

# Aortic Regurgitation

## Introduction

Aortic regurgitation (AR) occurs when blood flows retrograde across the aortic valve from the aorta into the left ventricle during diastole. This abnormal backflow of blood leads to pathologic changes in the heart in order to compensate for the decreased effective cardiac output that results. These pathologic changes are highly dependant on the severity and rapidity of onset of AR. They require a significant amount of time to take place; therefore if *acute* aortic regurgitation were to occur, compensation for the increased LV volume would not be able to take place. The small increase in LVEDV (left ventricular end diastolic volume) due to the acute AR would lead to a greatly increased LVEDP (left ventricular end diastolic pressure). This increased pressure is automatically transmitted to the pulmonary vasculature leading to severe symptoms of left heart failure including flash pulmonary edema. However in chronic AR, the LV dilates and hypertrophies (eccentrically) slowly over time. These changes help to maintain normal LV pressures in the setting of a significantly increased LV volume. In fact, patients with severe chronic AR may have the largest LV end diastolic volumes produced by any cardiac disease state and yet their LV diastolic pressures are not elevated significantly. The stroke volume increases greatly in these patients so that despite the severe AR, a normal *effective* forward cardiac output can be maintained. As the AR continues to worsen, the LV can no longer dilate or hypertrophy further leading to increased LV pressures, which are then transmitted to the pulmonary vasculature. Symptoms of heart failure then occur.

Also, as the AR worsens allowing larger volumes of blood to be regurgitated during diastole, the aortic diastolic pressures drop significantly. These low diastolic pressures, coupled with the increased systolic pressures produced by the hypertrophied LV to maintain cardiac output, produce a *wide pulse pressure*. The large stroke volume can produce signs and symptoms of a high flow state (see below) and the low diastolic pressures can significantly affect coronary perfusion pressures, since the coronary arteries fill during diastole.

## Etiologies

Aortic regurgitation can result from abnormalities of the aortic valve leaflets, dilation of the aortic root, or a severe increase in afterload. A complete list of etiologies is below:

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### Aortic leaflet/cusp abnormalities

- Infectious: Bacterial endocarditis, rheumatic fever
- Congenital: Bicuspid aortic valve calcification
- Inflammatory: SLE, RA, Behcet's syndrome
- Degenerative: Myxomatous (floppy valve), senile calcification
- Others: Trauma, post aortic valve valvuloplasty, diet drug valvulopathy

### Aortic root abnormalities

- Aortic root dilation: Marfan's syndrome, syphilitic aortitis, idiopathic aortitis, Ehlers-Danlos syndrome, relapsing polychondritis
- Loss of commissural support: Aortic dissection, trauma, ventriculoseptal defect (VSD)

### Increased afterload

- Uncontrolled systemic hypertension, supraaortic stenosis, coarctation of the aorta

## Signs and Symptoms

As chronic AR develops slowly over time, the LV can easily compensate as described above. Therefore patients remain asymptomatic for a long period of time early in disease. The first symptoms experienced may result from the large stroke volumes and forceful LV contractions causing the patient to experience palpitations. Angina may occur in the absence of atherosclerotic coronary disease since the low diastolic pressures in severe AR compromise coronary filling and the LVH results in increased oxygen demand. Signs of left and right heart failure occur late in disease (see Heart Failure).

Almost all patients with significant acute aortic regurgitation are symptomatic, unlike chronic AR. Symptoms of acute left heart failure occur and include severe dyspnea even at rest, orthopnea, and PND.

## Physical Examination

In chronic AR, visible cardiac and arterial pulsations are common due to the large stroke volume. The carotid pulse can commonly be seen. The PMI is displaced laterally and caudally due to the LV dilation and hypertrophy that occurs. On auscultation, the typical murmur of AR is a soft, high-pitched, early diastolic decrescendo murmur heard best at the 3<sup>rd</sup> intercostal space on the left (Erb's point) on end expiration with the patient sitting

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and leaning forward. This murmur is often difficult to distinguish from the Graham-Steel murmur of pulmonic insufficiency (see Heart Murmurs section). If aortic root disease is the cause of the AR, the murmur will be heard best at the right upper sternal border and not at Erb's point. In addition to the above murmur, a systolic ejection murmur may be present at the right upper sternal border simply due to the large stroke volume passing through the aortic valve with each systolic contraction of the LV. A diastolic rumble may also be heard at the apex due to the regurgitant jet striking the anterior leaflet of the mitral valve causing it to vibrate. This murmur is termed the Austin-Flint murmur. A widened pulse pressure is often present due to the high flow state as previously described. When severe heart failure develops, the pulse pressure will decrease and the below listed peripheral signs of AR are lessened. A fourth heart sound develops when LVH becomes severe and limits diastolic filling. A third heart sound is often present due to the increased early diastolic filling into a dilated LV.



<u>Name of Sign</u>	<u>Description</u>
<b>Corrigan's pulse</b>	<b>A rapid and forceful distension of the arterial pulse with a quick collapse secondary to the increased pulse pressure</b>
<b>De Musset's sign</b>	<b>Bobbing of the head with each heart beat</b>
<b>Muller's sign</b>	<b>Visible pulsations of the uvula</b>
<b>Quincke's sign</b>	<b>Capillary pulsations seen on light compression of the nail bed</b>
<b>Traube's sign</b>	<b>Systolic &amp; diastolic sounds heard over femoral artery ("pistol shots")</b>
<b>Duroziez's sign</b>	<b>Gradual pressure over femoral artery leads to a systolic &amp; diastolic bruit</b>
<b>Hill's sign</b>	<b>Popliteal systolic blood pressure exceeding brachial systolic blood pressure by 60 mmHg or greater</b>
<b>Shelly's sign</b>	<b>Pulsation of the cervix</b>
<b>Rossenbach's sign</b>	<b>Hepatic pulsations</b>
<b>Becker's sign</b>	<b>Visible pulsations of the retinal arterioles</b>
<b>Gerhardt's sign (aka Sailer's sign)</b>	<b>Pulsation of the spleen in the presence of splenomegaly</b>
<b>Mayne's sign</b>	<b>A decrease in diastolic blood pressure of 15 mmHg when the arm is held above the head</b>
<b>Landolf's sign</b>	<b>Systolic contraction and diastolic dilation of the pupil</b>

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Many peripheral signs of AR have been identified and named after the physician that first described it. These signs are listed above. A majority of these signs result from the large stroke volume and widened pulse pressure that occur in patients with severe AR.

In acute AR, the above listed peripheral signs are absent since the heart does not have time to compensate for the increased LV volume. Also, the murmur of AR is short in duration and may be difficult to hear since the aortic pressure and the LV pressure (which is elevated in acute AR) equalize fast in diastole. A soft first heart sound may be present due to early closure of the mitral valve.

### Diagnosis

The EKG in patients with AR is non-specific and may show LVH and left atrial enlargement. In acute AR, sinus tachycardia may be the only abnormality on EKG. The chest radiograph is also non-specific in AR. Cardiomegaly is present in patients with chronic AR. In acute AR, pulmonary edema is almost universally present. If the AR is due to an aortic dissection, the mediastinum may appear widened.

Echocardiography is crucial to the identification of AR, determining the etiology, and estimating the severity. Echocardiography is almost 100% sensitive and specific for the detection of AR. The actual regurgitant jet can be directly visualized using color flow Doppler. This is extremely important in patients with acute AR since the physical examination may not be revealing of any valvular abnormality.

The etiology of AR can often be determined using echocardiography. Structural abnormalities such as calcification or thickening of the AV can be seen. Vegetations on the AV may be identified (transesophageal echocardiography is more sensitive) indicating endocarditis as the cause. A bicuspid AV or prolapse of the AV may be seen. The size of the aortic root can be measured and aortic dissections can be identified.

The severity of AR can be estimated using three parameters: 1) Regurgitant jet size 2) Pressure half-time, and 3) Regurgitant fraction

Regurgitant jet size is helpful by measuring the ratio of the AR jet diameter just below the leaflets of the AV to the size of the LV outflow diameter. Ideally, the ratio should be zero since no regurgitant jet should be present. A ratio of  $< 24$  is mild, 25-45 is moderate, 46-64 is moderately severe, and  $> 65$  is severe.

The pressure half-time index is the time it takes for the initial maximal pressure gradient in diastole to fall by 50%. In patients with mild AR, this fall in pressure is gradual. In the setting of severe AR, a rapid drop in pressure occurs. A pressure half-time of  $> 500$  is

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considered mild AR, 500-349 is moderate, 349-200 is moderately severe, and < 200 is severe. The severity of AR assessed by using the pressure half-time is overestimated in patients with a significantly increased LVEDP.

The regurgitant fraction is perhaps a more straightforward means of assessing the severity of AR. The regurgitant fraction is the percentage of stroke volume that returns to the left ventricle from the aorta during diastole. For example, a regurgitant fraction of 33% would indicate that one-third of the total stroke volume returns to the LV retrograde across the AV during diastole. A regurgitant fraction of < 20% indicated mild AR, 20-35% indicates moderate AR, 36-50% indicates moderately severe AR, and > 50% severe AR.

It is important to note that the severity of AR assessed using echocardiography is dependent on the hemodynamic status of the patient at the time of evaluation, most importantly, the afterload.

	Jet size ratio	Pressure half-time (ms)	Regurgitant fraction (%)
Mild	< 24	> 500	< 20
Moderate	25-45	500-349	20-35
Moderate-Severe	46-64	349-200	36-50
Severe	> 65	< 200	> 50

Cardiac catheterization is not necessary in the majority of patients with AR because of echocardiography. During cardiac catheterization, AR can be detected by injecting contrast into the aortic root and visualizing the appearance of contrast in the left ventricle. Catheterization can also assess the ascending aorta for aortic root disease. A number of factors affect the grading of severity of AR during catheterization. If the catheter is position too closely to the AV, the amount of AR will be overestimated. The volume and rapidity of injection of contrast into the aortic root affects the amount of AR visualized. As previously mentioned, the hemodynamic parameters (mostly afterload) at the time of assessment also affect the severity of AR. The grading scale for AR used during catheterization is below. Cardiac catheterization can also measure LVEDV and LVEDP which can be helpful in determining the severity or AR. Cardiac catheterization is also indicated if aortic valve replacement is going to be performed so that the coronary arteries can be imaged. If significant coronary atherosclerosis is present, coronary artery bypass grafting can be done at the same time as the valve replacement.

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	Amount of LV contrast seen	Intensity of contrast	Clearance of contrast from LV
I (mild AR)	Some	Aorta > LV	Completely cleared on each beat
II (moderate AR)	Completely filled LV after many beats	Aorta > LV	Incomplete clearance on each beat
III ( moderate-severe AR)	Completely filled after several beats	Aorta = LV	Slow clearance
IV (severe AR)	Completely filled after only one beat	Aorta < LV	Very slow clearance

### Treatment

Acute aortic regurgitation carries a very high mortality if prompt surgical intervention in the form of aortic valve replacement (AVR) is not undertaken. Treatment of pulmonary edema and afterload reduction can help relieve symptoms and buy the patient some time before surgery is performed. Nitroprusside is the treatment of choice since it reduces both preload and afterload with great efficacy. Dobutamine may be needed if the patient remains hypotensive with a low cardiac output. The use of intraaortic balloon counterpulsation is not commonly used in acute AR as it is in acute MR. If the acute AR is found to be due to infectious endocarditis, seven days of intravenous antibiotics are often given before valve replacement.

In patients with mild to moderate chronic AR, no specific treatment is required. These patients should be monitored yearly to assess the progression of the disease. Antibiotic prophylaxis is recommended to prevent bacterial endocarditis.

Patients with moderate to severe AR that are symptomatic should undergo AVR if they fall into NYHA class III-IV heart failure. Those that are in NYHA class II should receive an exercise stress test to assess the endurance and exercise capacity. They should be treated with diuretics and afterload reduces (commonly ACE inhibitors) until AVR is indicated.

If moderate to severe AR is present but the patient is asymptomatic, AVR is not clearly indicated. Afterload reduces are given and frequent echocardiographic assessment is recommended to monitor for LV dysfunction. The ideal time for AVR is late enough in the course of the disease to justify the risk-benefit ratio of surgery, yet early enough to prevent potential irreversible myocardial damage from occurring. The LV ejection fraction has been shown to correlate best with the surgical outcome.