

Aortic Regurgitation

Chapter 273 | Part 6: Disorders of the Cardiovascular System

KEY CLINICAL POINTS

- 1 Aortic regurgitation (AR) results from primary valve disease, aortic root disease, or both.
- 2 Chronic severe AR causes LV volume overload, leading to dilation and eccentric hypertrophy.
- 3 Key physical findings include water-hammer (Corrigan), Quincke, Traube, and Duroziez signs.
- 4 Austin Flint murmur is a low-pitched mid-to-late diastolic rumble caused by mitral leaflet displacement.
- 5 Severe AR echocardiographic criteria: $V_c > 0.6 \text{ cm}$, $RVd \geq 160 \text{ mL}$, $RF \leq 50\%$, and $ERO \geq 0.3 \text{ cm}^2$.
- 6 Acute severe AR requires urgent surgery (within 24–48 h) due to rapid LV decompensation.
- 7 Surgical intervention for chronic cases is indicated by symptoms, $EF \leq 55\%$, $LVEDD > 50 \text{ mm}$ ($>25 \text{ mm/m}^2$), or $LVEDD > 65 \text{ mm}$.
- 8 Medical management includes diuretics and vasodilators; beta-blockers are used for hypertension or specific aortopathies.
- 9 TAVI is not recommended for surgical candidates with severe AR.
- 10 Patients with severe AR must avoid isometric exercises.

DEFINITION & CLASSIFICATION

- **Definition (Harrison's 22e):** Aortic regurgitation (AR) is a valvular heart disease characterized by the retrograde flow of blood from the aorta into the left ventricle during diastole.
- **Pathology Sources:**
 - Primary valve disease: Thickening, deformity, and shortening of individual aortic valve cusps.
 - Primary aortic root disease: Marked aortic annular dilation without primary involvement of valve leaflets.
 - Combined disease: Both valve and root pathology.
- **Associated Conditions:** May be associated with Marfan's syndrome, idiopathic dilation of the aorta, annuloaortic ectasia, osteogenesis imperfecta, or severe chronic hypertension.

EPIDEMIOLOGY

- **Gender Distribution:** Approximately 3/4 of patients with pure or predominant valvular AR are men.
 - Exception: Women predominate in cases with associated rheumatic mitral valve disease.
- **Congenital Bicuspid Aortic Valve (BAV):**
 - May develop predominant AR.
 - ~20% of these patients require aortic valve surgery between 10 and 40 years of age.

ETIOLOGY & PATHOPHYSIOLOGY

- **Hemodynamics:**
 - Increased total stroke volume in AR.
 - Severe AR: Regurgitant flow may equal effective forward stroke volume.
 - Compensation: LV dilation and eccentric hypertrophy (increased preload) allow larger stroke volumes without increased myocardial shortening.
 - Laplace's Law: LV dilation → increased LV systolic tension required for any given level of systolic pressure.
- **Chronic vs. Acute:**
 - Chronic: Long latent period (10–15 years); progression leads to failure of adaptive measures, rising end-diastolic volume, and falling EF.
 - Acute: LV is unprepared; diastolic pressures rise rapidly (often >40 mmHg); may cause premature mitral valve closure.
- **Myocardial Ischemia:**
 - Risk factors: Increased demand (dilation, hypertrophy, high systolic tension) + decreased supply (lower diastolic pressure/duration).
 - Can occur even without epicardial coronary artery disease (CAD).
- **Cardiac Size:** Significant LV wall thickening; hearts may be among the largest encountered, sometimes weighing >1000 g.

Etiologies

- **Valvular Lesions:** Congenital (bicuspid), Endocarditis, Rheumatic fever, Myxomatous (prolapse), Radiation, Trauma, Syphilis, Ankylosing spondylitis.
 - Table 273-1: Lists these as primary causes of valvular AR.
- **Aortic Root Disease:** Aortic dissection, Medial degeneration, Marfan syndrome, Bicuspid aortic valve, Nonsyndromic familial aneurysm, Aortitis, Hypertension.
 - Table 273-1: Lists these as primary causes of aortic root disease.

CLINICAL FEATURES

- **Symptoms:**
 - Early: Awareness of heartbeat (especially when lying down), palpitations, sinus tachycardia.
 - Progression: Exertional dyspnea → Orthopnea → Paroxysmal nocturnal dyspnea → Excessive diaphoresis.
 - Angina: May occur even without CAD; often poorly responsive to sublingual nitroglycerin; may be severe/prolonged.
 - Late: Systemic fluid accumulation (congestive hepatomegaly, ankle edema).
- **Acute Presentation:**
 - Elevated LV end-diastolic pressure → early mitral valve closure → soft S, narrow pulse pressure, and short, early diastolic murmur.

Physical Findings

- **Palpation:**

- LV impulse: Heaving, displaced laterally/inferiorly; prominent systolic expansion and diastolic retraction.
- Thrills: Diastolic thrill (left sternal border); Systolic thrill (suprasternal notch/carotid arteries).
- **Arterial Pulse:**
 - Water-hammer pulse (Corrigan's pulse): Rapid rise → sudden collapse.
 - Quincke's pulse: Capillary pulsations at the nail root.
 - Traube's sign: "Pistol-shot" sound over femoral arteries.
 - Duroziez's sign: To-and-fro murmur on light femoral artery compression.
- **Auscultation:**
 - Aortic valve closure (A) usually absent in severe AR.
 - Murmur: High-pitched, blowing, decrescendo diastolic murmur; becomes holodiastolic as severity increases.
 - Location: Left sternal border (valvular); Right sternal border (suggests aortic root dilation).
 - Austin Flint murmur: Soft, low-pitched, rumbling mid-to-late diastolic murmur → caused by displacement of anterior mitral leaflet; not associated with mitral obstruction.
 - Mid-systolic ejection murmur: Often heard in isolated AR; does not necessarily signify AS.

DIFFERENTIAL DIAGNOSIS

- **Aortic Stenosis (AS):** Coexistence of hemodynamically significant AS with AR usually excludes all rarer forms of AR (only occurs in rheumatic or congenital cases).
- **Murmur Dynamics:** Decrescendo nature is due to the decreasing reverse diastolic pressure gradient between aorta and LV.

1. Echocardiogram (Cornerstone):

- Assess LV size, systolic function (EF), and mitral leaflet fluttering.
- Determine cause: Detect aortic annulus dilation, dissection, or primary leaflet pathology.
- Severity Criteria:
 - Central jet width >65% of LVOT width.
 - Regurgitant volume ≥ 60 mL/beat.
 - Regurgitant fraction $\geq 50\%$.
 - Effective regurgitant orifice (ERO) ≥ 0.3 cm².
 - Diastolic flow reversal in proximal descending thoracic aorta.
 - Advanced metrics: Global longitudinal strain (GLS) to detect early dysfunction; 3D echocardiography for LV volumes.

2. Transesophageal Echocardiography (TEE):

Used for detailed anatomic assessment of valve, root, and aorta when TTE is limited.

3. Cardiac MRI (CMR):

Used for accurate LV volume measurement, aortic size/contour, and screening for fibrosis (late gadolinium enhancement).

4. Computed Tomography (CT):

Assessment of aortic valve, root, and thoracic anatomy.

5. Chest X-Ray:

Shows apex displacement; less sensitive than Echo/CMR/CT for root/ascending aorta enlargement.

6. Cardiac Catheterization & Angiography:

Confirms magnitude of regurgitation, LV function, and coronary artery status prior to surgery.

MANAGEMENT & TREATMENT

1. Acute Aortic Regurgitation:

- Treatment: Intravenous diuretics and vasodilators (e.g., sodium nitropride) for stabilization.
- Surgery: Required urgently (within 24–48 h).
- Contraindications: Intra-aortic balloon counterpulsation is contraindicated; Beta-blockers are best avoided to prevent reduced CO or slowed heart rate.

2. Chronic Aortic Regurgitation:

- Indications for Surgery: Symptom onset OR LV systolic dysfunction.
- Surgical Thresholds (Asymptomatic):
 - EF $\leq$ 50%
 - LVESD > 50 mm (> 25 mm/m²)
 - LVEDD > 65 mm.
- Monitoring: Patients not meeting criteria should be followed every 6–12 months.

3. Medical Therapy:

- Diuretics and Vasodilators (ACEi, ARBs, CCBs, or hydralazine) for symptom management and hypertension.
- Beta-blockers: Used for hypertension, to reduce sense of forceful heart action, or in specific cases like Marfan's syndrome/aortopathy.
- Specific conditions: Penicillin for syphilitic aortitis; avoid isometric exercises in severe AR.

Surgical Treatment

1. Aortic Valve Replacement (AVR):

- Indicated for symptomatic severe AR regardless of LV function.
- Options: Mechanical or tissue (biological) prostheses.
- TAVI: Not recommended for surgical candidates with severe AR due to limited success in dilated rings.

2. Valve-Sparing Procedures:

- David Procedure: Used when AR is due to aortic root/ascending aorta dilation without primary valve involvement; involves reimplantation of the native valve into a graft.

3. Prognosis:

- Isolated AVR for pure AR: Mortality approx 1–2%.
- Addition of aortic surgery: Mortality doubles.
- Advanced LV dysfunction: Mortality approx 5–10% (operative) and 3–5% per year (late).
- High-risk markers:
 - Abnormal LV GLS ($\geq -18\%$).
 - LV end-systolic volume index ≥ 45 mL/m².

PROGNOSIS & COMPLICATIONS

• Surgical Outcomes:

- Isolated AVR: Mortality approx 1–2%.

- With additional aortic surgery: Mortality doubles.
- Advanced LV dysfunction: Operative mortality 5 ext{--}10%; late mortality 3 ext{--}5% per year.
- **Risk Markers:**
 - LV GLS $\geq -18\%$ associated with excess hazard for death.
 - LV end-systolic volume index $\geq 45 \text{ mL/m}^2$ associated with reduced event-free survival.

SPECIAL POPULATIONS

- **General Precautions:**
 - Avoid isometric exercises in severe AR.
 - Treat arrhythmias and infections promptly and vigorously.
 - Syphilitic aortitis requires full course of penicillin.

KEY PEARLS & HIGH-YIELD POINTS

- **Clinical Signs:** Water-hammer (Corrigan) and Quincke's pulse are hallmark findings.
- **Austin Flint Murmur:** Low-pitched, rumbling mid-to-late diastolic murmur; indicates mitral leaflet displacement, not necessarily mitral stenosis.
- **Acute Management:** Surgery within 24–48 h; avoid beta-blockers to maintain heart rate/cardiac output.
- **Surgical Thresholds:** Symptomatic AR or asymptomatic with EF $\leq 55\%$, LVESD $> 50 \text{ mm}$ ($> 25 \text{ mm/m}^2$), or LVEDD $> 65 \text{ mm}$.
- **Echocardiographic Severity:** $V_c > 0.6 \text{ cm}$, RVd $\geq 160 \text{ mL}$, RF $\leq 50\%$, and ERO $\geq 0.3 \text{ cm}^2$.
- **Marfan's Syndrome:** Beta-blockers and ARB losartan may retard aortic root enlargement.

FLOWCHARTS & ALGORITHMS

Flowchart 1: Management of patients with aortic regurgitation

Step 1: Initial Severity Assessment

- Determine if AR is "Severe" or "Moderate".
- **Severe Criteria:** $V_c > 0.6 \text{ cm}$, holodiastolic aortic flow reversal, RVd $\geq 160 \text{ mL}$, RF $\leq 50\%$, and ERO $\geq 0.3 \text{ cm}^2$.

Step 2: Decision Path for Moderate AR

- If Moderate → Check if "Low surgical risk"?
- Yes → **AVR (2c)**.
- No → **Other cardiac surgery**.

Step 3: Decision Path for Severe AR

- Identify Symptom Status:
 - **Symptomatic (Stage D) → AVR (1).**
 - **Asymptomatic (Stage C) → Evaluate Echocardiographic parameters:**
 - If EF $\leq 55\%$ (Stage C2) → **Other cardiac surgery**.

- If EF > 55%, but (LVESD > 20 mm) and $LVESD/S^2 > 25 \text{ mm/cm}^2$ → **AVR (2a)**.
- If "Progressive changes" (geq 3 years: EF 55 ext{--}60%, EDD geq 65 ext{ mm}) → **AVR (2b)**.

REFERENCE TABLES

TABLE 273-1

Major Causes of Aortic Regurgitation

VALVE LESION	ETIOLOGIES
Aortic regurgitation	Valvular Congenital (bicuspid) Endocarditis Rheumatic fever Myxomatous (prolapse) Radiation Trauma Syphilis Ankylosing spondylitis Aortic root disease Aortic dissection Medial degeneration Marfan syndrome Bicuspid aortic valve Nonsyndromic familial aneurysm Aortitis Hypertension