INTRODUCTION

The aortic valve normally consists of three semilunar leaflets. It is designed to allow unrestricted blood flow from the left ventricle into the ascending aorta during systole and to prevent backflow during diastole. A pathologic state is established whenever each of these two functions is compromised: either aortic stenosis (AS) or aortic regurgitation (AR).

In AS, blood cannot flow freely out of the left ventricle during systole; in AR, the blood flows back into the left ventricle during diastole. AS or AR can be primary causes of severe illness, or they can exacerbate concomitant diseases in critically ill patients presenting to cardiac care units (CCUs).

Because there is no effective medical therapy for these conditions, severe AS and AR ultimately require surgical or transcatheter aortic valve replacement (TAVR) to achieve clinical stability and alter their natural progression. Rapid diagnosis and supportive medical therapy are necessary until definitive treatment can be provided.

With the introduction of TAVR, a true revolution has occurred. Patients with severe AS who were previously deemed too high risk to undergo surgery are now able to be treated safely and effectively. In the early days of TAVR, the procedure was done under general anesthesia and required postprocedural care with CCU observation. However, there has been a general trend to replace general anesthesia with moderate sedation, and CCU care is often shortened or even unnecessary.1

AORTIC STENOSIS

ETIOLOGY AND PATHOPHYSIOLOGY

In the adult CCU, the most common etiology of AS is calcific degeneration (Figure 35.1) of a previously normal trileaflet aortic valve (TAV) or a congenitally bicuspid aortic valve (BAV). Calcific TAV stenosis is typically encountered among the elderly and calcific BAV stenosis in middle-aged patients. It is estimated that moderate or severe AS is present in 0.4% of all Americans, and in 2.8% of people 75 years of age or older.2

Once thought to be the result of passive wear and tear, AS of a TAV is now understood to be a manifestation of generalized atherosclerosis, an active proliferative and inflammatory process.3,4 Thus, this form of AS and atherosclerosis share risk factors such as smoking, hyperlipidemia, and hypertension.5,6 In addition, disorders of calcium phosphate metabolism such as end-stage renal disease and Paget disease, as well as mediastinal radiation treatment, increase the risk for development of AS.7

BAV is the most common congenital heart defect, occurring in approximately 1% to 2% of all live births.8 BAV may evolve into

FIGURE 35.1 Aortic stenosis on echocardiography. Transesophageal echocardiogram (TEE) showing normal valve anatomy and a severely stenotic aortic valve, both in diastole and systole. Note how calcified and narrowed the stenotic valve appears.
AS or AR and may be associated with diseases of the aorta such as aortic aneurysm, aortic dissection, and coarctation.9

The most important physiologic adaptation to the pressure overload from AS is left ventricular hypertrophy (LVH). This leads to concentric hypertrophy, a process in which the left ventricular (LV) chamber becomes smaller and its wall thicker; such changes decrease the wall stress and preserve left ventricular ejection fraction (LVEF) for very long periods. When LVEF starts to decrease, the underlying mechanism is an afterload-LVH mismatch (insufficient hypertrophy for the degree of AS) rather than true cardiomyopathy.

**CLINICAL PRESENTATION**

The classic triad of symptomatic severe AS consists of angina, syncope, and dyspnea from heart failure. AS is usually asymptomatic unless severe. The importance of recognizing the symptomatic stage of AS, as established by the seminal work by Ross and Braunwald, is the rapid rise in mortality once symptoms develop.10 Following a latent asymptomatic period, patients who progress to severe symptomatic AS experience over 50% mortality in the next 2 to 3 years if no intervention is pursued.11

**DIAGNOSIS**

Although physical examination may establish the diagnosis, it is often unable to precisely grade AS severity. The typical findings in auscultation of AS include a systolic crescendo–decrescendo ejection murmur over the precordium that radiates to the neck, which becomes late peaking as the aortic valve becomes more stenotic. The prolonged ejection time leads to a single or, para-diastolic, split second heart sound (S2), with occasional absence of S2 in severe cases.12 Carotid upstroke is typically weak and delayed (*pulsus parvus et tardus*).

Electrocardiogram (ECG) often meets criteria for LVH and left atrial enlargement. Although chest X-ray (CXR) rarely provides direct evidence of AS, it occasionally shows aortic valve calcifications. In addition, pulmonary venous congestion is frequently seen in decompensated patients.

The primary means of diagnosing and grading AS is echocardiography, and aortic valve area (AVA) is the primary criterion of AS. When there is normal flow across the aortic valve, the magnitude of the peak and mean systolic gradient across the aortic valve is inversely related to AVA (Table 35.1). Additional echocardiographic findings in patients with significant AS include LVH, left atrial enlargement, and typically preserved LVEF.

A low transvalvular gradient (mean gradient ≤ 30 mm Hg) does not exclude severe AS. In patients with low transaortic flow rate, peak and mean systolic gradients do not accurately reflect severity of AS. Low flow may occur for two separate reasons: (1) low stroke volume in the setting of small hypertrophied left ventricle with preserved LVEF and (2) low stroke volume in the setting of LV systolic dysfunction and low LVEF. Low flow is typically defined as a low LV stroke volume index of < 35 mL/m².

In patients with low-flow, low-gradient severe AS with normal LVEF, echocardiography demonstrates small LV chamber size, abnormal diastolic filling, and increased LV relative wall thickness. In contrast, in patients with systolic dysfunction and depressed LVEF, low transvalvular gradients are found for two reasons: either afterload-hypertrophy mismatch or concomitant cardiomyopathy (such as ischemic heart disease). Differentiating between these two groups is extremely important because the patients in the first group will benefit from aortic valve surgery, whereas those in the second group may not be candidates (Figure 35.2).13

To differentiate between these two groups, various LV and aortic valve parameters are assessed at rest and following intravenous infusion of increasing amounts of dobutamine starting at 5 μg/kg/min and escalating up to 20 μg/kg/min.14 These parameters are usually measured by echocardiography (modified dobutamine stress echocardiogram). However, these parameters may also be evaluated during cardiac catheterization.

In patients with low-gradient AS with reduced LVEF, changes in the following three parameters are measured during dobutamine stress testing: LV stroke volume, AVA, and mean gradient. If the stroke volume increases ≤ 20%, the patient likely has severe cardiomyopathy and is usually not a candidate for aortic valve surgery. On the other hand, if the stroke volume increases more than 20%, two scenarios are possible: (1) AVA remains essentially the same but the mean gradient increases above 30 mm Hg and (2) AVA increases by 0.2 cm² or more but the gradient remains essentially unchanged. In the first scenario, patients have true severe AS and will benefit from aortic valve surgery.

### Table 35.1 Aortic Valve Stenosis Criteria in Patient With Preserved Transvalvular Flow

<table>
<thead>
<tr>
<th>NONSTENOTIC AORTIC VALVE</th>
<th>MILD AS</th>
<th>MODERATE AS</th>
<th>SEVERE AS</th>
</tr>
</thead>
<tbody>
<tr>
<td>Valve area (cm²)</td>
<td>2.0–4.0</td>
<td>&gt;1.5</td>
<td>1.0–1.5</td>
</tr>
<tr>
<td>Valve area index (cm²/m²)</td>
<td></td>
<td>&lt;0.6</td>
<td></td>
</tr>
<tr>
<td>Peak velocity (m/s)</td>
<td>&lt;2.5</td>
<td>2.5–3.0</td>
<td>3.1–4.0</td>
</tr>
<tr>
<td>Peak gradient (mm Hg)</td>
<td>&lt;25</td>
<td>25–36</td>
<td>37–64</td>
</tr>
<tr>
<td>Mean gradient (mm Hg)</td>
<td>&lt;25</td>
<td>25–40</td>
<td>&gt;40</td>
</tr>
<tr>
<td>ACC/AHA stage</td>
<td>A (at risk)</td>
<td>B (progressive)</td>
<td>C (asymptomatic)</td>
</tr>
</tbody>
</table>

ACC, American College of Cardiology; AHA, American Heart Association.
In the second scenario, patients have pseudosevere AS due to LV cardiomyopathy; they are unlikely to benefit from aortic valve surgery (Figure 35.3).

Cardiac catheterization is usually unnecessary because echocardiography is the gold standard for diagnosis and quantification of AS. However, because coronary artery disease is common in patients with AS, coronary angiography is usually performed before surgical or percutaneous AVR. In this setting, aortic valve gradients may be evaluated using invasive hemodynamic measurements.

When comparing aortic gradients obtained by cardiac catheterization, it is the mean but not the peak aortic gradient that correlates with echocardiography. Cardiac catheterization and echocardiography measure two separate peak gradients (Figure 35.4).

On catheterization, the so-called peak-to-peak (P2P) gradient is measured, which represents the pressure difference between the peak LV systolic pressure and the peak aortic systolic pressure. These two peaks do not occur simultaneously and are thus nonphysiologic; the more severe the AS, the greater the time difference between the two pressure peaks.

On echocardiography, the peak instantaneous pressure gradient (PIG) is measured instead; it represents the pressure difference between LV systolic and aortic pressure at the same time point. P2P is typically lower than PIG, largely due to the pressure recovery phenomenon (kinetic energy converted to potential energy in the ascending aorta).15

MEDICAL THERAPY

There are no proven medical therapies for prevention or treatment of AS, and routine antibiotic prophylaxis of bacterial endocarditis in patients with AS is no longer recommended.16

Usually, patients with severe AS present to the CCU with clinical signs of heart failure. Pharmacologic therapy including diuretics, angiotensin-converting enzyme (ACE) inhibitors, and digitalis may be used with caution. Although intravenous vasodilators used to be contraindicated in patients with severe AS, intravenous nitroprusside has been demonstrated to relieve symptoms of heart failure in patients with severe AS and severely reduced LV systolic function.17 In that study, intravenous nitroprusside was started at a mean dose of 14 ± 10 μg/min, and the dose was increased to a mean of 103 ± 67 μg/min at 6 hours and 128 ± 96 μg/min at 24 hours.

Patients with severe AS may also present with other critical illnesses, such as septic shock. Management of these patients can...
be extremely challenging owing to the narrow balance between preload and afterload. Hemodynamic compromise is common. Hypotension from severe sepsis or septic shock may initiate a spiral of death, in which low aortic pressure leads to low coronary perfusion, with further ventricular impairment that fails to pump blood against a severely stenotic valve. This negative feedback rapidly leads to cardiac arrest and should be promptly recognized so that vasopressors are administered before such cascade takes place.

**SURGICAL AND PERCUTANEOUS INTERVENTIONAL THERAPY**

AS is a mechanical problem that requires a mechanical intervention. AVR is the preferred therapeutic choice in patients with AS to improve symptoms and increase survival. Major indications for AVR are listed in Table 35.2.

Surgical aortic valve replacement (SAVR) is the standard approach, indicated for patients with acceptable surgical risk who meet indications for AVR. The technical aspects are beyond the scope of this chapter, but clinicians should be familiar with the decision process when choosing a surgical prosthesis. In addition to the appropriate size, such that there is no postoperative patient–prosthesis mismatch, the valve material has significant implications for the patient. Although a bioprosthesis is considered to be less durable than a mechanical valve, it offers the advantage of not requiring systemic anticoagulation. As such, in patients who are older or who have a contraindication for anticoagulation, a bioprosthetic implant is preferred. Conversely, in younger patients who have no contraindications for anticoagulation, a mechanical valve is reasonable. Above all, this should be a shared decision process between the surgeon and the patient.

**TABLE 35.2 Major Indications for Aortic Valve Replacement in Patients With Aortic Stenosis**

<table>
<thead>
<tr>
<th>SYMPTOMS</th>
<th>RECOMMENDATION FOR AORTIC VALVE REPLACEMENT</th>
<th>ACC/AHA STAGE</th>
<th>COR</th>
<th>LOE</th>
</tr>
</thead>
<tbody>
<tr>
<td>Symptomatic</td>
<td>Symptomatic high-gradient severe AS (by history or exercise testing)</td>
<td>D1</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>Symptomatic low-flow/low-gradient severe AS with reduced EF (confirmed by</td>
<td>D2</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>dobutamine stress echocardiography)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Symptomatic low-flow/low-gradient severe AS with preserved EF (if valve</td>
<td>D3</td>
<td>IIa</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>obstruction is considered the cause of symptoms)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Severe AS undergoing other cardiac surgery</td>
<td>C or D</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td>Asymptomatic</td>
<td>Asymptomatic high-gradient severe AS with LV dysfunction (EF ≤50%)</td>
<td>C</td>
<td>I</td>
<td>B</td>
</tr>
<tr>
<td></td>
<td>Asymptomatic high-gradient very severe AS (Vmax ≥5 m/s) and low surgical risk</td>
<td>C</td>
<td>IIa</td>
<td>B</td>
</tr>
</tbody>
</table>

ACC, American College of Cardiology; AHA, American Heart Association; AS, aortic stenosis; COR, class of recommendation; EF, ejection fraction; LOE, level of evidence; LV, left ventricular; Vmax, peak velocity.

For a complete list, refer to the 2014 AHA/ACC Valvular Heart Disease Guideline. Note the lower class of recommendation for symptomatic patients with low-flow severe AS, both with reduced and preserved EF.
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Complications such as vascular access complications, neurologic events, conduction abnormalities, coronary artery obstruction, low cardiac output, annular rupture, and paravalvular AR.

Besides femoral access bleeding and vascular complications from the large sheaths used, patients should be closely monitored for neurologic changes given the risk of stroke from the large-profile devices advanced through the arterial system and crushing the calcified native valve.

Patients are also monitored for conduction abnormalities, specifically complete heart block from extrinsic compression of the conductions system following valve deployment. These patients routinely receive temporary venous pacemaker placement preprocedurally. Persistent heart block requiring permanent pacemaker is more common in patients with preexisting conduction system disease, especially right bundle branch block. Need for permanent pacing was previously more commonly seen in the self-expandable valve. However, with the latest generation of TAVR valve, the rate of permanent pacemaker implantation is similar between self-expanding and balloon-expandable TAVR valves.

Periprocedural complications, usually diagnosed before transfer to CCU for monitoring, include coronary artery obstruction, low cardiac output, annular rupture, and paravalvular AR. Late complications include leaflet thrombosis, valvular degeneration, and infective endocarditis.

Currently, typical indications for balloon valvuloplasty in calcific AS include short-term relief of AS in patients undergoing noncardiac surgery (such as hip replacement), in those with terminal illness (such as cancer), or as a temporizing measure in patients who are rapidly decompensating from severe AS in whom durable interventions are being entertained.

TAVR has changed the landscape of therapy for AS, allowing for patients who were previously too ill for surgery to receive lifesaving therapy. Since the first percutaneous replacement was performed by Alain Cribier in 2002, there have been over 200,000 TAVR procedures performed worldwide. TAVR is now approved in patients with symptomatic severe AS who are inoperable, or are either high or intermediate risk for SAVR.

TAVR was considered experimental until 2010, when the first randomized trial showed a markedly improved survival in patients who were not surgical candidates. These findings were subsequently confirmed in head-to-head trials comparing TAVR with SAVR in high-risk and intermediate-risk surgical patients. In addition, TAVR is currently being investigated in low-risk patients.

There are currently two approved device types in the United States, a balloon-expandable (Edwards Sapien family of TAVR valves) and a self-expandable valve (Medtronic CoreValve family of TAVR valves), both of which have been updated with new design features to reduce paravalvular AR (Figure 35.5).

**FIGURE 35.5** Transcatheter aortic valve replacement valves. Two major types of transcatheter implantable aortic valves. The balloon-expandable valve has a short and straight frame (left), whereas the self-expandable valve is longer and hourglass-shaped (right). These different metallic frames facilitate identification in routine chest radiography. TAVR, transcatheter aortic valve replacement. (Images available at the manufacturer’s websites: www.tavrbyedwards.com and www.corevalve.com, respectively.)

**POST-TRANSCATHETER AORTIC VALVE REPLACEMENT Complications**

Patients who undergo TAVR are usually cared for in the CCU postprocedurally, so it is important to be familiar with the most common complications such as vascular access complications, neurologic events, conduction abnormalities, coronary artery obstruction, low cardiac output, annular rupture, and paravalvular AR.

**IMPACT OF SEVERE AORTIC STENOSIS ON PREGNANCY**

Patients with calcific AS are seldom of child-bearing age. In patients of child-bearing age, AS is usually caused by noncalcific
congenital abnormalities (such as unicuspid aortic valve). Young patients with moderate-to-severe AS should be advised against conception until AS is relieved. Those who nonetheless become pregnant may or may not develop severe symptoms. Pregnant patients with AS and mild symptoms may be managed conservatively during pregnancy with bed rest, oxygen, and β-blockers. Pregnant patients with severe symptoms may need percutaneous or surgical intervention. These procedures carry significant risk to both the mother and the unborn child.24

AORTIC REGURGITATION

ETIOLOGY AND PATHOPHYSIOLOGY

AR may be caused by disorders of the aortic root or the aortic valve leaflets. BAV, ascending aortic aneurysm, aortic dissection, and endocarditis are common causes of AR. Conditions predisposing the patient to aortopathies and AR include hypertension and connective tissue disorders (such as Marfan syndrome, Ehlers-Danlos syndrome, and ankylosing spondylitis). In less developed countries, rheumatic heart disease and tertiary syphilis remain important causes of AR.

In AR, the blood flows back into the ventricle through the aortic valve during diastole, joining the systemic volume that entered the left ventricle through the mitral valve. During the subsequent systole, both volumes leave the aortic valve together; the systemic volume continues into the aorta and its branches, whereas the regurgitant volume flows back into the ventricle. The cycle then repeats itself. Subsequent pathophysiology depends on whether AR is chronic or acute.

In chronic AR, the left ventricle progressively enlarges to accommodate the combined systemic stroke volume and the regurgitant volume. This remodeling often prevents significant elevation of left heart pressures. It may take years, if not decades, for patients with chronic AR to develop congestive heart failure owing to progressive LV systolic dysfunction. This adaptation contrasts with acute AR, in which there is sudden volume overload of a nondilated left ventricle, marked elevation of LV and left atrial pressures, life-threatening pulmonary edema, and even cardiogenic shock.

Patients with severe AR, whether acute or chronic, typically present to the CCU with signs and symptoms of heart failure. Although severe acute AR represents only a minority of AR cases, its often fulminant and life-threatening course necessitates CCU admission. The rest of the section on AR is devoted to severe acute AR.

CLINICAL PRESENTATION

The most common etiologies of severe acute AR are chest trauma, bacterial endocarditis, and dissection of the ascending aorta (Stanford type A aortic dissection). Less commonly, severe acute AR may result following balloon valvuloplasty for severe AS.

Patients with severe acute AR frequently present with fulminant pulmonary edema and cardiogenic shock. Patients with endocarditis will also have general signs and symptoms of a systemic bacterial illness. Patients with acute type A aortic dissection usually complain of severe chest pain, often in the setting of uncontrolled systemic hypertension.

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DIAGNOSIS

In severe acute AR, there may be few or no auscultatory findings of AR; the diastolic murmur is often soft, short, or even absent because of the rapid equilibration of aortic and LV pressures during diastole. This is due to the acute nature of the disease, which precludes increased compliance of the left ventricle. Therefore, the rapid rise in LV end-diastolic pressure limits the total regurgitant volume, explaining the underwhelming diastolic murmur and the lack of wide pulse pressure. There is, however, marked tachycardia and S3 gallop.

CXR in severe acute AR frequently reveals pulmonary congestion. ECG may show tachycardia; there may also be signs of myocardial ischemia (owing to high myocardial demand caused by very elevated LV pressures or when acute aortic dissection extends into the coronary artery ostia). When endocarditis leads to periaortic valve abscess, ECG may demonstrate varying degrees of atrioventricular conduction block.

Transthoracic and transesophageal echocardiography are the primary means of evaluating AR. Echocardiography may establish the etiology, mechanism, and severity of AR (Figure 35.6). The diagnostic criteria for grading the severity of AR are listed in Table 35.3.

MEDICAL THERAPY

Severe acute AR is a life-threatening medical emergency that necessitates highest levels of CCU care. Diuretic therapy, positive end-expiratory pressure, and supplemental oxygen administration with endotracheal intubation are used to treat pulmonary edema. Afterload reduction may be achieved with the use of intravenous vasodilators (such as nitroprusside). Disease-specific therapies, if available, should also be administered (such as antibiotic therapy for endocarditis).

SURGICAL AND PERCUTANEOUS INTERVENTIONAL THERAPY

In severe acute AR, aortic valve surgery should be performed as soon as possible, especially in cases of type A aortic dissection and infective endocarditis. The standard procedure of choice is surgical AVR. At present, there is no approved percutaneous option for patients with pure AR.

Aortic valve sparing surgeries may be an alternative to surgical AVR in specialized centers. This surgery, known as the David procedure (after Tirone David, the Brazilian surgeon who conceived and first performed this technique), involves ascending aorta repair with a vascular graft, followed by coronary artery reimplantation, reconstruction of the aortic root, and reimplantation of the aortic valve. This technique is better suited for young patients with genetic syndromes associated with aortic root dilatation, and it is still not employed universally.

In-hospital and long-term survival is significantly improved in patients who promptly undergo SAVR or repair. However, such intervention might be prohibitive in patients with severe embolic strokes or comorbid conditions that diminish the prospect of reasonable recovery.

Despite the excitement about transcatheter therapies in severe AS, use of TAVR in severe AR has been reported only anecdotally. A stenotic calcific aortic valve forms a solid structure for anchoring in transcatheter replacement, whereas a regurgitant
Section IV • Aortic, Pericardial, and Valvular Disease in the CCU

aortic valve—often associated with a dilated aortic root—poses a great challenge in holding a transcatheter valve. Still, TAVR may represent a last resort in patients who present with severe symptomatic AR who are not surgical candidates.

Registry data from self-expanding TAVR (which have a larger anchoring profile than balloon-expandable valves) in patients with predominant AR show significantly increased mortality when compared with patients undergoing TAVR for severe AS (around 20%), particularly in patients in whom valvular calcification was absent.27,28

Furthermore, intra-aortic balloon pump (IABP) is absolutely contraindicated when significant AR is present. Diastolic counterpulsations—which are the hallmarks of IABP function—worsen AR. This, in turn, decreases effective stroke volume and cardiac output, worsening cardiogenic shock. In addition, it increases LV end-diastolic pressure and myocardial oxygen consumption, worsening pulmonary edema and myocardial ischemia. Therefore, in conditions that usually require IABP for medical stabilization (such as cardiogenic shock and refractory ischemia), use of counterpulsation is prohibitive with moderate or severe AR.

IMPACT ON PREGNANCY

The hemodynamic changes associated with pregnancy (increased cardiac output from increased heart rate and stroke volume)
are more likely to decompensate patients with stenotic valvular diseases compared with those with insufficient valves, given the mechanical obstruction to blood flow in such conditions. Conversely, regurgitant lesions are better tolerated in pregnancy, and AR is generally tolerated unless it is severe.

Severe AR, whether acute or chronic, is one of the valvular heart lesions that may be associated with high maternal and/or fetal risk during pregnancy. Pregnant women with AR who have New York Heart Association functional class III to IV symptoms, severe pulmonary hypertension (pulmonary pressure >75% of systemic pressures), and/or LV systolic dysfunction are at particular risk for maternal and fetal complications. The same is true for pregnant women having Marfan syndrome with or without AR.\(^\text{24}\)

### References


Section IV • Aortic, Pericardial, and Valvular Disease in the CCU


GENERAL CONCEPTS OF AORTIC VALVULAR DISEASE

The heart is a muscular organ whose primary function is to pump blood in our body. To accomplish this goal, several valves are present in its different chambers to prevent backflow. The aortic valve is a valve composed of three thin leaflets, separating the aorta—the largest artery in the body from which all the blood pumped out of the heart flows—and the left ventricle—the chamber that pumps blood out of the heart to the rest of the body. When the aortic valve becomes too narrowed, doctors call it aortic stenosis. When it becomes leaky, it is called aortic insufficiency.

AORTIC STENOSIS

WHAT IS THE ILLNESS?

If the aortic valve becomes too narrow, pumping blood out of the heart becomes difficult. When patients feel symptoms from this valve narrowing (called stenosis), they usually report episodes of passing out, chest pain with exertion, and shortness of breath. Usually the cause of AS is wear and tear over many years, making it harder and less flexible with aging (the reason these patients are usually elderly). However, some patients are born with malfunctioning valves, and in these cases they develop valve narrowing at a younger age.

HOW WILL THE PATIENT BE TREATED?

There are no medicines that fix AS, so an invasive procedure is usually indicated in symptomatic patients. The standard procedure is to replace the valve with a prosthesis. In some patients this is done through open heart surgery, and in others the valve is replaced through catheters.

In patients with severe AS, the disease may progress rapidly and even cause death if not treated in time. Therefore, once diagnosed, planning for receiving a new valve should be undertaken.

WHAT IF THE PATIENT IS PREGNANT OR THINKING OF BECOMING PREGNANT?

If there is severe narrowing of the aortic valve, pregnancy may need to be delayed until the narrowing is relieved—usually through the use of balloon catheter inserted through the groin, threaded to the heart, and blown up inside the valve to make it larger. If already pregnant and there are symptoms (such as shortness of breath or chest pain), the patient may need to take certain medications that are effective for her but not harmful to the child. If the symptoms during pregnancy are severe, the balloon catheter treatment or even open heart surgery may be needed. These procedures carry significant risk to both the mother and the unborn child.

AORTIC REGURGITATION

WHAT IS THE ILLNESS?

AR is the medical term to describe a leaky aortic valve, allowing blood to backflow into the heart after it is pumped to the aorta. When this happens, there is extra work for the heart to pump blood forward, and this backflow may cause breathing problems from water buildup in the lungs and other parts of the body.

One of the most common causes of AR in the United States is a dilated aorta, where the valve sits. When this artery becomes too big, the valve leaflets become too far apart from each other and blood leaks back into the heart. This enlargement may happen from wear and tear in older patients, or from a loose buildup in patients born with genetic predisposition to aortic enlargement.

Another common cause for AR is infection in the valve, causing destruction of the leaflets and, consequently, leakage. Wear and tear of the valve leaflets may also cause AR, not only in older patients but also in young patients born with a malfunctioning valve. Finally, in patients born outside of the United States, long-standing syphilis and rheumatic fever may also lead to AR.

HOW WILL THE PATIENT BE TREATED?

In patients who develop AR in a short period of time, such that the heart does not have enough time to accommodate the leakage, quick medical stabilization in an intensive care unit is necessary. However, the ultimate treatment requires valve replacement with prosthesis.

Similar to AS, a severely leaky valve of AR may be replaced through open heart surgery. However, in contrast to AS, a severely leaky valve of AR cannot be replaced using catheters in most patients.

If the AR developed slowly, the heart eventually will become large and weak trying to accommodate the backflow of blood. However, in cases of rapidly developing severe AR, the disease may cause imminent death unless appropriate medicines and, eventually, surgery for valve replacement are employed.

WHAT IF THE PATIENT IS PREGNANT OR THINKING OF BECOMING PREGNANT?

Severe leakage of the aortic valve may not be well tolerated during pregnancy and the condition may harm the unborn child. Women with severe leakage who are considering pregnancy may need to delay it until the condition is treated. If already pregnant, significant heart problems may be experienced during the pregnancy.
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<th>Query</th>
<th>Remarks</th>
</tr>
</thead>
<tbody>
<tr>
<td>AQ1</td>
<td>References 21 and 22 are not cited in the text. Please provide in-text citation for the same.</td>
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