AORTIC STENOSIS (AS)

- **AS:** obstruction of Left ventricular outflow of blood across the aortic valve

ETIOLOGIES

1. ***Degenerative heart disease:*** calcifications (ex atherosclerotic/wear & tear) common in patients ***>70y****
2. ***Congenital heart disease:*** (ex. ***bicuspid Aortic valve***) common in patients ***<70y*.***
3. *Rheumatic heart disease:* AS may be accompanied by AR or mitral disease (but may be isolated).

PATHOPHYSIOLOGY

- Stenosis⇨ ***LV outflow obstruction (fixed CO)*** ⇨ ↑***afterload*** (***pressure overload)*** ⇨***LVH*** ⇨ ***LV failure.***

CLINICAL MANIFESTATIONS

- Most are asymptomatic. Usually becomes symptomatic when aortic valve area <1 cm^2 (Normal 3-4 cm^2)

Once symptomatic, patient's lifespan becomes significantly shortened if aortic valve replacement not performed.

1. ***Angina:*** (5y mean survival if valve replacement not done). ↑O_2 demand (LVH coupled with exertion) + ↓O_2 supply (2^{ry} to fixed cardiac output) ⇨subendocardial ischemia.

2. ***Syncope (exertional):*** (3y survival if valve replacement not done). Exertional peripheral vasodilation in the setting of a fixed CO ⇨ insufficient cerebral perfusion during exercise/exertion.

3. ***Congestive Heart Failure:*** (2y survival if valve replacement not done). ***Worst prognosis!***

PHYSICAL EXAMINATION

1. ***systolic "ejection" crescendo-decrescendo murmur @ RUSB radiates to carotid (neck)****.
   - ↓***murmur c ↓venous return: ex valsalva, standing, inspiration***.
   - ↑***murmur c ↑ venous return (ex squatting, leg raise, expiration); if patient sits & leans fwd.***
   - ***Signs of severity: late peaking murmur***, pulsus parvus et tardus**,** paradoxically split S_2, signs of LVH: LV heave & loud S_4 (due to contraction into stiff ventricle).

2. ***pulsus parvus et tardus: small, delayed, carotid pulse*.*** Narrowed pulse pressure, HTN.

DIAGNOSTIC STUDIES

1. ***Echocardiogram:*** ❶small aortic orifice ❷ L ventricular hypertrophy, ❸ thickened/calcified Aov.
2. EKG: ***LVH***; *Nonspecific changes* (LAE, LBB, left axis deviation, ±A fib or ischemic changes).
3. CXR: *Nonspecific changes* (poststenotic aortic dilation, AoV calcification, ±pulmonary congestion)

MANAGEMENT

A. **SURGICAL THERAPY:** ***Aortic valve replacement only effective treatment (tx of choice)!***

1. **AoV replacement (AVR):** symptomatic AS, asymptomatic severe AS (↓EF or AVA <0.6 cm^2).
   - **mechanical:** prolonged durability but thrombogenic (ex. stroke) so must be placed on anticoagulation therapy c warfarin (Coumadin).
   - **bioprosthetic:** less durable but minimally thrombogenic (usually used in patients that can't take warfarin). *Heterograft (porcine valve); pericardial.*
2. Percutaneous Aortic valvuloplasty (PAV): results in 50% ↑AV area, but 50% restenosis @ 6-12 mos, so used as a bridge to AVR, if pt not a surgical candidate or in pediatric pts
3. Intraortic balloon pump: stabilization, used as a bridge to AVR.

B. **MEDICAL THERAPY** ***no medical treatment truly effective***!

- No exercise restrictions in patients with mild AS.
- ***severe AS:*** because they are ***dependent on preload to maintain CO*** ⇨***avoid physical exertion/venodilators*** (ex. nitrates)/***negative inotropes*** (CCB, β-blockers)

AORTIC REGURGITATION (AR) or AORTIC INSUFFICIENCY (AI)

ETIOLOGIES

1. **Valve disease:** ***rheumatic heart dz*** (usually mixed AS/AR); ***Endocarditis,*** Bicuspid AoV.
2. **Aortic root disease/dilation:** ***hypertension***, ***Marfan syndrome, syphilis,*** rheumatoid arthritis, systemic lupus erythematosus, aortic dissection, ankylosing spondylitis.

PATHOPHYSIOLOGY

- Incomplete AoV closure during diastole ⇨ ***regurgitation of blood from Ao to LV (in addition to the normal antegrade flow from LA to LV)*** ⇨ ***LV volume overload**** ⇨ ***LV dilation*** ⇨ ***CHF.***

CLINICAL MANIFESTATIONS

Acute: (ex. acute MI, aortic dissection, endocarditis) ⇨ pulmonary edema, ±hypotension
Chronic: clinically silent while LV dilates ⇨ LV decompensation ⇨ CHF.

PHYSICAL EXAMINATION

1. ***Diastolic decrescendo, blowing murmur best at @ left upper sternal border (LUSB)***
   - ↑***murmur intensity c*** ↑**venous return:** ***sitting forward, expiration, handgrip, squatting. Severity of AR proportional to duration of murmur*** (except in acute/late disease); displaced PMI, ±thrill.
   - **↓ c ↓venous return (valsalva, standing, inspiration) & c ↓afterload (amyl nitrate).**
   - ±***Austin Flint murmur*** (mid-late diastolic rumble @ apex 2^{ry} to retrograde regurgitant jet mixing c antegrade flow from left atrium into the ventricle).
2. ***Bounding pulses*: 2^{ry} to ↑ stroke volume (↑SV).*** Laterally displaced PMI.
3. ***Wide pulse pressure*** (classic signs); ***Seen in chronic AI only.***

Classic Signs of WIDENED PULSE PRESSURE in AR/AI (seen ONLY with chronic AR/AI)	
SIGN	**DESCRIPTION**
Corrigan's (Water Hammer) pulse	***rapidly swelling & fall of radial pulse accentuated c wrist elevation***
Hill's sign	Popliteal artery systolic pressure > brachial artery by 60mmHg ***(most sensitive)***
Duroziez's sign	Gradual pressure over femoral artery ⇨ systolic and diastolic bruits
Traube's sound (pistol shot)	Double sound heard @ femoral artery c partial compression of femoral artery
De Musset's sign	***Head-bobbing*** c each heartbeat (low sensitivity)
Müller's sign	Visible systolic pulsations of the uvula
Quincke's pulses	Visible fingernail bed pulsations with light compression of fingernail bed

4. ***Pulsus Bisferiens: seen c AR + AS together or severe AR.*** Double pulse carotid upstroke.

DIAGNOSTIC STUDIES

1. ***Echocardiogram:*** regurgitant jet seen with Doppler flow.
2. EKG: nonspecific: (± LVH, LAD). CXR: nonspecific: ± cardiomegaly (due to LV dilation).

MANAGEMENT

Variable progression. CHF 2 year mean survival. Monitor for sx onset or progression of AR.

A. ***Surgical therapy: acute or symptomatic AR;*** *asymptomatic AR c LV decompensation* (LV ejection fraction <55%). Although 55% is a within normal LVEF range, ***patients with AR need a hyperdynamic ventricle to maintain CO.*** Aortic valve replacement preferred.

B. **Medical therapy:** ***afterload reduction c vasodilators*** (ACEI, ARB's, nifedipine, hydralazine) b/c afterload reduction ***improves ventricular performance by increasing forward flow.***

MITRAL STENOSIS (MS)

ETIOLOGIES

1. ***Rheumatic heart disease (RHD):*** ***almost always caused by rheumatic heart disease*.*** "fish mouth valve." MC in 3rd/4th decade
2. Congenital, left atrial myxoma, thrombus, valvulitis (SLE, amyloid, carcinoid).

PATHOPHYSIOLOGY

Obstruction of flow from LA to LV 2ry to narrowed mitral orifice ⇨ blood backs up into the L atrium ↕ ***L atrial pressure/volume overload*** ⇨ pulmonary congestion ⇨ ***pulmonary HTN**** ⇨ ***CHF.***

CLINICAL MANIFESTATIONS

Slow progression until symptoms. When symptoms occur, it is then associated c rapid progression.

1. ***Pulmonary sx:*** Dyspnea (MC sx), pulmonary edema, hemoptysis, cough, frequent bronchitis, pulmonary HTN.
2. ***Atrial fibrillation*:*** secondary to atrial enlargement ⇨ ***embolic events (esp CVA).***
3. ***Right-sided heart failure***: (due to prolonged pulmonary hypertension).
4. ***Mitral facies = ruddy (flushed) cheeks c facial pallor*** (chronic hypoxia).
5. Signs of left atrial enlargement: dysphagia (esophageal compression), hoarseness

PHYSICAL EXAMINATION

1. ***PROMINENT (LOUD) S_1***: due to delayed forceful closure of mitral valve. ± split S_2.

2. ***OPENING SNAP (OS)*:*** high-pitched early diastolic sound of the opening of stenotic valve. Valve area proportional to S_2-OS interval (tighter valve ⇨ shorter S_2-OS interval).
   Severity of MS: shorter S_2-OS interval & prolonged diastolic murmur

3. ***Early-mid diastolic rumble @ apex (low pitched) esp in LLD position:*** (±preceded by OS)
   - ↓ ***murmur intensity: ↓venous return (Valsalva, standing, inspiration).***
   - ↑***murmur intensity: ↑venous return (lying down, squatting, expiration), exercise,*** placing patient in ***left lateral decubitus position.***

DIAGNOSTIC STUDIES

1. ***Echocardiogram:*** narrowed mitral valve (normal LV function usually, ↑LA pressure, ± pulmonary HTN).
2. EKG: ***left atrial enlargement (LAE/P mitrale***); ±A fib or RVH (pulmonary HTN).
3. CXR: nonspp. LAE (straightening of L heart border, L mainstem bronchus elevation).

MANAGEMENT

A. **Surgical Management**:

1. ***Mitral valve repair or replacement***: ***symptomatic MS,*** pulmonary HTN. Mechanical better than porcine (porcine not as suitable in replacement).
   - **percutaneous balloon *valvuloplasty/valvotomy:*** ***best treatment of younger patients*,*** symptomatic isolated severe MS, asymptomatic patients with moderate to severe MS & good valve morphology, noncalcified valves.
   - Open valvotomy: if percutaneous is not successful or not possible.
   - mitral valve replacement: if unable to perform valvotomy

B. Medical Management: does not alter natural history nor delay need for surgery.

- Congestion (loop Diuretics & Na^+ restriction); β blockers; ±digoxin (if A fib).

MITRAL REGURGITATION

ETIOLOGIES

1. ***Leaflet abnormalities: mitral valve prolapse MC cause****, ***rheumatic heart disease, endocarditis,*** valvulitis, any cause of LV dilation (ex. Marfan syndrome).
2. ***Papillary muscle dysfunction: ischemia/infarction***, displacement 2ry to cardiomyopathy.
3. ***Ruptured chordae tendinae:*** collagen vascular disease, dilated cardiomyopathy, MVP

PATHOPHYSIOLOGY

- ***Retrograde blood flows from the LV into the LA*** (but the refluxed blood in LA returns to LV during diastole) ⇨***LV volume overload*** ❶***LA dilation*** as blood backflows to lungs (↑LA/pulmonary pressures) ❷↓***CO*** due to diminished effective forward flow.

CLINICAL MANIFESTATIONS

1. **Acute:** ***pulmonary edema*** (rapid volume overload on LA)***,*** hypotension. Dyspnea***,*** fatigue.
2. **Chronic:** ***A fib***, progressive Dyspnea on exertion, fatigue, CHF, pulmonary HTN, hemoptysis.

PHYSICAL EXAMINATION

1. ***Blowing holosystolic (pansystolic) murmur @ apex c radiation to axilla*** (high pitched).
   - ↓ ***(diminished) murmur: ↓venous return (valsalva, standing) inspiration; amyl nitrate***
   - ↑***(augmented) murmur: ↑venous return (squatting, laying down, inspiration) & handgrip, left lateral decubitus position***
2. ***Widely split S_2*** (↓LV ejection time results in early A_2, pulmonary HTN results in delayed P_2).
3. Laterally displaced PMI, ±thrill, ± S_3 (LV dysfunction), ± decreased S_1 if severe.

DIAGNOSTIC STUDIES

1. ***Echocardiogram:*** regurgitant jet, hyperdynamic LV (EF <60% = LV impairment)
2. ECG: nonspecific: LAE (P mitrale), LVH, ± A fib
3. CXR: nonspecific: cardiomegaly (dilated LA/LV), ± pulmonary congestion

MANAGEMENT

A. **Surgical:**
   1. Indications: acute or symptomatic MR; asymptomatic MR c ***LV decompensation/dilation*** (EF <55-60%). IABP for stabilization/bridge to surgery. ***Repair preferred over replacement.***

B. **Medical:** indicated if not operative candidate***. Vasodilators to ↓afterload*** (***ACEI,*** hydralazine/nitrates); ↓***preload*** (↓amount of MR – diuretics, nitrates); ± antiarrhythmics or digoxin to control A fib.

MITRAL VALVE PROLAPSE (MVP)

- Etiologies: myxomatous degeneration of the MV apparatus, assoc c connective tissue diseases (ex Marfan's, Ehlers-Danlos). ***MC in young women.***
- **CLINICAL MANIFESTATIONS:** ***most are asymptomatic!*** ❶***Autonomic dysfunction:*** anxiety, atypical chest pain, panic attacks; arrhythmias causing palpitations, syncope, dizziness, fatigue
  ❷***sx assoc with MR progression:*** fatigue, dyspnea, PND, CHF. ❸ stroke, endocarditis.
- **PHYSICAL EXAM:** *narrow AP diameter, low body weight, hypotension, scoliosis, pectus excavatum.*

- ***Mid-systolic click* best heard @ apex*** ±mid-late systolic murmur. ***Any maneuver, which makes the LV smaller (ex. valsalva, standing) results in earlier click*** & longer murmur duration (2ry to increased prolapse of abnormal valve c normal valve).

DIAGNOSIS: echocardiogram shows ***posterior bulging leaflets (with tissue redundancy).***
MANAGEMENT: ***reassurance (good prognosis). Beta blockers for autonomic dysfunction*.***