS in all leads; inverted T in several
Awareness of each contraction, especially if irregular beats lead to a pause with a further increase in preload. Low aortic diastolic pressure from the regurgitation of the incompetent valve into the left ventricle usually results in a wide pulse pressure. During exercise, systemic vascular resistance and diastolic filling period decrease, resulting in less regurgitation per cardiac cycle. This increases forward cardiac output without substantial increases in LV end-diastolic pressure. In some patients, the ability of the left ventricle to compensate for the chronic volume overload eventually is exceeded and LV failure develops. As the LV ejection fraction (EF) decreases, the ventricle dilates further, initiating a vicious cycle. Throughout this process, fibrosis of the myocardium slowly contributes to the development of irreversible LV dysfunction. Typical symptoms of CHF may become evident at this stage.

### Table 28-2
<table>
<thead>
<tr>
<th>Finding</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td>de Musset’s sign</td>
<td>Head bob with each systolic pulsation</td>
</tr>
<tr>
<td>Corrigan’s pulse</td>
<td>Bounding pulse, alternatively named “water-hammer pulse”</td>
</tr>
<tr>
<td>Traube’s sign</td>
<td>Booming systolic and diastolic sounds (“pistol shots”) over the femoral arteries</td>
</tr>
<tr>
<td>Muller’s sign</td>
<td>Systolic pulsation of the uvula</td>
</tr>
<tr>
<td>Duroziez’s sign</td>
<td>Systolic murmur over the femoral artery when compressed proximally, diastolic murmur when compressed distally</td>
</tr>
<tr>
<td>Quincke’s sign</td>
<td>Capillary pulsations noted in the nail beds or fingertips with each cardiac cycle</td>
</tr>
<tr>
<td>Hill’s sign</td>
<td>Popliteal systolic pressure exceeding brachial pressure by 30–60 mm Hg</td>
</tr>
</tbody>
</table>

**Physical Examination**

In acute severe aortic regurgitation, systolic BP is normal or decreased and diastolic BP is slightly elevated, resulting in a pulse pressure that is usually normal. Although tachycardia is usually present, the precordium is relatively quiet. The first heart sound is soft because of premature closure of the mitral valve and may be absent in severe acute regurgitation. The second heart sound is also soft, and a third heart sound is frequently present due to the rapid early diastolic filling of the ventricle. A fourth heart sound is uncommon. In contrast to chronic aortic regurgitation, the diastolic murmur of acute regurgitation is often short, ending well before the end of diastole, and soft in intensity. A systolic murmur is also present, but not particularly loud due to the reduced forward output. A second diastolic murmur, the Austin Flint murmur, is a mid diastolic rumble similar to mitral stenosis best heard at the apex. Possible mechanisms of this murmur include relative mitral stenosis from the regurgitant jet displacing the anterior mitral leaflet, impedance of left atrial outflow, or vibrations induced by the regurgitant jet. An Austin Flint murmur indicates that aortic regurgitation is severe.

In chronic, compensated aortic regurgitation, increased carotid pulse volumes may be accompanied by a bruit or transmitted systolic murmur. Peripheral pulses are bounding due to the wide pulse pressure, with systolic hypertension and a low diastolic BP. The LV apical impulse is enlarged and displaced inferiorly and laterally. The first heart sound is normal or soft and the second heart sound may be normal, single, or paradoxically split. Ejection clicks may be heard, especially in patients with a dilated aortic root. A fourth heart sound can be detected as LV hypertrophy develops and a third heart sound occurs when the left ventricle decompensates. The diastolic murmur of chronic aortic regurgitation is best heard at the base of the heart along the left sternal edge or in the second right intercostal space. It is best detected with the diaphragm of the stethoscope while the patient is leaning forward during held expiration. The etiology of the regurgitation is more likely to be valvular if the murmur is louder to the left of the sternum, whereas aortic root disease is often the cause if the murmur is louder to the right of the sternum.
The diastolic murmur begins at the second heart sound and continues for a variable portion of diastole. Severity of the regurgitation is better correlated with the length rather than the intensity of the murmur. However, when the left ventricle begins to fail and end-diastolic pressure increases, the murmur shortens again. A systolic murmur may be present from increased forward flow across the aortic valve or concomitant aortic stenosis. An Austin Flint murmur, if present, indicates severe aortic regurgitation.

**DIFFERENTIAL DIAGNOSIS**

The hallmarks of chronic aortic regurgitation are an increased pulse pressure and diastolic decrescendo murmur heard at the upper sternal border. Several other conditions can mimic aortic regurgitation and should be considered in the differential diagnosis. First, patients with pulmonic regurgitation have a blowing diastolic decrescendo murmur, but would not usually have a wide pulse pressure or bounding carotid pulse. The murmur of pulmonic regurgitation should increase with inspiration; the pulmonic valve closure sound is often increased in intensity and a right ventricular (RV) heave may be present. ECG would show signs of RV strain or hypertrophy rather than left-sided abnormalities and chest radiography would show signs of RV enlargement. In adults, there is usually a comorbid condition causing pulmonary hypertension and therefore the pulmonary regurgitation. Second, in those presenting at a younger age the diagnosis of a patent ductus arteriosus should be considered. It causes a wide pulse pressure, as seen in aortic regurgitation, but the murmur is continuous with a low-pitched diastolic component. ECG in this condition would be normal or show signs of LV hypertrophy and chest radiography would show signs of RV enlargement. In adults, there is usually a comorbid condition causing pulmonary hypertension and therefore the pulmonary regurgitation. Second, in those presenting at a younger age the diagnosis of a patent ductus arteriosus should be considered. It causes a wide pulse pressure, as seen in aortic regurgitation, but the murmur is continuous with a low-pitched diastolic component. ECG in this condition would be normal or show signs of LV hypertrophy and chest radiography would show increased flow in the pulmonary vasculature. Third, if symptoms of dyspnea and chest pain begin suddenly, a ruptured sinus of Valsalva aneurysm should be considered. The pulse pressure is usually increased, but the murmur would be continuous rather than diastolic only. Chest radiography would show signs of increased flow in the pulmonary vasculature. Finally, a coronary arteriovenous fistula may present with a murmur that can be confused with aortic regurgitation. The murmur should be continuous, but occasionally the diastolic component can dominate, mimicking aortic regurgitation. Echocardiography and, if necessary, cardiac catheterization can be performed to distinguish all of these conditions from aortic regurgitation.

**DIAGNOSTIC APPROACH**

With chronic aortic regurgitation, the ECG frequently shows left-axis deviation and LV hypertrophy. The findings are nonspecific and may include interventricular conduction defects, non-specific ST-segment and T-wave changes, and PR-interval prolongation, especially if the etiology is inflammatory. These findings are not accurate predictors of the severity of regurgitation.

Chest radiography in chronic aortic insufficiency shows LV dilatation that may be massive (so called “cor bovinum”). Enlarged aortic root size suggests the etiology of the regurgitation. The pulmonary vasculature may be engorged with fluid during a decompensated state. With acute aortic insufficiency, there is minimal cardiac enlargement, with florid pulmonary edema the only finding.

Echocardiography is valuable for the initial assessment of acute and chronic aortic regurgitation and for serial follow-up examinations (Fig. 28-4). Complete echocardiography provides information about the etiology and severity of aortic regurgitation, the presence of concomitant valve disorders, and the state of LV compensation assessed by chamber size, function, and wall thickness. The severity of regurgitation can be estimated semiquantitatively by measuring the width or cross-sectional area of the regurgitant jet in relation to the LV outflow tract cross-sectional area by the finding of holo-diastolic flow reversal in the descending aorta or by measuring the pressure half-time of the regurgitant jet. Severity can be determined quantitatively by the pressure half-time method or by the continuity equation that yields the regurgitant volume and fraction. Additionally, information from echocardiography, notably LVEF and chamber dimensions, can be followed serially to determine the timing of surgical intervention.

Aortic regurgitation can also be evaluated by cardiac catheterization. Hemodynamic tracings in severe aortic regurgitation show a wide pulse pressure and an elevated LV end-diastolic pressure (Fig. 28-3). Aortic root angiography provides
ECG, pressure tracings, M-mode echocardiogram (Echo), and phonocardiograms (PCG) from a patient with acute severe (left) and chronic severe (right) aortic regurgitation.

A indicates A point corresponding to the peak upward motion of the mitral valve leaflet after atrial systole; AML, anterior mitral valve leaflet; Ao, aorta; C, C point where the two mitral leaflets come together; DM, diastolic murmur; E, E point corresponding to the maximal excursion of the anterior mitral valve leaflet after early diastolic opening; EDP, end-diastolic pressure; f, flutter of anterior mitral valve leaflet; LA, left atrium; LV, left ventricle; PML, posterior mitral valve leaflet; S₁, first heart sound; S₂, second heart sound; SM, systolic murmur.


a semiquantitative assessment of severity, based on the speed and completeness of LV opacification. Quantitatively, regurgitant volume and regurgitant fraction are calculated using the stroke volume from LV angiography and forward cardiac output obtained by the thermodilution or Fick methods.

MANAGEMENT AND THERAPY

Acute Aortic Regurgitation

Whatever the etiology, acute aortic insufficiency necessitates rapid diagnosis, with aggressive medical and surgical therapy, if feasible. Medical stabilization includes afterload-reducing agents to augment forward cardiac output, but worsening hypotension may preclude implementation of afterload-reducing therapy. Intraaortic balloon counterpulsation is contraindicated because it increases regurgitation. Slowing the heart rate is not recommended because it prolongs the diastolic filling period and thus lengthens the time during which regurgitation can occur. However, if acute aortic dissection is the etiology of the regurgitation, β-blockers may reduce the force of LV ejection. Long-term administration of β-blockers is important in patients with Marfan’s syndrome because it slows the rate of aortic dilatation and progression to aortic complications, a favorable effect while awaiting definitive surgical intervention.

With acute aortic dissection, the clinical picture may be dominated by other sequelae, including myocardial infarction (MI) from compromise of a coronary artery (most commonly the right coronary artery), hemopericardium with tamponade, hemorrhagic shock, or stroke due to involvement of a great vessel. When aortic regurgitation occurs as a result of infective
endocarditis, surgery occasionally can be delayed a few days, allowing further antibiotic therapy, but should not be postponed if there is significant hemodynamic instability or CHF. Despite concerns about placing a prosthetic valve during an infection, the risk of recurrence of infection is fairly low.

**Chronic Aortic Regurgitation**

Valve replacement should be considered for most patients with symptomatic severe aortic regurgitation, unless comorbid conditions preclude surgery. Preoperative LV systolic performance is the major determinant of postoperative prognosis in terms of LV function, symptoms of heart failure, and survival rate. In general, symptomatic patients with reduced LV function who undergo valve replacement have reduced postoperative survival rates, whereas those with preserved LV function have an excellent prognosis. However, there is a subgroup of patients with reduced LV function who show substantial improvement in LV function after valve replacement. In this subgroup, improvement in LV function resulted from the elimination of valve regurgitation and left ventricle volume overload and reversal of the imbalance between excessive afterload and the combination of preload reserve and compensatory hypertrophy (so called “afterload mismatch”). Key factors for improvement in LV function and prognosis after surgery are early identification of patients with minimal or no symptoms yet early signs of LV dysfunction and potential valve replacement, before severe symptoms develop.

There is controversy, however, regarding the timing of surgery in asymptomatic patients with severe regurgitation. Vasodilator therapy can reduce the degree of regurgitation, increase forward cardiac output, and delay the necessity of valve replacement. Accordingly, vasodilator therapy is recommended for asymptomatic patients with severe aortic regurgitation who have hypertension or a normal EF with enlarged LV volumes. Vasodilators are not recommended in patients with lesser degrees of regurgitation or if the EF and cardiac volumes are normal. Surgery is recommended in asymptomatic patients with severe aortic regurgitation who have a depressed LVEF (<0.50), severe ventricular dilatation (LV end-systolic dimension >55 mm or end-diastolic dimension >75 mm) or are undergoing surgery on a different valve, the aorta, or coronary arteries. Data that suggest that an exercised-induced decrease in LVEF has independent prognostic value in patients undergoing surgery are inconsistent. The most common surgical procedure is valve replacement, but alternative approaches include use of the patient’s pulmonary valve (Ross procedure) or valve repair (see chapter 34). Valve repair is most feasible in patients with perforation of a leaflet due to endocarditis or when redundancy of the free edge of one of the leaflets results in leaflet prolapse. Concomitant aortic root reconstruction is often necessary in aortic dissection and Marfan’s syndrome.

**FUTURE DIRECTIONS**

Minimally invasive aortic valve replacement surgery is becoming more common as surgical techniques are perfected. The entire surgical procedure is performed through a small incision to the right of the sternum rather than the traditional median sternotomy. This approach appears to shorten length of stay and recovery periods before returning to work, but it is unclear whether there are any long-term advantages or hazards. Percutaneous alternatives to surgical valve replacement are also being developed.

**REFERENCES**


