obstruction of blood flow from left ventricle (LV) → increased LV pressure → left ventricular concentric hypertrophy, which leads to:
Increased LV oxygen demand
Impaired ventricular filling during diastole → left heart failure

aortic valve opens in systolic

Aortic Stenosis:-

- Causes obstruction to LV outflow, which results in LVH.
- When the aortic valve are a falls below 1cm2, cardiacoutput fails to increase with exertion, causing angina (but may be normal at rest).
- With long-standing AS, the LV dilates, causing progressive LV dysfunction.
- With severe AS, LV dilation pulls the mitral valve annulus apart, causing MR.

Course

- Calcification of a congenitally abnormal bicuspid aortic valve.
- Calcification of tricuspid aortic valve in elderly
- Rheumatic fever

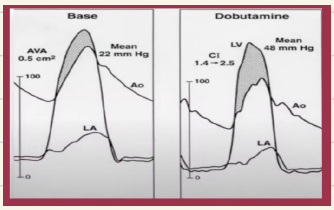
Altogether, only one-fourth of patients with symptomatic AS survives 3 years in the absence of aortic valve replacement (i.e., the 3-year mortality rate is 75% without surgery).

- Patients are often asymptomatic for years (until middle or old age) despite severe obstruction.
- Development of angina, syncope, or heartfailure is a sign of poor prognosis. Survival is similar to that of the normal population before the development of these three classic symptoms. Without surgical intervention, the survival is poor:
  Angina (35%)—average survival, 3 years
  Syncope (15%)—average survival, 2 years
  Heart failure (50%)—average survival, 1.5 years

a. Angina
b. Syncope—usuallyexertional
c. Heartfailure symptoms, such as dyspnea on exertion, orthopnea, or PND

- Harsh crescendo-decrescendo (diamond-shaped), late systolic ejection murmur that radiates bilaterally to the carotids (at 2nd ICS)
  - Handgrip decreases the intensity of the murmur.
  - Valsalva and standing from squatting decreases or does not change the intensity of the murmur (in contrast to hypertrophic cardiomyopathy).
- Soft S2
- S4 is best heard at the apex.
- Early systolic ejection click
- Small blood pressure amplitude, decreased pulse pressure
- Weak and delayed distal pulse (pulsus parvus et tardus) — diminished and delayed carotid upstrokes

Precordial thrill



Management of AS is straight forward:
If asymptomatic: No treatment
If symptomatic: Surgical aortic valve replacement or transcatheter aortic valve replacement (TAVR) depending on patient risk

Diagnosis

1. CXR findings: Calcific aortic valve, enlarged LV/LA (late)
2. ECG findings: LVH, LA abnormality
3. Echocardiography: Has replaced cardiac catheterization as standard test for hemodynamic and valve measurements for diagnosis of AS. Shows thickened, calcified aortic valve leaflets with limited mobility. Key measurements for diagnosis and severity are diminished valve area and increased ventricular-aortic pressure gradient
4. Exercise stress testing is indicated for asymptomatic patients with severe ASt o confirm asymptomatic status. These patients, despite having severe AS, do not require intervention. Should not be performed in symptomatic patients
5. Cardiaccatheterization
   a. Now used primarily in patients in whom echocardiography is nondiagnostic (i.e., poor visualization of valve, difficulty obtaining pressure gradients with Doppler)
   b. Useful in symptomatic patients before surgery because it can also reveal coronary anatomy, allowing the surgeon to do both CABG and aortic valve replacement in patients with both CAD and severe AS

Aortic Regurgitation

A. General Characteristics

1. Pathophysiology
   a. Also called aortic insufficiency; this condition is due to inadequate closure of the aortic valve leaflets. Regurgitant blood flow increases left ventricular end-diastolic volume.
   b. LV dilation and hypertrophy occur in response in order to maintain stroke volume and prevent diastolic pressure from increasing excessively.
   c. Over time, these compensatory mechanisms fail, leading to increased left-sided and pulmonary pressures.
   d. The resting left ventricular EF is usually normal until advanced disease.
2. Course
   a. For chronic aortic regurgitation, survival is 75% at 5 years.
      • After the development of angina, death usually occurs within 4 years.
      • After the development of heart failure, death usually occurs within 2 years.
   b. For acute aortic regurgitation, mortality is particularly high without surgical repair.

B. Causes

1. Acute
   a. Infective endocarditis
   b. Trauma
   c. Aortic dissection
   d. Iatrogenic as during a failed replacement surgery
2. Chronic
   a. Primary valvular: Rheumatic fever, bicuspid aortic valve, Marfan syndrome, Ehlers–Danlos syndrome, ankylosing spondylitis, SLE
   b. Aortic root disease: Syphilitic aortitis, osteogenesis imperfecta, aortic dissection, Behçet syndrome, Reiter syndrome, systemic HTN

C. Clinical Features

1. Symptoms
   a. May be symptomatic for many years
   b. Dyspnea on exertion, PND, orthopnea
   c. Palpitations—worse when lying down
   d. Angina
   e. Cyanosis and shock in acute aortic regurgitation (medical emergency)
2. Physical examination
   a. *Widened pulse pressure*—markedly increased systolic BP, with decreased diastolic BP.
   b. Diastolic decrescendo murmur best heard at left sternal border.
   c. *Corrigan pulse* (water-hammer pulse)—rapidly increasing pulse that collapses suddenly as arterial pressure decreases rapidly in late systole and diastole; can be palpated at wrist or femoral arteries.
   d. *Austin Flint murmur*—low-pitched diastolic rumble due to competing flow antegrade from the LA and retrograde from the aorta. It is similar to the murmur appreciated in mitral stenosis.
   e. Displaced PMI (down and to the left) and S_3 may also be present.
   f. Murmur intensity increases with sustained handgrip. Handgrip increases systemic vascular resistance (SVR), which causes an increased "backflow" through the incompetent aortic valve.

D. Diagnosis

1. CXR findings: Enlarged cardiac silhouette, dilated aorta
2. ECG findings: LVH
3. Echocardiogram—Perform serially in chronic, stable patients to assess need for

Additional sounds on cardiac auscultation			
Sound	Origin	Timing	Etiology
Aortic ejection click	• Opening of a stiff aortic valve • Heard best with the diaphragm of a stethoscope at the aortic region with the patient seated and leaning forward	• Early systolic sound (immediately after S1)	• Aortic stenosis
Mitral valve prolapse click	• Mitral valve prolapse into the left atria during systole • Heard best with the diaphragm of a stethoscope at the mitral region with the patient in left lateral position	• Midsystolic sound	• Mitral valve prolapse
Mitral valve opening snap	• Opening of a stiff mitral valve • Heard best with the bell of a stethoscope at the mitral region with the patient in a left lateral position	• Early diastolic sound (immediately after S2)	• Mitral stenosis
Mechanical valve clicks	• S1 and S2 sound like clicks. • Heard best with the diaphragm of a stethoscope	• Coincides with a normal S1 and S2	• Prosthetic valve
Pericardial friction rub	• Scratching sound due to friction between the visceral and parietal pleura • Heard best over the left sternal border during expiration with the patient sitting upright and leaning forward	• Systolic or diastolic sound	• Pericarditis
Pericardial knock	• Sudden cessation of ventricular filling against a rigid pericardial sack • Heard best at the left sternal border	• Diastolic sound	• Constrictive pericarditis